Deconstructing Chronic Low Back Pain in the Older Adult– Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment

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Introduction to Special Series: Deconstructing Chronic Low Back Pain in the Older Adult: Shifting the Paradigm from the Spine to the Person

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Conflicts of interest: The author has indicated that she has no conflicts of interest regarding the content of this article.

Key Words. Back Pain; Chronic Low Back Pain; Chronic Pain; Elderly; Geriatric; Homeostenosis; Low Back Pain; Lumbar; Magnetic Resonance Imaging; Older Adults; Pain Management; Treatment Outcome

Over the past decade, the estimated prevalence of low back pain (LBP) among older adults (typically defined as those ≥age 65) has more than doubled [1], and the utilization of advanced spinal imaging (e.g., computerized tomography (CT), magnetic resonance imaging [MRI]) and procedures guided by this imaging (e.g., epidural corticosteroids, spinal surgery) have continued to skyrocket [1–3]. Treatment outcomes, however, have not improved apace. Why? Part of the answer lies in the fact that treatment may in part be misdirected. This issue of *Pain Medicine* contains the first in a series of articles on how to systematically and comprehensively rethink our approach to evaluating and designing management for older adults with chronic low back pain (CLBP). The series is entitled "Deconstructing Chronic Low Back Pain in the Older Adult: Step-by-Step Evidence and Expert-Based Recommendations for Evaluation and Treatment" and the article in this issue focuses on hip osteoarthritis (OA), an important potential contributor to CLBP in older adults.

Current Practice

To understand how we might attempt to improve the care of older adults with CLBP, let us start by examining current approaches. Management of patients with CLBP often begins with a search for the cause of pain using spinal imaging. The vast majority of people with CLBP do not require imaging because they do not have "red flag" pathology, that is, serious disorders such as cancer or infection that require urgent treatment [4]. Spinal imaging in the older adult who does not have red flags on history or physical examination will almost certainly reveal "abnormalities." Imaging evidence of lumbar degenerative disc and facet disease is nearly ubiquitous in older adults, even those who are pain-free [5]. An estimated 20% of older adults without neurogenic claudication have moderate to severe lumbar spinal stenosis on magnetic resonance imaging [6]. Thus older adults may be especially susceptible to receiving invasive treatment guided principally by imaging-identified degenerative spinal pathology and it is not surprising that such treatment is often inefficacious. While CLBP can be a management challenge for patients of all ages, we focus on older adults because of their vulnerability to undergoing degenerative spine disease-focused procedures that may not be necessary, as well as adverse drug effects and invasive treatment-associated morbidity [7,8].

If we attempt to manage the older adult with CLBP solely using spinal imaging, there may be one of three results: 1) The physical cause of pain is identified and appropriately targeted treatment is prescribed

(e.g., severe central canal stenosis is identified and decompressive laminectomy results in reduction of pain and disability). 2) Pathology is identified that may be incidental (e.g., asymptomatic central canal stenosis, bulging discs, degenerative disc disease); the cause(s) of pain and disability lies outside of the lumbar spine (e.g., sacroiliac joint [SIJ] syndrome, iliotibial band pain, myofascial pain of the erector spinae or quadratus lumborum, hip OA), thus treatment may be inappropriately targeted. 3) Spinal pathology is identified that, when combined with biopsychosocial factors outside of the lumbar spine (e.g., anxiety, depression, fear avoidance beliefs, insomnia, fibromyalgia syndrome [FMS], hip OA), CLBP and disability results. If our treatment targets only degenerative spine disease in these patients, suboptimal outcomes are likely.

Deconstructing CLBP in Older Adults: A Geriatric Medicine Approach

The overarching goal when treating patients with chronic pain is to optimize their capacity to function despite the persistence of some pain. This aligns well with the philosophical practice of geriatric medicine, that is, optimizing patient function despite practitioners' inability to eradicate disease. At the core of aging is a concept called homeostenosis, defined as the progressive restriction of an organism's ability to respond to stress as it ages (the antithesis of homeostasis, an organism's capacity to maintain stability in the face of change; [9]). Older adults, by virtue of being alive, have accumulated a host of changes at the cellular and tissue level, such as decrease in bulk and quality of skeletal muscle (i.e., sarcopenia), loss of skin elasticity, and vascular stiffening, to name a few. These changes in and of themselves are often not associated with disease. Instead, they serve as a source of vulnerability or homeostenosis, that is, a "weak link" [10].

This series conceptualizes degenerative discs and facet joints of the lumbar spine as weak links instead of diseases and CLBP as a syndrome, that is, a final common pathway for the expression of multiple contributors [11]. A common syndrome familiar to those who care for older adults across multiple settings of care is delirium (acute confusional state), defined as an acute disorder of attention and cognition [12]. When a hospitalized older adult becomes acutely confused, the most likely culprit is an infection (e.g., urinary tract infections, pneumonia) or an adverse drug reaction [12]. Despite the patient's symptoms being indicative of brain dysfunction, brain imaging is not routinely recommended for those with delirium [12]. Instead, a search for causative factors outside of the central nervous system is recommended, that is, those factors that make the vulnerable aging brain overtly dysfunctional. Our goal is to move the treatment of CLBP in a similar direction by emphasizing identification and treatment of the multiple factors that when combined with degenerative disease of the lumbar spine, cause disability.

The Series

Our series of articles is written from the vantage point that the lumbar spine is a weak link or one of multiple treatment targets rather than the sole treatment target in older adults with CLBP. Each article published over the next months will contain a focused literature review and an illustrative clinical case. Our presentation of CLBP in older adults as a syndrome aligns with pain physiology. Pain is a complex physiological process contributed to by peripheral nociceptive stimuli and interpretation of those stimuli by the brain. In older adults with CLBP, factors outside of the lumbar skeleton that alter spinal biomechanics such as hip OA and leg length discrepancy (e.g., following joint replacement) may drive nociception. Factors that alter perception of nociceptive stimuli (i.e., top down inhibition) such as FMS, cognitive impairment, psychological maladaptation (e.g., fear avoidance beliefs, catastrophizing), anxiety, and depression also may contribute to pain and pain-associated disability.

Data indicate that older adults with chronic pain tend to be more psychologically robust than their younger counterparts [13], and interdisciplinary pain management programs that treat patients who fail first and second line therapy routinely use psychological interventions and other strategies to help patients improve their ability to cope and function with pain [14,15], focusing less on identifying and treating physical pain contributors. Older adults with CLBP often have multiple physical contributors to their pain and difficulty functioning [16], thus thorough hands-on physical assessment is critical to optimizing treatment outcomes. Because of an increased risk of social isolation and dementia, they also are likely to have unique psychosocial contributors to CLBP-associated disability that traditional pain management programs do not address.

Over the ensuing months, we will discuss conditions that occur commonly in older adults with CLBP $\pm$ leg pain and that should be evaluated routinely as potential contributors to pain and disability: 1) hip OA, 2) myofascial pain, 3) lumbar spinal stenosis, 4) SIJ joint syndrome, 5) lateral hip/thigh pain syndrome, 6) leg length discrepancy, 7) insomnia, 8) fibromyalgia, 9) depression, 10) anxiety, 11) psychological maladaptation, and 12) cognitive impairment. We have drawn from data within the pain, spine, orthopedics, pharmacology, physical therapy, psychology, psychiatry, rheumatology, rehabilitation, and gerontology literature, as well as expert opinion (when strong evidence was not available) to create algorithms usable in the clinical setting.

Methods

Each algorithm was created using a modified Delphi technique and takes into account resources within Veterans Health Administration (VHA) facilities to facilitate

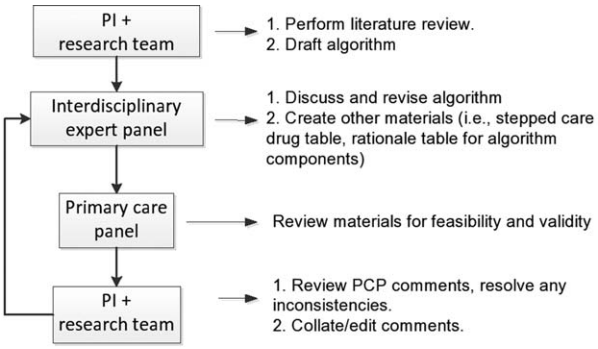

Figure 1 Schematic of modified Delphi process used to create algorithms. PI = principal investigator; PCP = primary care provider.

Please rate each statement using the following scale:

5=strongly agree, 4=agree, 3=no opinion, 2=disagree, 1=strongly disagree

1. The proposed treatment algorithm is evidence-based.
2. The treatment algorithm is easy to understand.
3. The proposed treatment algorithm can be readily applied within the VA.
4. The treatment algorithm appropriately balances the risks and benefits of available treatments for _____ in older adults.
5. There are important evidence-based treatments that have been omitted from the algorithm. (If so, please describe and provide supportive literature.)
6. If rigorously tested, evidence based treatments are lacking for older adults with _____, a rational approach has been presented. (If not, please suggest modifications.)

Additional Comments:_____

Figure 2 Questionnaire used to solicit feedback from primary care panellists.

broad uptake. As VHA facilities have a more restricted formulary than non-VHA facilities, we recommend medications that are available in both VHA and civilian settings. The Delphi technique is based on the premise that pooled intelligence enhances individual judgment and captures the collective opinions of a group of experts without being physically assembled [17,18].

An overview of the process used in this project is shown in Figure 1 and described here.

1. To begin, the Principal Investigator (DW) drafted an evidence-based treatment algorithm based on a comprehensive review of the literature and knowledge of medications and services available within VHA facilities.
2. The draft algorithm was distributed to an interdisciplinary expert panel, chosen based on recognition in their individual fields and/or their expertise in providing clinical care to older adults. This panel discussed the algorithm via teleconference, refined it and created accompanying tables (i.e., a table providing the rationale and corresponding citations for individual algorithm components; and for the majority of algorithms, a stepped-care drug table predicated on changes in aging-associated pharmacokinetics and pharmacodynamics). When strong evidence was not available, the expert opinion and clinical experience of panelists was drawn upon for guidance.
3. The refined materials were distributed to a 9-member primary care provider (PCP) panel who reviewed the materials using the questionnaire shown in Figure 2, focusing on the feasibility of implementing the algorithm in the primary care setting in general and in the VA in particular, as well as the validity of the recommendations for older adults.
4. A research assistant collated the PCP panel comments and the PI reviewed and clarified them as needed. For example, if one of the PCP reviewer's rating of an item in Figure 2 was an outlier as compared with that of the other reviewers, she communicated with them via e-mail to resolve any inconsistencies. Revisions to the collated comments were then made and distributed to the expert panel.
5. The expert panel reviewed the feedback and revised the algorithm and accompanying materials. They prepared a point-by-point response to the feedback that was distributed along with the revised materials to the PCP panel for review.
6. The entire process was repeated until no further revisions were recommended.

Our hope is that these algorithms and accompanying materials will provide a concrete framework to guide evaluating and managing the multiple contributors to CLBP ± leg pain in older adults. The ultimate goal is to optimize function and minimize morbidity in these vulnerable patients. Consider, for example, the 68-year-old woman with LBP and bilateral leg pain as well as FMS, fear avoidance beliefs, and a lumbar MRI that shows moderate central canal stenosis. She is treated with epidural corticosteroid injections and ultimately decompressive laminectomy, without relief. In fact, her pain worsens. Her FMS was never treated, despite the fact that axial pain is a central feature of FMS, as is pain in multiple body regions, including the legs. Her fear avoidance beliefs also were not addressed. Could surgery have been avoided if her treatment began with aerobic exercise and/or medication that targeted her FMS and cognitive behavioral therapy for her fear avoidance beliefs?

We present the algorithms individually for the sake of clarity, but practitioners will likely use them together, rather than as separate guidelines. Ensuring realistic treatment expectations lie at the core of each algorithm, along with a strong emphasis on patient education, so that each person being treated understands both the factors contributing to their symptoms and how to engage in self-management. These concepts are

fundamental to providing rational and high quality care to all patients, especially to those with chronic pain.

There is an art involved in caring for patients with chronic pain, a genuine therapeutic value of providers communicating positively with patients and instilling hope. Knowing how to prioritize the treatment of multiple contributors to pain and disability is also an art, and we hope to provide the artists' tools to as many providers as possible with the articles and algorithms that will appear in the following months. By deconstructing CLBP into separate components, we provide practitioners with a different framework to consider and utilize. Without a doubt, there will be subsequent projects that test the effectiveness of this kind of approach.

Epilogue: A Look to the Future

Why do nearly all of older adults have degenerative disease of the lumbar spine, but only a small fraction has CLBP, and still fewer are disabled by their pain? We know that many biopsychosocial factors influence the course and outcome of CLBP and that response to treatment of these factors is variable. It is likely that some of the discrepancy between imaging findings, symptoms and function, and the variability of treatment response has to do with as yet unidentified genetic and other biological factors, and one day, we may be able to more precisely prescribe biologic treatments based on one or more tests. That time is likely in the distant future.

How in the meantime should we rationally approach the evaluation and treatment of older adults with CLBP? The United States healthcare system is in crisis. Our excessive expenditures compared with other countries are generally not matched by superior quality or quantity of life [19]. Our society is aging rapidly and there is a burgeoning of older adults with CLBP for whom we must provide rational care. Our series of articles, "Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment," has been created to do exactly that.

Acknowledgments

This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The author thanks Dave Newman, Dr. Rollin Gallagher, and Dr. Eric Rodriguez for their thoughtful review of the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment

Part I: Hip Osteoarthritis

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Conflicts of interest: The authors have indicated that they have no conflicts of interest regarding the content of this article.

Abstract

Objective. To present the first in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. The series presents CLBP as a syndrome, a final common pathway for the expression of multiple contributors rather than a disease localized exclusively to the lumbosacral spine. Each article addresses one of twelve important contributors to pain and disability in older adults with CLBP. This article focuses on hip osteoarthritis (OA).

Methods. The evaluation and treatment algorithm, a table articulating the rationale for the individual algorithm components, and stepped-care drug recommendations were developed using a modified Delphi approach. The Principal Investigator, a five-member content expert panel and a nine-member primary care panel were involved in the iterative development of these materials. The algorithm was developed keeping in mind medications and other resources available within Veterans Health Administration (VHA) facilities. As panelists were not exclusive to the VHA, the materials can be applied in both VHA and civilian settings. The illustrative clinical case was taken from one of the contributor's clinical practice.

Results. We present an algorithm and supportive materials to help guide the care of older adults with hip OA, an important contributor to CLBP. The case illustrates an example of complex hip-spine syndrome, in which hip OA was an important contributor to disability in an older adult with CLBP.

Conclusions. Hip OA is common and should be evaluated routinely in the older adult with CLBP so that appropriately targeted treatment can be designed.

Key Words. Aged; Assessment; Hip Osteoarthritis; Chronic Pain; Elderly; Low Back Pain; Primary Care; Chronic Low Back Pain

Introduction

An estimated one in two people with hip osteoarthritis (OA) has low back pain (LBP) [1]. The Hip-Spine Syndrome (HSS) was first described by Offierski in 1983 [2]. Three types of patients were described – those with "simple" HSS who had pathology of both the hip and lumbar spine, but disability related to only one source; those with "complex" HSS who had symptoms from both the hip and spine without a clear single source of disability, such as patients with low back and leg pain and who have clinical evidence of both lumbar spinal stenosis and hip OA [3]; and those with "secondary" HSS who have inter-related pathology such as restricted hip motion from advanced OA causing abnormal biomechanics of the lumbo-pelvic-hip complex and consequent LBP [4]. Secondary HSS has been substantiated through studies demonstrating significant reduction or complete resolution of LBP in patients with hip OA following total hip arthroplasty (THA) [5–7].

Spinal pathology does not always lead to spinal pain. Much of magnetic resonance imaging (MRI)-identified degenerative lumbar pathology is incidental in older adults and its identification may lead to invasive procedures that are ineffective and sometimes dangerous. A study of individuals age 65 and older that included 162 participants with chronic low back pain (CLBP) and 158 pain-free revealed x-ray evidence of degenerative disc disease in 95%, both those with CLBP and those pain-free [8]. Jarvik et al. studied 148 pain-free individuals with lumbar MRI, and 21% of those $>$ age 65 (six of 29 individuals) had evidence of moderate or severe stenosis of the central canal, as compared with 11% of those age 55–65 and 6–7% of those $<$age 55 [9]. As noted in the series overview in this issue of *Pain Medicine*, CLBP associated with functional compromise in the older adult should be approached as a geriatric syndrome, that is, as a final common pathway fed by multiple contributors [10]. These patients should routinely be evaluated for contributors to pain and disability that lie outside of the spinal skeleton such as hip OA, as this and other extraspinal factors may directly cause part or all of the patient's LBP or they may independently contribute to disability.

Preliminary data indicate that as many as one in four older adults with CLBP may have physical examination evidence of hip OA that is typically one of multiple contributors to their pain and disability [11]. Patients with hip OA may experience pain in their groin, buttocks, thigh, and/or distal lower extremity [12], and this could be misconstrued as pain emanating from pathology of the lumbar spine (e.g., radiculopathy). Identifying the hip rather than the back as a key pain generator could substantively impact management. We present a patient who has CLBP with hip OA being at least one contributor to his pain and difficulty functioning. This case demonstrates the clinical complexity of older patients with chronic low back and/or leg pain, comorbid hip and spine pathology, and a pragmatic approach to management of their musculoskeletal pain.

Methods

A modified Delphi technique involving a content expert panel and a primary care review panel, as per the detailed description provided in the series overview [13], was used to create the algorithm (Figure 1), the table providing the rationale for the various components of the algorithm (Table 1), and the stepped-care medication table (Table 2). Expertise represented among the 5 Delphi expert panel members for the hip OA algorithm included geriatric medicine, geriatric pharmacology and rheumatology.

Case Presentation

Relevant History

The patient is an 85-year-old man who presented to the Pain Clinic in 2012 with a 50-year history of LBP that started with an injury in 1960. He was treated with a discectomy in 1961, repeat back surgery in 1963, and a L4–L5 laminectomy and fusion in 2008. He reports a several year history of increasing achy right-sided LBP with radiation into his buttocks and upper outer thigh. His pain severity ranges from 3–7/10 with an average of 5/10. Prolonged standing and/or walking exacerbates his pain and sitting alleviates it. He can stand 2–5 minutes and/or walk $\sim$100 feet before he has to sit down because of pain. He denies lower extremity weakness, change in his bowel or bladder habits, fever, trauma, weight loss, or recent cancer associated with the worsening of his pain. He has tried hydrocodone, amitriptyline, several different nonsteroidal anti-inflammatory drugs, acetaminophen, and physical therapy (PT), all without significant reduction in pain or improvement in function. The only thing other than sitting or lying down that reduces his pain is oxycodone 5 mg/acetaminophen 325 mg, one pill three times daily. This regimen results in pain reduction from an average of 8–5 (on a 0–10 scale) for about 3 hours.

Relevant Physical Examination

The patient is awake, alert, oriented $\times$3, cooperative and in no apparent distress. Gait: Antalgic, favoring the right leg. Lumbar Spine: There is no tenderness over the spinous processes. The patient reports pain with forward flexion localized to the left paralumbar and sacroiliac joint regions and pain with extension that he experiences bilaterally. Hips: Internal rotation of the right hip is 10° and painful. Internal rotation of the left hip is not painful and 30°.

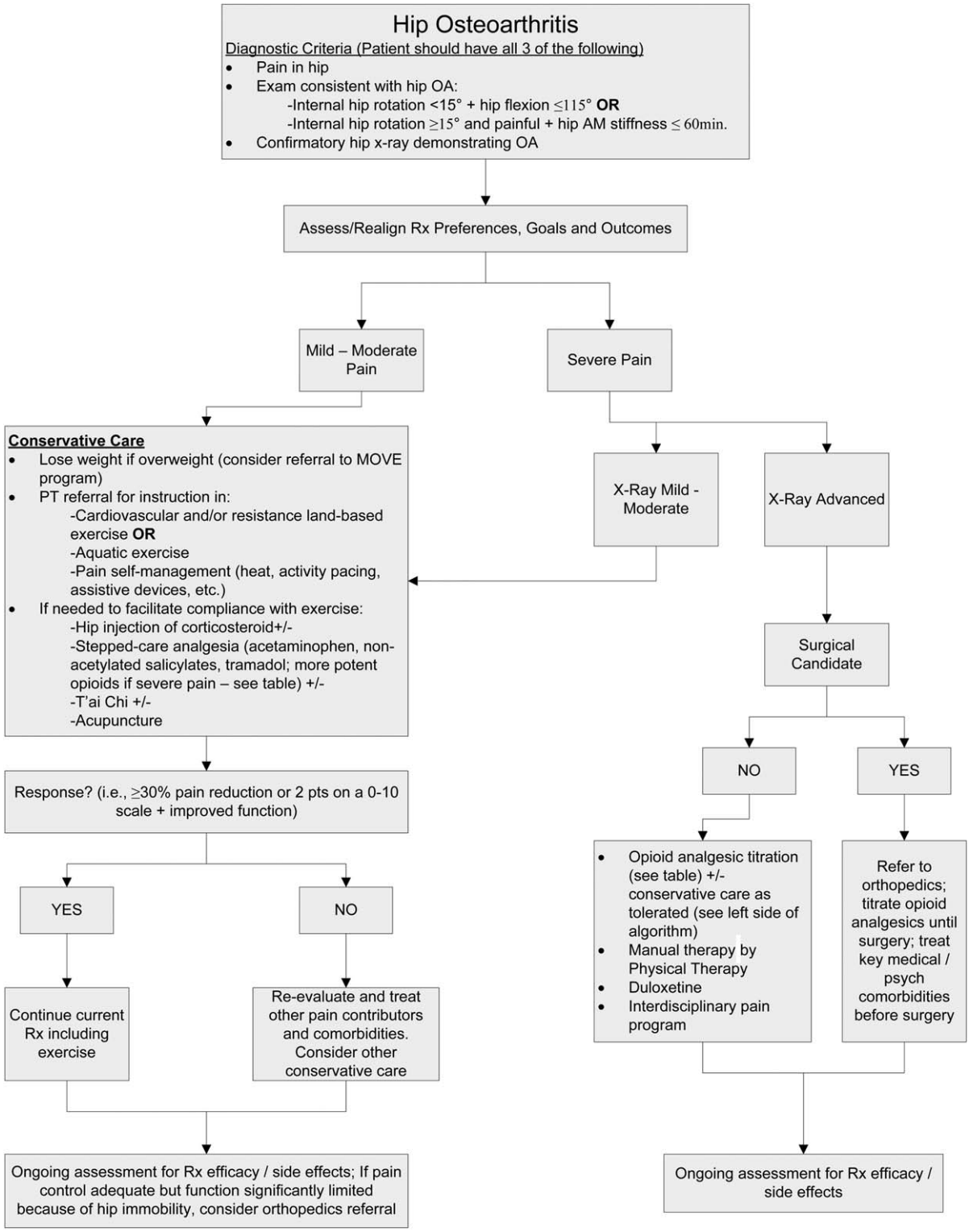

Figure 1 Algorithm for the evaluation and treatment of hip OA in an older adult.

Table 1 Hip OA: Theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm Component	Comments	References
Hip OA diagnostic criteria	We have not included ESR $<$ 20 mm/hr as a criterion given the nonspecificity of modestly elevated ESR in older adults. We also have not included age $>$50 as this applies to all older adults.	[14]
30% pain reduction as significant	Data on 2724 subjects from 10 placebo controlled trials of pregabalin in diabetic neuropathy, postherpetic neuralgia, CLBP, fibromyalgia, and OA.	[15]
Cardiovascular and/or resistance land-based exercise	ACR strongly recommends	[16]
Aquatic exercise	ACR strongly recommends. This should be recommended for people who prefer it to land-based exercise (LBE) or who are unable to tolerate LBE.	[14,17]
Pain Self-Management Program	ACR recommends conditionally. High quality evidence is lacking specifically in older adults. Arthritis pain self-management programs that contain some CBT elements demonstrate efficacy. Programs appear to benefit self-efficacy but not pain or physical functioning.	[16] [18] [19]
Weight loss	Prescribe for those who are overweight; ACR strongly recommends.	[16]
Acetaminophen	The 3000–3250 mg/day maximum is an FDA suggestion (not a mandate) and is based on no data in adults.	[20]
Nonacetylated salicylates	ACR conditionally recommends oral NSAIDs. Traditional NSAIDs (e.g., ibuprofen, naproxen) should not be used chronically in older adults because of the potential for multiple adverse effects including but not limited to gastrointestinal bleeding, renal insufficiency, and exacerbation of hypertension and congestive heart failure. As celecoxib also has many of these deleterious effects, it is not recommended for long-term use in older adults. NOTE: The 2012 Beers Criteria do not include celecoxib as a contraindicated drug; the AGS 2009 Pain Guidelines recommend against its chronic use in older adults.	[16] [21–23]
Tramadol	This agent is recommended prior to considering other opioids based on the combined evidence from two Cochrane reviews. Overall benefits from any opioid or opioid-like drug are modest. It is not clear that benefits outweigh risks.	[24,25]
Duloxetine	Duloxetine is FDA-approved for the treatment of OA pain. There is evidence of efficacy in older adults with knee OA pain, but not specifically for hip OA.	[26]
T'ai Chi	If available, T'ai Chi is recommended as the first CAM modality that should be tried because of strong evidence supporting its efficacy in preventing falls in older adults (an important comorbidity in older adults with chronic pain) and its modest efficacy evidence for reducing pain and improving function in older adults with hip OA.	[27–29]

Table 1: *Continued*

Algorithm Component	Comments	References
Acupuncture	As with most CAM interventions, further research is needed before any can be recommended strongly.	[30]
Acetaminophen with codeine not recommended.	Codeine's adverse effect profile argues against using it in older adults.	[31,32]

Imaging

An AP pelvis x-ray (Figure 2) revealed severe joint space narrowing and subchondral sclerosis of the right hip (arrow in Figure 2) and mild joint space narrowing of the left hip.

Clinical Course

The patient's oxycodone/acetaminophen was titrated to 2 pills four times daily and he experienced better pain relief and continued to be active. He was not interested in THA. Because of increasing pain and difficulty climbing stairs, 9 months later he underwent a fluoroscopically guided intra-articular hip injection with complete alleviation of his buttocks and leg pain and modest reduction of his LBP. He reported significant reduction of pain interference with his daily activities and sought consultation regarding THA.

Approach to Management

The patient presented has CLBP with hip OA being at least one contributor to his pain and difficulty functioning (Figure 1). The diagnosis of hip OA was made using a combination of clinical and radiographic criteria [14]. Because an estimated 50% or more older adults have radiographic evidence of hip OA that is asymptomatic [33] , the expert panel that created the hip OA algorithm (Figure 1) felt that using American College of Rheumatology (ACR) clinical criteria (i.e., the first two bullets in the box at the top of the algorithm) to guide the performance of x-rays is essential. See Figures 3A,B and 4A,B illustrating the physical examination techniques for assessing hip internal rotation and hip flexion.

As shown in Figure 1, treatment recommendations should be developed in collaboration with the patient to understand his treatment goals and preferences and assess his pain severity. For patients with unrealistic treatment goals, for example, complete pain relief, education should immediately ensue. In keeping with the core tenets of chronic pain rehabilitation, an eye toward optimizing function in those with CLBP should be the primary goal of treatment [34,35], thus pain reduction is a means to achieving that goal rather than a primary end point. Patients should expect, on average, 30% reduction in pain or 2 points on an 11 point (i.e., 0–10) scale [15]. For those with mild to moderate pain, we recommend conservative care that starts with weight loss for those overweight [16]. The MOVE!® Weight Management Program, designed by the VA National Center for Health Promotion and Disease Prevention, is recommended specifically for patients who receive their care within Veterans Health Administration facilities. The ACR recommends cardiovascular and/or resistance land-based exercise, or aquatic exercise as part of the foundation of hip OA management [16]. Aquatic programs are especially useful for people unable to tolerate land-based exercise [20]. Prescription of an assistive device for joint unloading also should occur.

Pain interventions that from the patient's perspective are passive, such as injections, are themselves inadequate to provide lasting benefits [36–38]. Patients who attend interdisciplinary pain management programs are taught to conceptualize these passive interventions as bridges to engaging in active pain self-management strategies [34,35,39], and that continued self-management will afford more robust benefits over time. Patients with hip OA as a contributor to their CLBP should be viewed similarly. The ACR recommends pain self-management conditionally because high quality evidence for the impact of pain self-management programs in reducing pain and improving function in older adults with hip OA is lacking, yet for patients with CLBP, active engagement in self-care is an important key to success [16,17].

What should be the role of pharmacological management? In keeping with the philosophy of chronic pain management articulated above, medication for older adults with hip OA and CLBP should be viewed as a means to an end (i.e., engaging in physical activity) rather than the end goal. Unless the patient has a contraindication, intra-articular hip injection for analgesia is an attractive option; in addition to the possibility of providing analgesia, it may help to ascertain the degree to which the hip itself is contributing to the patient's CLBP syndrome and associated disability. Note that the purpose of hip injection is to alleviate pain. If a patient has a contracted hip capsule and an associated flexion contracture, a hip injection will not treat the flexion contracture and its stress on the spine. In this situation, therefore, an injection may not be able to effectively ascertain the extent to which the hip is contributing to CLBP.

Guidelines for stepped care analgesia in the older adult with hip OA is provided in Table 2. In this table we include recommendations for starting doses and titration

Table 2 Stepped care drug management of hip OA pain

Drug	Dose/Titration (Note: Abbreviations such as bid should be avoided in an effort to reduce errors.)	Important Adverse Effects/Precautions
First Line Treatment		
Intra-articular corticosteroid	n/a	Diabetics must monitor glucose carefully following procedure.
Acetaminophen	325–1000 mg q4–6h while awake, max 3000–3250 mg/day Adjust dosing interval for renal function: CRcl 10–50: q6 hours; CRcl $<$ 10: q8 hours	Ask about all OTCs with acetaminophen; increased toxicity from chronic use if heavy EtOH use, malnourishment, preexisting liver disease—decrease maximum daily dose to 2 g. The manufacturer's label lists a maximum daily dose of 3000 mg for extra strength (500 mg) tablets or capsules, and 3250 mg for regular strength (325 mg) tablets and capsules. Health care professionals may still prescribe or recommend a maximum of 4000 mg per day.
Second Line Treatment		
Salsalate	500–750 mg twice daily; maximum dose 3000 mg/day	Does not interfere with platelet function; GI bleeding & nephrotoxicity rare; salicylate concentrations can be monitored if toxicity suspected. As these drugs are salicylates, providers should educate patients about symptoms associated with salicylism (e.g., nausea, vomiting, tinnitus, vertigo, reversible hearing loss, etc.). Avoid in patients with advanced renal disease or hepatic impairment.
Choline magnesium trisalicylate	750 mg three times daily; max 3 g/day	
Third Line Treatment		
Tramadol	Start 25 mg once a day; increase by 25–50 mg daily in divided doses every 3–7 days as tolerated to max dose of 100 mg four times a day. Renal dosing (CRcl $<$30 mL/minutes) 50–100 mg twice a day. Max. 200 mg/day.	Seizures and orthostatic hypotension. Other side effects similar to traditional opioids including constipation, sedation, confusion, respiratory depression. Potential for serotonin syndrome if patient is on other serotonergics. Do not use extended-release product if CR_{Cl} $<$30 mL/min.
Hydrocodone/ acetaminophen	2.5/325 or 5/325–10/325 mg q4–6h. Consider recommending a supplementary dose of APAP 325 with combination dose for additional analgesia before increasing the opioid dose. Total acetaminophen dose not to exceed 3000–3250 mg/day.	For all opioids, increased risk of falls in patients with dysmobility. May worsen or precipitate urinary retention when BPH present. Increased risk of delirium in those with dementia. Because of increased sensitivity to opioids older adults at greater risk for sedation, nausea, vomiting, constipation, urinary retention, respiratory depression, and cognitive impairment. Start stimulant laxative (e.g., senna) to prevent/treat constipation. Many would start at opioid initiation if patient has existing complaints of constipation or other risk factors. Some providers advocate ensuring that all patients in whom opioids are initiated have a stimulant laxative readily available and start it at the first sign of constipation.

Table 2: *Continued*

Drug	Dose/Titration (Note: Abbreviations such as bid should be avoided in an effort to reduce errors.)	Important Adverse Effects/Precautions
		Exercise caution and follow closely if opioids are started in patients who drive. Avoid concomitant prescription of opioids and other CNS depressants. Risk of addiction/diversion present with all opioids. Before starting an opioid, assess risk with the Opioid Risk Tool and during maintenance, monitor using tool such as Current Opioid Misuse Measure (COMM). Tools available at www.painedu.org.
Oxycodone or morphine	Start with 2.5 mg oxycodone or morphine q4h and titrate no more frequently than q 7 days; assess total needs after 7 days on stable dose, then convert to long acting. Morphine Dosing in Renal Impairment: Cl_{cr} 10–50 mL/minute: Administer at 75% of normal dose; Cl_{cr} <10 mL/minute: Administer at 50% of normal dose. Dosing in Hepatic Impairment: No dosage adjustment provided in manufacturer's labeling. Pharmacokinetics unchanged in mild liver disease; substantial extrahepatic metabolism may occur. In cirrhosis, increases in half-life and AUC suggest dosage adjustment required. Oxycodone Dosing in Renal Impairment: Serum concentrations are increased ~50% in patients with Cl_{cr} <60 mL/minute; adjust dose based on clinical situation. Dosing in Hepatic Impairment: Immediate release: Reduced initial doses may be necessary (use a conservative approach to initial dosing); adjust dose based on clinical situation. Controlled release: Decrease initial dose to one-third to one-half the usual starting dose; titrate carefully.	Side effects and risks of addiction/diversion as per hydrocodone. NEVER start long acting opioid before determining needs with short acting.
Other Considerations		
Duloxetine	Start 20–30 mg/day; increase to 60 mg/day in 7 days. Not recommended in ESRD or CLcr<30.	May precipitate serotonin syndrome when combined with triptans, tramadol, and other antidepressants. Key drug-disease interactions: HTN, uncontrolled narrow-angle glaucoma, seizure disorder. Precipitation of mania in patients with bipolar disorder. Important adverse effects include nausea, dry mouth, sedation/falls, urinary retention, and constipation. Contraindicated with hepatic disease and heavy alcohol use. Abrupt discontinuation may result in withdrawal syndrome. Contraindicated within 14 days of MAOI use.

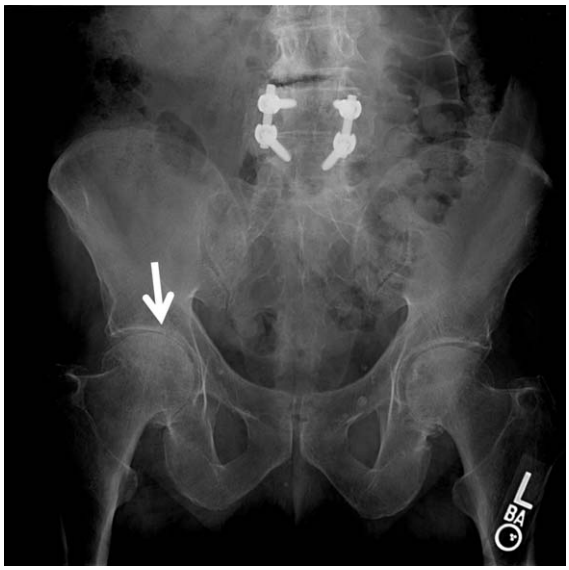

Figure 2 AP pelvis X-ray demonstrating severe joint space narrowing and subchondral sclerosis of the right hip (arrow) and mild joint space narrowing of the left hip

as well as dose-adjustments according to renal and hepatic function, and we highlight important drug-drug and drug-disease interactions in older adults. Note that we include nonacetylated salicylates but not nonsteroidal anti-inflammatory drugs (NSAIDs), in keeping with the 2012 Beers Criteria for potentially inappropriate medications for older adults [21]. Celecoxib has many of the same deleterious effects as other NSAIDs and also is not recommended [40]. While the 2012 American Geriatrics Society (AGS) Beers Criteria do not include celecoxib as a contraindicated drug; the AGS 2009 Pain Guidelines recommend against its chronic use in older adults [22]. Opioids are not part of the 2012 AGS Beers Criteria. We include specific opioid prescribing recommendations in Table 2. Our list includes those opioids with the greatest evidence for a reasonable benefit/risk ratio in older adults. We do not include methadone because of its complicated and variable pharmacokinetics and pharmacodynamics. We believe that its prescription should be overseen by a pain specialist. Meperidine, which is on Beers list, has a metabolite with significant risk of neurotoxocity and should be avoided in older adults [41]. Note also that sustained release opioids should only be started in patients only after a short acting agent has been initiated and titrated to effect.

Insufficient research has been performed to strongly recommend any one complementary or alternative intervention over another for the management of pain related to hip OA. We recommend T'ai Chi as the first CAM modality that should be prescribed for older adults with hip OA because of strong evidence supporting its efficacy in preventing falls in older adults [27], which are an important consequence of chronic pain [42]. There also is evidence for the modest efficacy of T'ai Chi for reducing pain and improving function in older adults with hip OA [28,29]. We highlight acupuncture because of the attention it has received regarding knee OA. Inadequate data exist to recommend for or against acupuncture for the treatment of hip OA [30].

For patients with severe pain and mild to moderate degenerative disease on x-ray, it is important to make sure that all pain contributors have been identified. For example, does the patient have widespread pain and a clinical picture consistent with fibromyalgia? Is there depression, anxiety, or maladaptive coping? If so, these

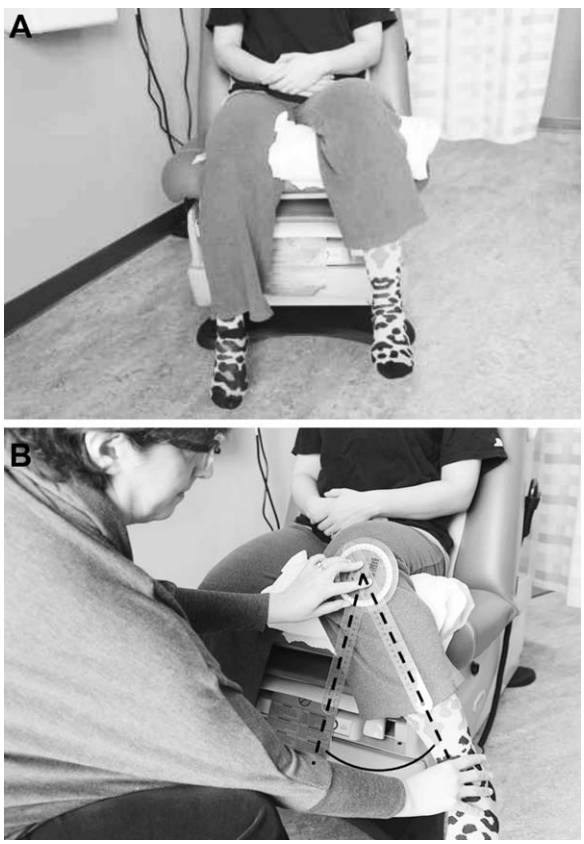

Figure 3 A: Internal Hip Rotation: Seated patient at rest, her leg elevated with a pillow to allow free movement about the femoral head. B: Internal Hip Rotation: Using a goniometer, an examiner measures internal hip rotation by placing the fulcrum on the patella, keeping the stationary arm perpendicular to the floor, and the movement arm along the midline of the tibia as she moves the foot as far away from the body as possible.

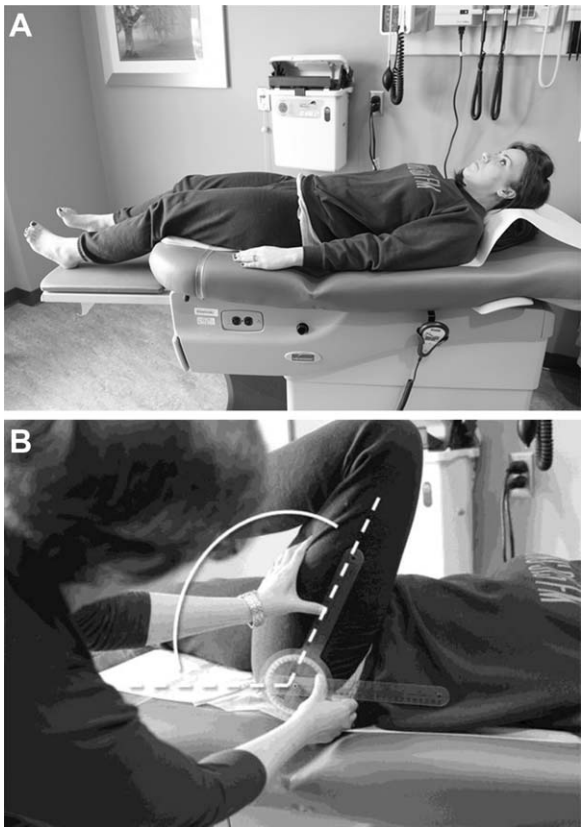

Figure 4 A: Hip Flexion: supine patient at rest, waiting to be examined for hip flexion. B: Hip Flexion: An examiner measures hip flexion with a goniometer on a supine patient by positioning the fulcrum over the greater trochanter, the stationary arm parallel with the patient's spine, and the movement arm along the femur.

conditions should be treated simultaneously with treatment of the hip OA, as we will outline in future algorithms of this series. At the same time, the conservative care listed in Figure 1 and described above should be followed. If the patient has severe pain, advanced degenerative disease on x-ray, and no contraindications to surgery, THA should be considered. If such a patient is not a surgical candidate, a discussion about initiating opioids will likely occur. Part of this discussion must include education about the risk of falls and hip fracture [43,44] and, therefore, the need to optimize mobility and stability prior to starting on these drugs. Preliminary data suggest that opioids may impair function in older adults with OA [45], thus the decision to start them must be accompanied by a frank discussion of their risks and that the main benefit is pain reduction, not improved function.

PT is recommended commonly for older adults with OA. While some data support the use of manual PT for patients with hip OA [46,47], recent data fail to support its efficacy in reducing pain or improving function [48]. In our practice, we have found that teaching caregivers how to perform simple hip distraction techniques may be helpful for short term pain relief.

Resolution of Case

The patient had what Offierski classified as "complex" HSS, that is, symptoms from both the hip and spine without a clear single source of disability. In such patients, injection of the hip and/or the spine may help to target treatment [2]. Following intra-articular hip injection, our patient experienced complete elimination of his buttocks and leg pain, modest improvement in his LBP, and marked improvement in his functional status, supporting that a large part of the patient's disablement was related to his hip OA. The patient underwent an orthopedic surgery consultation and was scheduled for THA. In the course of his preoperative evaluation, he was found to have aortic stenosis and valve replacement was recommended prior to THA, which the patient declined. The patient continues to be active and manages his pain with oxycodone. He has been educated about the risk of falls and hip fracture associated with opioids in older adults [43,44]. The risk-taking behavior that he describes is appropriate and he has not had any falls. He maintains realistic treatment expectations, focusing on his ability to function despite the persistence of some pain. Two years following initial Pain Clinic consultation, the patient continues to be functional and he is followed regularly by his primary care provider with as expected urine drug screen findings (i.e., presence of oxycodone and absence of nonprescribed drugs).

Summary

The lumbo-pelvic-hip complex should be considered as a unit when evaluating patients with CLBP. Because of the ubiquitous nature of imaging-identified degenerative spinal disease in older adults, examination of factors outside of the spine is critical in devising treatment strategies. Physical examination of the hips should occur routinely in these patients and when there is evidence of hip OA, x-rays should then be pursued to formalize diagnosis.

In the patient with "complex" HSS, that is, symptoms from pathology in both the hip and spine without a clear single source of disability, diagnostic blocks such as spinal and/or intra-articular injections may be helpful in identifying the main driver of disability. Given that these patients have chronic pain and multiple sites of pathology, realistic treatment expectations must be maintained.

Before starting oral analgesics in older adults, risks and benefits must be weighed carefully. Chronic NSAIDs, used commonly in younger patients, is not recommended for older adults [21]. This also is true for celecoxib [22]. Opioids are not without risk and prior to their

initiation, falls risk should be assessed and minimized where possible with the help of a physical therapist and an assistive device. There is no evidence that opioids improve function in patients with chronic pain. This fact must be emphasized to patients before starting these medications. All other reasonable treatment options, including evaluating and treating other contributors to pain and disability, should be exhausted before opioids are considered.

Older adults with CLBP often have multiple physical contributors to their pain and difficulty functioning including an estimated one in four likelihood of hip OA [11]. Thorough physical assessment that includes examination of the hips is critical to optimize treatment outcomes. Doing so has the potential to save substantial costs and morbidity and optimize function and quality of life.

KEY POINTS

1. Clinical evaluation of all older adults with CLBP should include evaluation (i.e., history and physical examination) of the hips.
2. X-rays should be used to formalize a diagnosis of hip OA, not to screen patients, as over half of pain-free older adults have radiographic evidence of degenerative hip disease.
3. Multimodal management that emphasizes non-pharmacological strategies and minimizes medications is preferred for older adults.
4. Nonsteroidal anti-inflammatory drugs used commonly for the treatment of hip OA in middle aged individuals should be avoided in older adults.
5. A decision to start opioids in older adults with hip OA and severe pain should be preceded by a discussion that highlights:

   a. Opioids may be associated with a number of potential adverse effects. Older adults should be educated specifically about the increased risk of falls and hip fractures.
   b. There is no evidence that opioids improve function, thus patients should recognize that their main purpose is for pain reduction.
   c. All other options, including treating other contributors to pain and disability should clearly have been exhausted before a decision is made to start opioids.

Acknowledgments

This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The author thanks Dave Newman, Dr. Rollin Gallagher, and Dr. Eric Rodriguez for their thoughtful review of the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult–Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment

Part II: Myofascial Pain

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Conflict of interest: The authors have indicated that they have no conflicts of interest regarding the content of this article.

Funding sources: Veterans Administration Rehabilitation Research & Development Service.

Abstract

Objective. To present an algorithm of sequential treatment options for managing myofascial pain (MP) in older adults, along with a representative clinical case.

Methods. A modified Delphi process was used to synthesize evidence-based recommendations. A multidisciplinary expert panel developed the algorithm, which was subsequently refined through an iterative process of input from a primary care physician panel.

Results. We present an algorithm and supportive materials to help guide the care of older adults with MP, an important contributor to chronic low back pain (CLBP). Addressing any perpetuating factors should be the first step of managing MP. Patients should be educated on self-care approaches, home exercise, and the use of safe analgesics when indicated. Trigger point deactivation can be accomplished by manual therapy, injection therapy, dry needling, and/or acupuncture.

Conclusions. The algorithm presented gives a structured approach to guide primary care providers in planning treatment for patients with MP as a contributor to CLBP.

Key Words. Low Back Pain; Myofascial Pain; Chronic Pain; Degenerative Disc Disease; Elderly;

Chronic Low Back Pain; Lumbar; Older Adults; Pain Disorder; Spinal Stenosis

Introduction

Myofascial pain (MP) as first described by Travell and Simons, is defined by a localized region of palpable tightness and tenderness within a muscle that is characterized by resistance to passive elongation, and reproduction of a predictable pattern of referred pain on palpation [1]. The pathogenesis of MP is not fully understood, but can be a local muscle response to underlying mechanical factors (postural abnormalities, biomechanical faults, chronic strain), or a response to altered neurotrophic factors secondary to spondylosis [2–4].

A characteristic feature of MP is the presence of localized palpable tender regions called trigger points (TrP). These have been identified on microscopic evaluation of involved muscles [5] and exhibit a distinct biochemical profile (i.e., inflammatory mediators, neuropeptides, cytokines, and catecholamines) as compared with normal muscles [6,7]. The elevated tissue tension in TrP was shown to be decreased by the administration of general anesthesia, supporting a spinal segmentally mediated etiology [8]. It has also been proposed that TrP may have a bidirectional relationship with central sensitization, being both a cause as well as an effect. Preliminary evidence suggests the prolonged nociceptive input from TrP can sensitize dorsal horn neurons, whereas the referred pain phenomenon seen in TrP may in fact be the result of central sensitization [9].

Studies have reported TrP prevalence ranging from 30% to 93% [10]. Using a structured examination, latent or active TrPs were identified in 93% of community-dwelling older adults with chronic low back pain (CLBP) attending a university-based pain management program [11]. Latent TrPs are painful when palpated, but the palpation-induced pain does not reproduce the patient's spontaneously reported pain, as occurs with active TrP. A subsequent study in older veterans with CLBP identified active TrP in approximately half of participants (Weiner, unpublished data), supporting MP as an important treatment target in older adults with CLBP.

