Current Pain and Headache Reports (2019) 23: 23
https://doi.org/10.1007/s11916-019-0757-1

OTHER PAIN (A. KAYE AND N. VADIVELU, SECTION EDITORS)

Low Back Pain, a Comprehensive Review: Pathophysiology, Diagnosis, and Treatment

Ivan Urits[1] · Aaron Burshtein[2] · Medha Sharma[1] · Lauren Testa[1] · Peter A. Gold[2] · Vwaire Orhurhu[1] · Omar Viswanath[3,4,5] · Mark R. Jones[1] · Moises A. Sidransky[6] · Boris Spektor[7] · Alan D. Kaye[8]

Published online: 11 March 2019

Abstract

Purpose of Review Low back pain encompasses three distinct sources: axial lumbosacral, radicular, and referred pain. Annually, the prevalence of low back pain in the general US adult population is 10–30%, and the lifetime prevalence of US adults is as high as 65–80%.

Recent Findings Patient history, physical exam, and diagnostic testing are important components to accurate diagnosis and identification of patient pathophysiology. Etiologies of low back pain include myofascial pain, facet joint pain, sacroiliac joint pain, discogenic pain, spinal stenosis, and failed back surgery. In chronic back pain patients, a multidisciplinary, logical approach to treatment is most effective and can include multimodal medical, psychological, physical, and interventional approaches.

Summary Low back pain is a difficult condition to effectively treat and continues to affect millions of Americans every year. In the current investigation, we present a comprehensive review of low back pain and discuss associated pathophysiology, diagnosis, and treatment.

Keywords Low back pain · Axial low back pain · Referred low back pain · Radiculopathy · Lumbosacral pain

This article is part of the Topical Collection on *Other Pain*

✉ Ivan Urits
iurits@bidmc.harvard.edu

[1] Department of Anesthesia, Critical Care, and Pain Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School, 330 Brookline Ave, Boston, MA 02215, USA

[2] Department of Orthopedic Surgery, Hofstra-Northwell Health System, Great Neck, NY 11021, USA

[3] Valley Anesthesiology and Pain Consultants, Phoenix, AZ, USA

[4] Department of Anesthesiology, University of Arizona College of Medicine-Phoenix, Phoenix, AZ, USA

[5] Department of Anesthesiology, Creighton University School of Medicine, Omaha, NE, USA

[6] Department of Anesthesiology, University of Arizona College of Medicine-Phoenix, Tyler, TX, USA

[7] Department of Anesthesiology, Emory School of Medicine, Atlanta, GA 30308, USA

[8] Department of Anesthesiology, Louisiana State University Health Science Center, New Orleans, LA 70112, USA

Overview of Low Back Pain

Low back pain encompasses three distinct sources of pain: axial lumbosacral, radicular, and referred pain [1••]. Axial lumbosacral back pain refers to pain in the lumbar, or L1-5 vertebral region, and sacral spine, or S1 to sacrococcygeal junction region [1••]. Radicular leg pain travels into an extremity along a dermatomal distribution secondary to nerve or dorsal root ganglion irritation [1••]. Referred pain spreads to a region remote from its source but along a non-dermatomal trajectory [1••].

Pain in these three locations is relatively common, both in the USA and worldwide. Overall, low back pain is the fifth most common reason for visiting a US doctor [2]. In the last 3 months alone, approximately 25% of adults in the USA experienced low back pain for at least 24 h [3]. Annually, the prevalence of low back pain in the general US adult population is 10–30% [2], and the lifetime prevalence of US adults is as high as 65–80% [4].

Low back pain is not only common, but also holds a significant cost and health care utilization burden in a country where rate of health care expenditures is skyrocketing in

relation to GDP growth and overutilization is a major concern. In 1998, total direct health care costs attributable to low back pain in the USA were $26.3 billion [5]. Apart from direct monetary health care expenditures, low back pain leads to significant opportunity cost as well; the 2010 Global Burden of Disease study identified low back pain as the leading contributor to disability and work days lost [6••].

In addition to being stratified by location of pain, the problem of low back pain can also be segmented based on chronicity into acute (< 6 weeks), subacute (6–12 weeks), and chronic (> 12 weeks) low back pain [2, 7]. While the majority of non-chronic patients are acute with pain self-limited to 6 weeks or less, 10–40% of patients develop symptoms lasting over 6 weeks [2]. Acute and subacute low back pain patients are managed differently from chronic patients.