Despite the commonplace nature of MP, allopathic medical education does not routinely include instruction in its evaluation and treatment. Thus, primary care physicians are often not confident in their ability to diagnose MP [12] and overlook it as a contributor to CLBP. This can result in misdirected, often suboptimal, unnecessarily invasive, and potentially dangerous treatments being prescribed [13]. This article presents an algorithm for managing MP in older adults, along with an illustrative case description.

Methods

This work was part of a larger project described previously [14]. We used a modified Delphi process to develop an algorithm (Figure 1) and evidence table (Table 1) providing the rationale for the individual algorithm components. The project principle investigator (DW) drafted an evidence-based treatment algorithm and evidence table, which were subsequently refined by an expert panel. The panel used the strongest available published evidence, supplemented by expert opinion and clinical experience as appropriate. The panel comprised five members, selected based on their recognition and expertise in their individual fields, representing geriatric medicine, pain medicine, physical medicine and rehabilitation, physical therapy and chiropractic.

The materials were then distributed to a 9-member primary care provider review panel that provided feedback using a structured questionnaire. The expert panel used this feedback to make additional modifications, and the process was repeated until no further revisions were recommended.

Case Presentation

Relevant History

The patient is a 72-year-old male presenting to his Department of Veterans Affairs (VA) primary care provider (PCP) with a 4-year history of low back pain attributed to a motor vehicle accident. He complains of a burning pain in the right lower lumbar region radiating to the right upper gluteal region with an average intensity of 5/10. The pain is present every day, approximately 50% of waking time each day, typically brought on by prolonged standing or walking, and relieved by sitting or lying down, although prolonged sitting also could cause pain. He denies lower extremity pain, weakness, numbness, tingling, unexplained weight loss, and bladder or bowel problems. He states, at the time of the accident, he was diagnosed with a bulging disc, and was prescribed ibuprofen, physical therapy (ultrasound, bicycle exercise, and core strengthening), chiropractic care (lumbar spinal manipulation and traction) and two rounds of epidural steroid injections. He reports no lasting improvement after any of these interventions. He continued to take over the counter ibuprofen 400 mg, two times per day. Over the past 4 months, his pain has been preventing him from bowling and playing with his grand-daughter, so he increased ibuprofen to 600 mg, two times per day, but he began to experience abdominal pain. He was prescribed oxycodone 5 mg/acetaminophen 325 mg, one pill every 6 hours as needed for pain, however, this

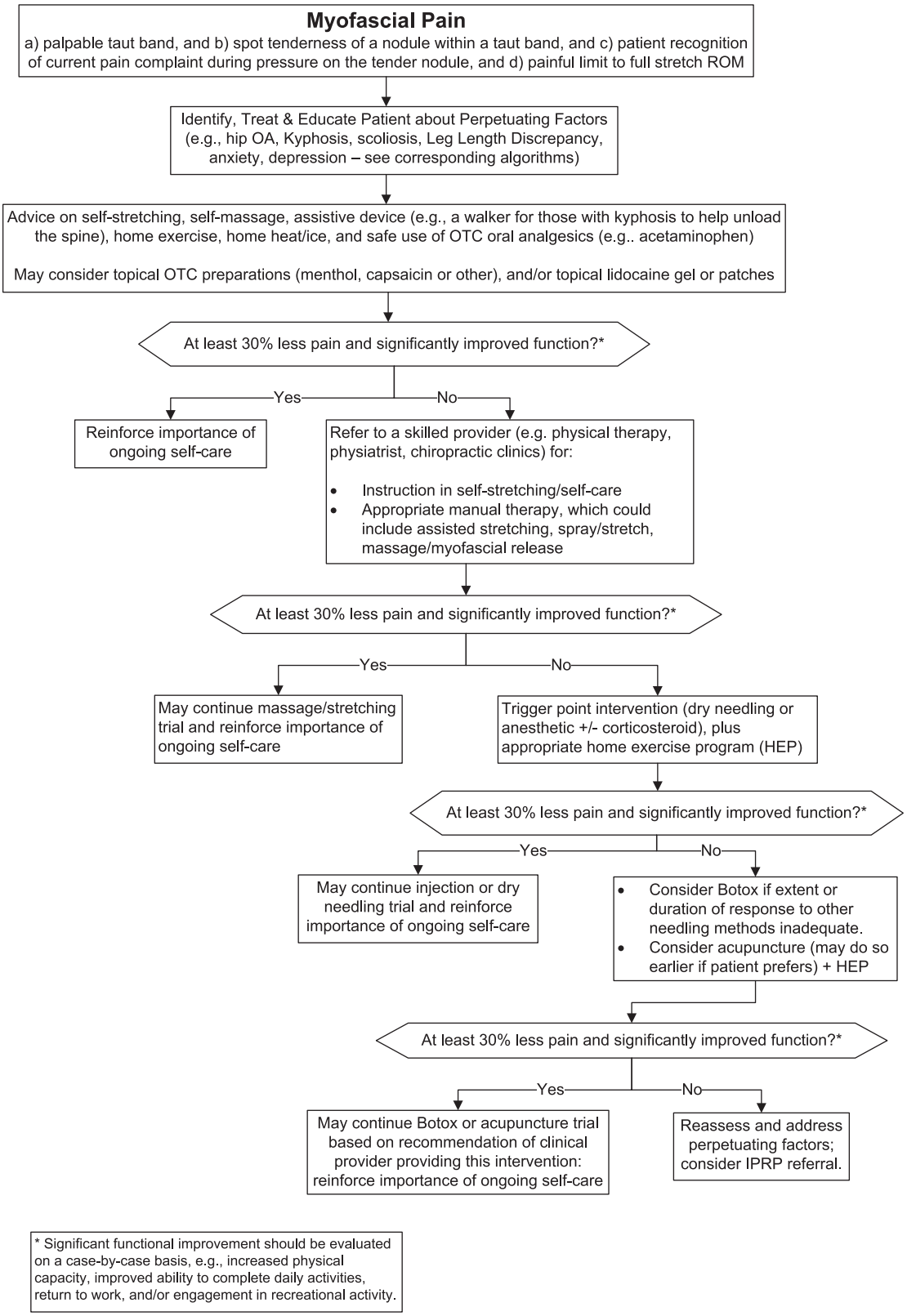

Figure 1 Algorithm for the evaluation and treatment of myofascial pain in an older adult.

Table 1 Myofascial pain: theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
30% pain reduction as significant	Data on 2724 subjects from 10 placebo controlled trials of pregabalin in diabetic neuropathy, postherpetic neuralgia, CLBP, fibromyalgia, and OA. Myofascial pain was not one of the conditions studied.	[15]
	In older adults with chronic low back pain myofascial pain is most often a pain comorbidity (i.e., accompanies the above conditions) rather than a sole contributor.	[11]
Importance of identifying and treating perpetuating factors Key role of manual therapy	Travell and Simons published the seminal and authoritative textbook on myofascial pain. Within this textbook, numerous treatment techniques are outlined in detail.	[16]
Trigger point deactivation with injection versus dry needling.	Numerous studies have been performed with highly variable quality, supporting trigger point injection, with or without injectate (i.e., dry needling). One study [17] purported that the critical therapeutic element is the local twitch response.	[17,18]
Botulinum toxin	There is neither strong evidence for nor against the use of botulinum toxin for the treatment of myofascial pain. Based on this, we recommend referral only if other interventions have failed.	[19]
Oral medications	A number of variable quality trials have been performed that suggest a number of oral agents may benefit those with myofascial pain including tizanidine, cyclobenzaprine, clonazepam, alprazolam, diazepam, and amitriptyline. Because of the potential for adverse CNS effects, these medications should be used with extreme caution in older adults.	[18,20]
Topical medications	As with oral medications, trials of topical medications have been of variable quality and, therefore, there is no strong evidence to recommend their use. Because of their favorable safety profile topical lidocaine, methylsalicylate, menthol, diclofenac, and thiocolchicoside can be tried.	[18]

caused sedation that interfered with activities of daily living.

Relevant Physical Examination

The patient is a pleasant, alert, and cooperative African American male in no apparent distress. His gait and station are unremarkable. There is increased low back pain at the end range of flexion and when returning to neutral from a flexed position. Straight leg raise, lumbar, hip, and sacroiliac orthopedic testing are painless [21]. Lower extremity motor strength, reflexes, and light touch are within normal limits. The lower lumbar paraspinal muscles are tight but not tender. There is a taut band in the right paraspinal musculature at the T12-L1 level, that when palpated reproduces the patient's spontaneously reported pain in the right lower lumbar and upper gluteal region.

Imaging

Lumbar magnetic resonance imaging (MRI) ordered by a previous provider revealed mild to moderate multilevel degenerative disc disease, and mild to moderate bilateral foraminal stenosis at L4-5 and L5-S1.

Clinical Course

The patient was referred to the VA chiropractic clinic, where he received manual myofascial release [22] of the thoracolumbar paraspinal muscles, and was instructed in self-massage with topical capsaicin cream. He was also taught a few key stretching and postural exercises. After four sessions, the patient reported pain had decreased to 2/10 intensity on average, and frequency had decreased to 3 days per week, with duration decreased to 10% of waking hours on those days. He was able to resume bowling and playing with his grand-daughter. He rated this as a 75% global impression of change. He was treated another two times with no further improvement, thus was offered a consultation for TrP injection. The patient was satisfied with the current outcome and declined any additional follow-up.

Approach to Management (Figure 1)

This patient with CLBP was presumed to have MP as a major contributor to his pain and functional limitations. Even the burning and radiating quality of his pain, which may indicate a neuropathic origin, are consistent with the diagnosis of MP. As is typical of most older adults with CLBP [11], there likely was more than one pain generator in this patient. Nevertheless, the key exam features seen here (muscle tightness [i.e., taut bands] with palpable point tenderness [i.e., TrP] as well as reproduction of distant [referred] pain that reproduces the patient's spontaneously reported pain) give the clinician a plausible mechanical target that can be approached in a systematic fashion. Common locations of TrPs thought to contribute to CLBP are the lower thoracic and lumbar erector spinae, the quadratus lumborum, and the gluteal muscles [1].

It is worth noting that before presenting to his VA PCP, this patient was initially managed by a private physician group that ordered the MRI and gave the patient the diagnosis of lumbar disc disease. In the majority of low back pain cases, advanced imaging is not indicated [23], and the findings have no bearing on the diagnosis of MP.

As shown in the algorithm (Figure 1), the initial approach to managing MP is to address any potential perpetuating factors such as scoliosis, leg length discrepancy, hip pathology, depression or anxiety, as these all may contribute to chronic muscle tightness. Appropriate treatment of any of these factors improves the likelihood of success of subsequent treatment of the MP. When possible, any factors that acutely precipitated an episode of MP also should be identified, and ameliorated to the greatest extent possible. Medication contributors to muscle pain and/or dysfunction, such as statins, also should be modified if possible [24]. Systemic illnesses such as Parkinson's disease that cause muscle dysfunction and may perpetuate myofascial pain also should be targeted as part of comprehensive treatment [25].

Providers should counsel patients on the importance of self-care including stretching, superficial heat/ice, self-massage, appropriate topical preparations, and the use of acetaminophen or other safe analgesics when indicated. These relatively simple measures are often beneficial and can empower patients to be active participants in their own health. However when these measures alone are insufficient, the next management step would be TrP deactivation. As the name implies, TrP deactivation aims to neutralize the chronically hyperactive/hypersensitive region of the involved muscle. This can be accomplished by manual therapy, injection therapy, and/or dry needling.

The manual therapy approaches to TrP deactivation essentially fall into two categories: stretching techniques (postisometric relaxation, spray and stretch, etc.) and/or massaging techniques (ischemic compression, myofascial release, etc.). In this case, the manual therapy was provided by chiropractors, yet these treatments also can be provided by medical/osteopathic physicians, physical therapists, and others with appropriate training. The choice of technique and degree of mechanical load applied must be tailored to the individual patient's tolerance. In this case, the patient reported satisfactory improvement after a short course of manual therapy and home instructions. Had there been little or no improvement to this initial intervention, management could have evolved in a stepwise fashion to include wet needling (i.e., injection of an anesthetic +/− corticosteroid) or dry needling, Botox injections or acupuncture. While wet needling is practiced commonly, evidence does not indicate that it is superior to dry needling, the introduction and subsequent manipulation of an acupuncture or hypodermic needle into a TrP, without any injectate [26]. The critical therapeutic element in both approaches is thought to be obtaining a local twitch response [27]. As depicted in Figure 1, all of these interventions should be accompanied by ongoing self-care and reassurance. Patients should be educated that judicious use of passive therapies can be appropriate, but that long-term benefit requires active patient engagement. As many factors may facilitate or impede a patient's engagement in self-care and physical activity [28], appropriate management should include collaborative goal setting [29].

Another important aspect of treating MP in older adults is building muscular resilience. As noted in the introduction to this CLBP series, a central concept in gerontology and in caring for older adults is *homeostenosis*, defined as the progressive restriction of an organism's ability to respond to stress as it ages [30,31]. Sarcopenia, diminished muscle bulk and quality associated with normal aging, is one component of *homeostenosis* in the muscular system [32,33]. It is possible that sarcopenia is an important perpetuating factor for MP in older adults, possibly accounting for the high prevalence of MP in these patients. Because of the undeniable presence of sarcopenia in all older patients, resilience building through a long-term home exercise program is essential.

The biopsychosocial model of chronic pain requires clinicians to consider other aspects in addition to the involved musculoskeletal target tissue. This patient exhibited no significant psychosocial contributors to his CLBP and difficulty functioning, For many patients with chronic musculoskeletal pain in general [34–36], and MP in particular [37], depression, anxiety, and other mental health conditions do contribute to pain and disability. Concurrent treatment of depression and pain has been shown to yield more favorable outcomes for both conditions [38]. Depending on the factors that have been

identified to perpetuate the patient's MP, optimal management may require a collaborative team approach including psychiatry, health psychology, addiction medicine, social work, and/or other disciplines [39,40]. A patient-centered approach including shared decision making has been shown to result in improved outcomes [41].

Resolution of Case

The patient continued to follow with his PCP and reported no significant back pain for the next 12 months. He remained fully functional in his desired activities of daily living. He was somewhat compliant with the active care instructions over this time, but he eventually discontinued his home exercise and self-management program. One year later he returned with increased symptoms and no new precipitating incident or perpetuating factors. His PCP advised the patient to resume home exercises and self-care, and if the pain remains bothersome he will be referred for manual therapy or TrP injection.

It is worth highlighting most nonsteroidal anti-inflammatory drugs (NSAIDs) are included in Beers Criteria for potentially inappropriate medications in older adults [42]. The older patient presented developed adverse effects associated with NSAIDs that could have been avoided had treatment been targeted specifically to his MP.

Summary

Providers should consider the contribution of MP in older adults with CLBP. Current evidence supports a number of interventions for CLBP but none have been shown to be clearly superior [23,43]. Identification and treatment of MP as part of the CLBP syndrome [14] requires neither imaging nor exposure to procedures with significant risk. Thus, prioritizing its identification and treatment, along with factors that precipitate and/or perpetuate it has the potential to substantially benefit quality of life with minimal associated risk. The MP algorithm presented in this article provides guidance for a stepwise approach that takes into account risk/benefit and patient preference. For optimal treatment planning, goals and expectations must be congruent among patients, primary care providers, and specialists.

Acknowledgments

This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The authors thank Dave Newman for his thoughtful review of the manuscript.

KEY POINTS:

1. Clinical evaluation of all older adults with CLBP should include muscle palpation to identify taut bands and trigger points associated with MP.
2. Characteristic features of MP include:
   a. History: acute, subacute or chronic regional muscle pain and/or stiffness
   b. Exam findings: Palpable taut band(s) within the muscle + spot tenderness of a nodule (i.e., trigger point), palpation of which reproduces spontaneous pain with a predictable referral pattern (i.e., active trigger point) ± a local twitch response.
   c. Painful limit to full stretch range of motion.
3. Deactivation of trigger points can be accomplished with manual therapy, dry needling, or wet needling. No one approach is clearly superior to another.
4. A key part of treating MP includes treating precipitating and perpetuating factors.
5. While some oral medications have demonstrated modest efficacy for treating MP, their risk-benefit ratio is prohibitive in older adults.
6. Active engagement in self-management that includes a home program to manage MP flares and build muscular resilience is a critical part of treating chronic MP in older patients.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment

Part III: Fibromyalgia Syndrome

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Conflicts of interest: The authors have indicated that they have no conflicts of interest regarding the content of this article.

Abstract

Objective. To present the third in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. The series presents CLBP as a syndrome, a final common pathway for the expression of multiple contributors rather than a disease localized exclusively to the lumbosacral

spine. Each article addresses one of 12 important contributors to pain and disability in older adults with CLBP. This article focuses on fibromyalgia syndrome (FMS).

Methods. A modified Delphi approach was used to create the evaluation and treatment algorithm, the table discussing the rationale behind each of the algorithm components, and the stepped-care drug recommendations. The team involved in the creation of these materials consisted of a principal investigator, a 5-member content expert panel, and a 9-member primary care panel. The evaluation and treatment recommendations were based on availability of medications and other resources within the Veterans Health Administration (VHA) facilities. However, non-VHA panelists were also involved in the development of these materials, which can be applied to both VA and civilian settings. The illustrative clinical case was taken from the clinical practice of the principal investigator.

Results. Following expert consultations and a review of the literature, we developed an evaluation and treatment algorithm with supporting materials to aid in the care of older adults with CLBP who have concomitant FMS. A case is presented that demonstrates the complexity of pain evaluation and management in older patients with CLBP and concomitant FMS.

Conclusions. Recognition of FMS as a common contributor to CLBP in older adults and initiating treatment targeting both FMS and CLBP may lead to improved outcomes in pain and disability.

Key Words. Fibromyalgia; Low Back Pain; Elderly; Chronic Pain; Back Pain

Introduction

Fibromyalgia syndrome (FMS) is a challenging diagnosis for many health care providers given the breadth of symptoms patients have on presentation and the paucity of specific objective findings. Twenty-five years ago, FMS was initially described as a syndrome characterized by widespread musculoskeletal pain that could not be explained by another diagnosis [1]. FMS has been increasingly recognized to encompass additional features such as fatigue and nonrestorative sleep, and these other symptoms are included in the updated 2010 American College of Rheumatology (ACR) criteria [2]. The prevalence of FMS increases with age, has a female preponderance, peaks in the seventh decade, and varies from $<1\%$ to 5% [3].

Prior reports have shown a relationship between chronic low back pain (CLBP) and widespread pain among patients in a variety of settings. A cross-sectional postal questionnaire study of musculoskeletal symptoms in the community reported that 893 of the 2,893 respondents (31%) experienced low back pain in the previous week with 222 (24%) of these individuals having localized low back pain and 281 (31%) reporting widespread pain in at least four other areas [4]. Recently, a large cross-sectional comparative analysis of 647 patients who were seen for CLBP in a primary care setting revealed that approximately 25% of these individuals also experienced chronic widespread pain as defined by the 2010 ACR criteria for fibromyalgia [5]. Those patients with CLBP and chronic widespread pain were more likely to be female and have more somatic symptoms and comorbidities than patients with only CLBP. Over 40% of patients with a primary spine diagnosis who presented to an academic outpatient pain clinic met survey criteria for FMS [6]. In our clinical experience, one in five older adults with CLBP has evidence of FMS, a prevalence that has also been suggested by other investigators [7].

Because of the high prevalence of chronic widespread pain among patients with CLBP, health care providers should perform a thorough history, physical examination, and limited laboratory testing to determine if FMS is a potential contributor to the pain and disability experienced by older adults with CLBP. Patients with FMS may experience pain in the low back, hips, and buttocks that could be mistaken for pain arising from disorders of the lumbar spine such as spinal stenosis or radiculopathy. FMS is under-recognized in older adults as Jacobson et al. found that in older adults, it took an average of 7 years for the diagnosis of FMS to be made from the time of symptom onset [8]. Moreover, many of these patients were treated with inappropriate medications such as opioids and had persistent, uncontrolled symptoms [8].

Early recognition of FMS and other extra-spinal factors that may directly contribute to CLBP or independently cause pain and disability may affect management and optimize patient outcomes. We present an older patient with chronic upper and lower back pain who had coexisting FMS. This case demonstrates the multitude of symptoms that patients with CLBP and comorbid FMS often report and the success that can be achieved with a multifaceted approach to treatment.

Methods

A detailed description of the modified Delphi method consisting of a content expert panel and primary care review panel is provided in the series overview [9]. This technique was used to create the algorithm (Figure 1), the table detailing the rationale behind the algorithm components (Table 1), and the stepped-care medication table (Table 2).

The five expert panel members for the FMS algorithm included a geriatrician, physical therapist, geriatric psychiatrist, pharmacist, and a rheumatologist.

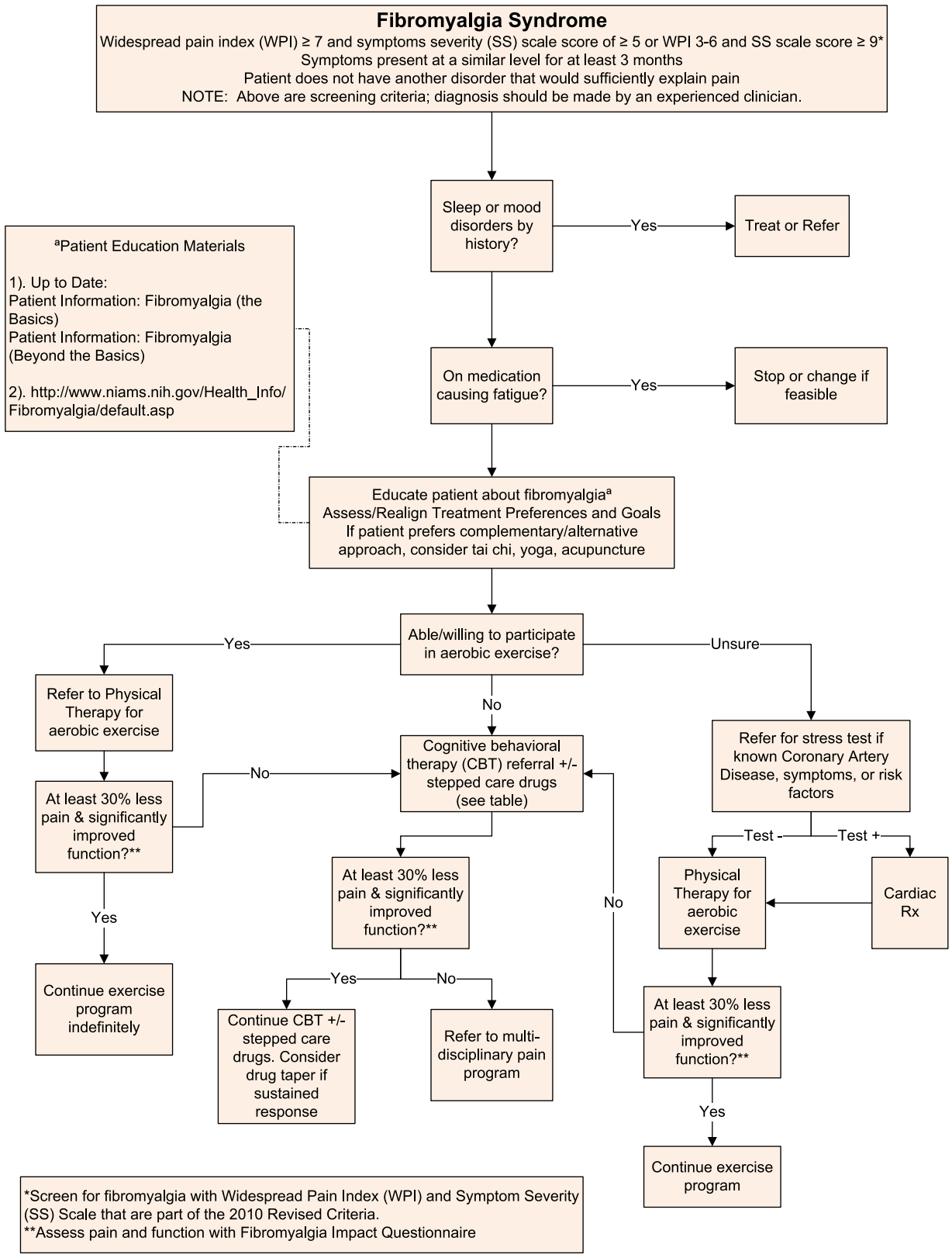

Figure 1 Algorithm for the evaluation and treatment of fibromyalgia syndrome in an older adult.

Table 1 Fibromyalgia syndrome: theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm Component	Comments	References
30% pain reduction as significant	Data on 2,724 subjects from 10 placebo controlled trials of pregabalin in diabetic neuropathy, postherpetic neuralgia, chronic low back pain, fibromyalgia, and osteoarthritis.	[21]
Aerobic exercise early in Rx	Strong efficacy evidence in general population of patients with fibromyalgia; not tested explicitly in older adults.	[41–43]
	Strong efficacy evidence of multiple exercise benefits in older adults in general (i.e., not specifically in those with fibromyalgia).	[26,44]
Cognitive behavioral therapy (CBT)	Strong efficacy evidence in general population of patients with fibromyalgia.	[41]
	High quality evidence is lacking specifically in older adults. Arthritis pain self-management programs that contain some CBT elements demonstrate efficacy.	[45]
Patient education	There is little evidence-based data on the optimal patient education program. Experts have recommended individual and/or group education presented by health professionals knowledgeable about fibromyalgia. Education sessions would cover information about symptoms, course of fibromyalgia, comorbid conditions, potential etiologies for fibromyalgia, role of psychosocial factors in contributing to pain, pharmacologic and nonpharmacologic therapy, and self-management approaches. Education could also focus on strategies to prevent nocebos. Suggested Patient Education materials: UpTo Date: Patient Information: Fibromyalgia (The Basics) Patient Information: Fibromyalgia (Beyond the Basics) National Institute of Arthritis and Musculoskeletal and Skin Diseases: http://www.niams.nih.gov/Health_Info/Fibromyalgia/default.asp	[22–24]
Sleep disorder evaluation	Sleep disorders are common in people with fibromyalgia and may be a risk factor for developing this condition.	[46,47]
Mood disorder evaluation	Fibromyalgia is frequently associated with psychiatric disorders such as anxiety and depression.	[15]
Medications causing fatigue	Chronic fatigue is a common problem in people with fibromyalgia. Minimizing the use of medications which cause fatigue may help alleviate symptoms of fatigue. Some medications that can cause fatigue include benzodiazepines, skeletal muscle relaxants, some antidepressants, antipsychotics, and anticonvulsants.	[19]
Gabapentin^{OL}	Not evaluated specifically in older adults with fibromyalgia. Recommended as first line in veterans as it is on formulary.	[48]
Duloxetine and Venlafaxine	Duloxetine is FDA approved for the treatment of FMS.	[43,49]
Milnacipran	FDA approved for the treatment of fibromyalgia. Not recommended as not available in VA, even nonformulary.	[43,50]
Pregabalin	FDA approved for treatment of fibromyalgia. Non-formulary in VA.	[51–53]
Nortriptyline^{OL} and Desipramine^{OL}	There is strong efficacy evidence for amitriptyline in the treatment of fibromyalgia, but this tricyclic antidepressant has strong anticholinergic side effects in older adults and is not recommended (on Beers list). Neither nortriptyline nor desipramine are on Beers list, thus if a tricyclic antidepressant is to be initiated, these are the preferred agents. Both are on formulary at the VA.	[39,41,43]
Cyclobenzaprine is absent from the algorithm.	There is strong efficacy evidence for cyclobenzaprine in the treatment of fibromyalgia, but because of strong anticholinergic side effects, it is on Beers list and not recommended for older adults.	[39,41]

Case Presentation

Relevant History: A 67-year-old female presents to the Pain Clinic in 2013 for evaluation of her chronic upper and lower back pain. During the 1980s, she injured her neck while playing volleyball. In 1999, she was involved in a car accident and experienced a whiplash injury to her neck. She has never experienced a spinal fracture and denies any history of back surgery. She continues to experience occasional neck pain. She also has shoulder pain that intermittently disrupts her sleep because she has to change positions in bed to feel better. For several years, she has suffered from bilateral right more than left sided upper back pain and lower back pain, as well as left-hand fourth and fifth digit numbness and tingling.

Last autumn, while she was working in her garden, she experienced worsening of her chronic upper back and lower back pain. The back pain is nonradiating and not associated with weakness in her upper or lower extremities. The pain is relieved with rest, exacerbated by activity, and does not awaken her at night. On occasion, she also notes right-sided abdominal pain and bilateral finger pain. She reports a history of chronic fatigue. She denies having any problems with her memory, fever, nightsweats, anorexia, weight loss, and changes in bowel or bladder habits. She tried tramadol without significant relief of her pain. Meloxicam relieves her back pain more effectively than tramadol. She also completed a course of physical therapy that alleviated her pain while she was participating in the program.

Relevant Physical Exam: The patient is awake, alert, fully oriented, cooperative, and in no acute distress. A physical exam is notable for several tender points of her neck and upper and lower back muscles after applying enough thumb pressure to cause the nailbed to blanch. The range of motion of her back and extremities is intact. Tenderness is noted over bilateral Heberden's and Bouchard's nodes.

Relevant Tests: The patient completes a FMS patient self-report survey [10] that reveals a score of 9 on the Widespread Pain Index (WPI) and 6 on the Symptom Severity (SS) score (see Figure 2). Complete blood count, chemistries, liver function tests, erythrocyte sedimentation rate, C-reactive protein, creatine kinase, and thyroid stimulating hormone were all unremarkable. The diagnosis of FMS was confirmed by a rheumatologist.

Clinical Course: We educated the patient regarding the diagnosis and treatment of FMS, her role in its management, and setting realistic treatment goals. We encouraged physical therapy specifically focused on aerobic exercise. She began gabapentin 100 mg by mouth every evening for a week that was titrated to 200 mg every evening. Gabapentin decreased her pain but this medication was eventually discontinued because of a 20 pound weight gain. Acupuncture, trigger point therapy, and aerobic exercise were recommended for the pain of her neck, upper back, and lower back. Topical lidocaine, transcutaneous electrical nerve stimulation, and acupuncture were ordered for the abdominal muscle pain. For her painful hand osteoarthritis, we prescribed acetaminophen, 1000 mg three times daily, and meloxicam as needed. She maintains realistic treatment expectations, has embraced self-management of her pain, and now focuses her energy and attitude on her ability to function despite the persistence of some pain.

Approach to Management

The patient presented came for evaluation of chronic upper and lower back pain and was ultimately diagnosed with FMS, a centralized pain disorder, that can contribute to pain and disability in people with CLBP. The finding of chronic widespread pain among patients with CLBP is common in the primary care setting [4–7]. Evaluating a patient for FMS should be performed in patients who have diffuse pain that is not entirely explained by injury or inflammation. Once the diagnosis of FMS has been made, a multidisciplinary approach to treatment is recommended. The expert panel that created the FMS algorithm (Figure 1) recommended assessing for the potential presence of a centralized pain disorder using ACR survey criteria for FMS. Unlike the original 1990 ACR survey criteria for FMS, the 2010 ACR criteria no longer require a tender point examination but do acknowledge that widespread musculoskeletal pain is accompanied by cognitive and somatic symptoms that are measured via physician assessment, the WPI, and SS Scale [2,3]. A 2011 modification of the 2010 ACR survey criteria for use in clinical and epidemiological studies led to the development of a patient self-report questionnaire (Figure 2) [10] that can be easily administered in the clinic setting and determines whether or not a patient meets ACR survey criteria for FMS. In this patient, the history, physical examination, and basic laboratory testing were performed to exclude other conditions that may mimic FMS. [6]. Evaluation with serologic tests such as the antinuclear antibody and rheumatoid factor is not indicated unless the patient has signs and symptoms suggestive of a systemic inflammatory rheumatic disease.

As shown in Figure 1, management of FMS in patients with CLBP starts with evaluating the patient for the presence of potentially modifiable risk factors, such as sleep and mood disorders, that are commonly seen in both CLBP and FMS. Over 80% of patients with FMS experience one or more sleep problems, such as difficulty falling asleep, staying asleep, or waking up too early and these sleep problems are associated with a decrease in health-related quality of life [11]. More than 60% of adults with CLBP have insomnia with pain intensity and fatigue as the main determinants associated with insomnia [12]. In general, insomnia is a common complaint in older adults with over 50% having difficulty

Table 2 Stepped care drug management of fibromyalgia*

Drug	Dose/Titration	Important Adverse Effects/Precautions
Gabapentin	Start 100 mg nightly. Increase by 100 mg weekly. Renal dosing: CLcr ≥30 mg/min, titrate to 600 mg BID; CLcr 15–29 mL/min, titrate to 300 mg twice a day; CLcr <15 mL/min, titrate to 300 mg daily. Supplement dose after dialysis.	Confusion, dizziness, somnolence, peripheral edema, weight gain. Withdrawal syndrome with abrupt discontinuation.
Nortriptyline or Desipramine	Start 10 mg at night. Increase by 10 mg weekly to max dose of 50 mg at night. Lower doses and slower titration recommended with hepatic impairment.	Constipation, orthostatic hypotension, urine retention. Anticholinergic; may exacerbate narrow-angle glaucoma, BPH; falls, delirium, seizures, BBB. Can prolong QT and cause Torsades de Pointes. Get EKG before starting.
Pregabalin	Start 25–50 mg at night. Increase by 25–50 mg weekly up to 100 mg twice a day. Max dose 300 mg daily. Renal dosing: CLcr 30–60 mL/min, adjust dose to 150–300 mg daily; Clcr <15 mL/min, use no more than 75 mg/d.	Confusion, dizziness, somnolence, peripheral edema, weight gain.
Duloxetine	Initiate 20–30 mg daily. Increase to 60 mg after 1–2 weeks. Max dose 60 mg daily. Not recommended in ESRD or with CLcr < 30 mL/min. Not recommended for use in hepatic impairment.	Nausea, dry mouth, sedation/falls, urinary retention, and constipation. Abrupt d/c may cause withdrawal syndrome; contraindicated with hepatic disease and/or heavy EtOH.
Venlafaxine ER	Initiate 25 mg daily. Increase by 25 mg weekly up to 225 mg/d. ESRD: reduce dose by 50% and give after dialysis. Reduce dose by 50% in mild-moderate hepatic impairment.	Nausea, dizziness, diaphoresis, dry mouth, insomnia, constipation

* 1. Prefer monotherapy starting with low doses.
•Initial trial of tricyclic antidepressant (nortriptyline or desipramine) OR initial trial of gabapentin depending on the patient's comorbidities, preference, cost, symptoms most concerning to patient.
•If pain or symptom reduction <30% with tricyclic antidepressant, try dual reuptake inhibitor (duloxetine or venlafaxine) and see #2 below.
•If pain or symptom reduction <30% with gabapentin, try pregabalin and see #2 below.
2. If pain or symptom reduction <30% with monotherapy, can try combination therapy using low doses of medications from different classes targeting the symptoms most concerning to the patient or referral to multidisciplinary pain treatment program.
3. If pain or symptom reduction >30% for an extended period of time, decrease to lowest effective dose of medication. Consider stopping medication to see if fibromyalgia can be controlled without pharmacologic therapy. Monitor for antidepressant discontinuation syndrome such as cholinergic rebound (nausea, sweating, urinary urgency) or serotonin/norepinephrine re-uptake inhibitor discontinuation syndrome (dizziness, weakness, nausea, headache, lethargy, insomnia, anxiety, poor concentration, and paresthesias). Monitor for symptoms of gabapentin or pregabalin withdrawal such as seizures.
CLcr = creatinine clearance; BID = twice a day; ESRD = end stage renal disease; BPH = benign prostatic hypertrophy; BBB = bundle branch block; EtOH = ethanol.

going to sleep or maintaining sleep [13]. Recent studies have indicated that there is a reciprocal relationship between sleep disturbances and pain and that sleep problems are an important contributor to the initiation and persistence of chronic pain [14]. Older patients with CLBP and comorbid FMS are at significantly increased risk for a sleep disorder. If a sleep disorder is suspected, evaluation should be undertaken to determine if they may have insomnia, sleep-related breathing disorder, or sleep-related movement disorder. Medical history or medications should be reviewed to assess for potential contributors to insomnia. Minimizing use of medications aggravating insomnia as well as treating medical and psychiatric conditions contributing to sleep difficulties are recommended. Theophylline, oral decongestants such as pseudoephedrine, and stimulants such as

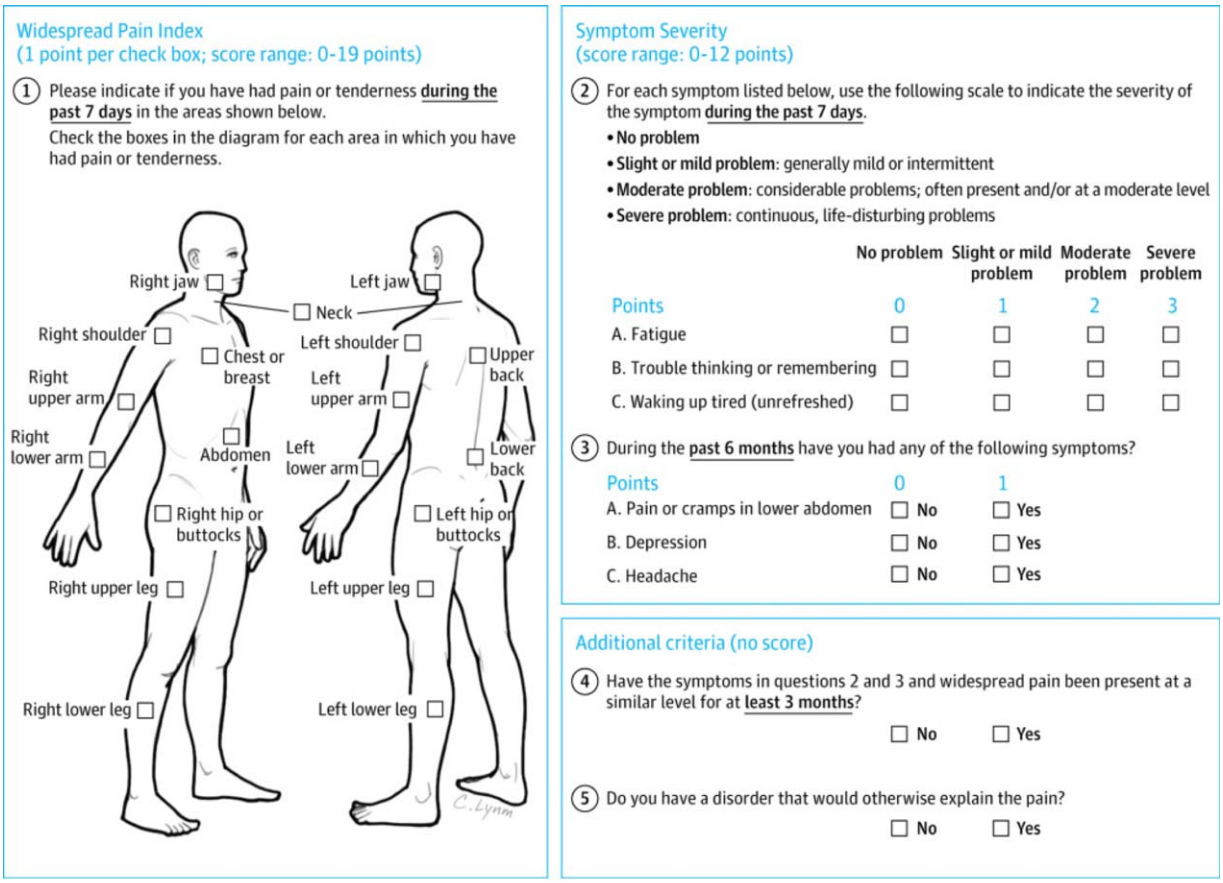

Figure 2 Patient self-report survey for the assessment of fibromyalgia based on criteria in the 2011 modification of the ACR preliminary diagnostic criteria for fibromyalgia. Scoring information is shown in blue. The possible score ranges from 0 to 31 points; a score ≥13 points is consistent with a diagnosis of fibromyalgia.

methylphenidate are examples of medications that may cause or worsen insomnia, especially in older adults. An Insomnia algorithm will be published later in this CLBP article series.

Psychiatric conditions such as depression, anxiety, obsessive-compulsive disorder, and posttraumatic stress disorder (PTSD) are often associated with FMS [6,15]. There is a higher prevalence of depression, anxiety, and PTSD in patients with FMS as compared with the general population [16,17]. In approximately 66% of patients with FMS, an experience believed to be the most burdensome traumatic event and PTSD symptoms developed prior to the onset of FMS [17]. Physicians should, therefore, screen for psychiatric conditions in FMS patients. Treatment of the psychiatric disorders should be initiated or appropriate referrals made. Algorithms on Depression and Anxiety will be published later in this CLBP series.

Fatigue is a distressing symptom among patients with CLBP and FMS and is likely influenced by the presence of pain, mood disturbance, unrefreshing sleep, and use of certain medications [18,19]. Because fatigue may occur as a result of medications that cause central or peripheral nervous system depression or lead to anemia, the patient's medication list should be thoroughly reviewed for offending drugs and stopped if feasible [19]. Examples of medications that can cause drug-related fatigue include benzodiazepines, skeletal muscle relaxants, anticholinergics, anticonvulsants, and sedating antidepressants.

The next steps in the management of CLBP complicated by FMS in older adults should focus on nonpharmacologic treatment (patient education, exercise, cognitive-behavioral therapy, complementary and alternative therapy). Patient education is aimed at helping patients set realistic treatment goals, determine

treatment preferences, reduce disability, and increase self-management skills. The primary objective of multidisciplinary treatment is aimed at shared decision making with identification of goals of therapy from the patient's perspective. The focus is on optimizing function with the understanding that it is unrealistic to expect complete resolution of pain [20]. Patients should be instructed to expect, on average, 30% reduction in pain or 2 points on an 11 point (i.e., 0–10) scale [21].

There is no consensus on an optimal patient education program. Experts have recommended individual or group education classes presented by health professionals knowledgeable about FMS. Patients with FMS have shown benefit from learning about its symptoms, the course of FMS, the role of psychosocial factors in contributing to pain, pain physiology, pharmacologic and nonpharmacologic therapy, and self-management approaches [22,23]. Patient education material can be obtained from online resources (Figure 1). Health care providers and patients should be aware of the nocebo effect, the expectation that medical intervention will cause harm, as it is prevalent in patients with FMS. Nocebos contribute to treatment failure, and patients should, therefore, be educated to help prevent poor treatment outcomes [24].

Various types of exercise (aerobic, resistance, and aquatic) are useful because they decrease pain and improve function in patients with FMS with successful exercise interventions usually involving 30–60 minutes of light to moderate intensity exercise occurring three times a week for 7 weeks [25]. Aerobic exercise is particularly recommended because it has both cognitive and physical benefit for older adults [26] and strong efficacy evidence for the treatment of FMS [27]. Depending on the older adult's experience with exercise and other comorbidities, it may be prudent to initiate such a program under the guidance of a physical therapist with the goals of self-maintained graded exercise and exercise pacing and to screen for cardiac and other medical conditions that would potentially contraindicate aerobic exercise. It is important that patients with FMS understand that the benefit of aerobic exercise diminishes if the exercise is discontinued [27]. Both land-based and pool exercises have been evaluated, and they appear to have equal efficacy and improve wellness and fitness [28,29].

For patients who are unable to participate in an exercise intervention, cognitive behavioral therapy (CBT) should be offered because it has some evidence for efficacy in the treatment of FMS with demonstrated improvement in pain, mood, and function [30,31]. Reviews specifically focused on CBT for the treatment of chronic pain in the elderly have found CBT to significantly decrease self-reported pain intensity without an effect on medication use or depressive symptoms [31].

Complementary and integrative nonpharmacologic modalities have also been evaluated in the treatment of FMS and are an attractive option for older adults because of the low risk of adverse effects. There is low to moderate evidence for acupuncture as a treatment to relieve pain and stiffness arising from FMS [32], and we have found it beneficial on a case by case basis. Tai chi, a Chinese martial art with slow meditative movements, has been promoted for its health benefits. Recent studies have shown the benefit of Tai Chi in FMS with reduced Fibromyalgia Impact Questionnaire scores and improvements in other patient-reported surveys of physical function and quality of life [33,34]. Yoga has also shown significant benefit for reducing pain and fatigue in patients with FMS [35].

If the older patient with CLBP complicated by FMS continues to have disabling widespread pain despite addressing sleep disturbances and mood disorders, providing patient education, and trying nonpharmacologic therapy, then pain medication would be the next step. The decision to prescribe medication for the treatment of FMS in older adults should be made judiciously and based on knowledge of potential drug–drug and drug–disease interactions as well as age-related changes in pharmacokinetics and pharmacodynamics. Guidelines for stepped care FMS pain management in older individuals is provided in Table 2 starting with low doses and titrating cautiously. This table includes starting dose and titration suggestions with adjustments for renal or hepatic dysfunction and highlights important adverse effects [36,37]. There are few studies that evaluate the use of pharmacotherapy in the treatment of FMS in older adults. Fitzcharles et al. note that the potential therapeutic benefit of medications used to treat FMS in the elderly is often overshadowed by adverse effects [38].

Only three medications (pregabalin, milnacipran, and duloxetine) are FDA-approved for the treatment of FMS. Despite its FDA approval for the treatment of FMS, milnacipran was not included in the stepped care drug management table because it is unavailable through the VHA. Amitriptyline and cyclobenzaprine have strong efficacy evidence in FMS, but are not included in the table because they meet Beers' criteria for potentially inappropriate medications in older adults due to their significant anticholinergic effects [39]. If prescribing a tricyclic antidepressant as step one in the treatment of FMS, nortriptyline, the active metabolite of amitriptyline, or desipramine are preferable options in older adults because they have less anticholinergic effects than amitriptyline and cyclobenzaprine.

Gabaergic analgesics such as gabapentin and pregabalin are second step agents. There is minimal evidence for gabapentin in reducing pain associated with FMS, and its use is limited because over 60% of people experience an adverse event [40]. If FMS does not respond to a step two agent, then a step three agent, a serotonin-norepinephrine reuptake inhibitor (SNRI), such as duloxetine or venlafaxine, could be tried. It is important to note that there are no studies that have

evaluated formally SNRIs in older FMS patients [22]. Nonsteroidal anti-inflammatory drugs (NSAIDs) and opioids should be avoided given their potential to do harm without clear benefit for the treatment of FMS [39].

Resolution of Case

The patient has FMS which is a major contributor to pain and disability from her CLBP. She is representative of a subset of CLBP patients with a centralized pain phenotype whose pain is amplified by central nervous system factors and unlikely to be alleviated with only peripherally directed analgesics [6]. Successful management of these patients requires evaluation for any sleep problems, mood disorders, and psychosocial stressors as well as patient education regarding the treatment goal of optimizing function despite the persistence of some pain. She was educated about the inappropriateness of opioid analgesics and the importance of self-management with physical therapy and massage. The patient participated in several weeks of aerobic exercise with great improvement in her neck and upper back pain and planned to join a club where she would have access to aquatic therapy. She tried acupuncture for her myofascial pain with benefit, albeit lasting only several days postprocedure. She was started on gabapentin 100 mg at night time but had to stop this medication because of weight gain. She has maintained realistic treatment expectations, embraced self-management of her FMS, and focused her energy and attitude on her ability to function despite the persistence of some pain.

Summary

Older adults who present for evaluation of CLBP should be assessed for FMS, as chronic widespread pain is a common finding in those with CLBP [4–6]. Importantly, FMS is a diagnosis that is often overlooked in older adults, resulting in a delay in treatment [6]. When the diagnosis of FMS is suspected, the FMS patient self-report questionnaire should be administered as a screening tool [2,10]. In the event that the patient screens positive, a specialist should be consulted to confirm FMS if doubt exists regarding the diagnosis. Important comorbidities such as depression, anxiety, sleep disorders, and PTSD should be addressed [10–18]. Medications should be reviewed to determine if they may be contributing to fatigue [19].

As is true for all patients with chronic non-cancer pain, educating patients with FMS about realistic treatment goals is critical, focusing on optimizing function [20]. A multifaceted approach to treatment can achieve significant improvements in the health-related quality of life of older adults with CLBP and coexisting FMS.