In the typical treatment paradigm, acute and subacute patients are first assessed for "red flags," indicating patients with more serious etiology who need further evaluation [2]. If there are no red flags, physicians may proceed to provide a patient education comprising of the general, non-specific etiology of their pain, favorable prognosis, likelihood of similar recurrences in most people, and reassurance to expect a favorable course. Physicians should encourage patient self-management, including minimizing bedrest, remaining active, and returning to work and normal activity as soon as possible [8]. Judicious short-term application of heat through pads or blankets is better supported in the literature than lumbar braces or cold packs [9]. Additionally, short-term application of a capsicum-based topical showed analgesia relative to placebo within the first week of use [10]. In terms of pharmacotherapy, acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), and muscle relaxants are first-line medicinal therapies that aim to minimize side effects [11]. Patients should be instructed to avoid opioids if possible, unless pain is severe in intensity and they are unresponsive to more conservative medications [11]. Patients with persistent pain beyond 1 month should be re-evaluated [12].

"Yellow flags" are risk factors for development of chronicity [13]. Psychosocial and emotional factors are strong predictors of low back pain chronicity [14]. When present, physicians may consider enhanced patient education and earlier-focused cognitive behavioral therapy approaches to target the following: anxiety and depression, catastrophizing, fear avoidance behavior (e.g., significant worry about aggravating low back pain by engaging in normal activities), passive coping strategies, job dissatisfaction, higher levels of disability, somatization, and disputed compensation claims [14].

Chronic back pain is defined as low back pain for over 12 weeks, with up to one third of all low back patients report moderate-intensity low back pain ongoing a year after an acute episode [2]. In chronic back pain patients, a multidisciplinary, logical approach to treatment is most effective, with medical, psychological, physical, and interventional approaches as outlined below [15].

Patient Assessment

History

When evaluating a patient with lower back symptoms, it may not be possible to define a precise cause, because up to 85% of patients will be diagnosed with non-specific lower back pain upon primary evaluation [16, 17]. Therefore, it is important to look for evidence of specific etiologies of back pain to adequately diagnose the type of lower back pain.

There are certain specific characteristics of lower back pain that should be elucidated. The duration of symptoms stratifies the patient into a particular group of acute, subacute, or chronic low back pain to help guide decision-making [16, 18•]. The location of pain and radiation, either axial or radicular low back pain, is also important to discern and clarify [19]. The severity of pain can be collected with a specific scale (i.e., visual analog scale or numerical rating scale score) and used to determine the current, average, worst, and best scores [4, 18•, 19]. To further develop a sense of the pain, characteristics such as burning, lancinating, aching, numbness, and electric shock sensations are of significance to ascertain [18•]. The circumstances that initiated the pain, if any, are also of note. Specifically, if the patient sustained a motor vehicle accident, understanding whether the patient was a driver or passenger, airbag deployment, site of impact, and type of vehicles involved may indicate the type of pain that may have developed. Moreover, alleviating and provoking factors, such as sitting, standing, walking, and laying down, help clarify the differential diagnosis [18•]. Likewise, the documentation of a previous history of similar episodes of lower back pain can clarify an intermittent recurrent nature of the symptoms [19]. Prior evaluation and pain management, such as noting previous diagnostic studies and interventions, is helpful in guiding future management [19]. Temporal changes in presentation indicate a progression of symptoms and can be used to assess symptom development [19]. Lastly, the functionality of the patient with the pain during work and activities of daily living may affect the degree of treatment [18•].

Additionally, the initial evaluation of a patient with lower back pain should include screening questions about concerning constitutional symptoms (red flags) that point to a potential progressive or unstable cause for pain such as cancer, infection, trauma, and neurologic compromise [20]. Among patients who present with lower back pain less than 1% will have a serious systemic etiology [17].

Having a history of cancer (excluding non-melanoma skin cancers) is the strongest risk factor for back pain from bone metastasis [18•, 19, 21]. Bone metastasizing cancers include

breast, lung, renal cell, and prostate cancers [17]. Special attention is needed on the type of cancer, location, and treatments of the cancer. Any indications by the patient, such as recent weight loss, worsening pain at night, and inability to attain relief at rest or in the supine position, are symptoms often seen in patients with spinal tumors or metastasis [16, 18•].

A history of infections stemming from a recent fever, malaise, spinal injection, epidural catheter placement, IV drug use, immunosuppression, and other concurrent infections specifies a potential cause of lower back pain and should be taken into account for treatment [16, 17].

Comparably, a history of recent or substantial trauma is of critical importance to discern [18•, 20]. Understanding the mechanism of traumatic injury can be used to treat ligamentous instability or fracture.

Lastly, neurologic compromise can be seen in patients with bladder or bowel control changes as spinal cord or cauda equina compression may lead to urinary retention followed by urinary and/or fecal incontinence [16, 18•, 20]. Any recent numbness, gait instability, or weakness in the legs are symptoms that may develop. Specifically, bilateral leg numbness with saddle anesthesia is indicative of cauda equina syndrome [16].

Patients with lower back pain should also be evaluated for social or psychological distress [18•]. An assessment of a history of substance abuse, disability compensation, work status, and symptoms of depression is an indicator of such psychosocial distress [20]. Likewise, psychiatric comorbid conditions, somatization, and/or maladaptive coping strategies are all associated with poorer outcomes in patients with low back pain and should also be established [18]. Examples of prefabricated questioners to assess some of these important aspects of pain management include the opioid risk tool (ORT), PHQ9 Questionnaire, and the current opioid misuse measure (COMM) score.

Physical Exam

A physical evaluation, either brief or extensive, is an essential part of the management of low back pain [20]. A general physical exam offers pertinent patient data, including vital signs, ambulation status (assistive devices, mobility, and gait), appearance, behavior, signs of distress, skin, mood and affect, judgement, and thought process [17].

A neurological examination should also be conducted, consisting of motor strength in the back and lower extremities, sensation, deep tendon reflex testing, and upper motor neuron reflexes [17, 19]. This will help the practitioner diagnose and/or rule out more specific causes of lower back pain such as spinal cord, nerve root, and peripheral nerve pathology.

Additional parts of the physical evaluation are an inspection of the thoracolumbar spine, palpation over the spinous process, range of motion movements, and tests for specific disorders. The initial inspection via evaluation of the thoracolumbar spine provides information on posture and alignment [22]. This includes a special focus on abnormal kyphosis, lordosis, or scoliosis. In addition, skin evaluation should focus on rashes, scars, swelling, and signs of trauma or inflammation [17].

Furthermore, palpation over the spinous processes can reveal localized tenderness which is seen in patients with abscess, epidural tumor, and vertebral compression fractures [22]. Special consideration should be given to tenderness in the paraspinal region, which can be seen in patients with facet arthropathy and myofascial-related pain [23]. Light palpation will help detect allodynia or hyperalgesia which will typically indicate neuropathic pain.

Pain related to range of motion movements and/or limitations provides additional information regarding the type of lower back pain [20]. Normal range of motion of the thoracolumbar spine is 90° of forward flexion, 30° of back extension, 60° of lateral rotation, and 25° of lateral flexion [24]. Pain that is provoked by lateral rotation and back extension is suggestive of facet arthropathy [25]. Pain that is provoked by forward flexion is suggestive of discogenic or vertebral body-related pathology because flexion of the lumbar spine causes axial loading [26]. However, pain on range of motion is not specific and may occur due to other causes.

The physical also consists of various tests for specified disorders. The Patrick's test evaluates hip and sacroiliac pathology, both of which are associated with lower back pain. With the patient in a supine position, the examiner should passively flex, abduct, and externally rotate the hip. Pain in the groin area suggests hip pathology, while pain in the back suggests sacroiliac joint pathology [27••].

Additionally, a straight leg raise test should be conducted to determine any involvement of the lumbar nerve roots or hamstring muscle in the lower back pain [22]. With the patient in a supine position, the examiner should lift the patient's leg at the heel while the knee is straight. The hip should be flexed to an angle of 70° to 90°. This test produces tension in the lumbar nerve roots. A positive straight leg raise reproduces radicular pain experienced by the patient radiating from his lower back or hip down to his ankle (the pain must occur in a radicular pattern). If pain remains localized to the posterior thigh area, it is most likely cause by tension on the hamstrings [16, 18•].

A Gaenslen's test should also be done to identify lower back pain related to the sacroiliac joints [28••]. With the patient in the supine position, the hip joint should be flexed maximally on one side and the opposite hip joint extended, stressing both sacroiliac joints simultaneously. This can be achieved by having the patient lift his knee to push toward the chest while the other leg is allowed to fall

over the side of an examination table, and is pushed toward the floor, flexing both sacroiliac joints. The test is considered positive if pain related to the SI joint is reproduced by this maneuver [28••].