Key Points

1. Older adults with CLBP should be evaluated for fibromyalgia as a contributor to their pain and functional impairment.
2. Older adults with widespread pain should not be automatically diagnosed with fibromyalgia. It is important to conduct a thorough history and physical exam as fatigue and widespread pain may indicate a serious underlying disorder such as malignancy or connective tissue disease.
3. Evaluation and treatment should include identification of conditions (e.g. mood disorders) and/or medications that may cause or exacerbate fibromyalgia symptoms.
4. Patients with fibromyalgia should be educated about their diagnosis to help create realistic treatment goals with an emphasis on reducing (not eliminating) pain and improving everyday function.
5. An array of treatment options is available for the treatment of FMS. Older adults with FMS should be educated and encouraged about the multiple treatment options available including aerobic exercise, cognitive behavioral therapy, judicious use of stepped-care medications, and interdisciplinary treatment.

Acknowledgments

This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government.

The authors wish to thank Dave Newman for his overall assistance with this project and for his thoughtful review of the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult: Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment

Part IV: Depression

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Conflicts of interest: Dr. Karp has received medication supplies for investigator initiated studies from Pfizer and Invidior. The other authors have no potential conflicts to report.

Abstract

Objective. To present the fourth in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. The series presents CLBP as a syndrome, a final common pathway for the expression of multiple contributors rather than a disease localized exclusively to the lumbosacral spine. Each article addresses one of twelve important contributors to pain and disability in older adults with CLBP. This article focuses on depression.

Methods. The evaluation and treatment algorithm, a table articulating the rationale for the individual algorithm components, and stepped-care drug recommendations were developed using a modified Delphi approach. The Principal Investigator, a three-member content expert panel, and a nine-member primary care panel were involved in the iterative development of these materials. The algorithm was developed keeping in mind medications and other resources available within Veterans Health Administration (VHA) facilities. As panelists were not exclusive to the VHA, the materials can be applied in both VHA and civilian settings. The illustrative clinical case was taken from one of the contributor's clinical practice.

Results. We present an algorithm and supportive materials to help guide the care of older adults with depression, an important contributor to CLBP. The case illustrates an example of a complex clinical presentation in which depression was an important

contributor to symptoms and disability in an older adult with CLBP.

Conclusions. Depression is common and should be evaluated routinely in the older adult with CLBP so that appropriately targeted treatments can be planned and implemented.

Key Words. Aged; Assessment; Depression; Chronic Pain; Elderly; Low Back Pain; Primary Care

Introduction

Major depressive disorder (MDD) has a reported 1-year prevalence of 6–12% in older adults in both Veterans Affairs and civilian settings. In addition to MDD, the prevalence of clinically significant subsyndromal depressive symptoms in late-life (generally defined as $\geq$65 years) is estimated to be even higher. This may be due to under-recognition in the context of complex comorbidities [1,2]. Depression is often a recurrent illness, triggered, and exacerbated by both psychological stress and medical illnesses. High medical burden in older adults contributes to treatment response variability such as delayed response to antidepressant pharmacotherapy and increased likelihood of recurrence [3].

Numerous studies suggest that depression worsens both the severity of and disability caused by chronic low back pain (CLBP) [4–8]. A large survey of community dwelling older adults found that mild to severe depressive symptoms increased the odds of disabling low back pain over a period of 2 years by 30–60% [8]. Similarly, baseline disabling low back pain ranging from a little of the time to all of the time increased the odds of depressive symptoms by 27.9–84.2%, respectively [8]. As depression is a treatable illness, a rational approach to reducing the burden of CLBP is to diagnose and treat comorbid depression. To date, there is little research published about how to assess and treat these conditions simultaneously.

There are several challenges related to identifying depressive symptoms. Depressed older adults frequently communicate emotional distress by focusing on somatic complaints and describing nonspecific symptoms. Rather than spontaneously reporting depressive symptoms, the older adult may describe feeling helpless due to unrelenting back pain, joint pain, or gastrointestinal distress [9,10]. Instead of reporting the cardinal symptoms of depression (i.e., depressed mood or anhedonia), many older adults with CLBP and other chronic pain conditions who are in a depressive episode often report non-specific symptoms such as irritability, insomnia, decreased energy, difficulty concentrating, and memory problems [10,11].

Reasons for this overlap in clinical presentation may be due to shared neurobiology and psychology between depression and CLBP [12] as well as genetic influences [13]. Areas of the brain which modulate mood also process pain and include the dorsolateral prefrontal cortex, anterior cingulate cortex, periaqueductal gray, insular cortex, and hypothalamus [14–16]. Psychological similarities between patients with depression and patients with CLBP include diminished self-efficacy and subsequent learned helplessness [17–21]. Older adults who have become disabled by either depression or CLBP often have a sense that they are unable to manage these and other chronic conditions. Both conditions frequently wax and wane, are exacerbated by environmental stressors, and may be responsive to similar pharmacologic (e.g., antidepressants) and behavioral treatments (e.g., cognitive behavioral therapy [CBT], mindfulness techniques) [12,22]. This overlapping neurobiology and psychology support the need for a shared approach to treatment.

Disability and loss of function are among the most feared consequences of medical problems and pain in late-life [23]. Pain-related disability is worse in patients with depression, further supporting the importance of its diagnosis and treatment [7,8]. Thus, to increase the likelihood that CLBP and associated disability will respond to treatment, it is vital to systematically screen for and treat clinically significant depressive symptoms in the older adult who presents with CLBP. We present a patient who has CLBP with depression being at least one contributor to his pain and difficulty functioning. This case demonstrates the clinical complexity of older patients with CLBP, depression, and a multidisciplinary approach to his clinical care.

Methods

A modified Delphi technique was used to create an algorithm for assessing and treating CLBP and depression (Figure 1), as well as a table providing the rationale for the various components of the algorithm (Table 1), and the stepped-care medication table (Table 2). This iterative approach to the development of the algorithm is described in detail in the first article of this series [45]. Expertise represented among the Delphi expert panel for the depression algorithm included geriatric psychiatry, geriatric medicine, and geriatric psychopharmacology.

Case Presentation

Relevant Pain and Functioning History

The patient is an 88-year-old widowed Caucasian man residing in an assisted living facility (ALF) who presented to his primary care physician complaining of a flare of his CLBP. He described having low back pain “as long as I can remember.” The pain is worse with walking but does not radiate into either leg. He states the pain can “hit me” at any time, including while sitting and at times while supine. He describes the pain as aching and heavy and at a 6/10 severity on average. However, over the past 2 weeks, he describes the pain as 10/10 severity more than 50% of the time.

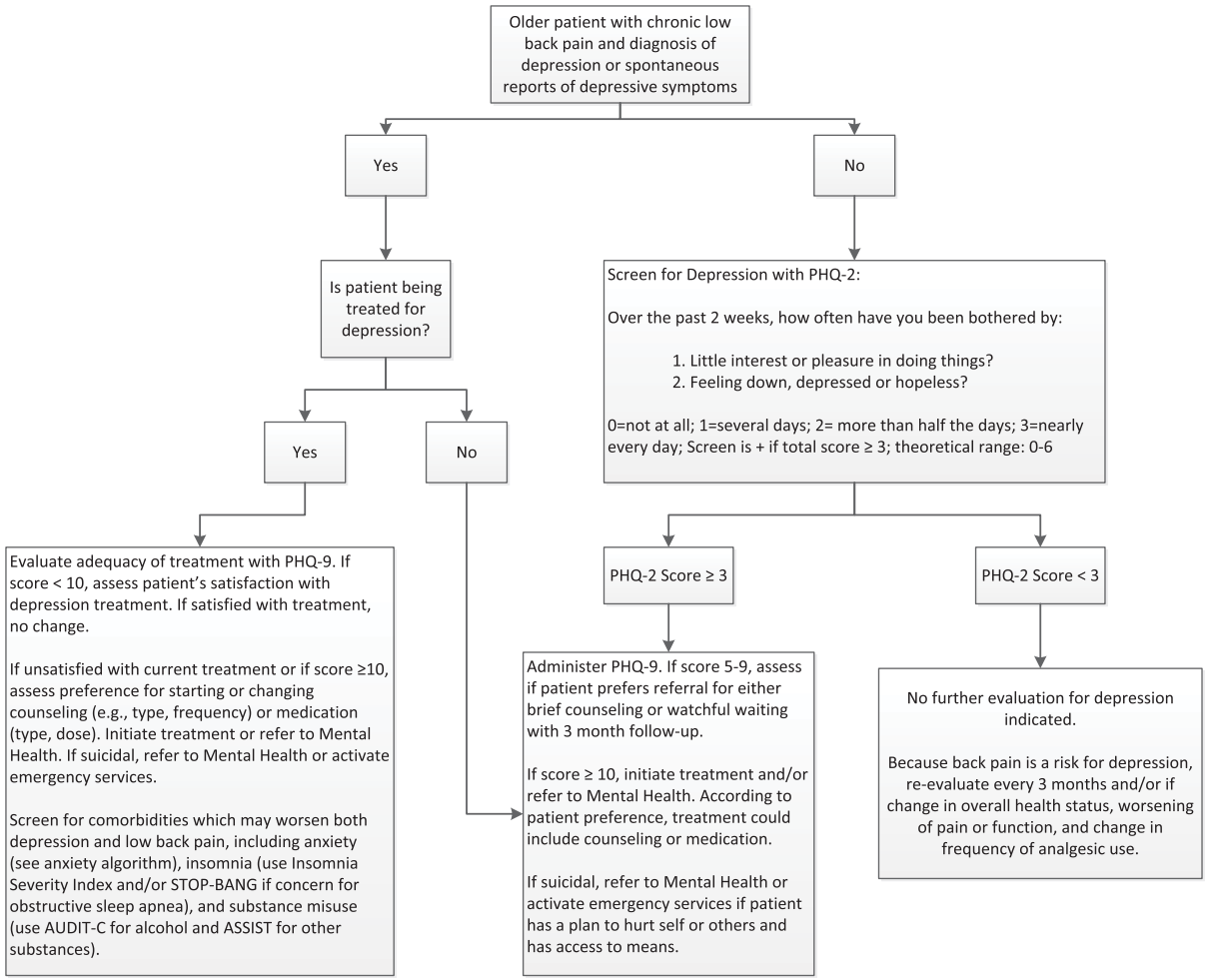

Figure 1 Depression algorithm.

He has had two spine surgeries. These included lumbar decompression (20 years ago) and laminectomy and fusion (8 years ago). Each of these surgeries improved his back pain for about 2 months, but the pain then returned at the same level of severity. In addition to these surgeries, he has had three epidural steroid injections, physical therapy, chiropractic manipulation, massage therapy, heat therapy, opioid, and nonopioid oral analgesics, topical analgesics, and been episodically compliant with a home-based stretching and core-strengthening program. He is currently prescribed oxycodone extended release 10 mg bid and oxycodone immediate release 5 mg every 4 hours as needed for pain. He has been taking additional oxycodone immediate release over the past 2 weeks. He has a TENS unit at home, and although it helps, he has trouble motivating himself to use it. He is unable to walk or stand for more than 15 minutes because of the pain. He has also curtailed attending church and reduced the number of community dinners he attends each week at the ALF. Although he has not fallen, he is fearful that he will.

Relevant Physical and Psychiatric Examination, and Review of Systems

He is alert and oriented × 5 with good fund of knowledge and no language deficits. His gait is notable for short step length and relatively slow gait velocity. He carries a standard straight cane in his left hand for balance. On physical exam there was no evidence of leg length discrepancy, scoliosis, sacroiliac joint pain, vertebral body pain, or pain and restricted motion of the hip with internal rotation. On palpation, there was mild myofascial pain of the paralumbar musculature. The Mini Mental State Examination score was 27 (theoretical range 0–30) [46]. As his wife died and his move to the ALF, he describes feeling lonely and does not see much reason for living as most of his friends and all of his siblings have died. He scores 20 on the Patient Health Questionnaire (PHQ)−9 (theoretical range 0–27), endorsing daily depressed mood, insomnia (with sleep continuity disturbance and early morning awakening), self-critical thinking, low appetite, trouble concentrating, and a passive death wish [25]. He denies active suicidal

Table 1 Depression and back pain: Theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm Component	Comments	References
Depression screening with PHQ-2 and PHQ-9	The Patient Health Questionnaire (PHQ) is a self-administered version of the PRIME-MD diagnostic instrument for common mental disorders. A PHQ-2 score $>$ or $=$3 had a sensitivity of 83% and a specificity of 92% for major depression. Likelihood ratio and receiver operator characteristic analysis identified a PHQ-2 score of 3 as the optimal cutpoint for screening purposes.	[24,25]
	The PHQ-9 is the depression module, which scores each of the 9 DSM-5 criteria as "0" (not at all) to "3" (nearly every day). PHQ-9 score $\geq$10 had a sensitivity of 88% and a specificity of 88% for major depression. PHQ-9 scores of 5, 10, 15, and 20 represented mild, moderate, moderately severe, and severe depression, respectively.	
Screening for psychiatric comorbidities and insomnia	Late-life depression rarely occurs in isolation. Thus, screening for and assessment of late-life depression should always involve screening for other psychiatric disorders, including anxiety, alcohol, and drug abuse. As comorbid psychiatric disorders affect clinical course and prognosis, and may worsen long-term disability and pain management, treatment is critical to optimize both psychiatric and pain outcomes.	[12,26–30]
	Untreated insomnia is associated with worse depression and low back pain treatment outcomes. We recommend screening for insomnia with the Insomnia Severity Index (available on myhealthevet.gov) and treating insomnia along with depression and low back pain to optimize outcomes.	
	If the patient is at high risk for obstructive sleep apnea (obese, male, African American, prescribed opioids, smoker, cardiovascular disease), consider administering the STOP-BANG questionnaire to assess whether referral for diagnostic polysomnography is indicated.	
CBT	Dissemination and implementation of cognitive behavioral therapy for depression in the VA system resulted in mean improvement in depression scores by about 40% from initial to later treatment phase. The effect size for improvement in quality of life ranged from $d = 0.39$ to $d = 0.74$. However, while a meta-analysis of trials using CBT for late-life depression found it to be more effective than waiting list or treatment as usual, greater efficacy than active controls was not observed.	[31–34]
	Despite the findings of this meta-analysis, CBT for pain has been shown to ameliorate pain-related symptoms for chronic back pain patients treated in an outpatient setting. CBT for pain provided in a group setting is associated with up to 5-year improved health and economic benefits compared with an information comparison group. Although CBT for depression in older adults may not be superior to other active controls, given the efficacy of CBT for pain and the superiority of CBT to treatment as usual, we recommend this as the psychosocial intervention of choice for older adults with low back pain and depression.	
Stepped care antidepressant treatment	According to expert consensus guidelines for unipolar nonpsychotic major depression, the preferred strategy is an antidepressant (selective serotonin reuptake inhibitors or venlafaxine XR are the preferred agents) plus psychotherapy. As these guidelines were prepared before the release of duloxetine, and given that duloxetine is approved for the treatment of chronic pain in addition to depression, we include	[35–41]

Table 1 *Continued*

Algorithm Component	Comments	References
	duloxetine as recommended for these patients. These guidelines also suggest that if the patient has a comorbid medical condition (e.g., chronic low back pain) that is contributing to the depression, both the depression and medical condition should be treated from the outset. The majority of experts would continue treatment with antidepressant medication for at least 1 year if a patient has had a single episode of severe unipolar major depression, for 1–3 years for a patient who has had two such episodes, and for longer than 3 years if there is a history of three or more episodes.	
	Beer's Criteria suggests monitoring for hyponatremia when starting an antidepressant in older adults.	
	Second line antidepressant pharmacotherapy may include bupropion, mirtazapine, and nortriptyline (with appropriate cardiac monitoring).	
	While not specific for older adults, the Sequenced Treatment Alternatives to Relieve Depression Study (STAR*D) showed that about half of participants are symptom-free after two treatment levels. Over the course of all four treatment levels, almost 70% of those who did not withdraw from the study became symptom-free.	
	The table included in the algorithm reflects best practice for the first three steps of depression pharmacotherapy that may be offered in primary care.	
Serial monitoring of progress	Monitor improvement in depression with the PHQ-9. As antidepressant pharmacotherapy may reduce both pain severity and pain interference, routinely assess these clinical outcomes with a numeric rating scale for both pain severity and pain interference. The use of the 24-item Roland Morris Back Pain Disability Questionnaire may also be used to assess improvement in functioning.	[42–44]
	Serial monitoring of adherence to both pharmacological and psychosocial treatment plans and management of barriers to compliance should be addressed.	

ideation or plan. He does not own any firearms and is not stockpiling opioids. Upon further questioning, he states he spends most of his day sitting in a chair watching television or staring out the window as he "feels sapped of energy." Associated symptoms he describes include chronic nausea and constipation, feeling cold, blurry vision, pain in other joints, urinary frequency, and dry mouth. He denies any weight change, fever, chills, or night sweats.

Clinical Course

The patient revealed that in addition to analgesia, he also used the oxycodone extended release for its calming and "numbing" effect, and was counseled that there were better and safer treatments available. The evening dose of oxycodone extended release was discontinued, and the oxycodone immediate release was reduced to 5 mg every 6 hours. To treat both the depression and back pain, pharmacotherapy with duloxetine was initiated at 30 mg for 1 week and increased to 60 mg starting week 2. The clinic social worker began to see the patient every other week to deliver supportive psychotherapy informed by CBT and Problem Solving Therapy techniques [47]. Their work together also focused on sleep consolidation techniques and increasing his participation in pleasurable activities [48]. With his permission, the social worker engaged both his daughter and the social coordinator from the ALF into treatment planning.

Approach to Management

Upon further history taking, it became clear that depression and bereavement were playing roles in this flare of CLBP. As he spontaneously reported some depressive symptoms after the passing of his wife, he was appropriately screened with the PHQ-9. If this patient had not spontaneously mentioned loneliness and some hopelessness, he could have been screened with the PHQ-2 which is shorter yet still has robust psychometric properties [49].

Table 2 Recommended early sequence of antidepressant pharmacotherapy*

Level (all levels are 6 weeks)	Medication	Target/Maintenance Dose	Notes
Level 1	SSRI:		May consider first line treatment with sertraline if there are concerns about prolonged QTc†.
	Citalopram	20 mg (for patients >60 y.o.)	
	Sertraline	100–200 mg	
Level 2	Taper and stop SSRI		Duloxetine is U.S. Food and Drug Administration (FDA)-approved for both major depression and chronic pain.
Non-response	Start duloxetine	60 mg	
to Level 1	Or		Duloxetine is not recommended for patients with end-stage renal disease or severe renal impairment (estimated creatinine clearance <30 mL/minutes)
	Start venlafaxine	Target dose 150–300 mg	
Level 2	Continue SSRI		Confirm patient has no history of seizure disorder and is not at increased risk of seizure
Partial response	Augment with		
to Level 1	bupropion SR	200 mg bid	(i.e., taking tramadol)
Level 2A	Continue SSRI		While low dose nortriptyline may reduce pain and help with sleep, (and, therefore, benefit mood), these low doses are unlikely to have specific antidepressant effects.
Partial response	Consider low dose		
to Level 1	nortriptyline to help with pain and sleep (if no contraindication, per below).	10–25 mg po qhs	
Level 3	Taper and stop all		Nortriptyline may have analgesic effects.
Non-response	Level 2 medications		Obtain Electrocardiogram (EKG) prior to use.
to Level 2	Start nortriptyline	Plasma concentration 80–120 ng/mL	Limit supply of medication if patient at high suicide risk.
		Steady state achieved in approximately 5–6 days.	Monitor for anticholinergic side effects.
		If possible, plasma levels should be "trough," drawn immediately before next dose.	
Level 3	Continue SSRI		Caution use of lithium in older adults with renal insufficiency (or using concomitant Nonsteroidal Anti-inflammatory Drugs (NSAIDs), Angiotensin Converting Enzyme (ACE) inhibitors, thiazide, or loop diuretics).
Partial response	Taper and stop		
to Level 2	bupropion SR	Plasma concentration 80–120 ng/mL	
	Augment with either:		
	Nortriptyline	Plasma concentration 0.6–0.8 mEq/L	Obtain Thyroid Stimulating-hormone (TSH) level before using lithium.
	Lithium		Note lower plasma levels than used in younger adults and/or with bipolar disorder. Refer for psychiatric co-management if desired.

This stepped care approach for the pharmacotherapy of depression and low back pain in older adults is blended from both the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) and Prevention of Suicide in Primary care Elderly-Collaborative Trial (PROSPECT). Duloxetine has been added to this updated algorithm given its dual approval from the FDA for both major depression and chronic pain. This algorithm is truncated at level 3 because beyond this level, primary care physicians should consider referring the patient for evaluation by mental health.

Nonresponse may be defined as <30% improvement in a depression rating scale after 6 weeks of treatment with a score still in the symptomatic range. Partial response may be defined as >50% response on a depression rating scale, with a score still in the symptomatic range.

* At all levels of this sequenced care, referral for cognitive behavioral therapy should be offered. This is, especially important given the significant proportion of older adults who will refuse or stop antidepressant pharmacotherapy. Given the added stress of chronic low back pain on depression, patients may benefit from learning more adaptive approaches to coping with these challenges, improving problem solving skills, planning pleasurable activities, increasing activity, and improving restorative sleep.

† The FDA has recommended that citalopram should no longer be used at doses greater than 40 mg/day because it could cause potentially dangerous abnormalities in the electrical activity of the heart, in particular prolonged QTc. The maximum recommended dose for patients older than 60 is 20 mg/day.

Ref. [38].

Ref. [39].

Ref. [40].

Ref. [41].

The algorithm presented in the Figure 1 provides guidance for the clinician as to when to use the PHQ-2 vs the PHQ-9. The PHQ-2 is best utilized when the older patient with CLBP has no prior diagnosis of depression and no spontaneous report of depressive symptoms. The PHQ-2 score of greater than or equal to 3 has a sensitivity of 83% and a specificity of 92% for major depression [24] (Table 1). Patients who screen positive with the PHQ-2 should be administered the PHQ-9. If clinical resources are available and the PCP feels a referral is indicated (i.e., because of high depression severity, history of treatment nonresponse, or complex psychiatric and psychosocial comorbidity) a referral to Psychiatry may be warranted. Because patients with CLBP are at increased risk of new onset depression, those who screen negative on the PHQ-2 should be reevaluated at 3-month intervals or sooner in the setting of worsening health status, pain, or increased analgesic use.

For older patients with CLBP who already carry a diagnosis of depression or are spontaneously reporting depressive symptoms, we recommend using the PHQ-9. The PHQ-9 scores each of the nine DSM-5 criteria for depression from 0 being "not at all" to 3 being "nearly every day." A score of 10 or higher has a sensitivity of 88% and a specificity of 88% for major depression [25]. PHQ-9 scores also provide a range of depression severity with mild, moderate, moderately severe, and severe, corresponding to scores of 5, 10, 15, and 20, respectively [25]. Based both on whether the patient is already receiving treatment for depression and the current PHQ-9 score, the algorithm provides directions for further evaluation and treatment including screening for psychiatric and sleep comorbidities, counseling, and medication management.

Once clinically significant depression is diagnosed, screening for comorbid psychiatric conditions should be undertaken, as comorbidity is the norm, not the exception [26]. Common comorbid conditions in older depressed patients include anxiety, cognitive impairment, and alcohol misuse [27], and in VA settings, PTSD is frequently comorbid. While data are limited, there is evidence of improved outcomes for depression with comorbid anxiety or alcohol use disorders when treated using CBT or substance use counseling, respectively [26]. Another common comorbid condition, which worsens both depression and CLBP, is insomnia [28]. A detailed algorithm guiding evaluation and treatment of insomnia will be published later in this series on CLBP in the older adult. The Insomnia Severity Index is a brief screen for insomnia and can be easily incorporated into routine assessments [50]. Sleep apnea is especially prevalent in patients who are obese, living with cardiovascular disease, and is worsened by the use of opioids and benzodiazepines. If sleep apnea is suspected, the STOP-BANG questionnaire can be used to assess the need for referral to sleep medicine and possibly diagnostic polysomnography [30]. Important other risks for poor response to treatment for both depression and CLBP include prolonged and/or use of high-dose opioids [51] and history of multiple spine surgeries [52]. Tailoring treatment based on an individual's psychiatric and medical comorbidities provides the best chance of improving both depression and CLBP.

CBT has demonstrated clinical benefit for both depression and CLBP [31–34]. The efficacy of CBT for depressed older adults is well established [53] with average effect sizes above 0.80 [54]. CBT is an established intervention for CLBP [55], and there is evidence for its use in older adults with pain syndromes. In general, studies of CBT in older adults with chronic pain support its efficacy for significantly reducing pain (with small to medium effect sizes) [56], with meaningful improvements in indices of adapting to and coping with pain, such as measures of depression, anxiety, pain catastrophizing, self-efficacy, and level of activity [57]. CBT for CLBP and MDD utilizes similar techniques such as learning to pace activities, involvement of spouses/caregivers, reinforcement of adaptive responses, reframing affective and cognitive responses, learning active coping skills and relaxation techniques, and problem solving skills training [56,58]. Using a personalized approach, CBT may be individualized for these complex patients. To our knowledge, there have not been randomized controlled trials of CBT for patients with both MDD and CLBP. In the context of coexisting CLBP and depression, CBT is our consensus recommendation. CBT targets the diminished self-efficacy and learned helplessness, which often occur in both depression and CLBP, particularly in older adults. CBT can be delivered by psychologists, social workers, and psychiatrically trained nurses. Computerized CBT programs are becoming increasingly available, may be equivalent to therapist-delivered CBT [59], and should be considered for patients who are computer savvy and/or have limited access to mental health care. There is also evidence supporting goal attainment as a measurable focus of treatment for older adults living with chronic conditions [60]. This practical approach enhances patient-centered care and may align well with the processes of CBT. In addition to CBT and pharmacotherapy, increased physical activity may improve outcomes for both CLBP and depression [61,62] Increasing physical activity is consistent with the behavioral activation focus of CBT and may be a patient-centered goal of treatment.

In addition to CBT, our expert consensus guidelines for depression recommend pharmacotherapy with selective serotonin reuptake inhibitors (SSRI's) or serotonin norepinephrine reuptake inhibitors (SNRI's) [35,36,39] (Table 2). In addition to venlafaxine, duloxetine has been shown to be effective as both an SNRI antidepressant and for chronic musculoskeletal pain [40,41]. When prescribing serotonergic and noradrenergic reuptake inhibitor treatment in older adults, monitoring for hyponatremia is recommended [37]. An accepted approach for duration of treatment is continuation of antidepressant pharmacotherapy for at least a year if this is the first episode of depression and at least 3 years if the patient has a history of recurrent

episodes [63]. Patient and caregiver education is important when antidepressants are administered for depression and/or analgesia. Instructing patients on both the importance of taking the medication every day as prescribed and that the medication is being prescribed for both conditions may improve adherence and reduce stigma.

Approximately 50% of older adults do not respond to first line pharmacotherapy and require subsequent treatment trials. While not specific to older adults, the Sequenced Treatment Alternatives to Relieve Depression Study (STAR*D) showed that about half of participants are symptom-free after two treatment levels [36]. Second line treatments include bupropion, mirtazapine, or nortriptyline (see Table 2). While amitriptyline has been studied for use in certain pain conditions and in depression, its severe anticholinergic side effect profile has led to its inclusion on the Beers list [37], and so is not recommended in older adults. Nortriptyline has a somewhat less severe side effect profile but this should be monitored.

Using a measurement-based approach, pharmacotherapy focused on both depression and CLBP may result in superior outcomes than focusing on only one condition [12]. Indeed, serial monitoring of progress has been shown to improve outcomes for both depression treatment and chronic pain management by allowing appropriate adjustments to treatment plans [42,43]. In addition to the PHQ-9 for serial measurement of depression, the 24-item Roland Morris Back Pain Disability Questionnaire is recommended for use to monitor change in functioning related to low back pain [44]. Alternatively, periodic monitoring of pain severity using a numeric rating scale may also inform measurement-guided treatment.

Resolution of Case

After titrating the duloxetine up to 60 mg, the patient began to gradually show improvement first in low back pain and then depression severity [64]. Screening for psychiatric comorbidities including alcohol use was negative, as was screening for sleep apnea with the STOP-BANG questionnaire. He engaged in eight sessions of supportive therapy informed by CBT (with a focus on increasing pleasurable activities, strategies to increase physical activity while pacing himself to avoid precipitating a pain flare, and reducing insomnia). Additional benefit was gained through treatment planning with his daughter and the social coordinator from the ALF. They successfully supported him in becoming gradually more engaged in community activities. It is worth noting that social workers can be trained to deliver CBT in primary care settings. This is significant, as a major barrier to the delivery of evidence based depression and pain interventions is limited access to psychologists. Serial monitoring of progress and gradual reductions in use of opioids over time further contributed to his overall improvement.

Summary

Because of the increased prevalence of mood symptoms in patients living with chronic pain, screening for depression should be routine in older adults with CLBP. As depression worsens both pain severity and pain-associated disability, treating this modifiable contributor to CLBP can improve analgesia and functioning as well as other health and disability related outcomes [65]. Stepwise screening with the PHQ-2 and PHQ-9 is a highly sensitive and specific approach for detecting clinically actionable depression. If depression is detected, screening for and treating common comorbidities such as anxiety, substance misuse, insomnia, and sleep disordered breathing is indicated and may result in a more durable treatment response.

Depression and CLBP in older adults are mutually exacerbating conditions that both contribute to patient and family suffering, increased disability, cognitive impairment [66], polypharmacy, and hastened mortality [67]. Because of an overlapping neurobiology and psychology, treating these disorders as linked conditions may spare use of opioids and encourage a parsimonious approach to prescribing. Using a shared decision to account for patient preference and an interdisciplinary approach, initiating treatment with a SSRI or SNRI is first line treatment and may improve outcomes for both conditions. CBT is the behavioral intervention with the greatest evidence base for both depression and CLBP. Social workers and chronic disease care managers are increasingly colocated in primary care practices and may be trained in the reliable delivery of CBT. These clinicians also play a key role in monitoring symptom change and medication adherence, information which can then be communicated to the prescribing PCP. Educating patients that these interventions target both mood and pain symptoms may enhance compliance, resulting in better outcomes for both conditions.

Key points

1. Older adults with CLBP should be screened for depression, and treated if depression is present.
2. Effective treatment of depression in older adults with CLBP will reduce pain-related activities interference and overall disability.
3. Older adults with depression also should be screened for important psychiatric comorbidities that also may worsen CLBP including alcohol use, anxiety disorders, cognitive impairment, insomnia, and sleep disordered breathing.
4. Depression comorbid with CLBP should be treated for at least 1 year to avoid recurrence. Behavioral interventions and pharmacotherapy such as SSRIs and SNRIs, chosen based on symptom severity and shared decision making, may improve long-term outcomes.

Acknowledgments

This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration,

Office of Research and Development, Rehabilitation Research and Development Service. Dr. Karp would like to acknowledge grant AG033575 (the ADAPT Project: Addressing Depression and Pain Together), which provided support for his work on this article. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The authors thank Dave Newman for his thoughtful review of the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult – Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment. Part V: Maladaptive Coping

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interest: The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The authors have no conflicts of interest to report.

Abstract

Objective. As part of a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults, this article focuses on maladaptive coping—a significant contributor of psychological distress, increased pain, and heightened disability in older adults with CLBP.

Methods. A modified Delphi technique was used to develop a maladaptive coping algorithm and table providing the rationale for the various components of the algorithm. A seven-member content expert panel and a nine-member primary care panel were involved in the iterative development of the materials. While the algorithm was developed keeping in mind resources available within the Veterans Health

Administration (VHA) facilities, panelists were not exclusive to the VHA, and therefore, materials can be applied in both VHA and civilian settings. The illustrative clinical case was taken from one of the contributors' clinical practice.

Results. We present a treatment algorithm and supporting table to be used by providers treating older adults who have CLBP and engage in maladaptive coping strategies. A case of an older adult with CLBP and maladaptive coping is provided to illustrate the approach to management.

Conclusions. To promote early engagement in skill-focused treatments, providers can routinely evaluate pain coping strategies in older adults with CLBP using a treatment algorithm.

Key Words. Aged; Assessment; Maladaptive Coping; Chronic Pain; Elderly; Low Back Pain; Primary Care; Chronic Low Back Pain

Introduction

Older adults who experience chronic low back pain (CLBP) develop behavioral and cognitive coping strategies to tolerate or reduce pain. These coping strategies have been shown to significantly predict pain, functional capacity, and chronification of LBP. For example, adaptive coping strategies are generally associated with reduced pain, positive affect, and better psychological adjustment [1], whereas maladaptive coping strategies have been linked with negative outcomes such as psychological distress, increased pain, and heightened disability [2–4]. Please see Table 1 for examples of maladaptive and adaptive coping strategies. Research has found age-related differences in pain coping strategies across the life span [5, 6]. While older adults are more likely than younger adults to use a narrower range of pain-related coping strategies (e.g., less cognitive and more emotion-focused strategies), they tend to use these strategies more consistently and effectively [7]. As aging is associated with significant heterogeneity, many older adults with CLBP are at risk of engaging in maladaptive coping strategies. Fortunately, coping remains malleable with age, and maladaptive coping strategies can be effectively changed with interventions [8,9]. Therefore, it is increasingly important for providers to assess pain coping strategies in this population.

The fear-avoidance model (FAM) is a theoretical model that describes how psychological factors affect the experience of pain and the development of chronic pain and disability [10]. Within the FAM, maladaptive coping is often characterized by helplessness or reliance on others and includes catastrophizing (i.e., exaggerated orientation toward pain stimuli and pain experience) [11,12], fear-avoidance beliefs [13,14], and behavioral disengagement [15,16]. Patients with CLBP catastrophizing responses often create disproportionately strong fears about the potential for physical activities to produce pain or further harm the spine (i.e., fear-avoidance beliefs), reinforcing the original negative appraisal in a deleterious cycle [10]. It should be noted that fear-avoidance beliefs can occur concurrently with or independently of catastrophizing. Owing to these fear-avoidance beliefs, patients with CLBP may also decrease their engagement in activities that bring a sense of enjoyment, meaning, or accomplishment (i.e., behavioral disengagement) [17]. Again, the use of maladaptive coping strategies (i.e., catastrophizing, fear-avoidance beliefs, and behavioral disengagement) has been shown to be associated with negative consequences in older adults, such as increased disability [11–16]. As such, CLBP treatment should emphasize teaching cognitive and behavioral skills to increase patients' ability to cope with and manage pain. Fortunately, there are effective skill-focused treatments, such as physical therapy [18,19] and psychotherapy [20–22], that emphasize adaptive coping strategies or an active approach to dealing with CLBP using techniques such as coping self-statements, re-interpreting pain sensations, and increasing activity level.

Cognitive and behavioral pain coping strategies have been found to be predictive of adjustment to chronic pain above and beyond what may be predicted on the basis of patient history variables (e.g., disability status, number of surgeries) and the tendency of patients to somaticize [23]. Therefore, to identify CLBP older adults in need of skill-focused treatments, clinicians must obtain a greater understanding of patients' pain coping strategies.

In the clinical management of patients with CLBP, clinicians often assess for a number of demographic and medical status variables, but rarely are pain coping strategies routinely considered. One factor that may greatly limit clinicians' comfort with assessing for psychosocial factors of CLBP, such as pain coping strategies, is that current assessment tools and treatment guidelines are too general and global. Treatment algorithms/guidelines help to minimize random variation in care, increase patient receipt of evidence-based care, and improve

Table 1 Examples of adaptive and maladaptive coping strategies [6]

Adaptive coping	Maladaptive coping
Task persistence	Guarding (e.g., avoiding movement because of fear of injury)
Relaxation	Pain-contingent rest
Pacing one's activities	Pain-contingent social support (e.g., expecting others to foresee and respond to pain complaints)
Coping self-statements	

patient outcomes [24,25]. With an algorithm to guide the management of maladaptive coping strategies in older adults with CLBP, clinicians may not only help promote early engagement in skill-focused treatments aimed at increasing positive coping strategies but, in turn, also reduce pain, decrease emotional distress, and increase functionality. The purpose of this paper is to present a treatment algorithm for clinicians treating older adults with CLBP who engage in maladaptive coping strategies. We will also present an older adult patient who has CLBP with maladaptive coping and describe the approach to the management and resolution of the case.

Methods

As per the detailed description provided in the series overview [26], a modified Delphi technique involving a content expert panel and primary care review panel was used to create the maladaptive coping algorithm (Figure 1) and a table providing the rationale for the various components of the algorithm (Table 2). The PI (DW) drafted an evidence-based treatment algorithm and supportive tables based on a comprehensive review of the literature and general clinical utility when strong evidence was not yet available. The algorithm and accompanying table were then refined by an expert panel. Expertise represented by the seven Delphi expert panel members included geriatric medicine, geriatric pharmacology, geriatric psychology, and occupational therapy.

Case Presentation

Relevant History

The patient is a 69-year-old white male who has been divorced once and married to his current wife for 20+ years, is retired, and has brittle type I diabetes. He has had five lumbar surgeries. The patient showed initial improvement from each surgery but then had worsening low back and leg pain after a fall, which he believes was related to low blood sugar. He describes worry about further injury. The patient has not had physical therapy recently but received such services in the past after surgery. He failed epidural steroid injections, facet injections, and medial branch block and declined radio frequency ablation. The patient is taking bupropion SA 150 mg every 12 hours for mood with some benefit and 200 mg of trazodone for insomnia. His analgesic regimen includes oxycodone 10 mg every 6 hours, fentanyl 50 mcg every 72 hours, and gabapentin 900 mg three times a day. The patient complains of low energy.

Pain Presentation

The pain is described as sharp and stabbing, rated at 8 to 10/10 on a Numeric Rating Scale. He has often expressed that his pain is greater than 10. The main pain location is the axial lumbosacral junction with radiation into the bilateral anterior and posterior thighs (left greater than right). The pain is better when lying down with left leg elevation (i.e., placing a pillow under the knee while supine) and with analgesics and is worse with walking more than one block, bending, twisting, lifting, and "any activity that lasts for more than 5 minutes."

Assessment of Coping Style

Through a clinical interview, the patient described the constructs of catastrophizing, fear-based avoidance, and behavioral disengagement. He endorsed a pattern of catastrophizing: "*I don't think that this will ever get better. It will only get worse. A lot of the time it is unbearable*." In addition, he provided many examples of disengaging from enjoyable, meaningful activities, such as working in his garden, because he believed that physical activity might harm his back and worsen his pain (i.e., fear-avoidance beliefs).

He also reports feeling angry and disgusted much of the time and often retreats to a dark room.

Physical Examination

The following aspects are notable in the patient's physical examination. GENERAL: No acute distress. MOOD/AFFECT: Sad, mildly irritable, and tearful at times. GAIT: Antalgic, slightly wide based with shortened step length. Able to briefly toe-and-heel walk limited by back pain without instability. HIPS: Internal and external range of motion within normal limits without pain at end range bilaterally. Hip scour (i.e., manipulating the joint to look for "clicks" or "chunks") negative bilaterally. No tenderness over greater trochanter. LOW BACK: Normal muscle bulk without asymmetry; well-healed surgical scar. Tenderness to palpation is diffuse without focal point tenderness reproducing symptoms. Palpation of the right mid-lumbar paraspinal process caused left leg numbness but no trigger point/nodule. Range of motion is moderately pain limited in all planes with guarding and minimal extension/rotation with guarding. Straight leg raise is negative bilaterally sitting and supine. FABER: Negative on the left and right. Piriformis Test, a standard provocative muscle test to see whether a tight piriformis is stimulating sciatic pain, is negative on the left and right. Popliteal angle mildly reduced. NEUROLOGICAL: Motor: 5/5 in all myotomes of the bilateral upper extremities except as pain limited. Three toe raises left and five toe raises right, but limited by pain. Extensor halluces longus (i.e., the muscle that extends the big toe) and dorsiflexion of the foot are 4/5 bilateral, otherwise 5/5 in all myotomes of the bilateral lower extremities, except as pain limited. REFLEXES: Biceps, brachioradialis, triceps, patellar, Achilles 2+ bilaterally.

Imaging

Lumbar MRI revealed multilevel lumbar spondylosis with laminectomies (L4-5 and L5-S1) without fusion. No areas of residual moderate or severe stenosis.

Figure 1 Algorithm for the evaluation and treatment of maladaptive coping in an older adult.

Table 2 Maladaptive coping: theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
Role of fear avoidance in CLBP	Fear of activity is an important contributor to disability in older adults. Previous studies have validated the FAB questionnaire; however, for the purpose of this study we have utilized a single question (taken from the 2014 NIH Task Force on Research Standards for chronic Low Back Pain) to practically assess fear avoidance that is generalizable to a clinical setting.	1. Camacho-Soto A, Sowa GA, Perera S, Weiner DK. Fear avoidance beliefs predict disability in older adults with chronic low back pain. PM R 2012;4(7):493–7. 2. Sions JM, Hicks GE. Fear-avoidance beliefs are associated with disability in older American adults with low back pain. Phys Ther 2011;91(4):525–34. 3. Deyo RA, Dworkin SF, Amtmann D, et al. Report of the NIH task force on research standards for chronic low back pain. J Pain 2014;15(6):569–585. 4. Wertli MM, Rasmussen-Barr E, Held U, et al. Fear-avoidance beliefs- a moderator of treatment efficacy in patients with low back pain: a systematic review. Spine J 2014;14(11):2658–78.
Role of catastrophizing in CLBP	Pain catastrophizing is negative and distorted thinking and worrying about the pain and one's inability to cope. Catastrophizing has also been shown to influence LBP-related disability in middle-aged patients. The catastrophizing subscale of the Coping Strategies Questionnaire (CSQ) has been previously validated; however, for the purpose of this study we have utilized a single question (taken from the 2014 NIH Task Force on Research Standards for Chronic Low Back Pain) to assess coping that is generalizable to a practical clinical setting.	1. Morone NE, Karp JF, Lynch CS, et al. Impact of chronic musculoskeletal pathology on older adults: A study of differences between knee OA and low back pain. Pain Med 2009;10(4): 693–701. 2. Wertli MM, Eugster R, Held U, et al. Catastrophizing- a prognostic factor for outcome in patients with low back pain: a systematic review. Spine J 2014;14:2639–57. 3. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: A critical review. Expert Rev Neurother. 2009;9:745–58.
Role of behavioral disengagement in CLBP	Unwillingness to remain active predicts poor outcomes in CLBP. Active interventions are considered a mainstay of pain treatment.	1. Hall AM, Kamper SJ, Maher CG, et al. Symptoms of depression and stress mediate the effect of pain on disability. Pain 2011;152(5):1044–51. 2. Basler, HD, Jakle C, Kroner-Herwig B. Incorporation of cognitive-behavioral treatment in the medical care of chronic low back patients: A controlled randomized study in German pain treatment centers. Patient Education and Counseling 1997;31(2):113–24. 3. Van Tulder MW, Ostelo R, Vlaeyen JW, et al. Behavioral treatment for chronic low back pain: A systematic review within the framework of the Cochrane Back Review Group. Spine 2000; 25(20):2688–99.
Screening for dementia	Patients with dementia, because of difficulty putting pain into context, may have difficulty with pain coping.	1. Farrell MJ, Katz B, Helme RD. The impact of dementia on the pain experience. Pain 1996; 67(1):7–15. 2. Jamison RN, Sbrocco T, Parris WC. The influence of problems with concentration and memory on emotional distress and daily activities in chronic pain patients. Int J Psychiatry Med 1988;18:183–91.

(continued)

Table 2 Continued

Algorithm component	Comments	References
Screening for depression and anxiety	Depression occurs commonly with maladaptive pain coping strategies. Anxiety and fear commonly overlap.	1. Keefe FJ, Williams DA. A comparison of coping strategies in chronic pain patients in different age groups. J Gerontol 1990;45(4):P161–5. 2. McCracken LM, Gross RT. Does anxiety affect coping in chronic pain? The Clinical Journal of Pain 1993;9:253–9.
Role of psychoeducation for patients and caregivers	Patients and families often have misunderstandings about CLBP and its treatments. Education about CLBP can improve pain management as part of a comprehensive biopsychosocial approach.	1. Engers AJ, Jellema P, Wensing M, et al. Individual patient education for low back pain. Cochrane Database Syst Rev 2008; 1. 2. Burton AK, Waddell G, Tillotson M, Summerton N. Information and advice to patients with back pain can have a positive effect: A randomized controlled trial of a novel educational booklet in primary care. Spine 2009;24:2484–91.
Skill-focused treatment	Recent treatment guidelines recommend that for patients who do not improve with self-care options, clinicians should consider the addition of other treatment options including: - Exercise therapy - Yoga - Cognitive behavioral therapy - Progressive relaxation	1. Chou R, Huffman LH, American Pain Society, American College of Physicians. Nonpharmacologic therapies for acute and chronic low back pain: A review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. Ann Intern Med 2007;147(7):492–504.
	Interdisciplinary rehabilitation (also called multidisciplinary therapy): an intervention that combines and coordinates physical, vocational, and behavioral components and is provided by multiple health care professionals with different clinical backgrounds. The intensity and content of interdisciplinary therapy varies widely.	1. Gregg CD, Hoffman CW, Hall H, McIntosh G, Robertson PA. Outcomes of an interdisciplinary rehabilitation programme for the management of chronic low back pain. J Prim Health Care 2011;3(3):222–7. 2. Kamper SJ, Apeldoorn AT, Chiarotto A, et al. Multidisciplinary biopsychosocial rehabilitation for chronic low back pain: Cochrane systematic review and meta-analysis. BMJ 2015;18:350–444. 3. Cassidy EL, Atherton RJ, Robertson N, Walsh DA, Gillett R. Mindfulness, functioning and catastrophizing after multidisciplinary pain management for chronic low back pain. Pain 2012;153(3):644–50.
	Physical Rx: Physical therapy alone can improve pain coping strategies.	1. Bunzli S, Gillham D, Esterman A. Physiotherapy-provided operant conditioning in the management of low back pain disability: A systematic review. Physiother Res Int. 2011; 16(1):4–19. 2. Evans S, Sternlieb B, Tsao JC, Zeltzer LK. Using the biopsychosocial model to understand the health benefits of yoga. J Complement Integr Med 2009;6(1):1–22.

(continued)

Table 2 Continued

Algorithm component	Comments	References
	Mind-based Rx: A variety of mind-based strategies can improve pain coping strategies.	1. McCracken LM. Behavioral and cognitive-behavioral treatment for chronic pain: Outcome, predictors of outcome, and treatment process. Spine 2002;27:2564–73. 2. Ostelo RW, van Tulder M W, Vlaeyen JW, et al. Behavioural treatment for chronic low-back pain. The Cochrane Library 2005 3. Eccleston C, Morley SJ, Williams AC. Psychological approaches to chronic pain management: Evidence and challenges. Br J Anaesth 2013;111(1):59-63. 4. Morone NE, Greco CM, Weiner DK. Mindfulness mediation for the treatment of chronic low back pain in older adults: A randomized controlled pilot study. Pain 2008;134(3):310–19. 5. Esmer G, Blum J, Rulf J, Pier J. Mindfulness-based stress reduction for failed back surgery syndrome: A randomized controlled trial. J Am Osteopath Assoc. 2010;110(11):646–52. 6. Schutze R, Slater H, O'SullivanP, et al. Mindfulness-based Functional Therapy: A preliminary open trail of an integrated model of care for people with persistent low back pain. Front Psychol 2014;5:839–48.

Approach to Management

The patient presented was told that his CLBP was caused by multilevel degenerative disc disease with lumbar and cervical spondylosis complicated by chronic stress and diabetes. In addition, his maladaptive coping contributes to his pain and difficulty functioning. To help clinicians assess for maladaptive coping in patients with CLBP, the algorithm provides three statements targeting fear-avoidance beliefs, catastrophizing, and behavioral disengagement. Clinicians can also assess for coping strategies with widely available instruments, such as the Pain Catastrophizing Scale (PCS) [27], Survey of Pain Attitudes–Revised (SOPA-R) [28], Fear Avoidance Beliefs Questionnaire (FABQ) [29], and Coping Strategies Questionnaire–Revised (CSQ-R) [30].

Depression and anxiety are related to engagement in maladaptive pain coping strategies [31,32], but the patient screened negative for these disorders. Widely accessible depression and anxiety screening instruments include the Patient Health Questionnaire- 9 (PHQ-9) [33] and Generalized Anxiety Disorder Screener (GAD-7) [34]. Dementia is also associated with maladaptive coping responses [35,36] but the patient screened negative for cognitive impairment on the St. Louis University Mental Status Examination (SLUMS) [37], and the index of suspicion for cognitive impairment clinically was low, so no further testing was performed. In other articles in this series, we provide guidance on a clinical approach to the older adult with CLBP and anxiety, depression, or dementia.

As shown in the algorithm (Figure 1), treatment recommendations should be guided by patient–provider collaboration with an understanding of the patient's treatment goals and preferences. Patients with CLBP who actively engage in self-management have been shown to have superior outcomes compared with those who take a passive approach [38]. For patients who prefer a passive pain-focused treatment or treatment that is performed by someone other than the patient (e.g., chiropractic adjustment, medication, injections), clinicians should educate the patient (and caregiver when appropriate) about 1) the association between maladaptive coping and disability/pain; 2) how medication will not fix maladaptive coping and how such coping strategies will reduce medication benefits; and 3) skill-focused treatments. Recent treatment guidelines recommend that for patients with CLBP who do not improve with self-care options, clinicians should consider the addition of other skill-focused treatments [39]. There is strong evidence supporting physical (e.g., physiotherapist-provided operant conditioning or yoga) and mind-based treatments (e.g., cognitive behavioral therapy or mindfulness-based treatments) for maladaptive coping (Table 1). Another option for patients with CLBP with maladaptive coping is to be referred to an

interdisciplinary team-based pain rehabilitation program that includes physical, vocational, and behavioral components [40,41]. The authors acknowledge that interdisciplinary team-based pain rehabilitation programs may not be available to the provider/patient. The American Chronic Pain Association provides additional resources that may be helpful to providers practicing in resource-challenged areas (see http://theacpa.org/).

Resolution of Case

The previously described patient declined a new referral for physical therapy, stating that it only helped for a while in the past. He also reported that he was concerned about increasing his pain from physical therapy. The patient inquired about additional surgery and injections, but when advised that these would likely not provide benefit, he stated that he would be interested in learning mind-based pain management skills. He reported that this stemmed from a positive response he had from group therapy for depression. The patient was referred to a standard 10-week cognitive behavioral therapy (CBT) group specific for pain supplemented by some components of Acceptance and Commitment Therapy (ACT) [42].

The patient completed and responded well to the CBT group. He reported daily use of breathing-relaxation techniques. He also reported improved anger management through the use of cognitive awareness and attention to patterns of negative thoughts. Specifically, he demonstrated awareness of the link between pain flares and increased anger. He reported that although he still feels angry about hurting, he does not become mean or irritated with others as he once did. The patient's wife also noted a decrease in his irritability. In the CBT group, the patient set specific behavioral SMART (specific, measurable, attainable, relevant, and time sensitive) goals. The patient's SMART goals involved walking more, doing some yardwork, participating in household chores, and going on another elk hunting trip with his friends (something he had not done for several years). He accomplished this hunting trip, which he determined would be his last owing to other health concerns. While he did not hunt, he did chores around the camp, and he was pleased about his participation. The patient also learned to use the skill of pacing by inserting timed rest breaks to increase his walking to two blocks without significantly increasing his pain. Despite initial hesitation, the patient also engaged in a re-trial of physical therapy and learned a home exercise program that he performs somewhat sporadically several times a week. He said this helped enable him to do some yardwork, but, owing to an ongoing tendency to overdo it, he remains somewhat guarded about physical activity.

Consistent with a model of relapse prevention [43], the patient continued in a twice-per-month support group that focused on the application of adaptive coping skills to pain management and stressors. He has increased his time spent reading, an activity he finds pleasant and enjoyable. He provides support to other members of the group and reports this to be a rewarding and meaningful experience. He has also had a 75% reduction in fentanyl (50 mcg every 72 hours to 12.5 mcg every 72 hours) and 25% reduction in oxycodone (10 mg every 6 hours to 10 mg every 8 hours). The patient reported that this medication reduction has helped improve his energy and concentration. He has also successfully refrained from increasing oxycodone when experiencing pain flares, especially related to weather changes, as he now has other active coping skills that he is able to implement. Rather than depending on external interventions, he stated it helps him to maintain hope that healthcare technology will provide some additional relief for him in the future "*if I can just hang in there using what I know*." The patient is now able to recognize the importance of hope versus catastrophizing and how this helps him effectively cope with pain. The patient had a positive response to treatment as defined by his satisfaction with functional improvement and no longer endorsing maladaptive coping skills (i.e., fear-avoidance beliefs, catastrophizing, and behavioral disengagement).

Summary

Maladaptive cognitive and behavioral pain coping strategies are associated with negative patient outcomes and have been found to be predictive of adjustment to chronic pain. Therefore, it is paramount that clinicians routinely assess for pain coping strategies as a way to identify patients with CLBP in need of skill-focused treatments. The goal of the presented treatment algorithm is to provide an evidence-based decision aid that integrates patient preferences for clinicians to use in the shared treatment decision-making process.

Older adults with CLBP commonly exhibit increased levels of emotional and cognitive distress. In addition, patients with CLBP engaging in maladaptive coping strategies are at increased risk for having co-occurring mental health conditions, such as depression, anxiety, and dementia. Given the complexity of such patients, it is important that clinicians assess and treat mental health conditions adequately before engaging the patient in skill-focused treatments aimed at increasing positive coping strategies.

Given that the treatment decision is guided by patient preferences, it is important that patient education be implemented for those with unrealistic treatment goals. In keeping with the core tenets of chronic pain rehabilitation, the general goals of treatment should be optimizing function and reducing engagement in maladaptive coping skills. Individual goals should reflect specific and reasonable objectives that are motivating and meaningful to the patient.

Key Points

1. Clinical evaluation of all older adults with CLBP should include accessing patients' pain coping strategies.
2. Maladaptive coping strategies (e.g., catastrophizing, fear-avoidance beliefs, and behavioral disengagement) have been linked with negative outcomes such as increased pain and heightened disability.
3. Treatment algorithms to guide the management of maladaptive coping strategies in patients with CLBP can help to increase patient engagement in skill-focused evidence-based treatments.
4. Depression, anxiety, and dementia may entail maladaptive coping strategies; separate treatment algorithms are available for each of these disorders.
5. Treatment recommendations should be guided by collaboration with the patient to understand his/her treatment goals and preferences. Treatment options include:
   a. Passive pain-focused treatments (e.g., medication, injections). If the patient prefers this approach, (s)he should be educated to encourage openness to skill-focused treatment.
   b. Skill-focused treatments (e.g., physical therapy, psychotherapy)
   c. Integrated-pain and skill-focused treatments

Acknowledgments

The authors wish to thank Dave Newman for his assistance with this project.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment. Part VI: Lumbar Spinal Stenosis

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interest: The contents of this report do not represent the views of the Department of Veterans Affairs or the U.S. government. The authors have no conflicts of interest to report.

Abstract

Objective. To present the sixth in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. This article focuses on the evaluation and management of lumbar spinal stenosis (LSS), the most common condition for which older adults undergo spinal surgery.

Methods. The evaluation and treatment algorithm, a table articulating the rationale for the individual algorithm components, and stepped-care drug recommendations were developed using a modified Delphi approach. The Principal Investigator, a five-member content expert panel and a nine-member primary care panel were involved in the iterative development of these materials. The illustrative clinical case was taken from the clinical practice of a contributor's colleague (SR).

Results. We present an algorithm and supportive materials to help guide the care of older adults with LSS, a condition that occurs not uncommonly in those with CLBP. The case illustrates the importance of function-focused management and a rational approach to conservative care.

Conclusions. Lumbar spinal stenosis exists not uncommonly in older adults with CLBP and management often can be accomplished without surgery. Treatment should address all conditions in addition to LSS contributing to pain and disability.

Key Words. Aged; Assessment; Lumbar Spinal Stenosis; Spinal Stenosis; Chronic Pain; Elderly; Low Back Pain; Primary Care; Chronic Low Back Pain

Introduction

Lumbar spinal stenosis (LSS) is a common source of pain and diminished function among older adults with chronic low back pain (CLBP). Lumbar spinal stenosis results from narrowing of the lumbar spinal canal, and/or intervertebral foramina most often resulting from degenerative changes in the spine including facet joint arthrosis, loss of intervertebral disk height, degenerative spondylolisthesis, ligament thickening, post-surgical fibrosis, etc. [1]. The prevalence of LSS based on imaging criteria is estimated to be almost 50% in individuals over age 60, but many older adults with imaging evidence of anatomical stenosis are asymptomatic [2]. Lumbar spinal stenosis is the most common indication for spinal surgery among Medicare recipients, [3,4] occurring at a rate of 135.5 surgeries per 100,000 Medicare beneficiaries in 2007 [5].

Symptomatic LSS is often characterized by neurogenic claudication which is defined as symptoms of pain, weakness and/or numbness emanating from the spine and radiating into one or both buttock, thigh, or lower leg [6]. It is theorized that since extension of the spine and weight-bearing forces cause greater narrowing of the spinal canals [7,8] that the symptoms of LSS are exacerbated by standing, walking and bending backwards and relieved by sitting, lying or forward flexion movements. Other common clinical findings can include a wide-based gait, positive Romberg sign, and sensory or motor deficits in one or both lower extremities [9]. Because these symptoms are frequently present in other conditions common among older adults (i.e., hip osteoarthritis, vascular claudication, etc.), careful differential diagnosis is important [10]. Lumbar spinal stenosis can co-occur with these and other chronic conditions and thus may be an important contributor to a syndrome of functional compromise in older adults [11].

Despite the prevalence of LSS, there remains a good deal of uncertainty and variability in clinical management of the condition. The natural history of LSS is not well-understood, but it appears that many symptomatic individuals remain stable or improve over time [12] and those with asymptomatic LSS often remain free from symptoms for many years [13]. Except in rare instances of progressive neurologic deficits or cauda equina involvement, a period of non-operative management is generally advocated as an initial strategy [14,15]. Various non-surgical approaches have been recommended including watchful waiting, medications, physical therapy using a variety of interventions, and epidural steroid injections [16,17]; however there is little evidence to inform the selection or sequencing of these options [18]. An increasing number of individuals with LSS receive surgery, particularly complex fusion procedures [4,5,19,20]. More than 37,000 surgical procedures for LSS were performed in 2007 among Medicare recipients at a total cost of $1.65 billion [5]. Despite this level of utilization, a lack of consensus regarding appropriate indications for surgery is evidenced by high rates of geographic variation in LSS surgical procedures [21].

The number of older adults living with degenerative LSS will continue to increase given the aging of the population. With the desire of older adults to remain active and independent, there will be an increasing need for effective management strategies to mitigate the pain and resulting disablement that can occur with LSS. We present a case of an older adult with chronic low back pain related to LSS. The case highlights the pragmatic application of an algorithm developed to guide the diagnostic and treatment processes for older adults with LSS.

Methods

A modified Delphi technique involving a content expert panel and a primary care review panel, as described in the series overview [23] was used to create the LSS algorithm (Figure 1), the table providing the rationale for the various components of the algorithm (Table 1), and the stepped-care medication table (Table 2). Expertise represented among the 5 Delphi expert panel members for the LSS algorithm included geriatric medicine, physical therapy, physiatry, pain medicine and chiropractic.

Case Presentation

Relevant History

This patient is a 74-year-old female who is a retired cashier/business manager. She presented to a physiatry clinic with complaints of chronic, recurrent low back and lower extremity pain with episodes dating back approximately 20 years. Her chief complaint is back and left lower extremity pain extending to her dorsal foot limiting her walking to 20-30 minutes and her ability to stand upright when walking. Her symptoms have been gradually worsening over the past 2-3 years, and her current episode began suddenly two months ago when she began having pain down to her left foot and ankle that was further limiting her ability to walk. Any prolonged standing or walking exacerbates symptoms while sitting, leaning on a shopping cart, and using a walking stick relieves her symptoms. She denies any recent falls or concerns for falls. Her medical history includes osteoarthritis at the cervical spine, lumbar spine, and hands. She has a remote history of a right lower leg fracture

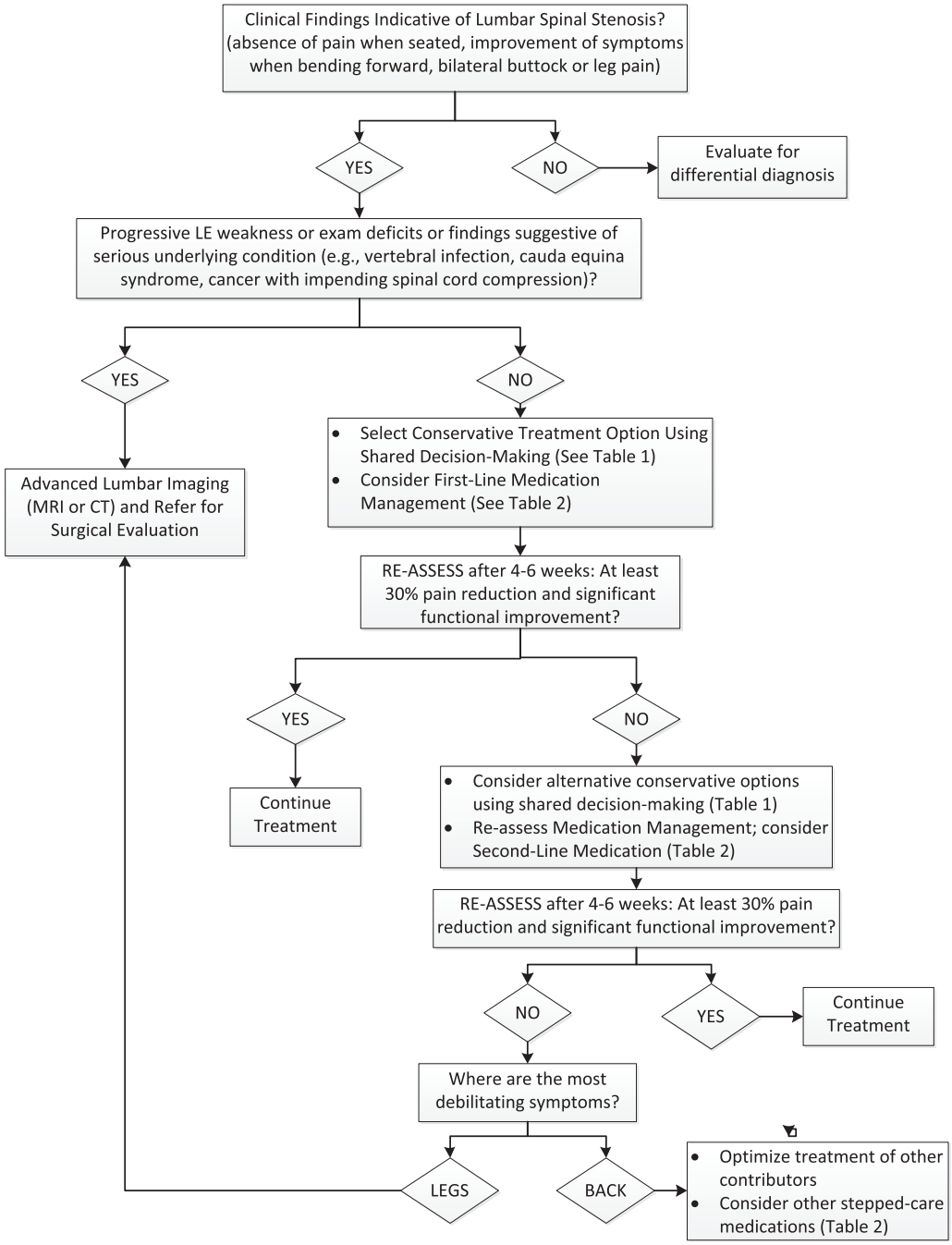

Figure 1 Algorithm for the evaluation and treatment of lumbar spinal stenosis.

and a resulting right knee flexion contracture. She has no history of any surgeries. Her current medications and supplements included: Lovastatin, Vitamin D-3; calcium citrate, fish oil, Coenzyme Q10, and Magnesium. She did not have any prescribed pharmaceutical pain management, but she uses non-prescription acetaminophen as needed several times a week to reduce pain with walking. She had three previous epidural steroid injections to manage past episodes. Her primary goal is to return to her usual routine of walking 45-60 minutes at least 4 days per week without pain or having to forward flex her trunk to limit symptoms.

Relevant Physical Examination

The patient is alert and oriented with no apparent distress. Her standing posture reveals increased flexion of lumbar spine and hips as well as the right knee due a flexion contracture of her knee. Her weight bearing is

Table 1 Lumbar spinal stenosis: Theoretical and pragmatic underpinnings of algorithm recommendations

Conservative treatment options		
1. Watchful waiting	Natural history of LSS can be favorable in 1/3 to 1/2 of patients. Catastrophic neurologic decline is rare.	[12,13]
2. Physical therapy	Improvements in pain and function with physical therapy including exercise and manual therapy. Can provide favorable results even in patients considered surgical candidates.	[36,42]
3. Manipulation	Improvement in function for patients with chronic low back pain. No studies specific to LSS.	[35]
4. Massage	Improvements in pain and disability for patients with chronic low back pain. Improvements enhanced when combined with exercise. No studies specific to LSS. No evidence of superiority for any specific massage technique.	
5. Acupuncture	Improvement in pain and function for patients with chronic pain. No studies specific to LSS.	[43]
6. Epidural steroid injection	Short-term improvement in pain and walking ability for patients with LSS.	[44]
7. Cognitive behavioral therapy	Short-term improvement in pain and function reported in comparison to controls for patients with chronic LBP. No studies specific to LSS.	[45]
8. Lumbosacral corset	Suggested to increase walking distance and decrease pain in patients with LSS. There is no evidence that results are sustained once the corset is removed.	[46]
9. Aquatic therapy	Exercises performed in the water may provide an opportunity for physical activity in patients with LSS whose symptoms substantially limit land-based exercise.	–

LSS = lumbar spinal stenosis. LBP = low back pain.

shifted to be greater on the left lower extremity. Her gait reveals increased forward flexion in the lumbar spine and hip flexion. Her spinal range of motion is full without pain in flexion; lumbar extension is limited to a few degrees with complaints of stiffness in her low back. There were no hip range-of-motion deficits. Her right knee range of motion reveals a 10 degree extension lag. Neurological assessment of her lower extremities reveals symmetrical strength and intact sensation with symmetrical Patellar and Achilles reflexes graded as 1+ bilaterally. Select muscle strength testing shows hip extension weakness graded as 4/5 bilaterally, and abdominal weakness graded as a 3/5 using Kendall's leg lowering test. Examination of muscle length indicates tightness of her hip flexors (iliopsoas and rectus femoris) and her iliotibial band bilaterally.

Imaging

Spinal radiographs were performed indicating multi-level degenerative disc disease and facet joint hypertrophy. There was no indication of degenerative spondylolisthesis. MRI was also performed and the results were characterized as severe central canal stenosis at the L3/4 and L4/5 spinal levels in the radiology report.

Clinical Course

The patient initially received an epidural steroid injection which abated her left lower extremity symptoms and back pain. However, she continued to have difficulty walking without leaning on a shopping cart, her exercise program was limited to walking 1.5 miles using a walking stick and approximately two rest breaks, and she was unable to stand fully upright after walking two blocks. Due to these continued activity limitations she began physical therapy care three weeks after her injection. Over six visits of physical therapy focused on manual therapy and exercises to improve hip extension and hip flexor flexibility, she was able to stand more erect statically, but she still needed to flex forward after approximately 5 minutes of walking. Her walking distance was still limited to 1.5 miles, but she indicated she needed breaks due to "breathing hard" rather than pain. Throughout her treatment, she continued going to a group exercise class for older adults

Table 2 Stepped-care drug management for lumbar spinal stenosis

Drug	Dose/titration	Important adverse events/precautions
First-line medication		
Acetaminophen	325-1000 mg q4-6h while awake, max 3000 mg/d Adjust dosing interval for renal function: CRcl 10-50: q6 hrs; CRcl $<$ 10: q8h	Ask about all OTCs with acetaminophen; increased toxicity from chronic use if heavy EtOH use, malnourishment, pre-existing liver disease–decrease maximum daily dose to 2 gm.
Salsalate choline magnesium trisalicylate	500-750 mg twice daily; maximum dose 3000 mg/day 750 mg three times daily	Does not interfere with platelet function; GI bleeding and nephrotoxicity rare; salicylate concentrations can be monitored if toxicity suspected. Providers should educate patients about symptoms associated with salicylism (e.g, nausea, vomiting, tinnitus, vertigo, reversible hearing loss, etc.).
Second-line medication		
Gabapentin	100 mg tid; consider 300 mg qhs if there is difficulty sleeping associated with pain	May cause dizziness and increase fall risk. May cause sedation and worsen peripheral edema. Withdrawal seizures possible with abrupt withdrawal from high doses.
Tramadol	25 mg once a day; increase by 25-50 mg daily in divided doses every 3-7 days as tolerated to max dose of 100 mg 4 times a day. Renal dosing (CRcl $<$ 30 ml/min) 100 mg twice a day.	Seizures and orthostatic hypotension. Other side effects similar to traditional opioids including constipation, sedation, confusion, respiratory depression. Potential for serotonin syndrome if patient is on other serotonergics.
Hydrocodone/ acetaminophen	2.5/325 or 5/325-10/325 mg q4-6h; max acetaminophen dose 3gm/d	For all opioids, increased fall risk in patients with dysmobility. May worsen or precipitate urinary retention when BPH present. Increased risk of delirium in those with dementia. Because of increased opioid sensitivity, older adults are at greater risk for sedation, nausea, vomiting, constipation, respiratory depression, urinary retention and cognitive impairment. Start stimulant laxative to prevent/treat constipation. Many would start at opioid initiation if patient has existing complaints of constipation or other risk factors. Some providers advocate ensuring all patients initiating opioids have a stimulant laxative available to start at the first sign of constipation. Exercise caution and follow closely if opioids are started in patients who drive. Avoid concomitant prescription of opioids and other CNS depressants. Risk of addiction/diversion present with all opioids. Before starting assess risk with the Opioid Risk Tool. During maintenance, monitor using tool such as Current Opioid Misuse Measure. (www.painedu.org)

(continued)

Table 2 Continued

Drug	Dose/titration	Important adverse events/precautions
Oxycodone or morphine	Start with 2.5-5 mg oxycodone or morphine q4h and titrate no more frequently than q7d; assess total needs after 7d on stable dose, then convert to long acting. Morphine *Renal impairment*: Cl_{cr} 10-50 mL/minute: Administer at 75% of normal dose; Cl_{cr} <10 mL/minute: Administer at 50% of normal dose. *Hepatic impairment*: No dosage adjustment provided in manufacturer's labeling. Pharmaco-kinetics unchanged in mild liver disease; substantial extrahepatic metabolism may occur. In cirrhosis, increases in half-life and AUC suggest dosage adjustment required. Oxycodone *Renal impairment*: Serum concentrations are increased ~50% in patients with Cl_{cr} <60 mL/minute; adjust dose based on clinical situation. *Hepatic impairment:* i*mmediate release:* Reduced initial dose may be necessary (use a conservative approach to initial dosing); adjust dose based on clinical situation. *Controlled release:* Decrease initial dose to one-third to one-half the usual starting dose; titrate carefully.	Side effects and risks of addiction/diversion as per hydrocodone. NEVER start long acting opioid before determining needs with short acting.
Duloxetine	Start 20-30 mg/d; increase to 60 mg/d in 7d. Not recommended in ESRD or CLcr<30.	May precipitate serotonin syndrome when combined with triptans, tramadol, and other antidepressants. Key drug-disease interactions: HTN, uncontrolled narrow-angle glaucoma, seizure disorder. Precipitation of mania in patients with bipolar disorder. Important adverse effects include nausea, dry mouth, sedation/falls, urinary retention, constipation. Contra-indicated with hepatic disease and heavy alcohol use. Abrupt discontinuation may result in withdrawal syndrome. Contraindicated within 14 days of MAOI use.

that involved sitting and standing exercises, which was very important to her.

Approach to Management

Diagnostic Considerations

The patient presented in this case was diagnosed with degenerative LSS based on clinical criteria that were consistent with imaging findings. The high false-positive rate for imaging in older adults [23] makes clinical correlation of any imaging findings a key consideration in the diagnosis of LSS as described in the expert panel algorithm (Figure 1). This patient was most debilitated by her leg symptoms, providing a rationale for the imaging that had been performed. As noted in the expert panel algorithm, imaging for patients in the absence of debilitating leg symptoms or other indications should be approached cautiously because of the risk for false positive results. Although rates of surgery of LSS are increasing [5], this patient responded favorably to a course of non-surgical management. Many patients diagnosed with LSS will have satisfactory clinical outcomes without surgery based on the natural history of the condition and/or benefits of various non-surgical treatment strategies [20].

The patient's complaints of leg pain that worsened with walking and was relieved by sitting (i.e., neurogenic claudication), and improvement of symptoms with forward flexion of her spine are important clinical findings that can be related to narrowing of spinal canals [6,24] and corroborate this patient's imaging findings. Claudication symptoms can also occur from arterial insufficiency. Vascular claudication, unlike claudication of neurogenic origin, is not affected by spinal position and can be relieved by ceasing activity and standing instead of having to sit down [25]. Further confirmation of the neurogenic versus vascular origins of this patient's claudication symptoms may have been accomplished by recording a normal ankle-brachial index, a measurement of the blood pressure in the lower leg compared to the arm [26]. Hip osteoarthritis is another differential diagnosis whose symptoms of increased pain while walking that is relieved by sitting can mimic LSS, and the two conditions frequently co-occur [10]. This patient however lacked hip pain and did not exhibit a loss of hip internal rotation range of motion at the time of evaluation. If present, findings of loss of hip ROM or provocation of pain would suggest need for further diagnostic work-up for hip osteoarthritis, as described in an earlier algorithm in this series [27].

Therapeutic Options

Several non-surgical treatment options have been recommended for patients diagnosed with LSS including watchful waiting, physical therapy, spinal injections as well as various complementary and alternative strategies such as chiropractic, acupuncture and massage [15,20]. Research is currently lacking to clarify the most effective options or specify optimal sequencing of treatments, thus a process of shared decision-making on the preferred options for an individual patient is recommended (Figure 1). Re-assessing the benefits of whichever non-surgical treatment approach is selected is an important consideration for effective management since there is little information to *a priori* identify which treatment may be helpful for any particular patient. The impact of treatment on patients' pain and function should be the focus of the re-assessment process. On average, an improvement of 30% in pain (2 points on a 0-10 numeric pain-rating scale) or function assessed with a validated questionnaire such as the Oswestry or Roland Morris can be considered as clinically meaningful improvement [28,29]. If meaningful improvement is not achieved, an alternative treatment strategy may be recommended. Surgery for degenerative LSS can be effective for many patients who fail to respond to non-surgical treatments [30]. In the absence of spondylolisthesis or instability, decompression surgery without fusion is recommended [20].

The patient in this case opted to first pursue epidural steroid injections based on receiving relief with these injections for prior episodes of LSS symptoms. Recent systematic reviews suggest epidural injections may provide short-term (2 weeks to 6 months) symptom relief in patients with neurogenic claudication due to LSS [20,31,32]. Existing evidence is limited and lacking in methodological rigor, and thus opinions on the efficacy of epidural injections differ. Despite this uncertainty, epidural steroids are commonly used for patients with LSS and utilization has been increasing rapidly [34]. This patient experienced a clinically meaningful reduction in pain following her epidural injection but her functional deficits persisted. Because her recovery was incomplete, the determination was next made to pursue physical therapy as a non-surgical treatment strategy.

The evidence for the benefits of physical therapy for LSS has also historically been sparse. Previous recommendations based largely on evidence from patients with chronic back pain have advocated that if physical therapy is chosen, an active approach focused on exercise with manual therapy and instruction for an ongoing self-monitored exercise program should be used, as opposed to physical therapy approaches focused on use of passive modalities (e.g, ultrasound, moist heat, etc.) [20,34,35]. A study by Delitto and colleagues [36] published since the most recent systematic reviews supports a physical therapy approach emphasizing exercise, specifically lumbar flexion exercises, exercises directed to patient-specific strength or flexibility deficits and general conditioning, as well as patient education about LSS, the favorable natural history in many patients, and the importance of remaining active. Results from this randomized trial found this physical therapy approach equivalent to surgical decompression after 2 years, although over half (57%) of the patients randomized to physical therapy crossed over to receive surgery by 2 years [36]. The patient in this case responded favorably to physical therapy with

improvement in her function. At the conclusion of her physical therapy treatment, she continued to experience deficits in endurance with physical activities, highlighting the need for an ongoing exercise and physical activity program.

The goal of pharmacologic management for patients with LSS is to manage pain so that function can be improved. The patient in this case opted to use over-the-counter nonsteroidal anti-inflammatories (NSAIDs) for this purpose. It should be highlighted that chronic use of NSAIDs is not recommended for older adults [38] and the patient presented used them only on an as needed basis, specifically to help her comply with her walking exercise program. Opioids should be considered a second-line analgesic (Table 2). Both opioids and NSAIDs have serious potential adverse effects in older adults (e.g., obstipation, falls, hip fractures, depression, disrupted sleep architecture, and delirium with opioids; renal failure, painless gastrointestinal bleeding, hypertension, congestive heart failure with NSAIDs) and careful monitoring with ongoing assessment of risk versus observed benefits is essential [36,37].

One of the most important aspects of treating older adults with LSS is to treat patients comprehensively, targeting all contributors to their CLBP and leg symptoms. We have gathered preliminary data suggesting that as many as half of older adults with CLBP and neurogenic claudication may have other important contributors to their symptoms, including hip osteoarthritis, sacroiliac joint syndrome, myofascial pain and fibromyalgia [38]. It is critical that providers exhaust treatments that address all functionally limiting conditions as a part of treating the older patient with LSS and neurogenic claudication [39,40].

Resolution of Case

This case represents a fairly typical presentation of an older adult with LSS. Her clinical presentation included pathognomonic findings for the condition including a long history of episodic back pain with postural-dependent symptoms that worsen with activities or postures that emphasize spinal extension or involve compression forces and ease with flexion positions. As is typical in cases of LSS, differential diagnosis is a key consideration as the symptoms can mimic other chronic conditions common in older adults such as hip osteoarthritis or vascular claudication and imaging is prone to false positive findings. The patient in this case, like many with LSS, was able to effectively manage her symptoms with pain relieving treatments including injections and nonsteroidal anti-inflammatory medication and maintain her function through an ongoing exercise program with assistance from physical therapy to work on specific impairments of strength and flexibility. Because of the episodic nature of LSS symptoms, this patient continues to experience intermittent back pain, however she is able to self-manage these symptoms with her exercise routine and occasional use of nonsteroidal medication.

Summary

Lumbar spinal stenosis is a very common source of pain and disability and should be considered as part of the differential diagnosis of older adults with low back pain. Due to the fact that degenerative changes in the lumbar spine are present on spinal imaging in most older adults, even those without any back pain, careful clinical examination is essential to accurate diagnosis. Differential diagnosis for conditions with similar symptom presentation is also an important consideration. Common conditions such as hip osteoarthritis and vascular claudication can present with similar symptoms and may co-occur with LSS.

Lumbar spinal stenosis is the most common indication for spinal surgery in the United States [4] and rates of surgery, particularly complex procedures involving fusion continue to increase [41]. The patient presented in this case however highlights the reality that many older adults with LSS can maintain function and manage their condition without surgery. A wide variety of non-surgical treatment options are available for LSS, and choosing among them for a particular patient can be difficult. The patient in this case found benefit in maintaining a regular exercise routine and intermittently using acetaminophen for symptom exacerbations, as well as epidural steroid injections and physical therapy for more severe symptoms. All of these strategies were used in an effort to maintain her function. The maintenance of function should be an overarching goal in developing a management plan for all older adults with LSS, and all contributors to pain and disability should be targeted.

Key Points

1. Lumbar spinal stenosis (LSS) is a clinical diagnosis that is corroborated by advanced imaging. Asymptomatic anatomical LSS is common in older adults, thus imaging should not be ordered without first conducting a thorough clinical assessment.
2. In the absence of progressive neurological deficits or cauda equina symptoms, management of LSS should begin conservatively (e.g., physical therapy, epidural injection, oral analgesics).
3. Many older adults with LSS can expect to remain symptomatically stable or improve over time, thus practitioners should educate patients about the importance of remaining active and attempt to quell fears of LSS-associated disablement.
4. LSS often co-occurs with other conditions that contribute to pain and disability in older adults, (i.e., the other conditions covered in this series such as hip osteoarthritis, depression, myofascial pain and fibromyalgia). Comprehensive assessment and treatment is needed to optimize outcomes.

Acknowledgments

The authors thank Dave Newman for his thoughtful review of the manuscript. The authors would also like to thank Kim Bennett, PT, PhD, for identifying the case presented.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment: Part VII: Insomnia

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interest: The contents of this report do not represent the views of the Department of Veterans Affairs or the U.S. government. The authors have no conflicts of interest to report.

Abstract

Objective. To present the seventh in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. This article focuses on insomnia and presents a treatment algorithm for managing insomnia in older adults, along with a representative clinical case.

Methods. A modified Delphi process was used to develop the algorithm and supportive materials. A multidisciplinary expert panel representing expertise in health psychology and sleep medicine developed the algorithm and supporting documents that were subsequently refined through an iterative process of input from a primary care provider panel.

Results. We present an illustrative clinical case and an algorithm to help guide the care of older adults with insomnia, an important contributor to CLBP and disability. Multicomponent cognitive behavioral therapy for insomnia (CBTI) and similar treatments (e.g., brief behavioral treatment for insomnia [BBTI]) are the recommended first-line treatment. Medications should be considered only if BBTI/CBTI is suboptimal or not effective and should be prescribed at the lowest effective dose for short periods of time (< 90 days).

Conclusions. Insomnia is commonly comorbid with CLBP in older adults and should be routinely evaluated and treated because it is an important contributor to pain and disability. The algorithm presented was structured to assist primary care providers in planning treatment for older adults with CLBP and insomnia.

Key Words. Low Back Pain; Chronic Pain; Chronic Low Back Pain; Insomnia; Sleep Disorders; Elderly; Older Adults

Introduction

Sleep problems are a highly prevalent comorbidity and consequence of chronic low back pain (CLBP), impacting an estimated 50–80% of individuals with CLBP [1–3]. Insomnia—dissatisfaction with sleep quantity or quality related to difficulty initiating, maintaining, and/or early morning awakenings [4]—is the most common sleep disorder in the general population and among those with CLBP [5]. Insomnia also significantly increases the risk of developing CLBP, even after controlling for socioeconomic, self-reported health, lifestyle behaviors, and anthropometric variables [6]. Prolonged sleep onset latency and poor sleep quality, key symptoms of insomnia, are associated with poor physical functioning and longer pain duration [7]. Also, self-reported insomnia severity is associated with pain intensity and vice versa [2]. The bidirectional relationship of pain and sleep is supported by multiple shared neurobiological underpinnings between the two disorders. Several studies have implicated dopaminergic signaling and opioidergic signaling, as well as negative and positive affect [8]. Structural and functional changes to similar brain structures, such as the activation to the limbic area, have been implicated in both pain and insomnia [9]. Also, dysregulation of the hypothalamus-pituitary-adrenal axis and decreased brain-derived neurotrophic factor have been linked to chronic pain and insomnia [9].

Comorbid chronic pain and insomnia is an important public health issue, as the combined impact magnifies the clinical and economic consequences and correlates related to each disorder [10]. Both insomnia and CLBP are independently related to significant reductions in quality of life [11,12], medical morbidity, and disability [13,14]. Furthermore, both are linked with a significant economic impact, each exceeding $100 billion annually [15–17]. Fortunately, effective pharmacological [18,19] and nonpharmacological treatments [20,21] are available that reduce pain, improve physical functioning, and decrease sleep disturbance. The most commonly prescribed medications for chronic pain and insomnia are opioid analgesics and sedative hypnotics [18,19], respectively. Unfortunately, these medications are linked with side effects, such as risk of falls and tolerance and withdrawal [22–24], all issues particularly important in older adults. Opioid analgesics may actually disturb sleep, change sleep architecture, and induce sleep disordered breathing [25,26]. Effective nonpharmacological treatments for insomnia with fewer associated risks are available but are often underutilized by clinicians. Assessment tools and treatment guidelines are often too general and global to meet the needs of a diverse patient population. Furthermore, the most commonly recommended nonpharmacological approach for managing insomnia—sleep hygiene [27]—lacks evidence [28] as a stand-alone treatment, especially in the context of chronic pain. Accordingly, there is a great deal of variation in how older adults with chronic pain and insomnia are treated, and there is limited evidence on effective strategies for the management of comorbid pain and insomnia in real-world clinical settings.

A substantial proportion of older adults with CLBP report insomnia. We present a pragmatic, evidence-based approach to the management of insomnia in older adults and a case illustrating the application and efficacy of nonpharmacological strategies.

Methods

A detailed description of the modified Delphi process used to create the algorithm (Figure 1), the table providing the rationale for the algorithm components (Table 2), and the medication table (Table 3) are provided in the series overview [29]. The expert panel team leader (ADB) drafted the initial algorithm and supportive tables based upon a comprehensive review of the literature and knowledge of insomnia treatments. The expert panel, which consisted of two health psychologists with expertise in behavioral sleep medicine (ADB, AG) and a sleep medicine physician (DJB), refined the algorithm and accompanying tables before receiving feedback from the primary care panel, as described previously [29].

Case Presentation

Insomnia Evaluation

The patient is a 66-year-old male veteran with a 20-year history of insomnia reported as secondary to chronic shoulder, low back, and knee pain. He used only over-the-counter analgesics and had declined a referral to the pain clinic in the past. He reported difficulty with maintaining sleep, often waking due to pain, with difficulty returning to sleep; symptoms occurred at least 4 nights per week. During the day, he reported fatigue, decreased motivation to complete tasks, and poor concentration. Estimated total sleep time ranged from 2–6 hours, usually no more than 2–3 hours at a time, with his bedtime from 10:30 PM–11:30 PM and wake time ranging from 3:30 AM–6:00 AM. Pre-bedtime activities included watching television, talking with his wife, smoking cigarettes, and taking sleep medication (trazodone, 50 mg). He reported minimal sleep onset latency, usually less than 10 minutes, but reported wake after sleep onset of 45 minutes or longer; after each nighttime awakening and about once a week, he was unable to return to sleep. On some nights during wake after sleep onset periods, he stayed in bed, and others he paced around the house, smoked a cigarette, read, watched baseball, or drank a glass of milk. He reported taking late-morning or early afternoon naps 1–2 times per week following nights with an early morning awakening. The patient denied symptoms of other sleep disorders (e.g., sleep apnea) or psychiatric disorders.

Medical History, Sleep Medication, and Relevant Substance Use

The patient's medical history is significant for insomnia, osteoarthritis, lung nodule, benign prostatic hypertrophy, contact dermatitis, erectile dysfunction, acne, tobacco

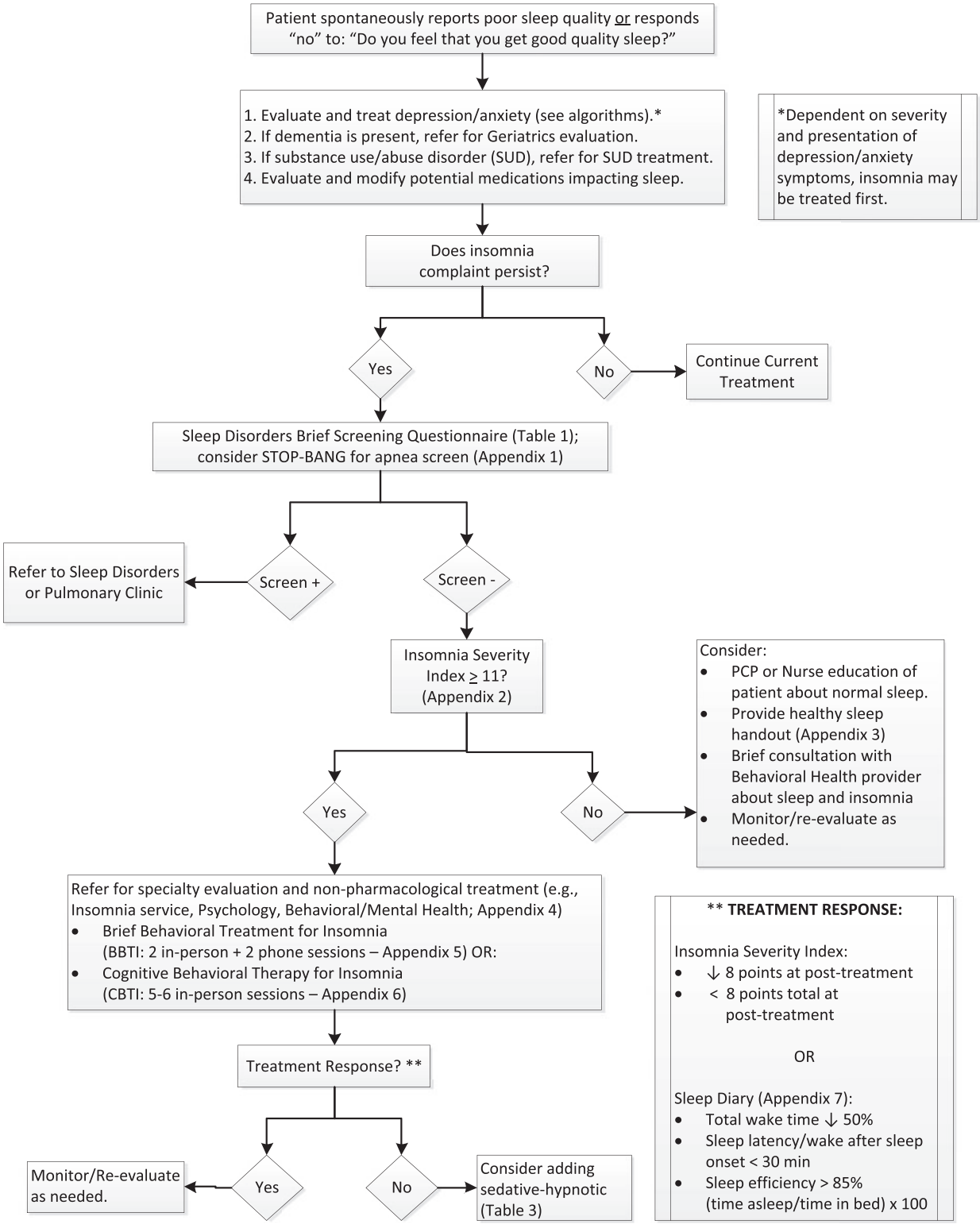

Figure 1 Algorithm for the evaluation and treatment of insomnia in an older adult.

Table 1 Sleep disorders brief screening questionnaire

Consider a referral to sleep disorders clinic, pulmonary clinic, or a sleep specialist for further evaluation if patient responds positively to any of the following questions:
In the past month...

Question	No	Yes
1. Have you had a problem with excessive sleepiness, including prolonged nighttime sleep (> 9 hours)? Is your sleep nonrestorative (wake up feeling unrefreshed)? Or do you sleep during the daytime almost daily?[Hypersomnolence Disorder]		
2. Have you noticed (or has anyone witnessed) the following: you snore, snort, have breathing pauses while sleeping, or wake up gasping for air?[Obstructive Sleep Apnea/Breathing-Related Sleep Disorder][If Yes, please complete the STOP-BANG]		
3. Have you ever been told, or suspected yourself, that you seem to act out your dreams while asleep (e.g., punching, flailing your arms in the air, making running movements, etc.)?[REM Behavior Disorder]		
4. Have you had irresistible attacks of sleep, such as suddenly lapsing into sleep or napping?[Narcolepsy]		
5. Is your sleep/wake schedule "out of sync" with other people? Do you have an unusual sleep/wake schedule (e.g., go to bed very late or sleep in very late)?[Circadian Rhythm Sleep-Wake Disorder]		
6. Have you had unpleasant feelings in your legs and an urge to move your legs as bedtime approaches (not pain)?[Restless Legs Syndrome]		
7. Have you noticed (or has anyone told you about) jerking arm/leg movements during sleep?[Periodic Limb Movements]		
8. Have you had (or has anyone told you about) abrupt awakenings from sleep beginning with a loud scream? Does this occur regularly/often?[Sleep Terrors]		
9. Have you had episodes of arising from bed during sleep and walking about?[Sleep Walking]		

Note: Items adapted from DSM-5 criteria for Sleep-Wake Disorders [4].

use disorder, and anger issues. He reported currently taking nightly trazodone 50 mg, which only occasionally helped to reduce the frequency of nighttime awakenings from 2–3 per night to 0–1 and did not help reduce early morning awakenings. He also reported a history of using flurazepam for several months (early 1990s), which he reported as helpful in improving his sleep. He also reported drinking large quantities of coffee each morning (twelve 6-oz cups ["a pot"]), but avoided caffeine in the afternoon due to concern it would interfere with nighttime sleep. He smoked one pack of cigarettes per day and half "a joint" of marijuana 2–4 times per week before bed, which he reported as helpful. Alcohol use was minimal, only a few times per year, and he denied ever drinking alcohol to assist with sleep.

Initial Clinical Course

The patient completed an insomnia evaluation, including week-long sleep diaries at baseline and throughout treatment [30], and five sessions of cognitive behavioral therapy for insomnia (CBTI) with a psychologist. When treatment began, he reported difficulty with maintaining sleep due to pain and frequent early morning awakenings. Treatment goals were: reduce wake after sleep onset, increase sleep quality, and reduce coffee consumption (morning) and cigarette use (night). The first three sessions focused on restructuring the sleep-wake schedule with stimulus control and sleep restriction, and reviewing and identifying relevant sleep hygiene factors. Session 3 also introduced concepts of cognitive therapy and further titration of the sleep-wake schedule. Session 4 continued the cognitive therapy work; session 5 emphasized self-management strategies and relapse prevention.

Approach to Management

The standard of practice for treating insomnia disorder is multicomponent CBTI [21,31–34]. However, the first step is to identify any organic and/or noninsomnia sleep

Table 2 Insomnia: theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
Evaluation and screening		
Evaluate for comorbid disorders that may require treatment and evaluate for potential sleep disturbing medications	Before considering a sleep disorder in the older adult with chronic pain, it is important to evaluate and potentially manage comorbidities and/or medications prior to addressing the sleep disorder. Dependent on comorbidity, the sleep problem may be able to be addressed prior to, or concurrently with, the management of a comorbidity or medication.	[4,25,26,43–45]
Sleep Disorders Brief Screening Questionnaire	This questionnaire (a compilation of elements of DSM-5) assesses for key diagnostic criteria for noninsomnia sleep-wake disorders. If any question is positive, further evaluation by a sleep specialist may be necessary.	[4]
STOP-BANG	The STOP-BANG is a brief screening measure to detect risks for obstructive sleep apnea (e.g., snoring, neck size, hypertension). A score ≥ 3 indicates intermediate to high risk of obstructive sleep apnea (OSA). In patients with mild, moderate, and severe OSA, this cut-off had sensitivity of 84.1%, 68.4%, and 94.8%, respectively, and specificity of 40.3%, 10.8%, and 27.6%, respectively. 0–2, low risk 3–4, intermediate risk 5–8, high risk	[36]
Insomnia Severity Index	Insomnia Severity Index (ISI)—a brief screening and outcome measure of insomnia severity. Insomnia causes nighttime sleep disturbance and clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important area of functioning. ISI score ≥ 11 indicates further assessment and/or treatment is needed. In a clinical sample, this cut-off had 97% sensitivity, 100% specificity, and a 97.9% correct classification rate. Categorical Scoring: 0–7, no clinical insomnia 8–14, subthreshold insomnia 15–21, clinical insomnia, moderate 22–28, clinical insomnia, severe	[37,38]
Treatment		
Nonpharmacological treatment of insomnia	Brief Behavioral Treatment for Insomnia (BBTI) and Cognitive Behavioral Therapy for Insomnia (CBTI) are the recommended first-line treatments for insomnia.	[28,55]
BBTI	BBTI emphasizes the behavioral components: stimulus control and sleep restriction. Stimulus Control, an American Academy of Sleep Medicine (AASM) Standard Recommendation, helps patients to re-associate the bed/bedroom with sleep and re-establish a consistent sleep-wake schedule. Sleep restriction, an AASM Guideline Recommendation, helps patients to re-associate the bed/bedroom with sleep and re-establish a consistent sleep-wake schedule.	[28,39,56]
CBTI	CBTI is a multicomponent therapy that includes stimulus control and sleep restriction plus cognitive therapy and relaxation training, both AASM Standard Recommendations. Cognitive therapy can further benefit treatment outcomes when combined with stimulus control and sleep restriction; cognitive therapy seeks to change maladaptive and/or dysfunctional beliefs about insomnia and	[28,39,56,57]

(continued)

Table 2 Continued

Algorithm component	Comments	References
	perceived daytime impact. Relaxation training helps reduce somatic tension or intrusive thoughts using active (e.g., progressive muscle relaxation) or passive (e.g., autogenic training) methods.	
Pharmacological treatment of insomnia	Pharmacological management of insomnia is recommended to be initiated after a nonresponse or suboptimal response to BBTI/CBTI if the patient is not a good candidate for, or refuses, nonpharmacological treatments. For all sedative hypnotic and/or prescription sleep medication, it is recommended that the lowest effective dose be used to avoid chronic use ($>$ 90 days). There is potential for habituation and tolerance for nonbenzodiazepine receptor agonists (BZRA). Activities that require concentration, such as driving, should be avoided; sedative hypnotics may cause sleep-related behaviors like sleep-walking, sleep-driving, making phone calls while sleeping, and eating while asleep. See Table 3 for more information about specific sedative-hypnotic medications.	[39,56,58]

Note: Standard Recommendation: a generally accepted patient care strategy with a high-degree of clinical certainty; Guideline Recommendation: a patient care strategy with a moderate degree of clinical certainty; SOL, sleep onset latency; TAU, treatment as usual; TIB, time in bed; TST, total sleep time; TWT, total wake time; WASO, wake after sleep onset.

disorders that require further evaluation and treatment (e.g., sleep apnea, narcolepsy) [35]. Comorbid depression, anxiety, and other psychiatric disorders, especially if severe and untreated, may need to be addressed prior to beginning an insomnia intervention. However, for patients with mild to moderate psychiatric symptoms, treatment for insomnia may occur prior to, or concurrent with, addressing comorbid symptoms. Symptoms of dementia and/or substance use disorder should also be addressed before proceeding with management of insomnia. Lastly, medications that may interfere with sleep (e.g., opioids, activating antidepressants) need to be identified and appropriately managed. In the algorithm (Figure 1), a positive screen on the Sleep Disorders Brief Screening Questionnaire (Table 1) and the STOP-BANG, a screening measure for risk of obstructive sleep apnea [36], if indicated, will lead to referral to a sleep disorders clinic or pulmonary clinic. A negative screen will lead to insomnia screening with the Insomnia Severity Index (Appendix 2; [37]), a psychometrically sound, brief screening tool and outcome measure. An Insomnia Severity Index score-$\geq$ 11 indicates at least minimal to moderate symptoms [38] and in a clinical sample showed 97.2% sensitivity and 100% specificity with 0% false-positive rate, 2.8% false-negative rate, and 97.9% correct classification rate [38]. An Insomnia Severity Index score $\geq$ 11 indicates a referral is needed to a clinician for further assessment and management of insomnia.