Diagnostic Testing

Diagnostic testing is seldom required in the course of lower back pain treatment [18•, 20]. Laboratory studies are rarely needed; yet, patients with suspected malignancy or infection can be tested with ERS and/or CRP, in addition to plain radiographs, to determine the need for advanced imaging [18•, 19]. Nonetheless, electrodiagnostic testing, consisting of electromyography (EMG) and nerve conduction velocity (NCV) testing, can help differentiate chronic from acute radiculopathy, localize the pathologic lesion, and in determining whether the radiologic abnormalities observed are the likely source of patient symptoms [18•].

Imaging studies are also only performed in certain circumstances [20]. Most of the patients with lower back pain of less than 4 weeks duration do not require imaging [19]. Imaging should only be performed when severe or progressive neurologic deficits are present or when serious neurologic disease is highly suspected (red flags) [19]. Patients with signs and symptoms or spinal stenosis and radiculopathy should only receive imaging if they are good candidates for surgery or minimally invasive interventions.

Imaging studies consist of either X-rays and/or advanced imaging. When there is a failure of medically directed conservative care of the lower back pain, and after making a decision to obtain imaging, the examiner should start with weight-bearing radiographs of the lumbar spine (AP and lateral) [19]. Advanced imaging, computed tomography (CT), or magnetic resonance imaging (MRI) are helpful if radiographs are not explanatory of unremitting lower back pain or there is substantial clinical suspicion for an underlying systemic disease, such as red flag signs [17, 19, 29]. MRI without contrast is generally considered the best initial test for most patients with low back pain who require advanced imaging. MRI with gadolinium allows the distinction of scar from disc in patient with prior back surgery [30]. In patients who require advanced imaging but cannot have an MRI, a CT scan is usually the next step.

Etiology of Low Back Pain

The etiology of lower back pain can often be differentiated based on a patient's history, physical exam and, in some cases, imaging. Myofascial pain is a commonly seen musculoskeletal complaint, especially after trauma or repetitive motion injury [23]. Myofascial pain is characterized by the presence of myofascial trigger points that are located in fascia, tendons, and/or muscle which, when triggered, result in a symptomatic pain response [31–34]. Patients will typically complain of paraspinal muscle discomfort and pain can radiate to the buttocks and thighs [16]. Physical examination may reveal localized, tender spots in a taut band with patient pain recognition, referred pain on palpation, and decreased range of motion [17, 33]. When inserting a needle or snapping a trigger point, a twitch response can be provoked [23].

Facet-mediated pain is a result of a multifactorial process associated with degeneration of the intervertebral discs that leads to lumbar facet joint degeneration [25]. Pain can be caused by osteoarthritis of the facet joints or by stress within the facet joint capsule [35]. Pain is often described by patients as a deep and aching sensation with unilateral or bilateral distribution. Occasionally, radiation to one or both buttocks, groins, and/or thighs can be present, but typically stops above the knee [26]. Factors that can exacerbate this facet-mediated pain include psychosocial stressors, increased or decreased physical activity, lumbar extension with or without rotation, and prolonged standing or sitting [25, 26]. Physical exam will often show pain on extension, lateral bending, and paraspinal palpation [36]. Imaging studies may be helpful in further identifying the pathology associated with facet-mediated pain [18•, 37, 38••].

Another common cause of low back pain is discogenic pain. According to Comer (2009), 39% of causes of lower back pain can be attributed to the intervertebral disc [36]. Internal disc disruption is primarily caused by degradation of the disc and its nuclear components and can be complicated by development of radial fissures that extend from the nucleus into the annulus [39]. The typical patient history is of pain in the center of their lower back with minimal radiation; however, if radiation is present, it typically locates to the buttocks or thighs [26]. This pain is commonly described as a deep, dull ache. Patients will often report that pain improves with standing and lying flat and may be reduced with extension [18•]. The pain is usually noted to worsen with sitting, driving, lumbar flexion, bending, twisting, Valsalva maneuver, and coughing [40]. A higher incidence of discogenic lower back pain occurs in patients who are obese and smoke tobacco products [26, 41]. In addition, patients with sedentary jobs that require prolonged sitting and patients with physical jobs that require lifting and vibration exposure have also been found to have a higher incidence of disease [42]. To aid in diagnosis, MRI can be performed to show disk degeneration [29, 40, 43]. Common findings associated with discogenic pain on MRI include high intensity signal in the posterior annulus on T2-weighted images, known as high intensity zone, bulging or protruding discs, and decreased disc signal intensity on T2-weighted images

suggesting dehydration [39]. Goals of management of discogenic pain should include improvement in pain threshold and improvement in function [20].