The core components of CBTI include [28,39]: education, stimulus control, sleep restriction, sleep hygiene, cognitive therapy, and relaxation training. Treatment is flexible and should be adapted to fit the needs of the patient; however, it is recommended to introduce stimulus control and sleep restriction as early in the course of treatment as is clinically feasible. Education provides basic facts about normal sleep versus insomnia and insomnia etiology and prognosis. Stimulus control helps to strengthen the association of sleep and the sleep environment: avoid nonsleep activities in bed, go to bed only when sleepy, and do not stay in bed if awake. An important factor to consider in older adults with CLBP is how resting—lying down without the intention to sleep—can impact sleep quality. Despite its potential benefit, it is important not to rest in bed as this is in opposition to stimulus control; resting should take place outside the bedroom if possible. Sleep restriction changes the sleep-wake schedule to match actual sleep time and establishes a consistent wake time, a key variable to developing an improved sleep-wake schedule. Cognitive therapy helps the patient identify maladaptive and dysfunctional beliefs about sleep, then challenge and change those beliefs to decrease worry and arousal. Relaxation training, which may be active (e.g., progressive muscle relaxation) or passive (e.g., guided imagery, mindfulness), can help reduce physical tension and/or cognitive hyperarousal. Practicing before bed and throughout the day can help further improve sleep when combined with other components of CBTI. Sleep hygiene involves assessing and modifying behavioral and environmental variables that may impair sleep quality. Common sleep hygiene topics include: avoiding clock watching; limiting caffeine intake, especially in the afternoon and evening; timing exercise properly; and addressing precipitants of nocturia, such as dietary and substance use changes. Improving sleep hygiene alone does not significantly improve sleep but may help enhance CBTI outcomes achieved through stimulus control and sleep restriction.

Brief behavioral treatment for insomnia (BBTI; [40,41]) is another evidence-based intervention based on the

Table 3 Drug management of insomnia

Drug	Dose/titration	Important adverse effects/precautions
Heterocyclics		
Doxepin is FDA approved for sleep maintenance insomnia; Trazodone and Mirtazapine are not FDA approved for treatment of insomnia. Less evidence than the non-BRZA but several randomized controlled trials provide evidence of effectiveness vs placebo or treatment as usual. More research is needed regarding long-term efficacy. Less risk for habituation or tolerance than non-BZRA. [59–63]		
Drug	Dose/titration	Important adverse effects/precautions
Doxepin	3–6 mg at bedtime	FDA approved for sleep maintenance insomnia. Side effects: drowsiness, dizziness, headache. May exacerbate restless legs, periodic limb movements, and REM sleep behavioral disorders. Contraindications: taking an MAOI, untreated narrow-angle glaucoma, severe urinary retention, and long QT syndrome.
Trazodone	25–50 mg at bedtime	Not FDA approved; off-label for sleep-onset insomnia. Side effects: drowsiness, dizziness, headache, blurred vision, dry mouth, arrhythmias, orthostatic hypotension, and priapism. Use with caution in patients with long QT syndrome and hepatic disease.
Mirtazapine	7.5–45 mg at bedtime	Not FDA approved; off-label for sleep-onset insomnia. Side effects: drowsiness, dizziness, dry mouth, increased appetite, weight gain, constipation, asthenia, and increased risk of hyponatremia. Use with caution in patients with renal impairment, hepatic disease, hypercholesterolemia, and hyperglyceridemia.
Melatonin-receptor agonists		
FDA approved for sleep onset insomnia. No habituation or tolerance risk and fewer morning adverse effects. May be less effective for reducing SOL in older adults. [39,58,59,64,65]		
Drug	Dose/titration	Important adverse effects/precautions
Ramelteon	8 mg at bedtime	FDA approved for sleep-onset insomnia. Side effects: drowsiness, dizziness, and interaction with fluvoxamine. Use with caution in patients with mild-moderate hepatic disease. Also be cautious with patients with schizophrenia, bipolar disorder, and major depression, as hallucinations have been reported. Increased risk of seizure activity related to melatonin and melatonin-receptor agonists. Avoid alcohol with ramelteon. Avoid activities that require concentration, such as driving.
Nonbenzodiazepine receptor agonists		
While the following medications are FDA approved for sleep-onset and/or sleep-maintenance insomnia, they are recommended to be avoided by the American Geriatrics Society 2015 Updated Beers Criteria for Potentially Inappropriate Medication Use in Older Adults. It is recommended that any use of these medications be done on an individual basis. No specific agent is recommended as preferable; each has shown to have positive effects on SOL, WASO, and/or TST in randomized controlled trials vs placebo or TAU. Patients may respond differently to different agents in this class. Factors to consider include symptom pattern, past response, cost, and patient preference. While there is evidence for long-term use for some agents, use $>$ 90 days should be avoided if possible. [39,50,56,58,64,66–73]		
Zolpidem	5 mg at bedtime	Side effects: drowsiness, dizziness, headache, amnesia, and GI symptoms. Eszopiclone is associated with unpleasant taste and dry mouth. Avoid driving or performing tasks requiring mental alertness and concentration. Risk for next-morning impairment is higher if less than 7–8 hours of sleep opportunity when taking at sleep onset; increased risk of impairment if $<$ 4 hours of sleep opportunity when taking for sleep maintenance insomnia. Patients should avoid alcohol when using these drugs. Patients with dementia, cognitive impairment, or delirium and with history of falls or fractures are at increased risk for adverse CNS effects.
Zolpidem CR	6.25 mg at bedtime	
Sublingual zolpidem	1.75 mg at bedtime or during nighttime awakenings (Intermezzo)	
	5 mg at bedtime (Edluar)	
Zaleplon	5–10 mg at bedtime	
Eszopiclone	1–2 mg at bedtime	

Note: SOL, sleep onset latency; TAU, treatment as usual; TIB, time in bed; TST, total sleep time; TWT, total wake time; WASO, wake after sleep onset.

behavioral principles of CBTI, stimulus control, and sleep restriction, and is delivered in a briefer format. Treatment consists of two in-person sessions and two phone calls: session 1 (week 1, in-person) provides treatment rationale and introduces stimulus control and sleep restriction, and the patient establishes a new sleep-wake schedule; in session 2 (week 3, in-person), the patient and clinician review the sleep diary, problem-solve barriers to BBTI implementation, and adjust the sleep-wake schedule as needed; relapse prevention is also discussed during session 2. Brief phone calls during weeks 2 and 4 are used to answer patient questions, make minor adjustments to the sleep-wake schedule, and manage adherence issues.

Not all patients are good candidates for BBTI/CBTI, or adaptations to treatment are necessary. While many patients with CLBP can engage in BBTI/CBTI without adaptation, one common change to CBTI involves utilizing a more passive relaxation method, as tightening and relaxing of muscle groups (i.e., progressive muscle relaxation) may cause discomfort/pain for some patients. Furthermore, the cognitive therapy portion will likely focus, in part, on pain-sleep or pain-specific thoughts and worries. Individuals with bipolar disorder, psychotic disorders, or seizure disorders are at risk of symptom exacerbation with severe sleep restriction or marked sleep-wake routine disruption. If BBTI/CBTI is initiated, it needs to be adapted appropriately and symptoms monitored. Sleep duration < 6 hours should be avoided in these patients in order to minimize excessive daytime sleepiness and potential adverse effects of sleep loss. Patients with bipolar disorder may benefit from BBTI/CBTI that integrates aspects of interpersonal and social rhythm therapy [42], an evidence-based treatment that focuses on the bidirectional relationship between mood, life events, and daily routines. Patients with active substance use disorder/abuse should be referred for appropriate substance treatment before participating in BBTI/CBTI, as sleep disruption related to initial stages of treatment (i.e., sleep restriction) may exacerbate substance use symptoms or interfere with recovery. However, BBTI/CBTI during the recovery and remission period may be beneficial [43,44]. Another population that may require treatment adaptation is patients with dementia. For patients that require a caregiver, the burden of establishing treatment may ultimately fall to them [45]. However, patients with mild dementia or mild cognitive impairment who are still able to manage aspects of their health independently may be able to successfully implement BBTI/CBTI. Older adults often deal with multiple chronic medical disorders and take numerous medications, many of which can impact sleep (e.g., diuretics contributing to nocturia and wakefulness). As part of the initial evaluation process, medical and psychiatric comorbidities and medications are always considered in regards to potential changes/adaptations to treatment. Any changes to medications will be a collaborative process between patient, prescribing provider, and insomnia clinician.

Access to insomnia care is a concern for many patients and providers, especially those in rural areas. Evidence-based insomnia care is most often available in urban settings. Even in large urban hospitals, providers trained in BBTI/CBTI may not be available. In the community, insomnia providers can be identified through publicly available resources such as the Society of Behavioral Sleep Medicine (http://www.behavioralsleep.org/index.php/society-of-behavioral-sleep-medicine-providers) and the Association for Behavioral and Cognitive Therapies (abctcentral.org/xFAT/). Within the Department of Veterans Affairs, therapists trained to deliver CBTI can be identified by contacting local Evidence-Based Psychotherapies Coordinators. One option for individuals who live a significant distance from a provider or are unable to travel are online CBTI programs. These programs often require a subscription or modest fee but are evidence-based and effective [46], and may significantly help increase access to care.

Patients with an Insomnia Severity Index score < 11 should be provided with information about healthy sleep behaviors and monitored/re-evaluated as needed. Patients who are not good candidates for BBTI/CBTI based on clinical presentation and/or an unwillingness to engage in behavioral therapies [35] should be referred to the appropriate specialist for consideration of pharmacological treatments.

Use of the algorithm should be a collaborative process between providers and patients to establish appropriate and realistic treatment goals. Although positive treatment response to CBTI or BBTI is common, $> 70\%$ for treatment completers [47–49], patients with unrealistic goals (e.g., no more nighttime awakenings, always feeling refreshed upon awakening) should be educated about appropriate treatment expectations. Many patients can expect to significantly reduce insomnia symptoms such as sleep onset latency and wake after sleep onset as well as increase sleep quality. Sleep onset latency and wake after sleep onset can often be reduced by approximately 50%, and sleep efficiency can improve by at least 10 percentage points, with many able to achieve sleep efficiency $\geq 85\%$, an indication of good sleep quality. A reduction of ≥ 8 points on the Insomnia Severity Index is also common and indicative of categorical symptom reduction (e.g., severe to moderate, moderate to mild, or mild to minimal). While total sleep time does not always increase, patients usually report improved sleep continuity and feeling more refreshed in the morning with less impairment during the day. Patient-determined expectations and goals, when realistic and feasible, are paramount and may be determined differently than sleep diary or Insomnia Severity Index outcomes. A key goal of the collaborative process is to identify the patient's definition of treatment success and attempt to work toward that goal.

The briefer treatment, BBTI [40,41], may be an appropriate treatment starting point for many patients, as it emphasizes the behavioral components—stimulus control

and sleep restriction—of CBTI [21] and can be delivered more efficiently than CBTI. For patients who respond more slowly and/or who have higher levels of anxiety and cognitive distortions that help to maintain their insomnia, treatment can be extended and include cognitive therapy and relaxation training (where indicated). Finally, relaxation training—active or passive—can help patients reduce arousal both before bed and during the day. After treatment is complete (BBTI or CBTI), if nighttime sleep is efficient and consolidated but remains insufficient in duration, nighttime sleep can continue to be extended, or time-limited naps during the day may be appropriate as long as they do not interfere with nighttime sleep. Patients who start BBTI/CBTI on a sedative hypnotic, like the case example, can initiate treatment while still using medication but may want to consider a taper during treatment under the guidance of the prescribing physician; however, other patients may prefer to taper off their medications prior to the start of treatment.

For patients not already using sedative hypnotic medications, if response to BBTI/CBTI is suboptimal (i.e., Insomnia Severity Index pre- to post-treatment reduction less than 8 points, sleep onset latency/wake after sleep onset reduced by less than 50%, and/or continued dissatisfaction with sleep quantity/quality), starting a sedative hypnotic medication may be indicated (Table 3). Furthermore, for patients who do not have access to BBTI/CBTI or who refuse treatment, sedative hypnotic medications may be appropriate. While medications can be effective, they should generally be considered a secondary option, as BBTI/CBTI provides patients with skills they can apply long after treatment ends and has a high rate of treatment response and a low risk profile. Furthermore, rebound insomnia may occur when medications are discontinued, which is less common following BBTI/CBTI. Many sedative hypnotics also are contraindicated in older adults per the American Geriatric Society Beers Criteria [50]. Recommendations for medication management of insomnia in older adults are provided in Table 3, including dosing guidelines, adverse effects, and precautions. Sedative hypnotics should be prescribed at the lowest effective dose for the shortest possible time, as even the nonbenzodiazepine receptor agonists have potential for habituation and tolerance in some patients. The recommendations in Table 3 are consistent with the 2015 Beers Criteria for potentially inappropriate medications for older adults [50].

Resolution of Case

The patient met DSM-5 criteria for Insomnia Disorder [4], persistent with comorbid chronic pain (shoulder, back, knee). He completed an insomnia evaluation and five sessions of CBTI with a psychologist. His sleep diary at session 1 indicated sleep onset latency of approximately 8 minutes with 1–2 nighttime awakenings, for an average wake after sleep onset of 70 minutes per night. Average time in bed (TIB) was 6 hours 53 minutes, and average total sleep time was 5 hours 33 minutes; sleep efficiency was 80.6% ([total sleep time/time in bed] $\times$ 100). Using the general guideline of total sleep time + 30 minutes [41], his new time in bed was 6 hours, 12:00 AM–6:00 AM. He was initially reluctant to accept this schedule, as he preferred to go to bed earlier and was also hesitant to purposefully wake up prior to 6:00 AM. Through education and a collaborative discussion of sleep preferences, as well as consistent tracking of sleep behaviors (via sleep diary), by session 5, he shifted and extended his sleep schedule from 12:00 AM–6:00 AM to 10:30 PM–5:00 AM.

Following treatment, the patient's sleep quality had improved. His sleep efficiency increased from 80.6% to 87.4%, wake after sleep onset significantly decreased (approximately 70 to 40 minutes per night), and he stopped taking nightly trazodone. He continued to experience 1–2 awakenings/night, but his ability to fall back asleep greatly improved. Furthermore, his early morning awakenings—waking at 2:00 AM–3:00 AM and being unable to return to sleep—had mostly stopped. He also no longer met criteria for insomnia per the Insomnia Severity Index [37]; from pre- to post-treatment, his score reduced to a 7 (no clinically significant insomnia) compared with a baseline score of 18 (moderate severity). In addition to improving his sleep, he was less fatigued during the day and reported fewer problems maintaining concentration. Unfortunately, he denied improvement in pain intensity.

The patient also greatly reduced coffee consumption (12 cups/day to 6–8 cups/day) and stopped smoking cigarettes before bed and during nighttime awakenings. He continued to smoke half "a joint" of marijuana approximately once per week and was encouraged to discontinue his marijuana use. The patient still napped periodically, usually if he wanted to stay up later to watch a hockey or baseball game, but he was mindful of the impact of daytime naps on his sleep and wake drive. He also maintained realistic expectations of additional improvements post-treatment, which focused on further reducing wake after sleep onset and caffeine intake and continuing to avoid cigarettes before bed. At a follow-up appointment 5 months post-treatment, he continued to deny clinically significant symptoms of insomnia (Insomnia Severity Index = 9). He also continued to report improved daytime functioning despite no reduction in pain.

Summary

Insomnia is highly prevalent and potentially disabling among older adults with CLBP, and they should be regularly assessed and treated appropriately for comorbid insomnia. The recommended first-line treatments are nonpharmacological—BBTI/CBTI. In the case example, treatment was guided by the patient's initial sleep complaints that were measured using prospective sleep diaries [30], which resulted in a new sleep-wake schedule. Further adjustments were made based on his adherence to recommended behavioral changes and his sleep-wake preferences. The case presentation provides an example of patient and clinician working

collaboratively to find a sleep-wake schedule that fits the patient's preferences but still results in good-quality sleep. As is typical of BBTI/CBTI, his total sleep time did not improve, but sleep quality and sleep efficiency improved and subjective symptoms per the Insomnia Severity Index decreased. While no sedative hypnotic medication was added to the treatment plan (the patient was already prescribed trazodone 50 mg), for patients who do not respond adequately to BBTI/CBTI, are not good treatment candidates, or refuse treatment, medications may be indicated. However, like with BBTI/CBTI, use of medications needs to include a discussion of realistic treatment outcomes and risks and benefits of the medications, including potential side effects. While not conclusive, there is evidence that improving sleep can reduce pain symptoms [51–54], and treating insomnia and other sleep complaints should be an integral part of managing CLBP as one method to improve functioning and quality of life.

Key Points

1. Insomnia can contribute to pain and disability in older adults with CLBP; thus, it should be screened routinely.
2. Before referring patients for insomnia evaluation and treatment, potentially offending medications should be modified.
3. Special populations that may require a customized approach to insomnia management include those with mood disorders, substance use, and dementia; these patients should be comanaged by appropriate specialists.
4. BBTI or CBTI are the recommended first choices for treatment. Prescription medications should only be considered if there is an inadequate response to nonpharmacological approaches.
5. A decision to start a hypnotic medication in older adults with CLBP should be preceded by a discussion that highlights the fact that hypnotics are often associated with adverse effects. Older adults should be educated specifically about the increased risk of falls and residual sedation the next morning.

Supplementary Data

Supplementary Data may be found online at http://painmedicine.oxfordjournals.org.

Acknowledgments

The authors thank Dave Newman for his thoughtful review of the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment. Part VIII: Lateral Hip and Thigh Pain

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interest: The contents of this report do not represent the views of the Department of Veterans Affairs or the United States Government. The authors have no conflicts of interest to report.

Abstract

Objective. This article presents an evidence-based algorithm to assist primary care providers with the diagnosis and management of lateral hip and thigh pain in older adults. It is part of a series that focuses on coexisting pain patterns and contributors to chronic low back pain (CLBP) in the aging population. The objective of the series is to encourage clinicians to take a holistic approach when evaluating and treating CLBP in older adults.

Methods. A content expert panel and a primary care panel collaboratively used the modified Delphi approach to iteratively develop an evidence-based diagnostic and treatment algorithm. The panelists included physiatrists, geriatricians, internists, and physical therapists who treat both civilians and Veterans, and the algorithm was developed so that all required resources are available within the Veterans Health Administration system. An illustrative patient case was chosen from one of the author's clinical practices to demonstrate the reasoning behind principles presented in the algorithm.

Results. An algorithm was developed which logically outlines evidence-based diagnostic and therapeutic recommendations for lateral hip and thigh pain in older adults. A case is presented which highlights the potential complexities of identifying the true pain generator and the importance of implementing proper treatment.

Conclusions. Lateral hip and thigh pain in older adults can contribute to and coexist with CLBP. Distinguishing the true cause(s) of pain from potentially a myriad of asymptomatic degenerative changes can be challenging, but a systematic approach can assist in identifying and treating some of the most common causes.

Key Words. Lateral Hip Pain; Greater Trochanteric Pain Syndrome; Chronic Low Back Pain; Thigh Pain; Iliotibial Band Pain; Lumbar Radiculopathy; Lumbar Radiculitis; Hip Osteoarthritis; Lumbar Spinal Stenosis; Meralgia Paresthetica; Diagnostic Algorithm; Elderly

Introduction

Many physicians assume that an older adult with low back pain (LBP) and concomitant lateral hip/thigh pain has lumbar spinal stenosis. However, in reality there are myriad causes of lateral hip/thigh pain in older adults and the diagnosis of this pain can be challenging due to pain referral patterns. First, the hip and nearby lumbopelvic structures share innervation from common nerve roots, so pain referral patterns from pathology of these structures overlap [1,2]. Second, faulty mechanics of the lumbar spine and/or hip can lead to compensatory movement patterns and eventually result in multiple pain generators. These challenges are illustrated in a study by Sembrano and colleagues. In a sample of 200 patients presenting for evaluation by a spine surgeon, only 65% had isolated spine pain, whereas 17.5% had a combination of hip, spine, and/or sacroiliac (SI) joint pain [3]. Lastly, diagnosing the etiology of hip and lumbopelvic pain in older adults is challenging in that many people have structural abnormalities on imaging studies that are asymptomatic. For instance, 93% of asymptomatic people 60–80 years old have MRI evidence of disc degeneration, 36% have a herniated disc, and 21% have spinal stenosis [4]. Additionally, only 46.5% of women ages 65 years and older who have radiographic evidence of hip osteoarthritis (OA) report hip pain "on most days for at least 1 month" [5].

This article is part of a series that addresses coexisting conditions and contributors to chronic low back pain (CLBP) with and without leg pain in older adults [6]. We present a diagnostic and therapeutic evidence-based algorithm designed for primary care clinicians to approach lateral hip and thigh pain in older adults. This is followed by a case that illustrates the challenges of identifying the cause of lateral hip pain and/or thigh pain in older adults and highlights the importance of a proper diagnosis to initiate appropriate management. Greater trochanteric pain syndrome (GTPS) and iliotibial band syndrome (ITBS) are two local causes of lateral hip and thigh pain; therefore, the management of these syndromes will be discussed in detail. Furthermore, because as many as one in four people with hip OA present with lateral hip pain [7] and because lumbar radicular pain and spinal stenosis affecting the lumbar nerve roots can also refer pain to this region, the algorithm will describe how to identify pain from these referred sources, as well. Finally, while meralgia paresthetica (MP) is less common and its clinical presentation is often clearly distinct from the conditions listed above, we will also briefly review its presentation, diagnosis, and management. Pathology of many other lumbopelvic structures, such as the sacroiliac joints and lumbar facets, also refers pain to the lateral hip and thigh. Discussion of these conditions is beyond the scope of this paper, and if a patient does not respond in a timely manner to management outlined in the algorithm in Figure 1, referral should be made to a musculoskeletal specialist who can evaluate for these conditions, as well.

Methods

As described in-depth in the series introduction [6], a modified Delphi approach was used by a content expert panel comprised of geriatric and physiatry specialists and physical therapists, in collaboration with a primary care panel. An evidence-based diagnostic and treatment algorithm (Figure 1) was developed iteratively. A corresponding table outlining the supporting evidence is presented in Table 1. The panelists included clinicians who treat both civilians and Veterans, and the algorithm was developed so that all required resources are available within the Veterans Health Administration system.

Case Presentation

Relevant History

The patient is a 90-year-old female with past medical history including a left sacral insufficiency fracture, thoracic vertebral compression fracture, and degenerative left medial meniscus tear, who presented with stabbing left lateral hip pain. The pain was chronic but had been progressing in severity over the past month. There was no inciting event or fall. The pain was exacerbated by lying on her left side and was affecting her sleep. At baseline, she ambulated with a rollator walker, but the pain was starting to limit her ability to walk. She had a history of low back and posterior left buttock pain, but she denied current pain in these regions. She also denied weakness or pain radiation into her lower extremities.

Relevant Physical Examination

On examination, she had no pain with lumbar or hip range of motion. She had full, symmetric, and pain-free lower extremity strength, and deep tendon reflexes and sensation to light touch were intact and symmetric in her lower extremities. She had a positive Trendelenburg sign with single-leg stance bilaterally,

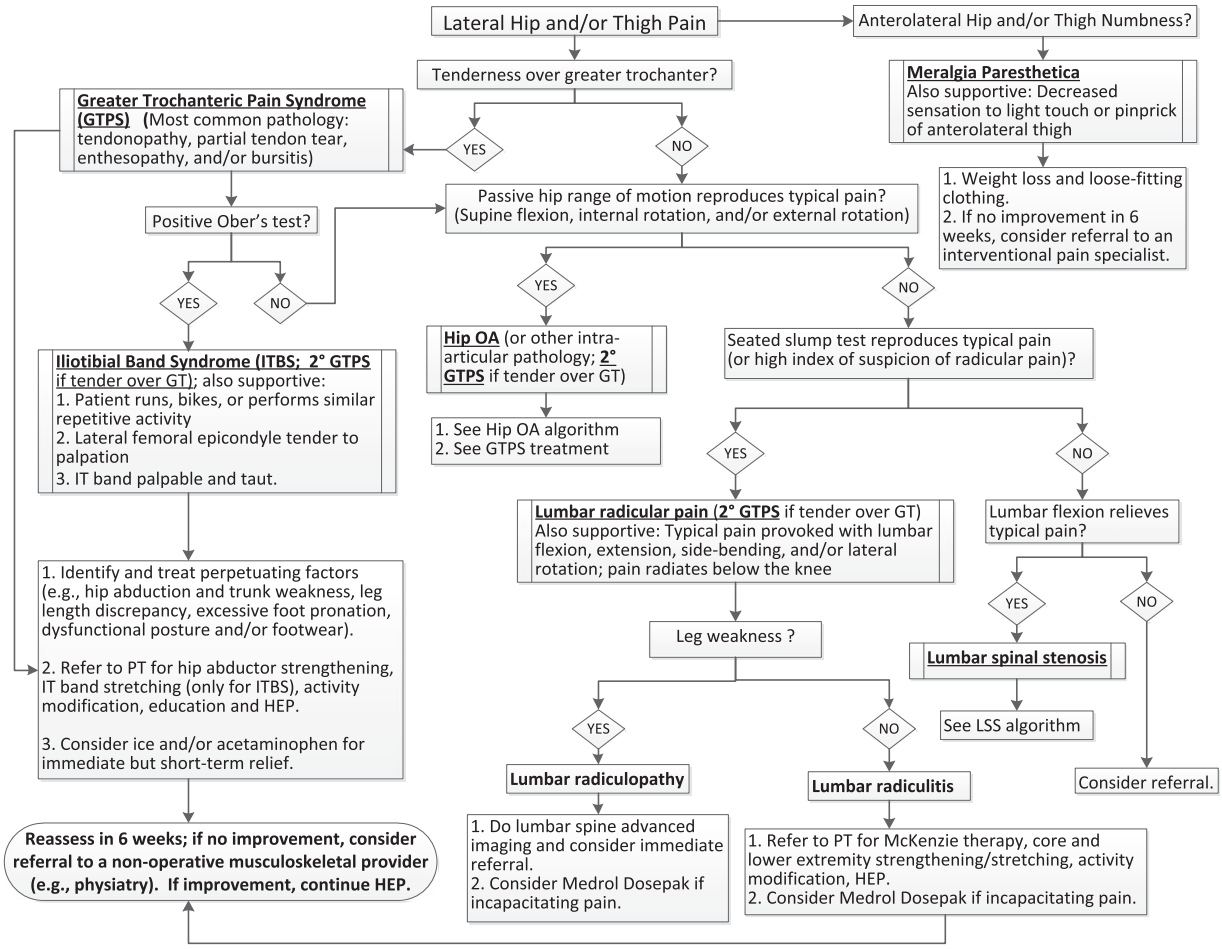

Figure 1 Algorithm for the evaluation and treatment of lateral hip and thigh pain in an older adult.

and she was tender to palpation over the left greater trochanter but not the right. Ober's, seated slump, and straight leg raise tests were negative. (See Figures 2 and 4 for descriptions of Ober's and the seated slump test.)

Imaging

Focused ultrasound performed by a musculoskeletal physiatrist with ultrasound training revealed evidence of bursitis of the left greater trochanter, which correlated with the patient's point of maximal tenderness.

Clinical Course

The patient was diagnosed with greater trochanteric pain syndrome (GTPS) that included an inflammatory component as evidenced by bursitis seen on ultrasound. She was educated on hip abductor strengthening exercises, but she was not interested in performing the exercises regularly. Instead she requested a corticosteroid injection, so an ultrasound-guided greater trochanteric bursa injection was performed by the ultrasound-trained physiatrist.

Approach to Evaluation and Management

Optimal management of patients presenting with lateral hip and/or thigh pain requires a systematic approach for accurate diagnosis of underlying pathology contributing to symptoms. This section describes evidence-based diagnostic and therapeutic approaches for the most common causes of lateral hip and thigh pain in older adults.

Greater Trochanteric Pain Syndrome (GTPS)

GTPS is defined as pain and tenderness to palpation over the greater trochanter (GT) [8]. Until the early 2000s this entity was most frequently called "greater trochanteric bursitis;" however, ultrasound and magnetic resonance imaging (MRI) studies have shown that approximately 20% of people with tenderness over the GT have imaging-proven bursitis. Non-inflammatory pathology is often seen on imaging, such as gluteal tendonosis, gluteal tendon tears, and/or enthesopathy at the GT [9,10]. GTPS is often caused by relative weakness of the hip abductor muscles, especially the gluteus

Table 1 Lateral hip and thigh pain: Theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
GTPS		
GTPS diagnostic criterion	Pain over the greater trochanter is best classified as a syndrome because multiple etiologies can lead to pain at the lateral hip	[8]
GTPS commonly coexists with chronic low back pain	Greater trochanteric tenderness was present in 44.9% of people with chronic low back pain, versus in 6.0% of controls	[12]
Trendelenburg sign—description	Trendelenburg's sign is positive if, during single-leg stance on the affected leg, the contralateral pelvis drops and/or the trunk shifts toward the stance leg	[30]
Trendelenburg sign is common in people with chronic low back pain	Trendelenburg sign was positive in 54% of people with chronic low back pain versus 9.7% of controls	[12]
Corticosteroid injection is not first-line treatment	An ultrasound study of 877 patients and an MRI study of 174 patients demonstrated that only approximately 20% of GTPS cases had true bursitis (i.e., inflammation). Additionally, corticosteroid injections are toxic to local tendon tenocytes and potentially contribute to further weakening of tendons	[9,10], [17–20]
Hip abduction strengthening is first-line treatment	In 229 patients, at 15 months gluteal strengthening (80% success rate) was superior to corticosteroid injection (48% success rate)	[16]
ITBS		
ITBS commonly co-exists with GTPS	Odds ratio of 2.54	[8]
Ober's test—reliability	Inter-rater reliability was 97.6%, and intra-rater reliability was 90%	[24,25]
Hip abduction strengthening is first-line treatment for ITBS	In a prospective trial, 22/24 runners treated with hip abductor strengthening were pain-free at 3 months	[27]
Hip OA		
Pain from hip OA can refer to the lateral hip	Symptomatic hip OA presented as lateral hip pain in 27% of patients	[7]
Lumbar radicular pain		
Lumbar radicular pain can refer to the lateral hip and thigh	In 48 subjects with lumbar disc herniation, 33% experienced pain in the lateral thigh and 46% had a herniation at the L1-2, L2-3, or L3-4 level	[29]
Seated slump test—sensitivity and specificity	For lumbar disc herniations, the seated slump test had a sensitivity of 0.84 and specificity of 0.83, which was overall superior to the straight leg raise test, which had a sensitivity of 0.52 and specificity of 0.89	[31]
McKenzie therapy for radiculitis	A positive pain response to repeated end-range lumbar motion (i.e., McKenzie therapy/mechanical diagnosis and treatment) predicted a positive response to non-operative care	[39]
Oral corticosteroids for radicular pain	In a randomized, double-blind, placebo-controlled trial of 269 patients with a lumbar disc herniation, a short course of oral corticosteroids resulted in modestly improved function but no improvement in pain	[40]
Lumbar spinal stenosis		
Pain from lumbar spinal stenosis can refer to the lateral hip and thigh	In 50 subjects with lumbar spinal stenosis, 42% experienced pain in the lateral thigh	[29]

GTPS = greater trochanteric pain syndrome; ITBS = iliotibial band syndrome; OA = osteoarthritis.

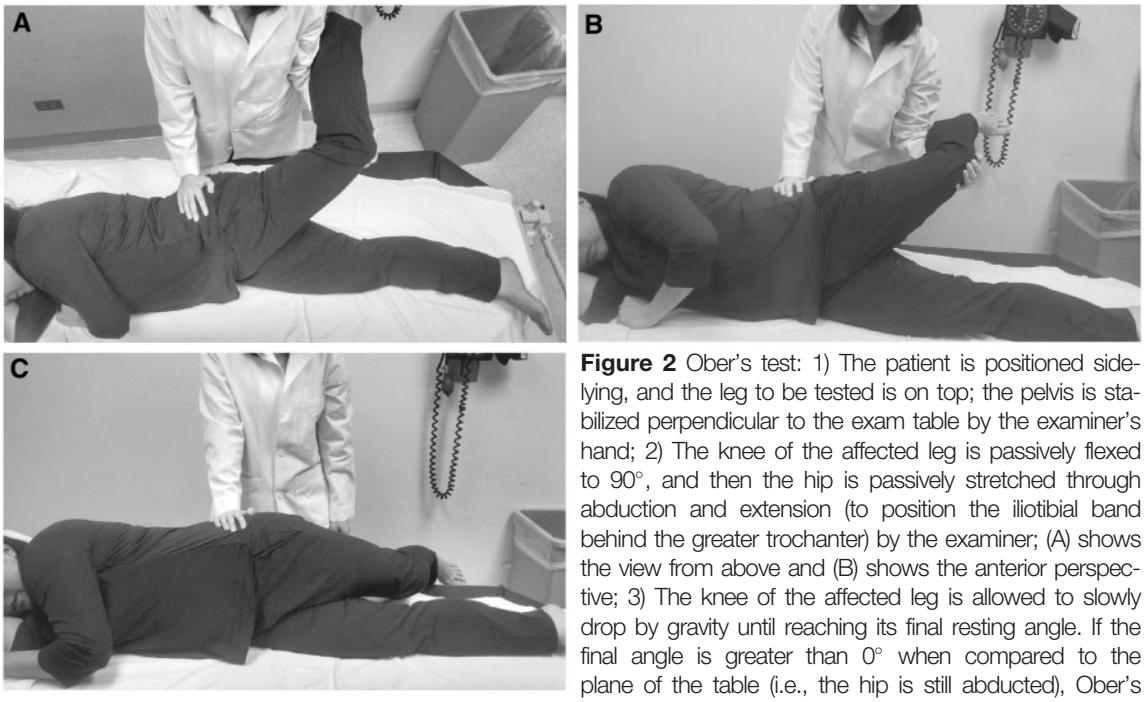

Figure 2 Ober's test: 1) The patient is positioned side-lying, and the leg to be tested is on top; the pelvis is stabilized perpendicular to the exam table by the examiner's hand; 2) The knee of the affected leg is passively flexed to 90°, and then the hip is passively stretched through abduction and extension (to position the iliotibial band behind the greater trochanter) by the examiner; (A) shows the view from above and (B) shows the anterior perspective; 3) The knee of the affected leg is allowed to slowly drop by gravity until reaching its final resting angle. If the final angle is greater than 0° when compared to the plane of the table (i.e., the hip is still abducted), Ober's test is positive; (C) highlights a negative Ober's test.

medius, which leads to excessive gluteal tendon shearing over the GT and eventual tendonopathy [11].

GTPS often coexists with other musculoskeletal lumbopelvic and hip conditions. Additionally, it is often a secondary pain generator resulting from suboptimal biomechanics. In fact, in people with low back pain, GTPS has a prevalence of up to 45% [12–14], and it commonly occurs in people with knee OA, as well [8]. GTPS is more common in women, possibly because they have a wider pelvis structure [14,15]. It may be associated with nighttime pain when lying on the affected side, pain with prolonged standing, and pain or paresthesias radiating down the lateral thigh along the course of the iliotibial band [13,15]. Table 2 reviews additional common pain characteristics which can be elicited during the patient history to help distinguish GTPS from other conditions.

Because GTPS is a clinical syndrome, the diagnosis can be confirmed with physical examination. Table 3 describes an efficient method to perform several exam maneuvers to assess for GTPS and other common lumbopelvic and hip conditions. To localize the GT, palpate the proximal lateral thigh of the limb being tested while the patient is standing on both feet and pivoting the leg being tested into hip internal and external rotation. The bony protuberance that is most prominent to the examiner's touch is the GT. Focal tenderness over this bony protuberance is the definition of GTPS. Another common finding in GTPS is a positive Trendelenburg sign because hip abductor weakness is often the leading biomechanical cause of GTPS. If the patient is able, a positive Trendelenburg can be determined by asking the patient to stand on one leg. If the pelvis contralateral to the stance leg drops, there is weakness of the stance leg hip abductor muscles. This is a positive Trendelenburg sign. If the patient intentionally shifts his/her trunk over the stance leg (thereby elevating the contralateral pelvis) in order to perform the task, this is considered a positive compensated Trendelenburg sign. This finding also corroborates hip abductor weakness of the stance leg. The presence of a Trendelenburg or compensated Trendelenburg sign can help a provider identify biomechanical issues to help direct treatment. However, a Trendelenburg's sign is positive in 54% of all people with CLBP [12] and it is not a finding specific to the diagnosis of GTPS, so it was not included in the diagnostic algorithm.

In the past, GTPS was commonly treated with corticosteroid injection; however, a recent study suggests that the most effective long-term treatment is hip abductor strengthening [16] in addition to management of any contributing underlying musculoskeletal conditions, such as gluteal tendinopathy. As discussed earlier, only 20% of GTPS cases have inflammatory pathology [9,10], thus corticosteroids would be expected to provide relief to this small subset of patients. Even in these patients the effect will likely only be temporary if the underlying biomechanical etiology is not also addressed. Furthermore, corticosteroids should be used with caution in older adults because of their well-known systemic side effects such as hypertension, hyperglycemia, increased appetite, edema, immune suppression, behavior and sleep alterations, and, with frequent repeated use, hormonal

Table 2 Key questions to ask a patient when evaluating lateral hip/thigh pain, organized by supported diagnosis

Questions to evaluate lateral hip/thigh pain
GTPS:
Pain when side-lying on the affected hip?
ITBS:
History of frequent running, biking, or other repetitive lower extremity activity?
Hip OA:
Pain in the groin?
Lumbar radicular pain:
Pain radiates below the knee?
Lower extremity numbness, tingling, and/or weakness?
Lumbar spinal stenosis:
Pain with walking, relieved with lumbar flexion (e.g., by pushing a shopping cart)?
Meralgia paresthetica:
Numbness +/− pain?

GTPS = greater trochanteric pain syndrome; ITBS = iliotibial band syndrome; OA = osteoarthritis.

Table 3 Physical examination maneuvers to evaluate lateral hip/thigh pain, listed in a sequence for maximum efficiency during a patient encounter

Efficient sequence of physical examination maneuvers to evaluate lateral hip/thigh pain
Standing:
1. Palpation of greater trochanter
2. IT band tightness appreciated on palpation (optional)
3. Pain with palpation of lateral femoral epicondyle (optional)
4. Active lumbar flexion
Seated:
5. Seated slump test
Supine:
6. Passive hip range of motion in supine (flexion, internal rotation)
Side-Lying:
7. Ober's test

IT = iliotibial.

and bone density effects. Evidence has demonstrated that corticosteroid injections are also toxic to local tenocytes and can potentially contribute to progressive tendonopathy and partial tears [17–20].

A hip-strengthening program is considered to be first-line treatment for patients with GTPS since it directly addresses the biomechanical cause of pain. If a patient is resistant to engaging in a strengthening program and/or is severely limited by pain from GTPS, if possible, we prefer referral to a musculoskeletal specialist prior to considering treatment with a corticosteroid injection for two reasons. First, using ultrasound or MRI to identify whether bursitis (i.e., active inflammation) is actually present helps the clinician determine how likely the patient is to benefit from an anti-inflammatory medication such as a corticosteroid. (When available and in the hands of an experienced ultrasonographer, ultrasound is superior to MRI because it is faster, cheaper, and provides real-time imaging which can also be used for visualization during injection.) Second, cadaveric studies have demonstrated that there are seven bursae surrounding the GT. Therefore, in order to maximize a clinical response to a corticosteroid injection, it should be performed under ultrasound guidance by an experienced provider into the appropriate bursa demonstrating inflammation. An experienced specialist can use ultrasound to evaluate for bursitis and accurately administer any indicated injection during a regular office visit, without the need for delay to obtain an MRI to confirm inflammation. Also, there is often concomitant gluteal tendinopathy in the form of tendon tears or tendinosis. It is possible that decreasing the inflammation of the bursa will result in incomplete pain relief. Of course, patients receiving a corticosteroid injection should still be encouraged to pursue physical therapy for hip abductor (especially gluteus medius) strengthening in order to minimize the chance for pain recurrence.

Iliotibial Band Syndrome (ITBS)

ITBS usually presents as distal lateral thigh pain, caused by friction from the iliotibial band (ITB), which rubs repeatedly over the lateral femoral epicondyle. The condition most commonly occurs in people who perform repetitive unidirectional activities such as running and cycling, have recently increased their training intensity, and who have relative hip abductor weakness [21,22]. The gluteus medius muscle is the primary stabilizer of the ITB during foot-strike, therefore weakness of the muscle can result in compensation and overuse of the tensor fascia latae. This, in turn, can lead to a shortened ITB and ITBS. Since similar suboptimal biomechanics predispose to both ITBS and GTPS, the two conditions can co-exist [8]. As a result of the location of the origin and insertion of the ITB, ITBS can also mimic lumbar radicular pain by presenting as lateral thigh pain radiating to the knee. Despite the increasing age of runners [23], it is important to note that ITBS more commonly occurs in younger runners and the syndrome is not commonly described in older adults. Older runners tend to be more limited by calf, Achilles, and hamstring injuries rather than knee injuries [21].

In the context of pain and tenderness over the lateral femoral epicondyle, a positive Ober's test is supportive of ITBS. The maneuver has a high inter-rater reliability of 97% and intra-rater reliability of 90% [24,25]. To perform the test, as demonstrated in Figure 2, the patient is positioned in side-lying with the affected leg on top, and the pelvis is stabilized perpendicular to the exam table by the examiner's hand. Next, the hip and knee of the affected leg are passively flexed to 90°, and then the examiner passively stretches the hip through abduction and extension in order to position the ITB behind the GT. Finally, the knee of the affected leg is allowed to slowly drop under the weight of gravity until it reaches its final resting angle. If the final angle is greater than 0°, meaning the hip is still abducted, Ober's test is positive and indicates the patient has a tight ITB [26]. Oftentimes in patients with ITBS, the examiner will also be able to palpate a taut ITB while the patient is standing or supine. Because the diagnosis of this syndrome is clinical, imaging is not necessary unless coexisting lumbar radicular and/or intra-articular hip pathology is suspected.

Similar to the treatment for GTPS, hip abductor strengthening is essential for long-term resolution of ITBS [27]. ITB stretching, in addition to correction of other contributing factors such as core weakness, suboptimal biomechanics, and improper footwear can be helpful, as well. Relative rest, ice, and acetaminophen are appropriate for acute management, but similar to the limitation of corticosteroid injections for GTPS, these treatments are unlikely to provide lasting relief unless the underlying biomechanical imbalance is also addressed.

Hip OA

Symptomatic hip OA is classically thought to present as anterior groin pain, but intra-articular hip pathology can also cause pain in the lateral hip, anterior thigh, knee, low back, buttock, and lower leg [7]. The diagnosis and management of hip OA is discussed in depth in another article in this series [28], but it is still important to consider this diagnosis when older adults present with lateral hip and/or thigh pain. In a study of 369 patients (443 hips) with symptomatic hip OA proven by pain relief after total hip arthroplasty, 27% of these patients presented with lateral hip pain [7]. Pain with passive hip range of motion while the patient is supine should prompt the clinician to consider the possibility of hip OA or other intra-articular hip pathology contributing to the patient's pain. If the patient is also tender to palpation over the GT, secondary GTPS is likely present and should be managed as detailed above.

Lumbar Radicular Pain

Lateral hip and thigh pain may be referred from irritation of the lumbar nerve roots [29]. Most commonly the L2 through L5 nerve roots refer pain to this distribution, but the L4 and L5 nerve roots are more likely to also refer pain *below* the knee (see Figure 3A). Based on the path of nerve roots, disc herniations or bulges of any of the L1-2 through L5-S1 intervertebral discs can potentially refer pain to this area. When a patient is describing the location of his/her pain, radiation below the knee is also suggestive, but not definitive for, lumbar radicular pain.

Physical examination supportive of lumbar radicular pain involves provocation of pain with isolated movements of the lumbar spine elements, especially the lumbar nerve roots. As an initial screen for lumbar radicular pain, the examiner should ask the patient to perform active lumbar flexion, extension, side-bending, and lateral rotation from a standing position to evaluate whether any of these movements reproduce and/or exacerbate the patient's lateral hip/thigh pain. For a more specific test, the seated slump test is designed to put selective tension

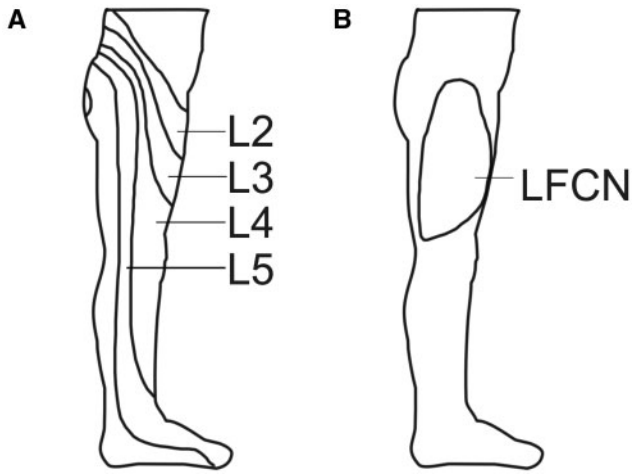

Figure 3 Sensory innervation of the lateral hip and thigh: Dermatomes L2-L5 (A) and the peripheral lateral femoral cutaneous nerve (LFCN) (B) provide the primary cutaneous innervation to the lateral hip and thigh.

on the lumbar nerve roots. To perform this maneuver, while seated the patient is instructed to slump into cervical, thoracic, and lumbar flexion. The examiner then extends the knee and dorsiflexes the ankle of the affected leg to the patient's end range of motion (see Figure 4). Of note, older adults often have stiff joints and muscle contractures which limit their passive range of motion, but the test can still be performed effectively. The test is positive if the patient's typical pain is reproduced with this maneuver and is subsequently relieved with neck extension [30]. Neck extension should result in less tension on the nerve roots but does not affect lumbar vertebral position or lower extremity muscle length, so if the patient's pain does not abate with neck extension, the pain may be due to myofascial low back pain or lower extremity muscle tightness rather than nerve irritation.

For lumbar disc herniations, the seated slump test has a sensitivity of 0.84 and specificity of 0.83, which is overall superior to the straight leg raise test (sensitivity of 0.52 and specificity of 0.89) [31]. The predictive value of the seated slump test for lumbar radicular pain is not ideal. In patients with a negative seated slump test but high clinical suspicion for radicular pain, conservative management can be pursued despite the negative test. Additionally, the patient can be referred to a non-operative musculoskeletal specialist for further evaluation. Of note, thorough discussion of the femoral nerve stretch test is beyond the scope of this article, but this test has also been shown to have a high likelihood ratio for mid-lumbar nerve root impingement [32–34].

Management of lumbar radicular pain depends on whether neurologic deficit is present. The definition of lumbar radiculopathy is radicular pain in the setting of focal weakness and/or asymmetrically diminished deep tendon reflexes. Objective sensory impairment may be evident on examination, but it is not a criterion for defining radiculopathy. Electrodiagnostic studies, although not necessary to make the diagnosis, are abnormal in lumbar radiculopathy. Deep tendon reflexes are commonly diminished bilaterally in older adults; therefore, in our algorithm we only recommend pursuing management of radiculopathy if the patient has objective evidence of asymmetric weakness on exam.

Patients with radiculopathy are at risk for progressive and permanent neurologic damage. Therefore, if a clinician is concerned for lumbar radiculopathy based on history and physical exam, the patient would benefit from urgent imaging and referral. Advanced imaging such as lumbar spine MRI (or CT if MRI is contraindicated) is useful to plan interventions such as epidural corticosteroid injections and/or surgical decompression. When referring the patient for specialist care, we recommend initial evaluation by a non-operative musculoskeletal provider if available, because not all radiculopathy requires surgical management. Seventy percent of patients with lumbar radiculopathy will improve with conservative care within 4 weeks of the onset of symptoms [35], and up to 90% of patients with lumbar radiculopathy from a herniated disc (in the absence of significant lumbar spinal stenosis) treated with core stabilization exercises and education eventually achieve good or excellent outcomes [36,37]. Especially in the aging population, avoiding unnecessary surgical procedures can help minimize associated morbidity and peri-operative complications. Nevertheless, this population will require close follow-up for patients who do not improve and in those with functionally significant weakness that may benefit from surgical decompression within the first 8 weeks of symptom onset [35].

Contrary to lumbar radiculopathy, lumbar *radiculitis* describes nerve root irritation *without* objective findings of

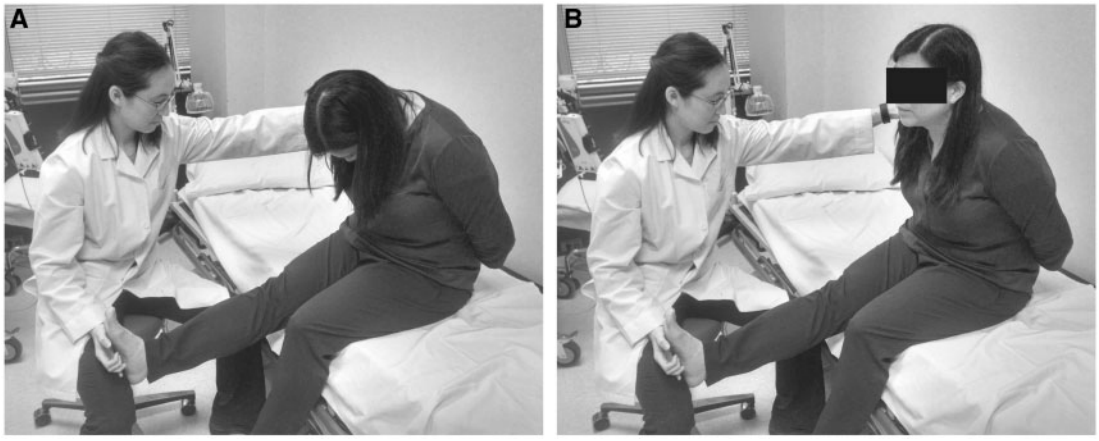

Figure 4 Seated slump test: 1) While seated, the patient is instructed to place her hands behind her back and then slump into cervical, thoracic, and lumbar flexion; 2) The examiner then extends the knee and dorsiflexes the ankle of the affected leg (A); 3) The test is positive if the patient's typical pain is reproduced with this maneuver and is subsequently relieved with neck extension (B).

nerve root damage (i.e., weakness or reflexes changes). Patients may complain of altered sensation (i.e., pain or pins and needles), but electrodiagnostic testing is normal and therefore not a recommended part of the work-up for lumbar radiculitis. For patients with lumbar radiculitis, we recommend a longer trial of conservative management prior to considering referral because, unlike in radiculopathy, these patients are not at immediate risk of progressive, permanent neurologic damage. For first-line treatment of radiculitis, we recommend McKenzie physical therapy, which is a standardized approach to the assessment and management of radicular pain. Also called Mechanical Diagnosis and Therapy (MDT), in this method McKenzie-certified therapists help patients identify a "directional preference" of lumbar spine motion (i.e., extension, flexion, and/or side-bending) which helps reduce radicular pain, centralize the pain to the back, and minimize pain altogether. McKenzie therapy teaches simple exercises which the patient should perform several times throughout the day, and it does not emphasize passive modalities such as manual massage or manipulation [38]. Patients who respond well to McKenzie therapy are likely to be able achieve adequate pain relief without operative intervention [39].

For radicular pain, whether from radiculitis or radiculopathy, we only recommend a short course of oral corticosteroids (such as a 6-day methylprednisolone taper) if a patient has incapacitating pain interfering with functioning and/or engaging in physical therapy. For lumbar disc herniation, oral corticosteroids have been shown to offer only modest improvement in function and have not been proven to reduce pain [40]. In older adults, the systemic side effects are often limiting and should not be prescribed without carefully considering the risks and benefits and involving the patient and/or caregiver in the decision-making process.

Another treatment option for patients with incapacitating pain limiting function is fluoroscopically-guided epidural corticosteroid injections. Full discussion of the indications and expected efficacy of these injections is beyond the scope of this paper, but it is important to appreciate that these injections must be utilized thoughtfully because in an improperly selected older patient, there is a risk for significant side effects from the procedure itself and from epidural corticosteroid administration, potentially without a high likelihood for clinical improvement. Based on a modified Delphi method, our expert panel believes that best practice is for primary care clinicians who are considering the use of an injection to first refer the patient to a non-operative musculoskeletal specialist (such as a physiatrist, anesthesia pain physician, or sports medicine physician), prior to ordering the injection, in order to evaluate for proper patient selection.

Lumbar Spinal Stenosis (LSS)

LSS is another common condition in older adults and can cause positional-dependent nerve root irritation due to degenerative changes resulting in narrowing of the spinal canal. The most common presenting symptom of LSS is progressive pain down the leg with continued walking that improves with sitting. In a study by Kalichman et al., up to 20% of 60–69-year-olds had CT-evidence of LSS, and while not everyone with the finding was symptomatic, people with imaging-diagnosed LSS were over three times more likely to have LBP [41]. When symptomatic, 42% of patients with LSS present with lateral thigh pain [29]. The diagnosis of LSS is important to consider when older adults present with lateral hip or thigh pain that improves by functionally increasing the spinal canal area with lumbar flexion [42]. Management of LSS is discussed in Part VI of this series [43].

Meralgia Paresthetica (MP)

In meralgia paresthetica, the lateral femoral cutaneous nerve (LFCN) is compressed, which results in sensory abnormalities along the lateral and anterolateral thigh. Since there is no motor component to the nerve, MP does not cause weakness. MP has been associated with metabolic factors such as diabetes mellitus and alcoholism, in addition to mechanical factors such as obesity, tight-fitting clothing around the waist (e.g., uniforms, jeans, belts, etc.), and lumbopelvic or lower extremity surgery as a result of direct trauma or positioning during surgery [44].

Clinically, MP is less common and tends to present much differently than the other conditions discussed thus far. Patients with MP often complain of numbness, tingling, and/or itching, more so than pain, and this is reflected in the algorithm in Figure 1. Additionally, whereas patients with lumbar radicular pain usually have difficulty describing the exact region of sensory symptoms, patients with MP can often precisely trace the affected cutaneous distribution of the LFCN (see Figure 3B). On physical exam, MP may cause dysesthesias over the entire anterolateral thigh, but it does not cause frank tenderness over the GT like GTPS does. It is important to note that as the prevalence of obesity rises, MP can co-exist with the other conditions that cause lateral hip and thigh pain.

To confirm the diagnosis of MP, patients can be referred for a nerve conduction study, but this test is technically difficult, particularly in obese individuals. Ultrasound, when performed by an experienced ultrasonographer [45], and magnetic resonance neurography (MRN) can also be useful diagnostically by detecting morphologic changes to the LFCN in people with MP [44].

Conservative management of MP involves losing weight, loosening clothing around the waist, and avoiding positions such as excessive hip extension, which can compress the LFCN under the inguinal ligament or elsewhere along its course. Additionally, KinesioTaping can be considered [44]. If conservative measures do not

result in symptom relief, patients can be referred to an interventional pain specialist for possible LFCN diagnostic nerve block, neurolysis, and/or neurectomy [46].

Resolution of Case

The patient experienced significant pain relief from the corticosteroid injection for a few weeks. However, 5 weeks after the injection her lateral hip pain returned, now with pain radiating into her left lateral thigh, knee, and lateral lower leg. On repeat exam, she now had tenderness over her left ITB, mild pain with passive hip range of motion and lumbar extension, and a positive seated slump test on the left. At this time, she was felt to have GTPS and ITBS with superimposed lumbar radicular pain. Ultrasound confirmed the recurrence of bursitis in the GT. She continued to be resistant to physical therapy, and 1 month later her pain had not improved so she requested a repeat ultrasound-guided greater trochanteric bursa corticosteroid injection.

The clinical course of this case highlights that corticosteroid injections for GTPS, even when an inflammatory bursitis is present, usually only provide temporary relief, and more definitive management of hip abductor strengthening must be strongly encouraged. In the setting of true bursitis and severe pain, a corticosteroid injection can help facilitate active engagement in a rehabilitation program. However, in older adults who are at risk for co-morbidities such as osteoporosis, hypertension, diabetes, and delirium, minimizing the use of corticosteroids is advantageous. This case also demonstrates that evaluation of lateral hip and thigh pain is often not straightforward. In this particular patient, she likely developed secondary lumbar pain, possibly in part due to compensatory mechanical changes in an attempt to relieve pain from her sub-optimally treated GTPS.

Summary

The evaluation of lateral hip and thigh pain in an older adult can be challenging. Potential etiologies include local and referred causes, and clinicians must consider a myriad of lumbopelvic and hip conditions such as GTPS, ITBS, hip OA, lumbar radicular pain, and LSS. Identifying a patient's true pain generator(s) enables initiation of proper treatment and avoidance of unnecessary treatments and their potential adverse effects. Therefore, if a patient is not responding adequately to the conservative measures described in the algorithm, we recommend considering referral to a non-operative musculoskeletal specialist, who has expertise in evaluating the entire lumbopelvic region. We do not recommend prescribing opioids before the patient has completed a trial of conservative management and has been evaluated by a specialist as these medications often place the patient at risk of adverse drug reactions. These side effects may be avoided if the underlying etiology of the pain is addressed.

Perhaps the most common local cause of lateral hip and thigh pain in older adults is GTPS. Gluteus medius weakness is often a significant contributor to the development of GTPS. As a result, hip abductor strengthening exercises should be the first-line treatment of this syndrome in conjunction with medication. While medications and modalities may provide temporary symptomatic relief, pain will likely recur if the underlying weakness is not addressed. Additionally, since GTPS and other lumbopelvic conditions often coexist, it is important to perform a thorough history and physical examination to evaluate for the possibility of multiple pain generators, as well.

Key Points

1. Lateral hip and/or thigh pain in the older adult with CLBP can be from a number of causes, with those most common being greater trochanteric pain syndrome (GTPS), iliotibial band syndrome (ITBS), hip osteoarthritis, lumbar radiculopathy/radiculitis, and lumbar spinal stenosis. Meralgia paresthetica also should be considered in the differential diagnosis. Often lateral hip and/or thigh pain is multifactorial.
2. Greater trochanteric pain syndrome (GTPS) is a common cause of lateral hip pain and is usually due to weakness of the gluteus medius muscle. Hip abductor strengthening is the first-line treatment for GTPS.
3. True GT bursitis causing GTPS occurs in a minority of cases; therefore, corticosteroid injections should be used sparingly, and ideally only when there is imaging evidence of bursitis. Injection should always be accompanied by a hip abductor strengthening program.
4. In older adults with CLBP, iliotibial band syndrome (ITBS) is less commonly described than GTPS. However, hip abductor strengthening is part of the first-line treatment for both GTPS and ITBS.
5. Lumbar radiculopathy (i.e., pain + weakness) requires aggressive evaluation and treatment to avoid progressive and permanent neurologic damage. Lumbar radiculitis (pain without weakness) can be evaluated and managed more conservatively.

Acknowledgments

The authors wish to thank Dave Newman for his valuable assistance in preparing the manuscript.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment: Part IX: Anxiety

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interest: The contents of this report do not represent the views of the Department of Veterans Affairs or the United States government. There are no conflicts of interest to report.

Drs. Jordan F. Karp and Elizabeth A. DiNapoli are co-first authors on this article.

Abstract

Objective. As a part of a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults, this article focuses on anxiety—a significant contributor of reduced health-related quality of life, increased use of medical services, and heightened disability in older adults with CLBP.

Methods. A modified Delphi technique was used to develop an algorithm for the screening and clinical care of older adults with CLBP and anxiety. A 4-member content expert panel and a nine-member primary care panel were involved in this iterative development process. Evidence underlying the recommendations is not strictly based on VA populations; therefore, the algorithm can be applied in both VHA and civilian settings. The illustrative clinical case was taken from one of the contributor's clinical practice.

Results. We present a treatment algorithm and supporting tables to be used by providers treating older adults who have anxiety and CLBP. A case of an older adult with anxiety and CLBP is provided to illustrate the approach to management.

Conclusions. To promote early engagement in evidence-based treatments, providers should routinely evaluate anxiety in older adults with CLBP using a screening and treatment algorithm.

Key Words. Anxiety; Low Back Pain; Chronic Pain; Avoidance Behavior; Cognitive Behavior Therapy; Fear

Introduction

Patients with chronic low-back pain (CLBP) commonly exhibit increased levels of emotional distress [1]. For example, anxious mood and other symptoms of anxiety are commonly seen in patients with CLBP [2]. Prevalence of anxiety disorders in CLBP patients (19–31%) has been found to be greater than that of the general population (10–25%) [3–5]. Polatin and colleagues (1993) also found that approximately 95% of adults with a lifetime history of anxiety disorders experienced these symptoms prior to the onset of low back pain, with only 5% reporting the development of anxiety after the onset of low-back pain [3]. Additionally, symptoms of psychological distress (e.g., anxiety and somatization) have been found to predict subsequent onset of new episodes of low back pain [6,7]. Therefore, anxiety disorders often occur prior to and may predict the onset of CLBP. This is important from a public health perspective because co-occurring anxiety and CLBP are associated with reduced health-related quality of life, greater functional disability, increased use of medical services, and exerts a greater negative impact on patient health than either condition alone [8–11].

According to the *Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition* [12], anxiety disorders include syndromes that share features of excessive, persistent fear, anxiety and related behavioral disturbances that cause clinically significant distress or impairment in important areas of functioning. Depending on the type of object or situation that induces the fear or anxious response and the content of the associated thoughts or beliefs, anxiety disorders may be classified into: generalized anxiety disorder (GAD), separation anxiety, phobic avoidance, social anxiety, and panic disorder with or without agoraphobia. Anxious mood, tension, and general somatic symptoms of the sensory type have been found to be more common than other types of anxiety symptoms in CLBP patients [2].

Anxiety and CLBP may commonly coexist due to shared neurobiology, such as common areas of brain activation [13,14] and similar dysregulation of neurotransmitters in the central nervous system (e.g., GABA and glutamate) [15,16]. Similar psychological mechanisms may also explain the relationship between pain and anxiety, such that catastrophizing and hypervigilance may lead to amplification of physical and psychological symptoms. Indeed, high anxiety levels may heighten perception of pain and alter pain behavior in acute and chronic pain patients [17,18]. Patients with co-existing anxiety and pain experience more severe pain and greater pain interference with activities compared to patients with pain only [8]. Kinesiophobia and pain catastrophizing (two specific forms of anxious behaviors) predict more severe pain and disability in patients with CLBP [19].

Age can substantially influence the presentation of anxiety, including its symptomatology, prevalence, and treatment response. Late-life anxiety is frequently undiagnosed and thus untreated, particularly in patients suffering from chronic pain. This may be due to the way older adults express their anxiety which is often as general concerns about physical health, somatic symptoms, or worsening disability, and not describing physical symptoms of anxiety [20]. For example, older adults tend to worry less frequently than younger adults and have more medical and somatic complaints as opposed to psychological distress [21,22]. Additionally, fear of falling is a common and uniquely geriatric anxiety syndrome marked by fear and avoidance of movement and physical activities [21]. Despite one in three community-dwelling older adults experiencing CLBP [23] and another 7% experiencing an anxiety syndrome [24], clinicians lack well-established guidelines for evaluation and treatment of anxiety in older adults with CLBP.

The purpose of this paper is to present an evaluation and treatment algorithm for clinicians treating older adults with co-occurring anxiety and CLBP. We also will present a case example to highlight the clinical complexities of evaluating and treating anxiety in older adults with CLBP. Since generalized anxiety disorder (GAD) is the most common anxiety syndrome in older adults [25], it is the focus of the case example.

Methods

As per the detailed description provided in the series overview [26], a modified Delphi technique involving a content expert panel and primary care review panel was used to create the CLBP and anxiety algorithm (Figure 1), the table providing the rationale for the various components of the algorithm (Table 1), and medication management recommendations (Table 2). The PI (DW) drafted an evidence-based treatment algorithm and supportive tables based upon a comprehensive review of the literature and general clinical utility when a strong evidence-based approach was not yet available. The algorithm and accompanying tables then were refined by an expert panel. Expertise represented among the 4 Delphi expert panel members included geriatric medicine, geriatric psychiatry, and geriatric psychology.

Case Presentation

Relevant Pain and Functioning History: The patient is a 76-year-old widowed black female living alone in a senior high rise who presented to her primary care physician complaining of severe low back pain. She described the pain as a constant ache (6/10 severity) that can become a sharp and stabbing pain (8/10 severity) while walking, bending, twisting, or lifting objects. Her back feels especially stiff in the morning. Her first episode of low back pain began without obvious cause 10 years ago. She has had low back pain episodically since then, however during the past 2 years the low back pain has worsened and is present more days than not. She is unable to walk more than two blocks at a

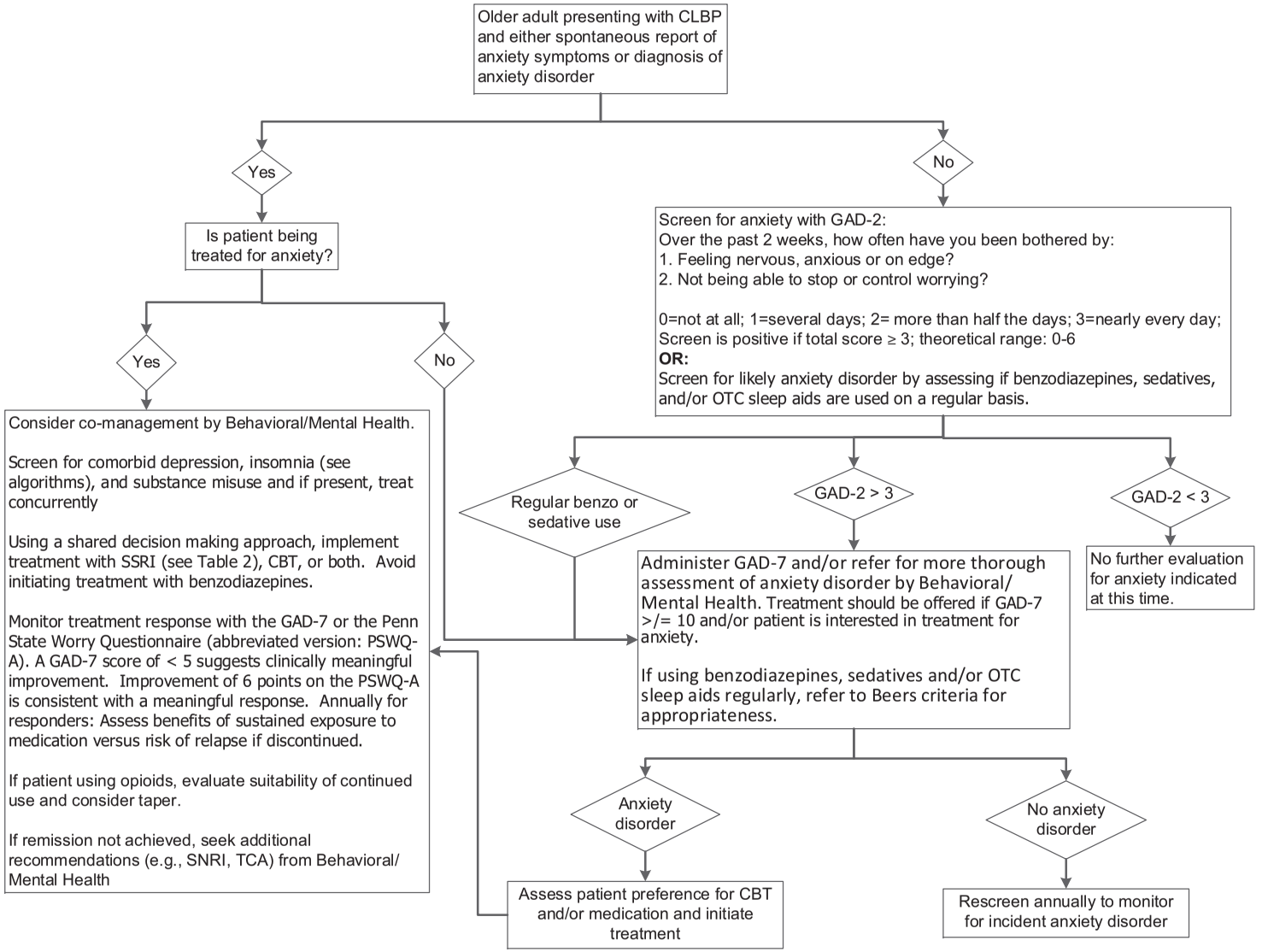

Figure 1 Algorithm for the evaluation and treatment of anxiety in an older adult with CLBP.

Table 1 Anxiety: Theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
Dual screening: for anxiety and for benzodiazepine or sedative use.	Anxiety is more common than is depression in older people. Similar to depression, it is associated with adverse effects on health and cognition. Many older adults with clinically significant anxiety have not been diagnosed in the past and may not spontaneously report anxiety. Given the high comorbidity of low back pain and anxiety and the potential benefits of treatment, we recommend screening for anxiety disorders with the GAD-2 in all older adults with CLBP. Brief screeners (2 items) have been found to be equally sensitive and specific at detecting anxiety in CLBP patients as widely used longer-form "gold standards."	Wolitzky-Taylor KB, Castriotta N, Lenze EJ, Stanley MA, Craske MG. Anxiety disorders in older adults: a comprehensive review. Depress Anxiety 2010;27(2):190–211. Airaksinen O, Brox JI, Cedraschi C et al. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. Eur Spine J 2006;15(2):192–200. Reme SE, Lie SA, Eriksen HR. Are 2 questions enough to screen for depression and anxiety in patients with chronic low back pain? Spine 2014;39(7):455–462.
	Long-term benzodiazepine and sedative use is common yet unsafe for older adults. Inappropriate prescribing of these agents for depression or analgesia should be avoided.	Campanelli CM. American Geriatrics Society updated Beers Criteria for potentially inappropriate medication use in older adults: The American Geriatrics Society 2012 Beers Criteria Update Expert Panel. J Am Geriatr Soc 2012;60(4):616–631.
GAD-2 for anxiety screening, using 3 as cutpoint.	GAD-2 is a good screening instrument with high sensitivity and specifity. Lower cutpoints may be used for older adults because they may report lower scores than younger patients.	Mohlman J, Bryant C, Lenze EJ, et al. Improving recognition of late life anxiety disorders in Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition: Observations and recommendations of the Advisory Committee to the Lifespan Disorders Work Group. Int J Geriatr Psychiatry 2012;27(6):549–556. Wild B, Eckl A, Herzog W, et al. Assessing Generalized Anxiety Disorder in Elderly People Using the GAD-7 and GAD-2 Scales: Results of a Validation Study. Am J Geriatr Psychiatry 2013;22(10):1029–1038.
If a benzodiazepine or sedative is prescribed, refer to Beers criteria for appropriate use. The Beers criteria suggest that in older adults, benzodiazepines may be appropriate for seizure disorders, rapid eye movement sleep disorders, benzodiazepine	Sedative hypnotics are frequently prescribed for older adult with anxiety in primary care settings. Older adults have increased sensitivity to benzodiazepines and decreased metabolism of long-acting agents.	Campanelli CM. American Geriatrics Society 2012 Beers Criteria Update Panel. American Geriatrics Society updated Beers Criteria for potentially inappropriate medication use in older adults. J Am Geriatr Soc 2012;60(4):616–631.
	Even low doses of benzodiazepines (such as frequently prescribed in primary care) in older adults increases the risk of falls, fractures, cognitive impairment, and delirium.	Simon GE, Ludman EJ. Outcome of new benzodiazepine prescriptions to older adults in primary care. Gen Hosp Psychiatry 2006;28(5):374–378.

(continued)

Table 1 Continued

Algorithm component	Comments	References
withdrawal, ethanol withdrawal, severe (disabling) generalized anxiety disorder, peri-procedural anesthesia, and end-of-life care. In general, all benzodiazepines increase risk of cognitive impairment, delirium, falls, fractures, and motor vehicle accidents in older adults.		
Especially in cases of dependence, consider working with a specialist to reduce benzodiazepine use.	Discontinuing benzodiazepines that have been prescribed long-term is frequently difficult. Issues with which to contend include minimization of physiological withdrawal, addressing psychological dependence, and monitoring for the return of the underlying anxiety disorder.	Parr JM, Kavanagh DJ, Cahill L, Mitchell G, McD Young R. Effectiveness of current treatment approaches for benzodiazepine discontinuation: a meta-analysis. Addiction 2009;104(1):13–24. Baillargeon L, Landreville P, Verreault R, et al. Discontinuation of benzodiazepines among older insomniac adults treated with cognitive-behavioural therapy combined with gradual tapering; a randomized trial. Can Med Assoc J 2003;169:1015–1020.
If GAD-2 is >/= 3, conduct a more thorough assessment using GAD-7.	Using the threshold score of 10, the GAD-7 has a sensitivity of 89% and a specificity of 82% for generalized anxiety disorder. It is moderately good at screening three other common anxiety disorders – panic disorder (sensitivity 74%, specificity 81%), social anxiety disorder (sensitivity 72%, specificity 80%), and post-traumatic stress disorder (sensitivity 66%, specificity 81%).	Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder. Arch Intern Med 2006;166:1092–1097. Kroenke K, Spitzer RL, Williams JBW, Monahan PO, Löwe B. Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. Ann Intern Med 2007;146(5):317–325.
If more thorough assessment indicates the presence of significant anxiety symptoms or disorder, initiate treatment. Strongly consider treatment if GAD-7 $\geq$10.	Untreated anxiety can lead to decrements in health and cognitive function. SSRIs and, to a lesser extent, CBT are efficacious for treating anxiety in older adults. Both of these interventions are safer than benzodiazepines. While SSRIs are superior to placebo, the benefit may not be as durable as the skills acquired with CBT. For example, CBT may boost response among older adults with GAD who are partial responders to an SSRI. In addition, those who receive CBT may be able to discontinue their SSRI and maintain adequate symptom control.	Gonçalves DC, Byrne GJ. Interventions for generalized anxiety disorder in older adults: systematic review and meta-analysis. J Anxiety Disord 2012; 26(1):1–11. Gould RL, Coulson MC, Howard RJ. Efficacy of cognitive behavioral therapy for anxiety disorders in older people: a meta-analysis and meta-regression of randomized controlled trials. J Am Geriatr Soc 2012;60(2):218–229. Brenes GA, Danhauer SC, Lyles MF, Hogan PE, Miller ME. Telephone-delivered cognitive-behavioral therapy and telephone-

(continued)

Table 1 Continued

Algorithm component	Comments	References
		delivered nondirective supportive therapy for rural older adults with generalized anxiety disorder: A randomized clinical trial. JAMA Psychiatry 2015;72(10):1012–1020. Lenze EJ, Rollman BL, Shear MK, et al. Escitalopram for older adults with generalized anxiety disorder: a randomized controlled trial. JAMA 2009; 301(3):295–303. Schuurmans J, Comijs H, Emmelkamp PM, et al. A randomized controlled trial of the effectiveness of cognitive-behavioral therapy and sertraline versus a waitlist control group for anxiety disorders in older adults. Am J Geriatr Psychiatry 2006;14(3):255–263. Lenze EJ, Mulsant BH, Shear MK, et al. Efficacy and tolerability of citalopram in the treatment of late-life anxiety disorders: results from an 8-week randomized, placebo-controlled trial. Am J Psychiatry 2004;162(1):146–150. Wetherell JL, Petkus AJ, White KS, et al. Antidepressant medication augmented with cognitive-behavioral therapy for generalized anxiety disorder in older adults. Am J Psychiatry 2013;170(7):782–789.
SSRIs are first line pharmacotherapy. If no response to SSRI, consider switch to SNRI or other agent such as mirtazapine or nortriptyline. There is no evidence to suggest augmentation is superior to switching strategies for pharmacologic treatment of anxiety in older adults.	Use with caution in patients taking other highly serotonergic agents or those with history of hypertension. If SNRI medication does not lead to improvement, consider stepwise trials of mirtazapine (15–45 mg/qhs) or nortriptyline 10–50 mg qhs. EKG should be obtained before exposure to tricyclic antidepressants. If anxiety is disabling and/or does not respond to these interventions, referral to Behavioral Health/Psychiatry is indicated.	Katz IR, Reynolds CF 3rd, Alexopoulos GS, Hackett D. Venlafaxine ER as a treatment for generalized anxiety disorder in older adults: pooled analysis of five randomized placebo-controlled clinical trials. J Am Geriatr Soc 2002;50(1):18–25.
Once treatment is initiated, monitor response with the GAD-7. Scores of </= 5 on the GAD-7 may suggest clinically meaningful improvement. The Penn	SSRIs are effective maintenance treatment for late-life anxiety disorders. Reassess at annual intervals to determine if continued treatment is warranted. For example, if patients have engaged in CBT and acquired improved coping skills, a slow taper of the SSRI may be attempted. If a	Wetherell JL, Petkus AJ, White KS, et al. Antidepressant medication augmented with cognitive behavioral therapy for generalized anxiety disorder in older adults. Am J Psychiatry 2013;170:782–789.

(continued)

Table 1 Continued

Algorithm component	Comments	References
State Worry Questionnaire (abbreviated version) may also be used to monitor improvement. Improvement of 6 points on the PSWQ-A is consistent with a meaningful response. Measurement-based behavioral health is the preferred approach for monitoring response to interventions.	taper is attempted, reassess frequently for re-emergence of anxiety symptoms.	Lenze EJ, Mulsant BH, Shear MK, et al. Efficacy and tolerability of citalopram in the treatment of late-life anxiety disorders: results from an 8-week randomized, placebo-controlled trial. Am J Psychiatry 2004;162(1):146–150. Roy-Byrne P, Craske MG, Sullivan G, et al. Delivery of evidence-based treatment for multiple anxiety disorders in primary care: A randomized controlled trial. JAMA 2010;303(19):1921–1928.
Annually for responders: Assess benefits of sustained exposure to medication versus risk of relapse if discontinued.		
Consider taper of opioids prescribed for CLBP in patients with anxiety syndromes.	Anxiety may worsen the experience of pain. Opioid analgesics have both anxiolytic and mood elevating properties. Some older adults with comorbid CLBP and anxiety may be misusing opioids in an effort to reduce their burden of anxiety. Given the risks of prolonged exposure to opioids in older adults, and that opioids are not an approved treatment for anxiety, a taper of opioids may be indicated.	Wasan AD, Michna E, Edwards RR, et al. Psychiatric comorbidity is associated prospectively with diminished opioid analgesia and increased opioid misuse in patients with chronic low back pain. Anesthesiology 2015;10(123):861–872.
If GAD-2 is 3 or greater but more thorough assessment is negative, reassess with GAD-2 at least annually.	A stepped care protocol with at-risk primary care patients has been shown to reduce the incidence of anxiety disorders and depression by 50% over 24 months. Annual screening of these at risk patients is efficient and may identify syndromal anxiety in patients who may not spontaneously report it.	van't Veer-Tazelaar PJ, van Marwijk HW, van Oppen P, et al. Stepped-care prevention of anxiety and depression in late life: a randomized controlled trial. Arch Gen Psychiatry 2009;66(3):297–304. van't Veer-Tazelaar PJ, van Marwijk HW, van Oppen P, et al. Prevention of late-life anxiety and depression has sustained effects over 24 months: a pragmatic randomized trial. Am J Geriatr Psychiatry 2011;19(3):230–239.

time or stand for more than several minutes without severe pain. The low back pain limits her ability to function independently and her two daughters have been assisting her with transportation, groceries, and accompanying her to doctor appointments. When asked how she spends her day, she replied, "*I used to be more active, now I am alone puttering at home more often than not*." The patient described being fearful of walking up the stairs to her bedroom because of fear of falling because *"the pain might overwhelm me, making me feel weak*," and she is concerned that excessive activity will damage her spine.

Table 2 Recommended early sequence of pharmacotherapy for treatment of generalized anxiety disorder

Medication	Target/maintenance dose	Notes
Citalopram	20 mg (for patients > 60 y.o.)*	May consider first line treatment with sertraline or escitalopram if there are concerns about QTc.*
Sertraline	100–200 mg/day	
Escitalopram	10–20 mg/day	

*The FDA has recommended that citalopram should no longer be used at doses greater than 40 mg per day because it could cause potentially dangerous abnormalities in the electrical activity of the heart, in particular prolonged QTc. The maximum recommended dose for patients older than 60 is 20 mg/day. If no response to SSRI, consider switch to SNRI or stepwise trials of mirtazapine (15–45 mg/qhs) or nortriptyline (10–50 mg qhs).

She has consulted both a neurological surgeon and orthopedic surgeon during the past 5 years to inquire if surgery may help with her low back pain. While equivocal, both surgeons suggested she consider laminectomy and fusion. Because of anxiety about being able to manage on her own after surgery and worries that the surgery might not work, she has never had spine surgery. Over the past 4 years she episodically consulted pain medicine specialists at a pain clinic where she was treated with opioid analgesics, non-opioid analgesics, and muscle relaxants. She tried physical therapy 2 years ago, but felt it exacerbated the pain. She has used heat therapy, massage therapy, and topical analgesics. Currently she is prescribed cyclobenzaprine 10 mg TID, gabapentin 900 mg TID, oxycontin 10 mg BID, and alprazolam 0.5 mg up to TID as needed.

Relevant Physical and Psychiatric Examination, and Review of Symptoms: She was alert and oriented X 5. Her thought process was tangential but redirectable. She carried a quad cane in her left hand, and her stride length was short with a slow gait. Examination of paralumbar erector spinae musculature was notable for exquisite tenderness with taut bands and trigger points bilaterally that when palpated reproduced the patient's pain. She appeared frail and her body mass index was 20. Her Mini Mental State Examination score was 29 (theoretical range is 0–30), missing 1-point for not knowing the county [27]. She scored a 15 on the Generalized Anxiety Disorder 7-item scale (theoretical range 0–21) endorsing daily anxiety, worrying too much about different things, being unable to stop worrying, and feeling afraid as if something awful might happen [28]. More than half the days she has trouble relaxing. She spontaneously reported that she has always been a poor sleeper, but is now having increasing difficulty falling asleep despite taking the 0.5 mg of alprazolam before bed every night as she has for many years. She said that she worries most at night while she is trying to fall asleep. She described using alprazolam and prayer to help manage excessive worry. The Patient Health Questionnaire (PHQ-9) was administered to assess for depression [29]. She scored a 9 (theoretical range 0–27), which is indicative of mild depressive symptoms. She endorsed the following: insomnia and poor appetite more than half the days, and mild symptoms of low energy, poor concentration, self-critical thinking, and restlessness. She did not meet criteria for a depressive disorder, denying depressed mood, anhedonia, or suicidal ideation, saying, "*I love being alive, I'm just so worried that something bad is going to happen, or I will fall*." She denied stockpiling pills or misusing her medications. She completely abstains from alcohol. Upon further discussion, she reported that she is increasingly worried about her health and what will happen "*when my pain gets too bad that I can't walk—my daughters will have to push me in a wheelchair*." In addition to her CLBP, she described concerns about bowel functioning, frequent nausea, headaches, fatigue, and tingling in her hands and feet. She denied fever, chills, night sweats, or weight change. The main identified contributors to the patient's CLBP were Generalized Anxiety Disorder (GAD) and myofascial pain syndrome.

Approach to Management

Many older adults with clinically significant anxiety have not been previously diagnosed with an anxiety disorder and may not spontaneously report symptoms of anxiety [30]. Given the high comorbidity of anxiety and low back pain and the potential benefits of treatment, we recommend screening for anxiety with the GAD-2 [28] in all older adults with CLBP. Brief screeners (2-items), such as the GAD-2, have been found to be equally sensitive and specific at detecting anxiety in CLBP patients compared to widely used longer-form "gold standards" [28,31–33]. If patients score a 3 or greater on the GAD-2, providers should conduct a more thorough assessment of anxiety severity by using the GAD-7 [28], which provides more granular details about anxiety that is not routinely ascertained during medical visits. Using the threshold score of 10, the GAD-7 has a sensitivity of 89% and a specificity of 82% for GAD [34]. For patients that were positive on the brief screener (i.e., GAD-2 $\geq$ 3) but more thorough assessment was negative for anxiety (i.e., GAD-7 $<$ 10), it is recommended that providers administer annual GAD-2 screenings to these at-risk patients as an efficient way to identify syndromal anxiety in patients who may not spontaneously report it. The patient presented above spontaneously reported anxiety symptoms, thus the GAD-7 was performed without first doing the GAD-2. Somatic complaints accompanying anxiety often support a diagnosis of GAD rather than

other anxiety disorders. When excessive somatic complaints are present, patients may also meet criteria for Illness Anxiety Disorder [12]. It is not uncommon for anxious older adults to have more than one anxiety disorder, which may be treated simultaneously.

Another approach for providers to screen for anxiety in older adults with CLBP is to assess whether the patient is taking benzodiazepines, sedatives, and/or over-the-counter sleep aids on a regular basis. Long-term benzodiazepine and sedative use is common, particularly in primary care settings, yet considered to be relatively unsafe for older adults [35,36]. Even low doses of benzodiazepines in older adults may lead to negative outcomes, such as increased risk of falls, fractures, cognitive impairment, and delirium [37]. If benzodiazepines or sedatives are prescribed, we recommend providers refer to the Beers criteria for appropriateness of prescribing as well as consider working with a specialist to reduce benzodiazepine use in cases of dependence. Discontinuing benzodiazepines that have been prescribed long-term is often challenging because of physiological withdrawal, psychological dependence, and need to monitor for the return and clinical management of anxiety symptoms [38,39].

When treatment is warranted (i.e., current diagnosis of anxiety or GAD-7 $\geq$ 10), both selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapy (CBT) are effective for treating anxiety in older adults [40–42]. Using a shared-decision making approach (i.e., a collaborative process where patients and their providers make health care decisions together), providers may implement treatment with a SSRI (Table 2), CBT, or both. While SSRIs are superior to placebo [43,44], the benefits of pharmacotherapy may not be as durable as the skills acquired with psychotherapy. CBT may not only boost treatment response for partial responders to an SSRI but may also allow for patients to discontinue their SSRI while maintaining adequate symptom control [21]. When pharmacotherapy is the preferred treatment method, it is recommended that SSRIs be used first-line. If patients do not respond, providers may then consider switching to a serotonin-norepinephrine reuptake inhibitor (SNRI) or another agent such as mirtazapine or nortriptyline (i.e., consider stepwise trial of mirtazapine (15–45 mg/qhs) or nortriptyline (10–50 mg qhs)). When treatment is initiated, whether it is pharmacotherapy or psychotherapy, providers should utilize a measurement-guided approach, monitoring for clinically meaningful response with the GAD-7 (scores of $\leq$ 5) or the Penn State Worry Questionnaire-abbreviated version (improvements of 6 points) [45]. If anxiety is disabling and/or does not respond to these interventions, providers should consider a referral to Behavioral Health or Psychiatry. On an annual basis, providers should assess benefits of sustained exposure to medication versus risk of relapse if discontinued.

Older adults with an anxiety disorder are frequently diagnosed with a comorbid depressive or insomnia disorder [46,47] and if present, such disorders should be treated concurrently as described in the Depression and Insomnia articles in this series [48,49]. In addition, some older adults may misuse opioids in an effort to alleviate anxiety. Given the risks of prolonged exposure to opioids in older adults (e.g., falls, sedation, respiratory depression), and that opioids are not an approved treatment for anxiety, a taper of opioids may be indicated.

Resolution of Case

The patient's primary care physician reviewed the common symptoms of GAD with her and her daughter. The patient was resistant to the idea that anxiety may worsen her experience of pain, but her daughter reinforced that she has observed this pattern—that when her mother is more anxious, often in response to family stress and concerns about money, she often experiences a pain flare and becomes less physically active. The provider also explained to the patient that her current medications—cyclobenzaprine, alprazolam, and oxycontin—may increase her risk of falls and cognitive impairment and pointed out that these medications do not seem to be providing adequate analgesia. The patient stated "*Yes, but they take the edge off. Without them I would be in even worse shape*."

After describing the various treatment options for anxiety, the patient and provider mutually decided that to treat her symptoms of anxiety she would be prescribed escitalopram 10 mg/day. At first, the patient was apprehensive about taking an antidepressant, claiming "*I'm not depressed*." She was fearful about side effects (e.g., weight gain and interactions with other medication) and if the medication would be addictive. Her provider explained to her that antidepressants are approved for the treatment of anxiety in addition to depression. The daughter agreed to provide ongoing encouragement to her mother to take the escitalopram every day. The patient was offered a referral to a psychologist for CBT, but she refused. She did agree, however, to join a gentle yoga and meditation group at their church, and the daughter agreed to attend with her on a weekly basis. Relaxation approaches, such as yoga and meditation, have been found to be the most effective component of CBT for anxiety in older adults [50].

At the first follow-up visit 2 weeks later, the patient reported experiencing nausea, "palpitations," and "wooziness" after taking the first dose of escitalopram 10 mg and stopped taking it after that first dose. She agreed to a much slower titration, starting at 2.5 mg for the first week, increasing by 2.5 mg every 7 days. She tolerated this slow titration quite well, and after taking the 10 mg dose for 6 weeks, stated that she felt that her "*mind is not racing all the time—I'm settled now*." She continued to endorse daily low back pain, but denied any recent flares and was less fearful about movement and the future. She attended the gentle yoga/meditation group with her daughter and was following recommendations of the instructor to use deep breathing and progressive muscle relaxation techniques for at least 15 minutes

every day. After 2 months of treatment, the provider assessed the patient's progress with treatment with the GAD-7 and she scored a 5, which indicated an improvement from her initial assessment. She described feeling *"peaceful"* after practicing the deep breathing and progressive muscle relaxation exercises, and was committed to continuing this practice. While she refused to stop taking the alprazolam for sleep, she did agree to decrease daytime use. Although the patient was fearful of stopping use of opioids over the course of four months, the long acting oxycontin was gradually discontinued. Since there is insufficient evidence to support the efficacy of cyclobenzaprine for myofascial syndromes [51], and cyclobenzaprine is listed on the Beers list as a medication to avoid in older adults, this medication was also discontinued over the course of four months. This slow taper of these medications did not result in worsening of her pain. Despite limited evidence that gabapentin improves myofascial pain, and acknowledging that it is not approved for treatment of anxiety [52], since the patient did not want to discontinue the gabapentin and was tolerating it well, because of perceived benefit, her physician agreed to continue its use.

It also should be highlighted that this patient had both anxiety and myofascial pain (MP) as important contributors to her CLBP. Anxiety is thought to be an important perpetuating factor in patients with MP [53]. Anxiety symptoms are common in patient with MP [54], and our own preliminary data (not shown) suggest that one in two older veterans with MP as part of their CLBP also has significant anxiety symptoms. Anxiety may cause muscle tension [55] that contributes to MP, and it can contribute to central sensitization, thought to be an important contributor to MP pathogenesis [56]. The pharmacological and behavioral treatment of our patient's anxiety likely contributed to improving her MP and the lessening frequency of her pain flares.

Summary

Symptoms of anxiety are common in older adults with CLBP and the co-occurrence of these disorders is associated with negative patient outcomes such as amplified disability and overuse of opioids and benzodiazepines. Unfortunately, late-life anxiety is often undiagnosed and thus undertreated, particularly in chronic pain patients. Therefore, it is paramount that providers routinely screen for anxiety in older adults with CLBP to identify patients who may benefit from treatment (i.e., SSRIs or CBT). Recently, the US task force released recommendations for screening of depression in adults [57]. Even though this statement did not include assessing for other commonly occurring symptoms of emotional distress, such as anxiety, we believe joint screening (i.e., anxiety and depression) may be efficiently done with this high-risk group of older adults. The goal of the treatment algorithm (Figure 1) is to provide an evidence-based decision aid for clinicians to use in the shared treatment decision-making process with older adults with co-occurring anxiety and CLBP.

Older adults with CLBP commonly exhibit increased levels of emotional and cognitive distress. In addition, patients with anxiety and CLBP are at increased risk for having co-occurring mental health conditions, such as depression, insomnia and substance use. Given the complexity of such patients, it is important that providers assess and treat these mental health conditions while concurrently treating anxiety. Because they are associated with negative patient outcomes, providers also need to assess for whether the patient is taking long-term benzodiazepines, sedatives, and/or over-the-counter sleep aids on a regular basis. If such medications are prescribed in older adults with co-occurring anxiety and CLBP, it is recommended that providers refer to the Beers criteria for appropriateness and consider discontinuing their use, possibly with guidance from Behavioral Health specialists.

Key points:

1. Co-occurring anxiety and CLBP have been linked with negative outcomes such as greater functional disability, reduced health-related quality of life, overuse of benzodiazepines, and misuse of opioids. Therefore, clinical evaluation of all older adults with CLBP should include screening for anxiety.
2. Treatment algorithms to guide management of anxiety in patients with CLBP may increase recognition of patients in need of treatment as well as increase their engagement in evidence-based treatments.
3. Long-term use of benzodiazepines, sedatives, and OTC sleep aids for treatment of anxiety or pain should be avoided in older adults. They also may be indicative of an underlying anxiety disorder.
4. Treatment recommendations should be guided by collaboration with the patient to determine shared treatment goals and preferences. First-line treatment options include pharmacotherapy with SSRIs or psychotherapy using a cognitive behavioral approach.
5. Anxiety can contribute to muscle tension and myofascial pain. Thus, anxiety is an important treatment target in patients with a myofascial contributor to CLBP.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment: *Part X: Sacroiliac Joint Syndrome*

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Conflicts of interest: There are no conflicts of interest to report at this time.

Disclosure: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the United States government.

Abstract

Objective. To present an algorithm of sequential treatment options for managing sacroiliac joint (SIJ) pain in the setting of chronic low back pain (CLBP) in the older adult. This is the tenth part in a series, and includes an illustrative clinical case.

Methods. The stepped care drug table and evaluation and treatment algorithm were created following a thorough literature review of approaches and subsequent analysis through a modified Delphi process. The principal investigator developed the initial draft, which was refined for content by an interdisciplinary panel of five experts. The refined materials were then assessed for the feasibility of implementation and validity of recommendations for older adults in a primary care setting by a panel of nine primary care providers. While not exclusive to Veteran's Health Administration (VHA) facilities, an emphasis was made to include resources and medications available to providers in the VHA.

Results. The algorithm and drug table developed to systematically identify and address SIJ pain in the older adult is presented here. The process should begin with recognizing the presenting symptoms of CLBP stemming from the SI region, and supporting physical exam testing using the compression test and thigh thrust maneuver. Identification of the SIJ as a pain generator is followed by assessment and treatment of contributory factors. SIJ pain treatment should begin with education and self-management including exercise, and may escalate to include interventional procedures and/or referral to a pain rehabilitation program.

Conclusions. Pain originating from the SIJ is often under-recognized, but a structured and consistent approach can help identify older patients who would benefit from treatment of this contributor to CLBP.

Key Words. Chronic Low Back Pain; Sacroiliac Joint Pain; Older Adults

Introduction

The understanding of the sacroiliac joints (SIJs) has a complex history stretching back centuries [1]. Debate over their form, function, specific contribution to CLBP, and how to effectively identify and treat pain originating from the SIJ continues today.

The first of a two-part study by Fortin et al. in 1994 [2] helped establish the fact that pain can arise from the SIJ itself through intra-articular injections of contrast in asymptomatic controls. The second part sought to develop a screening tool to aid in the clinical diagnosis of SIJ pain [3]; more than two decades later, the ideal evaluation methods have yet to be defined.

Complicating the effort to make a diagnosis is the well-established recognition that pain that appears in the region of the SIJ can originate from other sources, including herniated discs and facet joints [4,5]. Conversely, pain originating from the SIJ can refer to the groin and adjacent areas such as the gluteal muscles and lower extremity, even as far as the foot [6]. Furthermore, as described by King et al., consistency within the literature on the precise usage of "SIJ pain" as intra- vs extra-articular in origin is lacking; the former, they describe as true SIJ pain, while the latter would more properly defined as "sacroiliac complex" pain, which would include pain originating from the supporting ligaments [7].

Imaging, including computed tomography (CT) and magnetic resonance imaging (MRI), can be useful in the identification of sacroiliitis associated with trauma or ankylosing spondylitis (AS) [8]; the latter rarely presents symptomatically in patients beyond their fifth decade [9]. However, in the absence of these conditions, imaging findings are rarely helpful in identifying the sacroiliac joint as a pain generator. Further complicating radiographic diagnosis is the common co-existence of degenerative changes observed in lumbar spine imaging of older adults. As discussed earlier in this series [10,11], Hicks et al. performed radiographic assessment of 320 subjects over 65, 162 with CLBP alongside 158 without pain, and discovered that 95% of subjects in both groups showed evidence of degenerative disc disease [12], while Jarvik et al. identified moderate or severe stenosis in the lumbar spine in 21% of a small group [13] of pain-free adults over 65 when examined by MRI. The near-ubiquity of degenerative findings on plain radiographic [12] or advanced imaging [13] suggest that these modalities have little if any value used as a screening test establishing a specific cause and guiding treatment of CLBP in the older adult.

This article aims to define an algorithm for treatment of patients with suspected or confirmed LBP that originates in the SI joints. We present a case adapted from a patient cared for by one of the contributors. We describe what may be a familiar patient presentation to a clinician managing the health and wellbeing of adults in the later stages of life, and how to systematically address the often complex problem of CLBP.

Methods

As part of the special series addressing CLBP in older adults, this work utilized the modified Delphi technique described in detail in the introduction to this series [10]. Through that process, an algorithm (Figure 1) and corresponding evidence table (Table 1) were developed, along with a stepped care drug management table (Table 2). The panel of five experts was comprised of two physiatrists, a physical therapist, internal medicine physician, and a pain anesthesiologist. One member of the team had a primary appointment in the VA Healthcare System. The completed algorithm was reviewed by a panel of nine primary care physicians, followed by feedback and modification to result in the final algorithm.

Case Presentation

Relevant History

The patient was a 69-year-old female with a 2-year history of low back/buttock/hip pain radiating to the left lower extremity that began after surgical treatment for chronic left foot pain. Three years prior she was diagnosed with posterior tibial tendon dysfunction, and subsequently underwent triple arthrodesis at the subtalar, talonavicular, and calcaneocuboid joints of the left foot, with a revision due to nonunion 6 months prior to presentation. Her low back pain began 1 year after her initial arthrodesis, which she felt was due to her foot pain causing irregularities in her gait. Pain was reported as always present, with a baseline severity of 4/10 and frequent increases to 10/10. She states that the pain was worsened by transitions between sitting and standing, walking long distances, and lying for long periods on her back or side. She was no longer able to vacuum

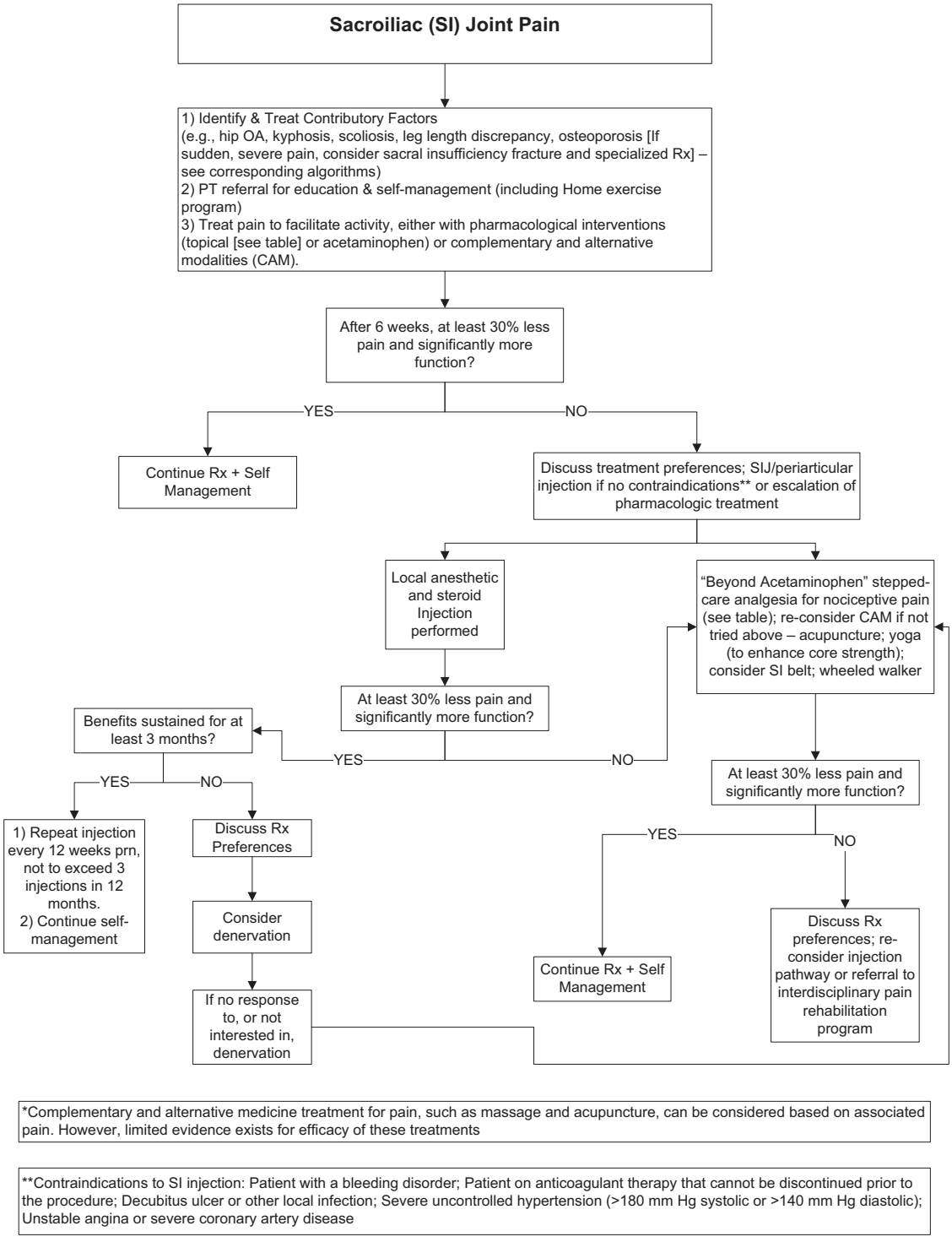

Figure 1 Algorithm for the evaluation and treatment of Sacroiliac Joint Syndrome in an older adult with CLBP.

her home. The pain was mildly relieved by heat, rest, and forward flexion. She denied bladder or bowel incontinence, spinal trauma, weakness, numbness, tingling, or falls. Prior treatments included gabapentin, acetaminophen, topical lidocaine, several non-steroidal anti-inflammatory drugs (NSAIDs), L4-L5 epidural steroid injection, and physical therapy (PT) directed toward core strengthening. None of these interventions provided

Table 1 Sacroiliac joint syndrome: Theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm component	Comments	References
30% pain reduction as significant	Data on 2724 subjects from 10 placebo controlled trials of pregabalin in diabetic neuropathy, postherpetic neuralgia, CLBP, fibromyalgia, and OA.	[23]
	SIJ pain was not one of the conditions specifically studied; in older adults with CLBP it is often one of several contributors.	[28]
SIJ injection with local anesthetic and steroid	While no single aspect of the history or physical examination can reliably identify pain originating from the SIJ, a battery of three or more provocation tests can predict response to diagnostic blocks. Evidence supports both intra- and extra-articular causes for SIJ pain, and clinical studies demonstrate intermediate-term benefit for both intra- and extra-articular steroid injections.	[29]
SIJ denervation	In those who fail to experience sustained relief from SIJ injections, radiofrequency (RF) denervation may provide significant relief lasting up to 1 year. Conventional RF denervation has been compared to a cooled RF technique that results in a wider ablation diameter. The results do not demonstrate a clear advantage of one technique over the other.	[30–32]
Pain self-management program	High quality evidence is lacking specifically in older adults. Arthritis pain self-management programs that contain some CBT elements demonstrate efficacy.	[21]
Non-acetylated salicylates	Non-cyclooxygenase selective NSAIDs (e.g., ibuprofen, naproxen) should not be used chronically in older adults because of the potential for multiple adverse effects including but not limited to gastrointestinal bleeding, renal insufficiency, and exacerbation of hypertension and congestive heart failure.	[25]
Opioids as part of stepped-care management	American Geriatrics Society pain guidelines recommend opioids over non-selective NSAIDs.There are no data specifically for the treatment of SIJ pain.	[25]

significant pain relief or functional improvement. She owned a cane and a walker, but felt steadier without them. She never used shoe orthoses. She was recently prescribed hydrocodone 10 mg/acetaminophen 325 mg to be taken every 4 hours as needed, which she typically took to get out of bed in the morning and to fall asleep at night.

Relevant Physical Examination

The patient was alert, oriented, pleasant, cooperative, and in no apparent distress. She had an antalgic gait that was slightly wide-based. Lumbar range of motion was pain limited, provocative in flexion, but not extension. There was no evidence of scoliosis or kyphosis. Significant tenderness was present on palpation of the left greater trochanter, no tenderness on the right greater trochanter. No leg length discrepancy was observed in the supine position. Anterior superior iliac spines were symmetric when standing. The left SIJ was tender to palpation but there was no tenderness on the right. Compression test (Figure 2A) and thigh thrust (Figure 2B) maneuvers were provocative of low back pain symptoms on the left side and non-provocative on the right. Internal and external hip range of motion were normal and non-provocative of pain. The seated slump test, to evaluate for radiculopathy [14], was provocative of left posterior thigh pain, but demonstrated no change with release of dural tension (i.e., cervical extension), and non-provocative on the right. Straight leg raise was negative bilaterally. Bilateral strength, sensation, and reflexes in the lower extremities were symmetric and within normal limits. She had a pes planus deformity of the left foot, and impaired active and passive range of motion for left ankle plantarflexion and dorsiflexion.

Imaging

Lumbar x-rays from a previous provider obtained approximately 6 months prior to presentation showed

Table 2 Stepped care drug management of sacroiliac joint pain

Drug	Dose/titration*	Important adverse effects/precautions
Topical preparations	Lidocaine 5% bid-tid; diclofenac gel 1% bid.	Efficacy evidence is limited.
Acetaminophen	325–1000 mg q4–6h, max 3 gm/d. Adjust dosing interval for renal function: CRcl 10–50: q6 hrs; CrCl $<$ 10: q8 hours.	Ask about all OTCs with acetaminophen; increased toxicity from chronic use if heavy EtOH use, malnourishment, pre-existing liver disease—decrease maximum daily dose to 2 gm.
Salsalate	Salsalate: 500–750 mg bid; maximum dose 3000 mg/day.	Does not interfere with platelet function; GI bleeding & nephrotoxicity rare; salicylate concentrations can be monitored if toxicity suspected.
Choline magnesium trisalicylate	Choline magnesium trisalicylate: 750 mg tid; maximum dose 3000 mg/day.	
Tramadol	Start 25 mg daily; increase by 25–50 mg daily in divided doses every 3–7 days as tolerated to max dose of 100mg QID. Renal dosing (CRcl $<$ 30 ml/min) 100mg bid.	Seizures and orthostatic hypotension. Other side effects similar to traditional opioids including sedation, confusion, respiratory depression. Potential for serotonin syndrome if patient is on other serotonergics such as triptans, duloxetine, and other antidepressants.
Hydrocodone/ acetaminophen	2.5/325 or 5/325–10/650 mg q4–6h; max acetaminophen dose 3gm/d. Consider recommending a supplementary dose of APAP 325 or 500 mg with combination dose for additional analgesia before increasing the opioid dose.	For all opioids, increased risk of falls in patients with dysmobility. May worsen or precipitate urinary retention when BPH present. Increased risk of delirium in those with dementia. Because of increased sensitivity to opioids older adults at greater risk for sedation, nausea, vomiting, constipation, urinary retention, respiratory depression, and cognitive impairment. Start stimulant laxative at first sign of constipation. Some might start at initiation if patient has existing complaints of constipation or other risk factors.
Oxycodone or morphine	5–10 mg oxycodone q4h (begin with 2.5–5mg q4h) OR morphine 2.5–5 mg q4h; assess total needs after 7d on stable dose, then convert to long acting.	Side effects as per hydrocodone.Start stimulant laxative at first sign of constipation.NEVER start long acting opioid before determining needs with short acting.
Duloxetine	Start 20–30 mg/d; increase to 60 mg/d in 7 d. Not recommended in ESRD or CLcr $<$30.	May precipitate serotonin syndrome when combined with triptans, tramadol, and other antidepressants. Key drug-disease interactions: HTN, uncontrolled narrow-angle glaucoma, seizure disorder. Precipitation of mania in patients with bipolar disorder. Important adverse effects include nausea, dry mouth, sedation/falls, urinary retention, constipation. Contraindicated with hepatic disease and heavy alcohol use. Abrupt discontinuation may result in withdrawal syndrome. Contraindicated within 14 days of MAOI use

*Abbreviations such as bid should be avoided in an effort to reduce errors.

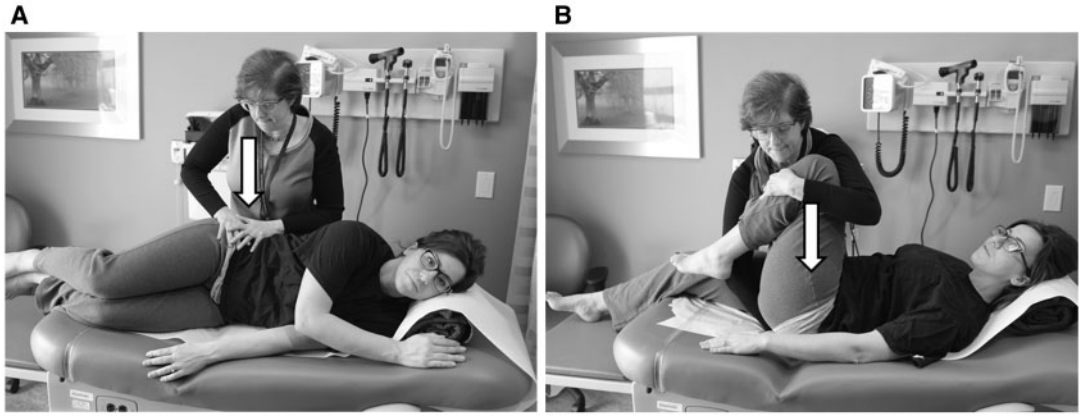

Figure 2 (A) Compression test. The patient lies on the unaffected side with hips and knees flexed to approximately 90 degrees. The examiner, positioned above the patient, applies a force vertically downward on the uppermost iliac crest. (B) Thigh thrust. The patient lies supine with the hip and knee flexed with the thigh perpendicular to the exam table. One of the examiner's hands cups the sacral base and the other arm and hand wraps around the flexed knee to apply pressure in the anterior to posterior direction along the long axis of the femur. It is frequently helpful for the examiner to be positioned above the subject to maximize the applied force.

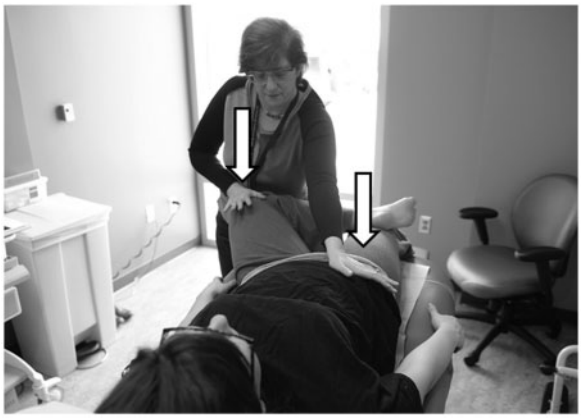

Figure 3 FABER test (Patrick's sign). With the patient supine, bring the hip into flexion, abduction and external rotation by placing the ipsilateral heel against the knee of the opposite leg. Provide overpressure to the medial knee and contralateral anterior superior iliac spine.

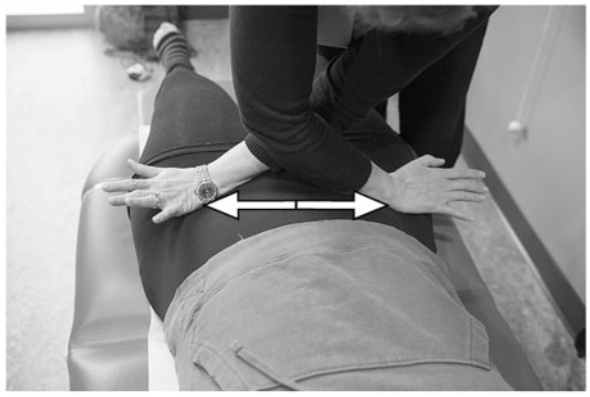

Figure 4 Distraction test. With the patient supine on the exam table, the examiner with crossed arms applies pressure to the anterior superior iliac spines posteriorly and laterally.

grade 1 (mild) anterolisthesis of L4 on L5, preserved disc height and vertebral body height, and mild bilateral sclerosis of the sacroiliac joints, with left worse than right.

Clinical Course

The patient was started on a PT program with an emphasis on hip abductor strengthening and gait normalization, along with a home exercise program (HEP). Her hydrocodone/acetaminophen was discontinued, and tramadol was initiated. However, the tramadol precipitated migraine headaches, and was discontinued. She began using over-the-counter analgesics only as needed. Her back pain and functional status continued to decline over the following 4 months despite compliance with her HEP. She began having difficulty walking in the community. She was counseled on the risks and benefits of a fluoroscopically guided intra-articular SI joint injection, and decided to pursue this procedure. Following anesthetization of the skin, a 22-gauge, 3.5-inch spinal needle was advanced under continuous fluoroscopy into the left sacroiliac joint. Contrast agent was infused to ensure proper placement and a typical arthrographic joint pattern was produced. Depo-Medrol

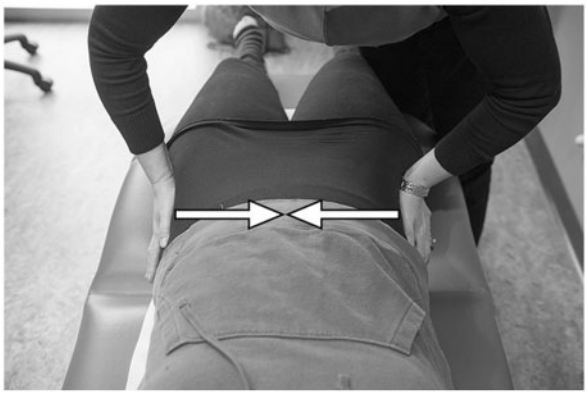

Figure 5 Compression test. With the patient supine on the exam table, the examiner places his/her hands on the iliac crests and applies an inward/downward force. Pain indicates a positive test.

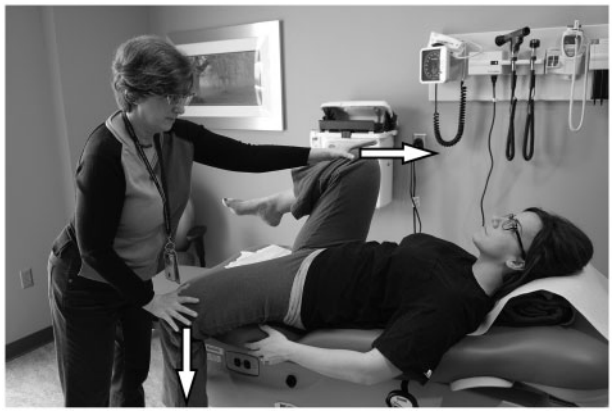

Figure 6 Gaenslen test. With the patient supine, and the tested side extending partially over the edge of the table, the examiner guides both knees to chest then lowers the leg on the tested side unsupported over the edge of the table while maintaining flexion of the opposite side.

and 2% lidocaine were then infused. This injection reproduced her concordant back pain, and was therefore considered diagnostic. Following the procedure, the patient reported an immediate pain reduction from 10/10 to 4/10, with a 50% reduction in pain severity present at 6-week follow up. She was able to walk for considerably longer distances and sleep comfortably on her back and sides. With symptoms more effectively controlled, she was able to restart physical therapy along with her home exercise program, and continued to progress toward achieving her functional goals.

Approach to Management

This patient has CLBP that is likely multifactorial and has been aggravated by compensatory change in gait due to her chronic lower extremity pathology. Her foot pain had been present for nearly her entire adult life, but she experienced a significant increase in severity over the past decade, further exacerbated after a corrective surgical procedure. Her CLBP had previously been attributed to a variety of sources, including sciatica, lumbosacral degenerative disc disease, lumbar spinal stenosis, spondylolisthesis and lumbosacral neuritis before presenting to our clinic. Her initial exam provoked pain consistent with trochanteric bursitis [15], adding to the list of potential contributory sources. This complex history highlights the fact that CLBP is frequently multifactorial.

Van der Wurff et al. examined the diagnostic accuracy of a multi-test regimen for the sacroiliac joint, compared to fluoroscopically controlled double SIJ blocks [16]. This study of 27 patients suggested that a test regimen with three or more positive physical exam tests is indicative of SIJ pain and may be used to reduce diagnostic SIJ injections, corroborating previous studies [17]. Similarly, Szadek et al. described positive results on two pain provocation tests, the compression test (Figure 2A) and thigh thrust (Figure 2B) [18], as helpful in diagnosing SIJ pain. They also reported that three or more positive pain provocation tests is preferred to achieve a high diagnostic odds ratio (DOR), calculated as 17.2. However, they stressed that even this testing should be regarded cautiously, based on the potential for inter-practitioner variability, and acknowledged that the studies included were performed at academic centers or spine treatment units. Based on the available data and relative ease of performing the tests in a primary care practice, we recommend the compression test (side-lying) (Figure 2A) and thigh thrust to satisfy consideration of the SIJ region as a pain generator in primary practice. Additional exam maneuvers, such as Patrick's sign (FABERs) (Figure 3), distraction (Figure 4), compression (supine) (Figure 5), Gaenslen (Figure 6), and sacral thrust [16,17] can be added to improve diagnostic accuracy based on the comfort level of the practitioner. These are shown in Figures 3–6. The sacral thrust test is not shown, as this is performed with the patient prone, a position that may be difficult for many older adults to comfortably assume. Of note, these physical exam maneuvers are performed most effectively using an adjustable or mat table. However, due to the frequent lack of availability of these exam tables in primary care offices, the exam technique is shown in its modified form using standard exam tables.

The algorithm presented (Figure 1) shows that the first step in addressing SI joint pain begins with identifying and treating potential contributory factors, including but

not limited to hip osteoarthritis, kyphosis, scoliosis, leg length discrepancy, and osteoporosis.

This process should inform the next step: a referral to physical therapy. The therapist should educate the patient and teach them how to begin to manage their pain on their own, including instruction for a home exercise program. The therapist should target strengthening the hip abductors, correcting any impairments of strength or flexibility that may exist, and building both core and pelvic stability. These can include side-lying resistive exercises (clamshell, i.e., hip abduction with knees bent maintaining contact between the ankles, and straight leg abduction), weight-bearing exercises such as single leg standing, balance-related exercises, water-based training, or Tai Chi [19]. Some evidence also suggests that increasing balance may have the added benefit of reducing falls in older individuals [20] and there have been some promising results in programs integrating elements of CBT, specifically in patients with arthritis [21,22].

The third part of the initial plan should address pain directly, working with the patient to develop an approach to pain management that is sufficient to begin and maintain their therapeutic activity regimen. The patient should be counseled that the combination of physical therapy, self-care, and pharmacologic intervention should reduce pain by 30% at 6 weeks, noted previously to be a clinically important difference [23], and have a significant positive impact on their daily function. We recommend beginning with topical lidocaine or diclofenac gel alone or in combination with acetaminophen. It is also worth considering complementary and alternative medical (CAM) therapy such as acupuncture or massage for pain management. We do caution that limited evidence exists to confirm the efficacy for treating SI joint pain utilizing CAM interventions or the aforementioned topical preparations. Improvements in self-reported disability and pain have been reported with the use of sacroiliac joint belts (pelvic belts) [24] though this has not been explicitly studied in an older adult population. A wheeled walker may be prescribed if there is concern regarding stability or balance.

If these initial pharmacological interventions do not produce sufficient relief to allow for increased activity, the stepped care process outlined in Table 2 should be followed. Salsalate is recommended over other NSAIDs due to a lower risk of GI bleeding and renal toxicity from lack of effect on platelets [25]. If opioids are indicated, we recommend tramadol as an initial intervention, with appropriate precautions taken, particularly related to potential drug-drug interactions (e.g., serotonin syndrome when combined with other serotonergic medications), sedation, and fall risk. Opioids of increasing strength may be necessary to facilitate therapeutic interventions, though patient education on the risks, benefits, and expectations is critical and should be used with caution.

When pharmacological management is insufficient to permit return to activity or participation in an active physical therapy program, additional pain generators should be addressed (e.g., greater trochanteric bursitis), and interventional management may be considered. In a recent systematic review of the literature, it was concluded that it is unclear whether image-guided intra-articular diagnostic injections of local anesthetic predict positive responses to therapeutic injections [26]. The same systematic review revealed moderate quality of evidence of the effectiveness of therapeutic SIJ injection. Because of the low intra-articular placement of blind SIJ injections [27], fluoroscopic guidance is recommended. Pain can originate from the ligamentous structures of the sacroiliac complex in addition to the intra-articular structure. Sacral lateral branch thermal radiofrequency ablation has been evaluated for treatment of pain originating from the posterior elements of the sacroiliac complex. While the literature is limited, some evidence of moderate quality exists to support the use of sacral lateral branch thermal radiofrequency ablation when other treatments have been ineffective, though the specific indications for this procedure remain unclear [7]. Interdisciplinary pain rehabilitation programs, where available, may also be a viable option for patients in whom insufficient improvement has been achieved.

Resolution of Case

Following the reduction in pain and improvement in functional activities with physical therapy, the patient began an introductory yoga program to further strengthen her core. She reported ability to walk for longer distances and sleep was improved. She was independent in her activities of daily living and instrumental activities of daily living, and her improvements remained at 6 months.

Conclusion

When seeing patients presenting with CLBP, the SI joint should be considered during any comprehensive evaluation. While pain may be correctly identified as originating from the SI joint, there can be a variety of contributing factors, which should be identified and addressed. This patient suffered from chronic lower extremity pain that caused a gait deformity leading to added stress and physical imbalances, contributing to SIJ symptoms.

Treatment focuses on increased movement and, therefore, interventions to address physical deficits are paramount. Pharmacological interventions should target pain control to allow increased mobility and interventional management may be necessary. The algorithm described here can guide those steps, and prevent misdiagnosis and unnecessary procedures with associated potential morbidity.

Key Points

- Physical examination of all older adults with CLBP should include examination of other potential pain generators, including sacroiliac joint provocative tests.
- While x-rays can be used to determine the extent of SI joint involvement with systemic diseases such as ankylosing spondylitis, imaging should not be routinely used for evaluation of patients with SI joint symptoms.
- Management begins with identifying and treating contributing factors, physical therapy and a home exercise program to address underlying weakness, and pharmacological intervention when needed to facilitate activity.

Pharmacologic Recommendations

- Topical treatments, acetaminophen, and salsalate, in that order, should comprise initial pharmacological pain management.
- Non-cyclooxygenase-selective NSAIDs are included in Beers' Criteria for Potentially Inappropriate Medication Use in Older Adults [25]. They are not recommended for chronic use in these patients.
- When opioids are indicated, patient education on the risks and benefits must be thorough.
- Interventional management may be necessary and should be used to facilitate physical therapy when feasible.

Acknowledgments

Thanks to William George of the VA Pittsburgh for creating the photographs that accompany this article. The authors wish to thank Dave Newman for his valuable assistance in coordinating this project.

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Deconstructing Chronic Low Back Pain in the Older Adult–Step by Step Evidence and Expert-Based Recommendations for Evaluation and Treatment: Part XI: Dementia

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service.

Disclosure and conflicts of interests: The contents of this report do not represent the views of the Department of Veterans Affairs or the US government. The authors report no conflicts of interests.

Drs. Rollin Wright and Monica Malec are co–first authors on this article.

Abstract

Objective. To present the 11th in a series of articles designed to deconstruct chronic low back pain (CLBP) in older adults. The series presents CLBP as a syndrome, a final common pathway for the expression of multiple contributors rather than a disease localized exclusively to the lumbosacral spine. Each article addresses one of 12 important contributions to pain and disability in older adults with CLBP. This article focuses on dementia.

Methods. A modified Delphi technique was used to develop an algorithm for an approach to treatment for older adults living with CLBP and dementia. A panel of content experts on pain and cognition in older adults developed the algorithm through an iterative process. Though developed using resources available within Veterans Health Administration (VHA) facilities, the algorithm is applicable across all health care settings. A case taken from the clinical practice of one of the contributors demonstrates application of the algorithm.

Results. We present an evidence-based algorithm and biopsychosocial rationale to guide providers evaluating CLBP in older adults who may have dementia. The algorithm considers both subtle and overt signs of dementia, dementia screening tools to use in practice, referrals to appropriate providers for a complete a workup for dementia, and clinical considerations for persons with dementia who report pain and/or exhibit pain behaviors. A case of an older adult with CLBP and dementia is presented that highlights how an approach that considers the impact of dementia on verbal and nonverbal pain

behaviors may lead to more appropriate and successful pain management.

Conclusions. Comprehensive pain evaluation for older adults in general and for those with CLBP in particular requires both a medical and a biopsychosocial approach that includes assessment of cognitive function. A positive screen for dementia may help explain why reported pain severity does not improve with usual or standard-of-care pain management interventions. Pain reporting in a person with dementia does not always necessitate pain treatment. Pain reporting in a person with dementia who also displays signs of pain-associated suffering requires concerted pain management efforts targeted to improving function while avoiding harm in these vulnerable patients.

Key Words. Dementia; Chronic Pain; Low Back Pain; Lumbar; Primary Care

Introduction

Pain and dementia are common conditions that increase in frequency with age [1–3]. Pain in older adults tends to be underreported and undertreated compared to pain in younger adults [4]. At the same time, dementia is common and often under-recognized by clinicians. Dementia afflicts as many as 50% of those age 85 and older in the United States [5] and it can directly impact pain assessment and management [3,6].

The cornerstone of chronic pain management is an assessment to identify the multiple medical and psychosocial factors potentially contributing to pain and functional impairment [7], and this includes assessment of cognitive function for many older adults. In patients not already known to have dementia, indicators that should prompt cognitive assessment to screen for dementia include: self or family report of memory loss, difficulty processing information during the clinical encounter, inability to report specific details during the pain history necessitating additional input from a caregiver, older than age 85, or a discrepancy between reported pain and observed pain-related behaviors (see Figure 1 and Table 1) [5,8–10]. Patients can be screened for dementia using one of several measures that are brief and easy to use, such as the Mini-Cog, Montreal Cognitive Assessment (MoCA), and Saint Louis University Mental Status examination (SLUMS) (see Table 1) [11–13]. Patients with a positive screen merit further evaluation for possible dementia and may include a referral for a more comprehensive cognitive evaluation that would be incorporated as part of the pain management plan.

The burden of chronic low back pain (CLBP) in older adults with dementia is unknown. However, dementia is likely to alter pain management because it alters pain reporting, pain behaviors, and pain coping [14]. Some evidence suggests that older adults with dementia report pain nearly as often as cognitively intact older adults [15,16]. Furthermore, the multidimensional experience surrounding pain appears similar in persons with and without dementia [17]. The expression of pain or behaviors surrounding pain, however, may differ meaningfully. Many patients with dementia can report pain reliably [18], and we are taught that patient self-report is the gold standard. In all patients with CLBP, however, validation of pain self-report with behavioral observation is critical. If the patient with dementia reports 8/10 pain and demonstrates no behavioral manifestations of pain, it is possible that their pain verbalization represents simply perseveration, and additional pain treatment may not be required. Patients in pain may present in myriad ways, and no presentation either validates or invalidates the pain experience. Any disconnect between pain reporting and pain behavior should be further explored. In such patients, real-time assessment of pain during daily activities that the patient reports cause pain, such as walking, can provide key information regarding whether pain-focused treatment is needed.

Evidence also indicates that some persons with dementia and pain exhibit agitation and anxiety more often than persons with dementia who are not in pain [19]. Persons in the moderate-to-advanced stages of dementia may lose language skills and ability to verbally express their pain, and they may communicate pain through behaviors (e.g., grimacing, yelling, or bracing), anxiety, and/or agitation. In fact, anxiety and agitation may represent the only clues that a person with dementia is in pain. Failure to consider pain as a potential source of agitation can lead to the overuse of anxiolytics and antipsychotics to control these behaviors [20]. For these reasons, assessment of pain in persons with dementia must extend beyond a simple “yes or no” question about pain. Based on our clinical experience caring for patients with CLBP and dementia, this type of oversimplification often leads to inadequate pain assessment and management. A standardized approach to assessment in nonverbal dementia patients includes evaluation during painful conditions or procedures, observation of pain behaviors using a validated assessment tool, proxy report of behaviors that indicate pain, and an analgesic trial [21].

The presence of dementia significantly impacts pain management because it alters pain reporting, pain behaviors, and pain coping. It also may alter treatment compliance, expectancy, and response. Unfortunately, a readily available evidence base to inform clinical practice in this growing population does not exist. We present an algorithm (Figure 1) to help health care providers caring for older adults with CLBP to recognize which patients to screen for dementia and what dementia

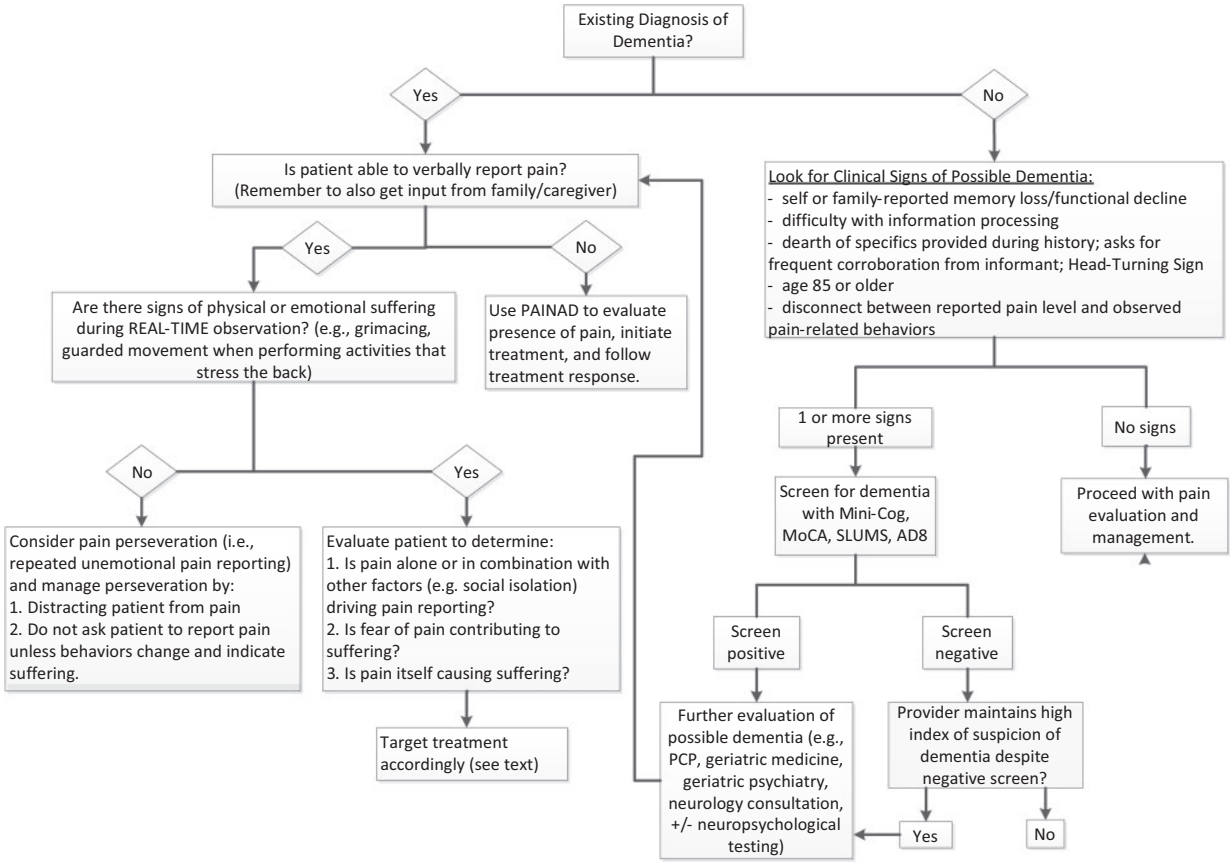

Figure 1 Algorithm for the evaluation and treatment of dementia in an older adult with CLBP. AD8 = Eight-item Interview to Differentiate Aging and Dementia; MoCA = Montreal Cognitive Assessment; PAINAD = Pain Assessment in Advanced Dementia Scale; PCP = Primary Care Physician; SLUMS = Saint Louis University Mental Status examination.

screening tools to use. The algorithm also highlights clinical considerations regarding pain management in people who may have dementia. To illustrate how to apply the algorithm, we present a case with CLBP and underlying dementia. Ultimately, the purpose of this algorithm is to help detect the presence of cognitive impairment in older patients who report pain so that the provider knows to look for evidence of pain-related suffering rather than rely solely on self-report to develop a pain treatment plan.

Methods

An interdisciplinary panel of eight experts used a modified Delphi technique, described in the CLBP series overview [22], to create an algorithm for the approach to the patient presenting with CLBP and signs of dementia. The clinical indicators of dementia relevant to pain practice were identified through a literature review, with ultimate inclusion decided upon by the consensus of the expert panel. The content expert panel refined the algorithm (Figure 1) and the rationale for the various components of the algorithm (Table 1) based on feedback from a primary care provider panel as part of an iterative process [22].

Case Presentation

Relevant History

An 87-year-old woman presents with low back pain for seven years. She is two years status post-L2-4 laminectomy after physical therapy and conservative pain management measures failed. She now has persistent pain and difficulty functioning. She is accompanied by her daughters.

The patient is a retired homemaker who used to garden and play tennis in her free time. For the past five years, she has become increasingly sedentary. Fifteen months ago, she moved to an assisted living facility because of difficulty maintaining her two-story home. She still

Table 1 Dementia: theoretical and pragmatic underpinnings of algorithm recommendations

Algorithm Components	Comments	References
Clinical signs of possible dementia	Age 85 and older: The disease is highly prevalent (~50%) in this age group but often overlooked by primary care providers, even in its later stages. Experts recommend a very low threshold to screen for dementia even if symptoms of decline (any change in performance of familiar tasks, activity, memory, hygiene, accuracy in bill paying, driving, or taking medications, or symptoms of anxiety, judgment, apathy, depression) are subtle.	[5,23,24]
	Disconnect between reported pain and pain behaviors: Some data point to exaggerated pain behaviors and attention to pain in those with dementia.	[8–10,46]
Dementia screening tools	Mini-Cog: 2 items, 3 minutes; max score = 5; score of ≤ 3 = positive screen; assesses short-term verbal recall and visuospatial skills (includes clock drawing). Developed for and validated in primary care with minimal education or ethnicity bias. Very short administration time. One limitation is that the Mini-Cog is a less sensitive test for mild-to-moderate impairment.	[12,13,47–49] 1. Mini-Cog website: www.mini-cog.com
	MoCA (Montreal Cognitive Assessment): 12 items, 10 minutes (longer for severe impairment); max score = 30; score of < 26 = positive screen. Visuospatial/executive functioning (includes clock drawing); naming; attention; repetition; verbal fluency; abstraction; short-term verbal recall; orientation. Developed and validated for mild cognitive impairment and tests many domains. Educational and ethnicity bias exists.	2. MoCA website: www.mocatest.org
	SLUMS (Saint Louis University Mental Status Examination): 11 items, 5–7 minutes; max score = 30; score of < 27 with high school education = positive screen. Orientation; calculation; verbal fluency; short-term verbal recall; attention; visuospatial (includes clock drawing). Assesses multiple cognitive domains and no apparent educational bias. Primarily studied in VA populations.	3. SLUMS website: http://aging.slu.edu/index.php?page=saint-louis-university-mental-status-slums-exam
	AD8 (Eight-item Interview to Differentiate Aging and Dementia): 8 items, ~3 minutes; score of > 1 = positive screen. Assesses intra-individual change across a variety of cognitive domains. Developed as an informant interview; also can be administered to patient. Sensitivity > 84%; specificity > 80%. Can be completed in person or over the telephone.	[32]
Verbal pain reporting in those with dementia	Evidence indicates that many patients with dementia can reliably report pain, that is, that their pain reporting is consistent over time.	[18]
Need to identify suffering before treating pain	Chronic pain does not disappear; thus reporting the presence of pain is expected in all patients with chronic pain, regardless of cognitive function.	[14,44,50]
	Pain reporting should not automatically be equated with pain-related suffering.	
Use of PAINAD in those unable to report pain	This scale requires 5 minutes of observation during activity before scoring.	[38]

manages her own medications. She reports low back pain on most days, currently 8/10, but cannot describe her pain quality or precipitating factors. She reports pain alleviation with sitting or lying down. She is prescribed tramadol 50 mg two to three times per day as needed but is unable to report how many times she has taken it

over the past week. She cannot say whether it is helping her. Her pain worsened during physical therapy, so she stopped going after two or three sessions. Her daughters took over her finances when she moved to assisted living. They also report that the patient had not filled her medications properly for several months prior to moving into the facility and that she had stopped participating in the activities she once relished like gardening, playing Scrabble, and going out to dinner with friends, saying that she no longer enjoyed them.

Relevant Physical Examination

On exam, the patient is neatly dressed and appears comfortable. She has a sad affect and makes poor eye contact. She has mild thoracic kyphosis, no taut bands or trigger points (i.e., no myofascial dysfunction), and full/painless internal hip rotation bilaterally. Her strength is 5/5 in all extremities. Her sensory exam is normal. When asked to walk, she says that she has too much pain to do so. When the examiner offers her hand, she takes it and walks 15 feet without difficulty. When asked to walk farther, she grimaces, begins rubbing her back, and says that she needs to sit down because she is experiencing pain. Because of her advanced age and inability to provide sufficient pain-related detail during the history, a Montreal Cognitive Assessment exam is performed and reveals a score of 21/30.

Clinical Course

Based upon the history and physical examination, the following recommendations are made: 1) Neuropsychological testing to evaluate for possible dementia and/or possible depression; 2) discontinue tramadol; 3) begin acetaminophen 1,000 mg by mouth three times per day; 4) collaborate with the assisted living facility director and/or nursing staff to devise a supervised medication administration program to ensure this resident takes scheduled analgesia while retaining some self-efficacy and independence; 5) physical therapy with specific attention to reducing fear avoidance beliefs; 6) request that staff (nursing, aides, direct care workers) at assisted living facility record pain behavior observations, specifically activity engagement, grimacing during ambulation, and pain verbalizations to help track treatment outcomes; 7) activities staff to engage patient in a gardening group.

Approach to Management

As shown in the algorithm (Figure 1), the first step in the approach to this patient is to look for signs of possible dementia, and our patient exhibited several indicators. The patient in the case is 87 years old and presents with a report of refractory back pain. She does not have known dementia. The prevalence of dementia in adults aged 85 and older has been reported to be as high as 50% [5]. Therefore, clinical suspicion, particularly when even very subtle changes in memory, behavior, activity, or performing usual tasks are detected, supports screening for dementia in the oldest old (e.g., age 85 and older) [23,24]. While other guidelines do not recommend routine screening in those age 85 or older because of ineffective pharmacologic and/or nonpharmacologic dementia treatment, we recommend routine screening for those with CLBP because of the profound impact that dementia can have on the experience, expression, and treatment of pain.

A second clinical sign in our case that should lead to dementia screening, as shown in Figure 1, is the dearth of specifics provided during the pain history, with frequent corroboration from an informant when present. In the early stages of dementia, patients are often able to participate in conversation without any obvious indication of cognitive impairment as retained social skills can mask the presence of cognitive deficits [18]. However, underlying dementia may unmask itself as details surrounding the pain history are explored and the patient is unable to provide sufficient information. Cognitive impairment is so common in people 85 and over that providers would be well advised to encourage these patients to bring an advocate or family member who knows them to participate in the appointment [25]. At the same time, patients often defer to informants during the interview to compensate for immediate and remote memory difficulties. Some patients with dementia have the "head-turning sign"; that is, they repeatedly turn to their family member when the examiner asks them a question [26]. Our patient reports severe pain on most days, including at the time of the encounter, and relief with inactivity. She cannot provide additional details about her pain experience, and her daughters fill in the remainder of the pain history, particularly surrounding pain-related disability [27].

Difficulty with information processing is another sign of dementia. This can be uncovered during the physical examination as an inability to follow complex commands such as the directions for the Timed Up and Go Test that is used commonly to assess fall risk: stand up, walk to the line, turn around, walk back to the chair and sit down [28]. The patient may stand up and walk to the line, then stop and wait for further directions rather than completing the task. Difficulty with information processing makes it hard for a patient to independently participate in a pain management plan and typically requires family or caregiver support to follow through.

Our patient's withdrawal from daily activities represents another potential clue of an underlying dementia. She reports that she no longer enjoys long-standing hobbies, which she and her daughters attribute to persistent pain. Another pain-related explanation for disengagement from previously enjoyable daily activities could be

attributed to maladaptive pain coping, where fear avoidance beliefs lead to disengagement over time [29]. However, decreased engagement in usual activities and hobbies can also be indicative of dementia [30]. Research demonstrates that older adults across the spectrum of cognitive impairments demonstrate greater variability in performance of daily activities and increasing disability as they progress [31]. Impaired cognition can have a significant effect on medication compliance, as well as impact one's ability to participate in activities secondary to progressive loss of problem-solving skills and memory, which represents another clinical indicator to support a screen for dementia, per Figure 1. It also should be highlighted that such symptoms may be indicative of underlying depression that may be comorbid with dementia.

Our patient was screened for dementia as she exhibited several signs of dementia (listed in Figure 1). The expert interdisciplinary panel evaluated multiple screening tools for dementia and ultimately recommended three based upon their ease of use in the clinical setting, time to complete the measures, and their psychometric properties for accurately identifying dementia (Table 1). The suggested tools include the Mini-Cog, MoCA, and SLUMS [11–13]. The Eight-item Interview to Differentiate Aging and Dementia (AD8) is another brief dementia screening tool that can be administered either to an informant (preferred) or the patient. This instrument differs from the other screening measures in that it queries intra-individual change across a variety of cognitive domains [32]. Our patient completed the MoCA, for which she scored 21/30, indicating a positive screen for dementia (i.e., score < 26).

For patients who screen positive, and for patients who screen negative but for whom a high clinical suspicion of dementia remains, a referral for formal dementia evaluation may be warranted. A formal dementia evaluation starts with a standard clinical assessment for change in cognition, which can be performed either by a primary care provider or by a subspecialty dementia expert such as a neurologist, geriatrician, or geriatric psychiatrist. Reasons to pursue a more comprehensive workup by a subspecialty expert, including comprehensive assessment of cognition and mood by a neuropsychologist, include uncertainty about the diagnosis or type of dementia, early onset, rapid progression, and the need for a clear understanding of a patient's retained and lost cognitive abilities [24,33–35]. A negative screen in conjunction with a low index of suspicion for an underlying dementia supports continuing with comprehensive pain evaluation.

Per Figure 1, once a diagnosis of dementia is established, the next step is to determine whether the patient can report pain verbally. The majority of patients in the outpatient setting have retained ability to report pain. If the patient lacks this ability, a nonverbal pain assessment tool should be used to evaluate for pain-related behaviors. Although multiple validated tools are available, no tool is appropriate for every setting and current evidence does not support recommending one tool over another [36,37].

One of the most commonly used tools, the Pain Assessment in Advanced Dementia Scale (PAINAD), can be used to systematically evaluate pain-related behaviors [38]. The PAINAD requires five minutes of observation during activity and incorporates five indicators of discomfort rated on three levels: 0 = absent; 1 = present but not constant or severe; and 2 = severe/constant. The scoring was intended to help delineate pain severity. While the tool can be used to determine the presence or absence of pain behavior, its use to determine severity is not supported [39]. If the evaluation supports the presence of significant pain, then the patient should be evaluated to identify sources of pain so that appropriate treatments can be initiated. When a patient maintains the ability to report pain, similar to our patient, the observation of pain-related behaviors (for example, grimacing and vocalizations), especially during physical activity, should be integrated into the assessment to help validate the reported experience and inform the need for additional interventions.

Next, physical assessment is used to corroborate the patient's self-report. The patient exhibits reluctance to ambulate during the encounter but can be coaxed to do so with encouragement and support from the provider. Once up, she is able to ambulate a short distance without pain. When asked to ambulate further, she grimaces and braces her back, possibly indicating the presence of pain.

The absence of pain during ambulation, coupled with ongoing report of severe pain, represents inconsistency between patient self-report and pain-related behavior. This real-time patient observation while performing an activity reported to be associated with pain (i.e., if the patient says walking is too painful, observe her while she walks and ask her about her pain at that moment) provides valuable information about the pain history as well as allows comparison of self-reported pain and pain behaviors. The presence of such inconsistency (i.e., "a disconnect") represents another possible sign of dementia (see Figure 1). Moreover, pain behaviors and attention to pain may be exaggerated in patients with dementia [40]. Our patient appears to demonstrate fear avoidance beliefs with her initial reluctance to ambulate and yet ambulates when holding the hand of the examiner. Fear of pain may have contributed to our patient's decreased activity level and indicates a sign of maladaptive coping as well [29]. Management of maladaptive coping (e.g., fear-avoidance beliefs, catastrophizing) is addressed in a separate algorithm in this series [29].

When there are signs of physical or emotional suffering, such as in our patient during continued ambulation, then the source of the suffering must be clarified. If suffering is clearly related to pain, then appropriate pain interventions should be initiated. If suffering does not appear to be related to pain or as in the case of our patient not solely due to pain, then other causes should be sought and managed as well. Social isolation, depression, and fear of pain can all drive pain reporting. Often maladaptive coping with fear avoidance leads to inactivity and social isolation. Depression commonly coexists with pain in older adults and can also lead to withdrawal from activities and social isolation. Assessment of depression is addressed in another algorithm in the series [41]. Social isolation can be relieved with enrollment in a day program or a move to an assisted living facility. We recommended that our patient participate in a gardening group. Our patient exhibited signs of physical pain as well as fear avoidance in her reluctance to ambulate. This fear of causing pain resulted in her withdrawal from activities including previous physical therapy. Her management plan included both an analgesic trial with scheduled acetaminophen, physical therapy with specific attention to reducing fear avoidance beliefs, and participation in a gardening group to encourage a pleasurable activity that included socialization. We wish to highlight that while nursing, physical therapy, and activities therapy were provided on-site for our patient, these resources are not available at many assisted living facilities. For facilities that do not provide nursing care or rehabilitation services, the provider may wish to collaborate with home health for nursing care and physical therapy either through home health or outpatient-based services. Similarly, for engagement in a gardening group without on-site activities therapy, one might consider a nearby senior center or community volunteer organization.

If no sources of suffering are identified, then pain perseveration should be considered as a potential explanation of our patient's pain report, as indicated in Figure 1. Pain perseveration is the repeated reporting of pain that occurs without any sign of associated distress. Like other forms of perseveration, it occurs despite the absence or cessation of a stimulus and is a common characteristic of dementia [42]. Patients with pain perseveration will not exhibit nonverbal pain behaviors. Pain perseveration becomes the most likely explanation when the family or caregiver reports frequent pain report by a patient who does not appear to be in pain or when a patient who frequently talks about pain during the clinical encounter does not demonstrate objective signs of pain during the evaluation.

An empiric analgesic trial may or may not be helpful in distinguishing physical pain from pain perseveration [43]. Patients with chronic noncancer pain will not become pain-free with treatment [44]. They can be expected to continue to report pain, as can those with pain perseveration. As noted earlier, it is critical to ensure that persistent pain reporting is not associated with suffering before ascribing it to perseveration and to note whether the persistence of pain reporting indicates lack of analgesic efficacy. Thus an analgesic trial must be coupled with the other assessment approaches described in this paper. When pain perseveration is suspected to be the driver of pain reporting, the best intervention may be distraction, a strategy that can have analgesic benefits as well [45]. Another strategy providers and caregivers can use to manage patients with pain perseveration is to avoid asking about pain unless nonverbal indicators or other signs of pain-related suffering emerge.

Resolution of Case

Three months later, the patient and her daughters return for follow-up. Neuropsychological testing confirms a diagnosis of dementia secondary to probable Alzheimer's disease. The patient continues to benefit from acetaminophen 1,000 mg three times a day. Her supervised medication administration program ensured that she took the acetaminophen regularly, and that seemed to help increase participation in physical therapy. The physical therapy program was recently completed, and the patient is now able to walk at least 20 minutes at a time on most days of the week. The patients' daughters and staff have learned to avoid initiating conversations about pain, particularly in the absence of signs of suffering. Both daughters report rare spontaneous complaints of back pain when they visit. They have observed their mother to be much more animated, and they recently attended the assisted living's annual garden show that featured a display their mother had helped to create. The pain logs indicate that the assisted living staff has observed much less frequent pain reporting and pain behaviors.

Summary

Recognizing that the older adult with CLBP also has dementia can substantively shape the approach to pain management. Dementia impacts multiple aspects of pain evaluation and management, including pain reporting, treatment compliance, pain coping, treatment expectancy, and treatment response. The dearth of literature on nonpharmacological approaches to chronic pain treatment in those with dementia underscores the need for a rational clinical approach to this common conundrum. The approach outlined here has been informed by the clinical experience and expertise of geriatricians.

Dementia often goes unrecognized by many practitioners, resulting in overly pain-focused treatment. Chronic pain cannot be eradicated [44], but it can be managed. Patients with chronic pain always will have pain on which they can report. The job of the provider evaluating the older patient with a pain complaint is to identify whether the pain itself should be the focus of treatment (i.e., evidence that the patient is suffering from pain), whether dementia and dementia-related behaviors should be the focus of treatment, and whether

both pain and dementia require intervention. This approach truly reflects patient-centered care.

Key Points

1. Dementia impacts pain reporting (especially historical details), treatment compliance, pain coping, treatment expectancy, and treatment response.

2. Older adults with CLBP should be screened for dementia when:
   a. They're older than age 85 years.
   b. They have difficulty providing logical details during the history.
   c. There is self- or caregiver report of observed memory or functional decline.
   d. There is observed difficulty with information processing during the history and/or physical examination.
   e. There is discrepancy between self-reported pain/pain interference and observed pain interference in real time.

3. Pain report should not be equated automatically with pain-related suffering. Other causes of pain report may include perseveration, pain-related fear, or a substitute for other unmet needs. Ascertaining the cause will profoundly impact management.

4. Pain self-management in the older adult with dementia should involve the caregiver(s). When referring the older adult with CLBP and dementia for treatment (e.g., physical therapy), always encourage the caregiver to attend as a way to optimize compliance.

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Deconstructing Chronic Low Back Pain in the Older Adult—Step-by-Step Evidence and Expert-Based Recommendations for Evaluation and Treatment: Part XII: Leg Length Discrepancy

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Funding sources: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government.

Conflicts of interest: The authors have no conflicts of interest to report.

Abstract

Objective. To present the last in a 12-part series designed to deconstruct chronic low back pain (CLBP) in older adults. This article focuses on leg length discrepancy (LLD) and presents an algorithm outlining approaches to diagnosis and management of LLD in older adults, along with a representative clinical case.

Methods. Using a modified Delphi approach, the LLD evaluation and treatment algorithm was developed by a multidisciplinary expert panel representing expertise in physical therapy, geriatric medicine, and physical medicine and rehabilitation. The materials were subsequently refined through an iterative process of input from a primary care provider panel comprised of VA and non-VA providers. The clinical case was taken from one of the authors.

Results. We present an algorithm and illustrative clinical case to help guide the care of older adults with LLD, which can be an important contributor to CLBP. Firstline assessment includes referral to physical therapy or orthopedics, depending on the context of the LLD. A variety of nonsurgical interventions may ensue depending on the etiology of the LLD, including shoe inserts, customized shoes, manual therapy, or a combination.

Conclusions. To promote a patient-centered approach, providers should consider evaluating for leg length discrepancy when treating older adults with CLBP to help diminish pain and disability.

Key Words. Leg Length Discrepancy; Leg Length Inequality; Chronic Low Back Pain; Chronic Pain; Elderly; Older Adults

Introduction

One in three community–dwelling older adults experiences low back pain [1]. As part of the series to deconstruct chronic low back pain in older adults, we highlight leg length discrepancy (LLD; also referred to as leg length inequality) as a possible piece of the puzzle. Studies have shown that 60–90% of the general population is affected by LLD of 5 mm or more [2]. In 2004, Juhl and colleagues reported that 68 percent of 421 patients with low back pain had radiographically identified pelvic asymmetry, suggesting LLD [3]. While they commonly coexist, a causative association between LLD and CLBP has not been demonstrated in the context of high-quality studies [4–6]. It has been shown, however, that LLD is more common in those with CLBP than in those without CLBP [5,7]. In studies that focus specifically on sacroiliac joint syndrome as a cause of low back pain, LLD is an accepted contributor [6,8–12].

The magnitude of LLD that is clinically significant is debated. Some authors hold the view that LLD of less than 20 mm is clinically insignificant [13,14], but others suggest that any LLD is of clinical significance [2,7,8,15,16]. Despite the debate, inclusion of LLD in the evaluation of patients with low back pain is widely accepted [9,17] and, based on the collective experiences of the expert panel, we recommend screening for LLD in all older adults that present with CLBP. There are significant potential benefits for those who are diagnosed and treated for LLD, and the downside to screening is minimal, save for the negligible loss of time. Depending on the etiology of the LLD, intervention can be as simple as a heel lift.

There are two main classifications of LLD: structural and functional. Structural discrepancies can result from an actual anatomic shortening of one or more bones of the lower extremity from congenital, traumatic, or diseased origins. Surgical procedures such as total hip and knee replacements also can be responsible for acquired structural discrepancies and are an important consideration in older adults given the frequency at which these patients have comorbid knee and/or hip osteoarthritis and undergo total joint replacement [18–24]. Functional LLD, more common than structural LLD, may be due to altered mechanics of the lower body such as foot hyperpronation/supination, scoliosis, pelvic obliquity, muscle imbalance, poor trunk stabilization, genu varum, valgum, and/or genu recurvatum [18–20,25,26]. Both structural and functional LLD will lead to anatomical compensation and potential pain of the low back, hip, knee, and/or ankle as well as associated functional impairments.

We present a clinical case and offer an algorithm to assist in the evaluation and treatment of the older adult with LLD and CLBP.

Methods

A detailed description of the modified Delphi process used to create the algorithm (Figure 1) is provided in the series overview [27]. The expert panel team leader (MH) drafted the initial algorithm based upon a comprehensive review of the literature and his experience in clinical practice. The expert panel, which consisted of geriatricians, physical therapists, and a physiatrist, refined the algorithm then distributed it to the primary care panel for feedback, as described previously [27].

Case Presentation

Relevant History

The patient is an 84-year-old male who lives independently with his wife. He was referred to physical therapy by his primary care physician who had concerns about the patient's balance and potential for falls. The patient described the presence of low back pain as a constant nag and is concerned about his ability to stand up straight. It is affecting his ability to garden, specifically with his ability to get on and off the ground. He is not using an assistive device. He rates the pain intensity at 3–4/10 when standing and describes it as a constant aching along the lumbosacral junction with occasional radiation into the posterior thighs. The pain becomes progressively more intense the longer he stands in one place. He notices approximately 30 minutes of morning stiffness. Sitting or lying down relieves the pain. He denies change in his bowel or bladder habits, fever, trauma, weight loss, or recent cancer associated with the worsening of his pain. He has tried aspirin, ibuprofen, naproxen, and acetaminophen. He has also tried chiropractic care, without improvement in function or reduction of pain. A previous provider had recommended off-the-shelf heel lifts for both shoes to improve his balance.

Relevant Physical Examination

A thorough objective functional evaluation was completed by a physical therapist. The patient is awake, alert, and oriented x3. He is cooperative and in no apparent distress. Standing forward flexion, extension, right side bending, and right/left axial rotation all were painless. Left side bending was associated with low back pain. Ankle range of motion reveals a 10-degree deficit in dorsiflexion bilaterally. Quadriceps and foot dorsiflexion/plantar flexion strength are 5/5. Hip flexion strength is 5/5 bilaterally, hip extension 3/5 bilaterally, and hip abduction 4/5 bilaterally. Single-limb heel raises to test functional calf strength are normal [28]. Light touch in all dermatomes was normal. Deep tendon reflexes are intact and equal bilaterally. The unipedal stance test to measure postural stability (i.e., balance) is administered and the patient is able to stand on his right

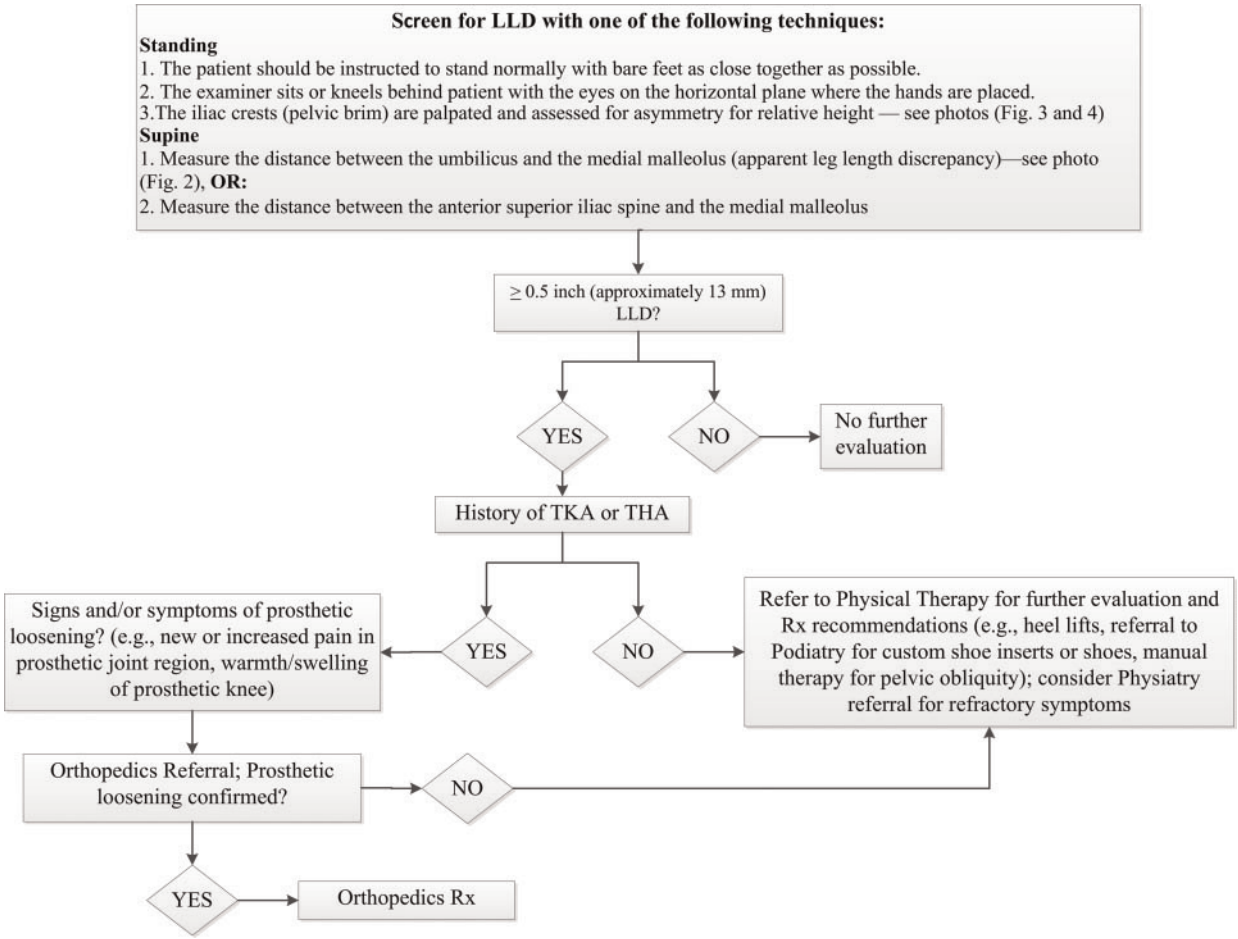

Figure 1 Algorithm for the evaluation and treatment of LLD in an older adult with CLBP.

leg for 25 seconds and on his left leg for 18 seconds, which is within normal limits for his age [29]. The left quadratus lumborum is tender to palpation. Measurement of leg length in supine position (from anterior superior iliac spine to medial malleolus) demonstrated the left leg to be 0.5 inches (approximately 13 mm) shorter than the right. When comparing the patient's iliac crest heights in the standing position, it was noted that the right side was higher than the left. Sacroiliac (SI) joint provocation tests (compression/distraction, FABER, thigh thrust), Scour test (for hip capsule), straight leg raise (for sciatica), and FAIR test (for piriformis pain) were all negative.

Clinical Course

Following evaluation by the physical therapist, the patient was educated on the diagnosis and treatment of LLD (Figure 1). The patient was very motivated to improve his low back pain and balance in order to maintain an active lifestyle. He was given a 7/16th-inch heel lift to be worn in the left shoe only. He started his first of eight physical therapy sessions to restore lower extremity strength, improve balance, and to ensure independence in a home balance and lumbar stabilization program. He also was assigned daily flexibility exercises to be performed each morning.

Approach to Management

Our algorithm suggesting approaches to screening and managing LLD is shown in Figure 1. Methods of assessing LLD include radiographic and direct and indirect assessment during physical examination. Radiographic measurements performed with the patient in either the supine or standing position are the most accurate [10,27], but radiography is expensive, not without risk (i.e., exposure to radiation), and may be time consuming [30]. Direct assessment of LLD during physical examination is performed with the patient supine and the distance between the anterior superior iliac spine and either the medial or lateral malleolus (identified by palpation) is measured [30,31]. While this method is clinically practical, it is less reliable than radiographic

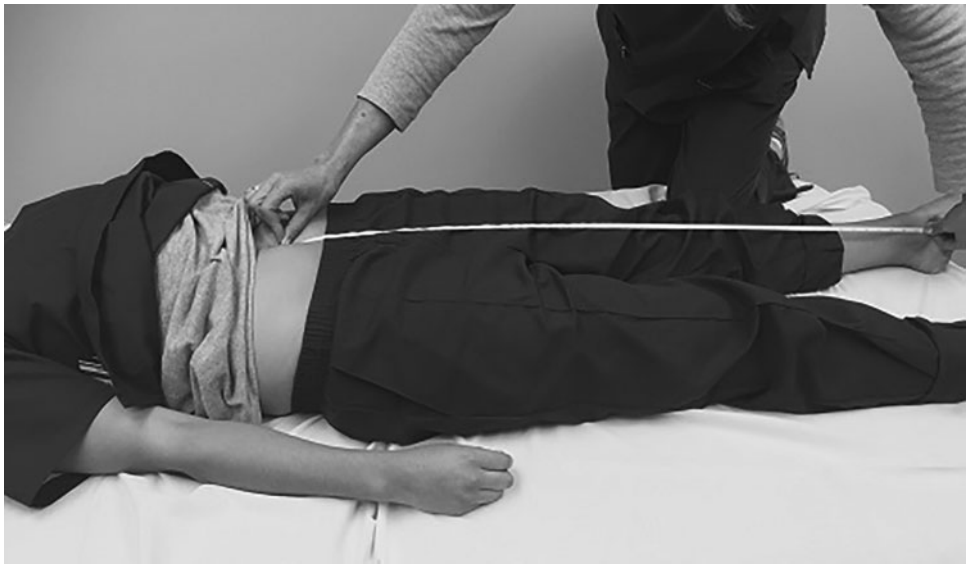

Figure 2 Screen for LLD: With the patient supine, the examiner uses a tape measure to assess the distance between the umbilicus and the medial malleolus.

measurement [32–34]. Direct assessment of apparent LLD during physical examination is performed with the patient supine and involves measuring from the umbilicus to the medial malleolus (Figure 2). Apparent LLD measurement removes the potential uncertainty of accurate identification of the anterior superior iliac spine [35]. Indirect assessment of potential LLD during physical examination is performed with the patient standing. While kneeling or squatting behind the patient, the clinician places their palms on the patient's left and right pelvic brim (iliac crests) and observes for the presence of symmetry (i.e., thumbs are even) or asymmetry (i.e., thumbs are uneven) [36]. This method is shown in Figures 3 and 4.

If at least 0.5 inches (approximately 13 mm) of LLD is suspected based on one of the above methods, further evaluation is warranted. When older adults have had prior total hip or knee arthroplasty and there are signs or symptoms of prosthetic loosening (e.g., new or increased pain in the prosthetic joint region and/or warmth/swelling in the case of a total knee arthroplasty), they should be evaluated by an orthopedic surgeon. If prosthetic loosening is identified, surgical treatment is warranted. If surgical assessment indicates no such loosening, patients should be referred to the physical therapist (PT), who may recommend heel lifts or orthoses or may employ manual techniques, depending on the cause. Depending on the outcome, the PT may further recommend referral to a podiatrist and/or physiatrist.

Results associated with shoe inserts vary widely [37]. Correction of pelvic obliquity appears to improve pain and functioning in patients with CLBP [38]. Relief of back pain has been documented in descriptive studies, case reports, and one small uncontrolled trial, but well-controlled trials are lacking [36,38–40]. Preliminary data by Golightly and colleagues demonstrated that heel lifts for those with LLD and CLBP are associated with significant reduction in pain and disability [41]. It should be highlighted that heel lifts and shoe inserts do not necessarily need to equalize the LLD completely to provide benefit. There appears to be consensus among authors that heel lifts should be implemented gradually and in small increments, especially for older adults [42–44]. This may be important for older patients with lifelong LLD as heel lifts could disrupt postural compensation and temporarily increase muscle soreness as the individual adapts to new position. Our patient was educated to ease into use for a few hours at a time. If an acute LLD is present following surgical procedure or trauma, heel lifts should be implemented quickly and without gradual implementation.

Practitioners should use their clinical judgement when selecting heel lifts, always weighing their risks and benefits. As noted earlier, potential benefits in patients with CLBP are reduction in pain and disability. Golightly and colleagues demonstrated in patients with CLBP and LLD substantial pain reduction (i.e., 75% less intense pain in the standing position) and clinically significant improvement in function as measured by the Modified Oswestry [41]. Potential risks include presetting the ankle in a plantar-flexed position that may predispose to ankle injury because of the relative instability of the lateral joint and weakness of the lateral ankle ligaments [45]. Increased callusing also may develop. In

Figure 3 Symmetry indicates that the patient does NOT have LLD.

Figure 4 Asymmetry indicates that the patient does have LLD.

addition, tightness/shortening of the Achilles and/or hamstring tendons may result from the preset plantarflexion. The patient's existing footwear also should be considered carefully, as well as whether a lift may cause excessive foot pressure. This may be relevant for those with diabetes mellitus or skin vulnerability related to other conditions.

The patient with LLD may benefit from a number of other interventions depending on the cause of the discrepancy, and treatment decisions should be determined collaboratively with the patient and be guided by their goals. Structural discrepancy related to prosthetic loosening requires orthopedic surgery evaluation and treatment as outlined in Figure 1. Functional LLD can be caused by alterations related to positioning of the foot (i.e., hyperpronation/supination), pelvic obliquity (e.g., related to muscular imbalance involving the quadratus lumborum, hamstrings, rectus femoris), and other muscle and/or joint imbalances (i.e., tightness or weakness), and treatment should be tailored accordingly. Some patients may benefit from custom shoe inserts or shoes that have been externally modified by an orthotist or a combination of shoe modification and manual therapy. It is recommended that clinicians with expertise in manual techniques treat patients with a goal of improving spine and hip mobility and that manual therapy should be combined with exercise [41]. To address any strength deficits in patients with CLBP, moderate- to high-intensity exercise is recommended. Progressive, low-intensity submaximal fitness and endurance activities also should be incorporated both for pain management and overall health promotion [41].

No matter what the intervention, a patient-centered treatment approach should drive decision-making, with careful consideration of risks, benefits, and costs. Patients should be reexamined four to six weeks following initiation of any new modified footwear to determine outcome of the intervention, assess skin integrity, and adjust the treatment plan if necessary. Similarly, careful follow-up during manual therapy or a multifaceted approach represents standard of care.

Resolution of Case

The patient has LLD, left leg shorter than right, and with correction there was some improvement in his constant back pain and, more importantly, function. The patient's LLD is considered functional. During his final physical

therapy visit, the patient reports 0/10 pain in his lower back while sitting and 3/10 with prolonged standing. The patient is able to demonstrate a safe independent floor-to-stand transfer. He has been educated about the importance of self-management, and he continues with his morning stretches. He is independent in all exercises and plans to continue to perform his daily early-morning stretching program and a lumbar stabilization and balance program three to four times per week. He maintains realistic treatment expectations, he has embraced self-management of his pain, and he is satisfied with his increased function. He was pleased with his decrease in pain from a constant state to only with prolonged standing.

Summary

Leg length discrepancies are associated with numerous postural alignment challenges that may lead to low back pain. The goal of the presented algorithm is to provide an evidence-based instrument to aid the clinician in a practical approach to evaluation and treatment. The case presented underscores that the older adult with CLBP may prioritize goals other than pain management per se, which include improving balance, reducing stiffness, and improving function. As with all older adults, utilization of a patient-centered approach is critical. We have developed this series of algorithms on CLBP to facilitate such an approach.

Key Points

1. Older adults with CLBP should be screened for leg length discrepancy (LLD) as a possible treatment target to reduce pain and, most importantly, to enhance function.
2. Leg length discrepancy can be structural or functional; both can cause postural compensations that contribute to pain and/or functional compromise.
3. Except in postsurgical patients, heel lifts should be implemented gradually and in small increments, especially for older adults.
4. Treatment of LLD should be tailored to the causative factors and the magnitude of the discrepancy and may include physical therapy to address muscular imbalance, a shoe lift or customized orthotics, and surgical intervention.

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Deconstructing Chronic Low Back Pain in Older Adults: Summary Recommendations

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Disclosure: This material is based on work supported by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, Rehabilitation Research and Development Service. The contents of this report do not represent the views of the Department of Veterans Affairs or the US government.

Conflicts of interest: Dr. Weiner has indicated that she has no conflicts of interest regarding the content of this article. Dr. Marcum is a consultant for Purdue Pharma. Dr. Rodriguez has indicated that he has no conflicts of interest regarding the content of this article.

In this issue of *Pain Medicine*, we publish the final algorithm in our 12-part series, "Deconstructing chronic low back pain in older adults—Step-by-step evidence and expert-based recommendations for evaluation and treatment" [1–12]. On behalf of the 42 interdisciplinary pain management experts and primary care providers that created the algorithms, we would like to recap the genesis of this series and the key learning points we hope that we communicated. A number of clinical observations coupled with evidence from the literature spurred us to action. Our clinical care of older adults with chronic low back pain (CLBP) continues to teach us that these patients are not simply a chronologically older version of younger patients with chronic pain. Many factors set older adults apart, including aging-associated vulnerabilities such as increased risk of mobility dysfunction and falls [13], polypharmacy [14], increased burden of asymptomatic degenerative pathology [15,16], social isolation [17], increased risk of dementia [18], restricted physiological reserves that enhance risk associated with invasive procedures [19], and multisource pain generation as the rule rather than the exception [20]. These facts underscore the need for a patient-centered approach when treating older adults with CLBP. As such an approach is taught uncommonly during medical training, we developed this series of articles funded by a Merit Review grant from the Veterans Health Administration Rehabilitation Research and Development Service. Contributors to the series have included interdisciplinary pain experts and primary care providers, with a goal of producing materials that are both evidence- and expert opinion–based, as well as feasible to implement in clinical practice.

We also were inspired to develop these decision support materials to address important gaps in the literature: 1) Deficiencies in imaging-directed assessment of LBP have been well highlighted [21–26], but alternative strategies that acknowledge the multifactorial contributors to pain and disability in older adults have not been proposed. An evidence base for many of the individual contributors to CLBP has been published, but it is scattered throughout multiple silos (e.g., rheumatology, pain medicine, psychiatry, orthopedics, neurosurgery, rehabilitation medicine, psychiatry, psychology). As a result, CLBP is typically referred to as a "nonspecific" condition and treatments are often generically prescribed. The library of algorithms that we have created has synthesized important scattered information in an effort to encourage providers to think more critically and specifically about this complex condition. 2) Existing evidence often has not been tailored to older adults, taking into account aging-related physiologic declines, the impact of comorbidities on treatment, or what treatments are readily available in clinical practice. Interdisciplinary pain management experts joined forces with practicing geriatricians to develop algorithms that were informed by principles of pain medicine and geriatric medicine. The resulting materials represent sound clinical practice

specifically for older adults with CLBP. 3) Referral of older adults to interdisciplinary pain programs and development of new and alternative treatments assume the failure of existing, evidence-based strategies. But, an inadequate evidence base exists for older adults. Future research will evaluate outcomes associated with our patient-centered treatment approach that is solidly grounded in the principles of gerontology, geriatric medicine, pain physiology, and pain medicine and create the evidence against which other new treatments can be benchmarked.

Underlying Model

Our patient-centered model of care asserts that CLBP is a geriatric syndrome rather than a nonspecific pain condition, that is, CLBP represents a final common pathway for the expression of numerous contributors *in addition to* degenerative disease of the lumbar spine (i.e., degenerative disc and facet disease), as shown in Figure 1. If degenerative disc and/or facet disease were the primary pain generators in older adults with CLBP, then virtually all older adults would have this condition [16]. And if anatomical stenosis of the lumbar spine were the only contributor to pain and disability in older adults with neurogenic claudication, an estimated 20% of older adults would require treatment for lumbar spinal stenosis [15]. Clearly these figures overestimate the magnitude of symptomatic degenerative lumbar spine disease in older adults.

Depicted in Figure 1 are several important concepts: 1) The majority of older adults with CLBP have more than one physical contributor to pain and disability [20]. Figure 1 identifies six such contributors—sacroiliac joint syndrome, leg length inequality, myofascial pain, fibromyalgia, hip osteoarthritis, and lumbar spinal stenosis. We focused in our series on these particular contributors because in our clinical experience, these conditions are those that are most common and most commonly overlooked or misdiagnosed in older adults with CLBP. 2) In keeping with pain pathophysiology, routine assessment of nonphysical factors that impact pain modulation also must be assessed and, not uncommonly, include dementia, depression, anxiety, maladaptive coping, and insomnia in older adults. These same conditions can independently contribute to disability and should be assessed routinely. 3) Leg pain often coexists with low back pain. Too often patients with CLBP and leg pain are assumed to have lumbar spinal stenosis as the pain generator, while in fact a number of conditions must be considered in the differential as shown in Figure 1. As noted above, asymptomatic lumbar spinal stenosis is not uncommon in older adults, thus a thorough history and physical examination should precede the ordering of imaging. The components that we recommend including when evaluating the older adult with CLBP ± leg pain, along with an approach to screening, are listed in Table 1. We have provided Table 2 as a summary of the algorithms created, their mode of contribution to CLBP, and the corresponding article in the series that describes each condition.

Pharmacological Pain Management: An Update

Since publication of the initial recommendations in this series, two key guidelines have been released of relevance to the treatment of CLBP in the older adult—the American Geriatrics Society 2015 Updated Beers Criteria for Potentially Inappropriate Medication Use in Older Adults [27] and the CDC Guideline for Prescribing Opioids for Chronic Pain [28]. For the Updated Beers Criteria, opioids were added as a medication class to avoid in older adults with a history of falls or fractures (excluding pain management due to recent fractures or joint replacement) [27]. Importantly, older adults receiving multiple central nervous system active medications should have their total exposure to these fall risk–increasing medications (e.g., anticonvulsants, antipsychotics, antipsychotics, antidepressants, benzodiazepines, other sedatives

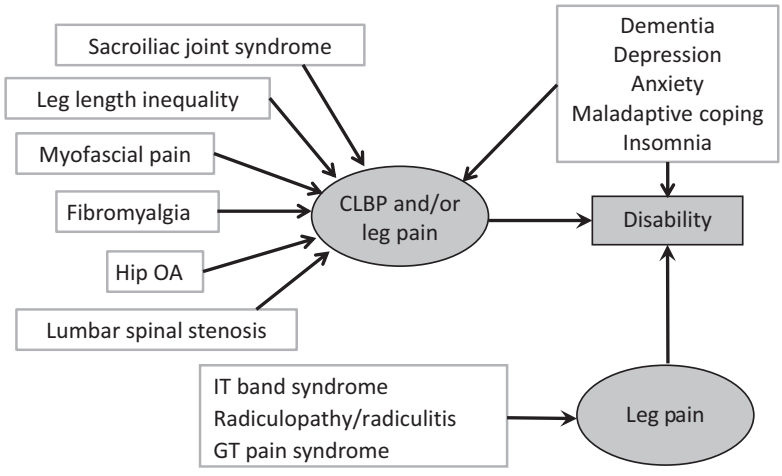

Figure 1 Chronic low back pain (CLBP) illustrated as a syndrome, a final common pathway for the expression of multiple contributors.

Table 1 Chronic low back ± leg pain in older adults: Essential clinical contributors screen

History

Question	Disorder screened	Interpretation/question source
Do you often feel like you hurt all over?	Fibromyalgia	Sensitivity of "hurt all over" validated in older adults with low back pain and depression, with American College of Rheumatology 1990 Criteria as comparison standard [32]. A positive response should prompt further evaluation for fibromyalgia, starting with the fibromyalgia self-report survey [33]. If positive, see fibromyalgia algorithm [3].
Over the last 2 weeks, how often have you been bothered by:		0 = not at all 1 = several days 2 = more than half the days 3 = nearly every day
1. Feeling nervous, anxious or on edge? 2. Not being able to stop or control worrying?	Generalized anxiety disorder (GAD)	Screen is positive for GAD if response to questions 1 + 2 ≥ 3: See anxiety algorithm [9].
3. Little interest or pleasure in doing things? 4. Feeling down, depressed, or hopeless?	Depression	Screen is positive for depression if response to questions 3 + 4 ≥ 3: See depression algorithm [4].
Note: Questions 1 and 2 are the GAD-2 [34]; questions 3 and 4 are the PHQ-2 [35]. Together, the 4 questions represent the PHQ-4 [36].		
Do you agree or disagree with the following statements?		If patient agrees with any of these statements, they may have maladaptive pain coping skills: See maladaptive coping algorithm [5].
1. It's not really safe for a person with my back problem to be physically active.	Fear-avoidance beliefs	
2. I feel that my back pain is terrible and it's never going to get any better.	Catastrophizing	
3. Due to my chronic back pain, I no longer engage in activities that are enjoyable and pleasant.	Behavioral disengagement	
Do you feel that you get good quality sleep?	Insomnia	If patient responds "no," see insomnia algorithm [7].
1. Does your back/buttocks/leg hurt when you are sitting? 2. Do you have pain in your buttocks/legs when standing or walking? 3. Does your pain lessen when you bend forward?	Lumbar spinal stenosis (LSS)	Supportive of lumbar spinal stenosis: • Absence of pain with sitting • Pain with standing/walking that is alleviated with forward lumbar flexion. If history is consistent with LSS, see lumbar spinal stenosis algorithm [6].
Do you have hip pain?	Hip osteoarthritis (OA)	If patient responds "yes," evaluate for hip OA: See hip exam below.

(continued)

Table 1 Continued

History

Question	Disorder screened	Interpretation/question source
Mini-Cog (3-word recall + clock drawing test [CDT])	Dementia	0 recalled words OR 1–2 recalled words + abnormal CDT suggests possible cognitive impairment: Should refer these patients for further evaluation; see dementia algorithm [11]. 3 recalled words OR 1–2 recalled words + normal CDT suggests lack of cognitive impairment: No further assessment needed unless high index of suspicion based on clinical data.

Physical examination

Physical exam component	Disorder screened	Associated findings/comments
Examination for taut bands/trigger points of erector spinae, quadratus lumborum, gluteus medius	Myofascial pain	*Active trigger points* are those that when palpated reproduce patient's spontaneously reported pain. See myofascial pain algorithm [2]. *Latent trigger points* are those that are not associated with spontaneously reported pain.
Internal hip rotation Hip flexion	Hip OA	Clinical criteria for hip OA include: 1) hip pain (patient report) AND EITHER: 2) internal hip rotation $< 15^\circ$ + hip flexion $\leq 15^\circ$ OR 3) internal hip rotation $\geq 15^\circ$ and painful + hip AM stiffness ≤ 60 minutes. If clinical criteria fulfilled, then confirm with hip x-ray. If hip OA present, refer to hip OA algorithm [1].
Standing: Assess symmetry of pelvic brim height. *Supine* (direct): Measure distance between anterior superior iliac spine and medial malleolus. *Supine* (apparent): Measure distance between umbilicus and medial malleolus.	Leg length discrepancy (LLD)	If LLD is found, refer to physical therapy for further assessment and treatment recommendations. See LLD algorithm [12].
Compression test Thigh thrust FABER Gaenslen test Distraction test	Sacroiliac joint (SIJ) syndrome	Perform physical exam maneuvers if supportive history for SIJ syndrome—i.e., pain in the SIJ region ± pain referred to the buttock, groin, or proximal leg—worse with transitions (e.g., sit to stand, stepping off curb) and without radicular symptoms. Presence of ≥ 3 positive physical exam tests provides optimal diagnostic sensitivity and specificity. If supportive history and physical examination findings, see SIJ syndrome algorithm [10].
Palpation over greater trochanter Ober's test	Greater trochanteric pain syndrome Iliotibial band syndrome	Evaluate in CLBP patients that also report lateral hip and/or thigh pain. See lateral hip/thigh pain algorithm for approach to differential diagnosis and treatment [8].

These should be performed on all older adults with chronic low back pain in whom red flags of serious underlying illness have been ruled out (e.g., fever, weight loss, suspicion of metastatic disease, rapidly progressive weakness, cauda equina syndrome, antecedent trauma, changed character or location of typical pain).

Table 2 Common contributors to chronic low back pain (CLBP)/leg pain in older adults

Condition	CLBP generator	Leg pain generator	Contributor to altered pain modulation	Reference Note: All articles are part of the series: Deconstructing chronic low back pain in the older—step-by-step evidence and expert-based recommendations for evaluation and treatment.
Hip osteoarthritis	X	X		Weiner DK, Fang M, Gentili A, et al. Deconstructing... Part I: Hip osteoarthritis. Pain Med 2015;16(5):886–97
Myofascial pain	X	X		Lisi AJ, Breuer P, Gallagher RM, et al. Deconstructing... Part II: Myofascial pain. Pain Med 2015;16(7):1282–9
Fibromyalgia syndrome	X	X	X	Fatemi G, Fang M, Breuer P, et al. Deconstructing... Part III: Fibromyalgia syndrome. Pain Med 2015;16(9):1709–19
Depression			X	Carley J, Karp JF, Gentili A, et al. Deconstructing... Part IV: Depression. Pain Med 2015;16(11):2098–108
Maladaptive coping			X	DiNapoli EA, Craine M, Dougherty P, et al. Deconstructing... Part V: Maladaptive coping. Pain Med 2016;17(1):64–73
Lumbar spinal stenosis	X	X		Fritz JM, Rundell SD, Dougherty P, et al. Deconstructing... Part VI: Lumbar spinal stenosis. Pain Med 2016;17(3):501–10.
Insomnia			X	Bramoweth AD, Renqvist JG, Germain A, et al. Deconstructing... Part VII: Insomnia. Pain Med 2016;17(5): 851–63
Lateral hip and thigh pain		X		Rho M, Camacho-Soto A, Cheng A, et al. Deconstructing... Part VIII: Lateral hip and thigh pain. Pain Med 2016;17(7):1249–60.
Anxiety			X	Karp JF, DiNapoli E, Wetherell J, et al. Deconstructing... Part IX: Anxiety. Pain Med 2016;17(8):1423–35
Sacroiliac joint syndrome	X	X		Polsunas PJ, Sowa G, Fritz JM, et al. Deconstructing... Part X: Sacroiliac joint syndrome. Pain Med 2016;17(9):1638–47
Dementia			X	Wright R, Malec M, Shega JW, et al. Deconstructing... Part XI: Dementia. Pain Med 2016;17(11):1993–2002
Leg length discrepancy	X	X		Havran M, Scholten JD, Breuer P, et al. Deconstructing... Part XII: Leg length discrepancy. Pain Med 2016;17(12):2230–37

Table 3 Key excerpts from the CDC guideline for prescribing opioids for chronic pain*

- When opioids are started, clinicians should prescribe the lowest effective dosage. Clinicians should use caution when prescribing opioids at any dosage, should carefully reassess evidence of individual benefits and risks when considering increasing dosage to 50 morphine milligram equivalents or more per day, and should avoid increasing dosage to 90 MME or more per day or carefully justify a decision to titrate dosage to 90 MME or more per day.
- Clinicians should evaluate benefits and harms with patients within one to four weeks of starting opioid therapy for chronic pain or of dose escalation. Clinicians should evaluate benefits and harms of continued therapy with patients every three months or more frequently. If benefits do not outweigh harms of continued opioid therapy, clinicians should optimize other therapies and work with patients to taper opioids to lower dosages or to taper and discontinue opioids.
- Clinicians should avoid prescribing opioid pain medication and benzodiazepines concurrently whenever possible.

*Not the entire guidelines.

and hypnotics) reduced. Clinicians should be aware of the occasional inclusion of Beers Criteria medications in our CLBP series, highlighting the fact that these medications are not absolutely contraindicated in older adults, but should be considered cautiously and in the context of the patient's comorbidities and other medications. The 2012 Beers Criteria did not include tricyclic antidepressants with a low side effect profile, that is, nortriptyline and desipramine. These medications have been added to the 2015 Beers Criteria. We have included nortriptyline as a second line consideration for the management of depression [4] and anxiety [9] and nortriptyline or desipramine as possibilities for the management of fibromyalgia [3].

We also wish to highlight that the 2015 Beers Criteria do not include celecoxib as a contraindicated drug [27], although the American Geriatrics Society 2009 Pain Guidelines recommend against its chronic use in older adults [29]. We advise that it be used cautiously and for short intervals if at all possible. If longer duration of use is deemed necessary, patients should have their blood pressure and renal function monitored carefully. It is important to note that all nonsteroidal anti-inflammatory drugs carry risk for kidney injury and should be used cautiously in older adults.

In addition to the main update to the Beers Criteria, for the first time a list of alternative medications to those included in the criteria was released [30]. For example, the list includes alternatives to tricyclic antidepressants for the treatment of neuropathic pain. Regular updates to the Beers Criteria are expected and should be anticipated by clinicians.

The US Centers for Disease Control guideline on prescribing opioids for chronic pain provides 12 recommendations about opioid prescribing for primary care clinicians [28]. Key excerpts from the guidelines are listed in Table 3. Overall, the guideline is intended to improve communication about the benefits and risks of opioids for chronic pain, improve the safety and effectiveness of pain treatment, and reduce risks associated with long-term opioid therapy [28]. We include opioids in the stepped care pharmacological pain management approach for several of the algorithms in our series—that is, hip osteoarthritis, lumbar spinal stenosis, and sacroiliac joint syndrome. All health care providers that prescribe opioids for older adults are obligated to educate them about all potential side effects, including the increased risk of falls and fractures, as highlighted in the 2015 Beers Criteria [27].

Conclusion

The age of precision medicine is upon us. This emerging approach to prevention and treatment of illness aims to tailor treatment according to "individual variability in genes, environment, and lifestyle for each person" [31]. In the case of CLBP, we advocate more precise treatment targeting by "unpacking the black box" of this syndrome to identify its amplifiers and generators. Unpacking is achieved through a focused history and physical, directed by the algorithms presented in this series. We advocate an analytic approach to what is often regarded as a medical monolith in order to discover specific remediable targets for treatment. Especially in chronic pain, the black box usually contains not only anatomic and physical generators, but also psychological amplifiers such as depression, anxiety, dysfunctional beliefs, and sleep disorders. Each patient represents a specific constellation of syndrome generators and amplifiers, so that identifying the patient-specific targets for treatment spares patients the burdens and risks of misdirected treatments. As the algorithms demonstrate, we do not completely reject steroid injections and opioid prescribing; we advocate discriminating use in circumstances in which safety concerns are outweighed by a likelihood of benefit.

Treatment targeted precisely to the identified components of the syndrome is likely to be both more effective and less fraught with risk. Because these targets for treatment interact through bidirectional cause-and-effect pathways to create and sustain the syndrome, effectively treating even a few components may implode the entire interdependent complex of symptoms.

We direct our approach primarily, but not exclusively, to primary care providers not only because chronic low back pain most often makes its first clinical appearance in primary care settings, but also because the object of treatment is not simply the pain, but the whole person, and primary care is the discipline generally held responsible for the whole person. The algorithms direct the

primary care provider in making judicious use of specialty providers and services, matching consultations to the identified targets for treatment in the particular patient. Our hope is to empower providers with useful tools for the gratifying work of disassembling the burdensome syndrome of CLBP safely and effectively.

Acknowledgment

The authors thank Dave Newman, MSW, MFA, for his thoughtful review of the manuscript.

Key Points

1. While older adults may identify CLBP as the cause of their disability, it is incumbent on the pain care provider to explore and validate all potential disability contributors (e.g., depression, anxiety, insomnia, dementia). Failure to do so may result in patient harm and/or misappropriation of health care resources.
2. We propose that CLBP should be approached not as a "nonspecific" condition but as a multifactorial syndrome comprised of a number of conditions outside of the lumbar skeleton. Degenerative disease of the lumbar spine should be considered the weakest link, but not the sole treatment target.
3. Comprehensive assessment of all factors contributing to pain and disability should guide treatment prescribing. Precise and comprehensive treatment targeting is the most rational approach to optimizing treatment outcomes and avoiding exposure of older adults to potentially dangerous interventions such as spine surgery and high-dose opioids.
4. Essential constructs to evaluate in the older adult with CLBP include dementia, depression, anxiety, maladaptive coping, insomnia, fibromyalgia, hip osteoarthritis, sacroiliac joint (SIJ) syndrome, myofascial pain (MP), and leg length discrepancy.
5. When the older adult with CLBP has leg pain, in addition to being evaluated for the above, the patient also should be evaluated for lumbar spinal stenosis, radiculopathy, iliotibial band pain, and greater trochanteric pain syndrome (the latter two when pain is on the lateral hip/thigh). Fibromyalgia, hip osteoarthritis, SIJ syndrome, and MP can also be associated with leg pain.
6. Nonpharmacological approaches to treatment, including thorough education and instruction in pain self-management techniques, should form the basis of treatment.
7. For older adults with dementia, a caregiver should be involved at all stages of evaluation and treatment.

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