After one or more spinal surgeries, patients are at risk of developing persistent or recurring low back pain with or without radicular symptoms, termed lumbar post-laminectomy syndrome [44]. According to Waguespack (2002), the incidence of lumbar post-laminectomy syndrome is between 10 and 40% [45]. The etiology of lumbar post-laminectomy syndrome is based on preoperative, intraoperative, and postoperative factors. Preoperative risk factors include patients with a history of anxiety, depression, and poor coping strategies [46]. Additionally, patient's perusing litigation and workers compensation are at increased risk of developing lumbar post-laminectomy syndrome [45]. Intraoperative factors include poor surgical technique, surgery at the incorrect level or site of the spine, and the inability to achieve the anticipated surgical goal [47]. Postoperative factors include surgical complications, the rate of disease progression, epidural fibrosis, new instability, and the development of myofascial pain syndrome [48].

Spinal stenosis is a condition in which degenerative changes of the lumbar spine lead to decreased available space for neural and vascular elements. Symptoms of lumbar spinal stenosis include gluteal and lower extremity pain, and/or fatigue that may or may not occur in conjunction with lower back pain [49]. There are several provocative and palliative features. Provocative features include upright exercise such as walking and positional changes such as lumbar extension producing neurogenic claudication symptoms. Palliative features include symptom relief with rest, sitting, and lumbar flexion [49]. In patients with a history and physical examination consistent with lumbar spinal stenosis, an MRI is suggested as the most appropriate test to evaluate for the presence of spinal canal narrowing or nerve root impingement [22, 50].

Sacroiliac joint pain typically occurs in the lower back or upper buttock overlying the joint [51]. The sacroiliac joint itself is a diarthrodial synovial joint with profuse innervation and thus has the capability of being a source of lower back pain [51]. Currently, there are no historical, physical, or radiologic features to provide definitive diagnosis of sacroiliac joint pain [28••]. However, several physical exam findings are suggestive of sacroiliac joint pain such as pain to palpation directly over the sacroiliac joint. The Patrick's and Gaenslen's test may also be used to clinically reproduce the pain [52]. There is moderate evidence for the use of diagnostic fluoroscopic CT-guided intra-articular joint injection, and lateral branch blocks may provide therapeutic pain relief in some patients [53, 54]. Massage may be of benefit in relaxing strained or spasmed muscles associated with the sacroiliac joint.

Some of the rarer causes of axial spine pain, with or without radiation, to also consider in the differential diagnosis of low back pain include fibromyalgia, piriformis syndrome, hip osteoarthritis, tumor, infection, aortic aneurysm, sickle cell crisis, and retroperitoneal mass, among many others [16, 18•, 21].

Treatment for Low Back Pain

Multidisciplinary Approach to Treatment

Lower back pain management varies from person to person, as not all patients respond to the same treatment approach, and no single intervention is generally completely effective for all patients. Consequently, limited trials of one or more interventions guided by evidence and effectiveness are utilized to manage the pain, while aiming to decrease overall costs. Pertinent courses of care include pharmacological treatments, psychological treatments, physical and rehabilitation treatments, complementary and alternative medicine approaches, and minimally invasive percutaneous approaches. Figure 1 represents an algorithmic approach to the appropriate treatment plan for patients with low back pain.

Pharmacologic treatments are fundamental for both acute and chronic lower back pain. Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown effective for short-term relief [16, 18•, 55]. Acetaminophen, for acute pain, does not have a clear difference in analgesia at dosages up to 4 g/day as compared to NSAIDs. However, regarding chronic lower back pain, acetaminophen is slightly inferior to NSAIDs for pain relief [18•]. Acetaminophen's benefits include favorable safety profile and low cost; yet, it has uncertain clinical significance of generally asymptomatic aminotransferase elevations above 4 g/day [19] and its use should be cautioned with other acetaminophen-containing drugs.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are also used for acute and chronic low back pain, and non-selective and COX-2 selective NSAIDs have been shown to be superior to placebo with no clear difference in efficacy between NSAIDs [20, 55]. Use of NSAIDs are cautioned with regard to renal, cardiovascular, and gastrointestinal systemic side effects, and use of the lowest effective dose for shortest duration possible is recommended [19, 20].

Skeletal muscle relaxants have also been shown to be effective for acute lower back pain. Short-term studies, of 2-week duration, show analgesia superior to placebo, yet with no clear difference between specific muscle relaxants [18•, 20]. The primary associated side effects of skeletal muscle relaxant use are the central nervous system (CNS) sedation and risk for falls [18•]. One particular relaxant, carisoprodol,

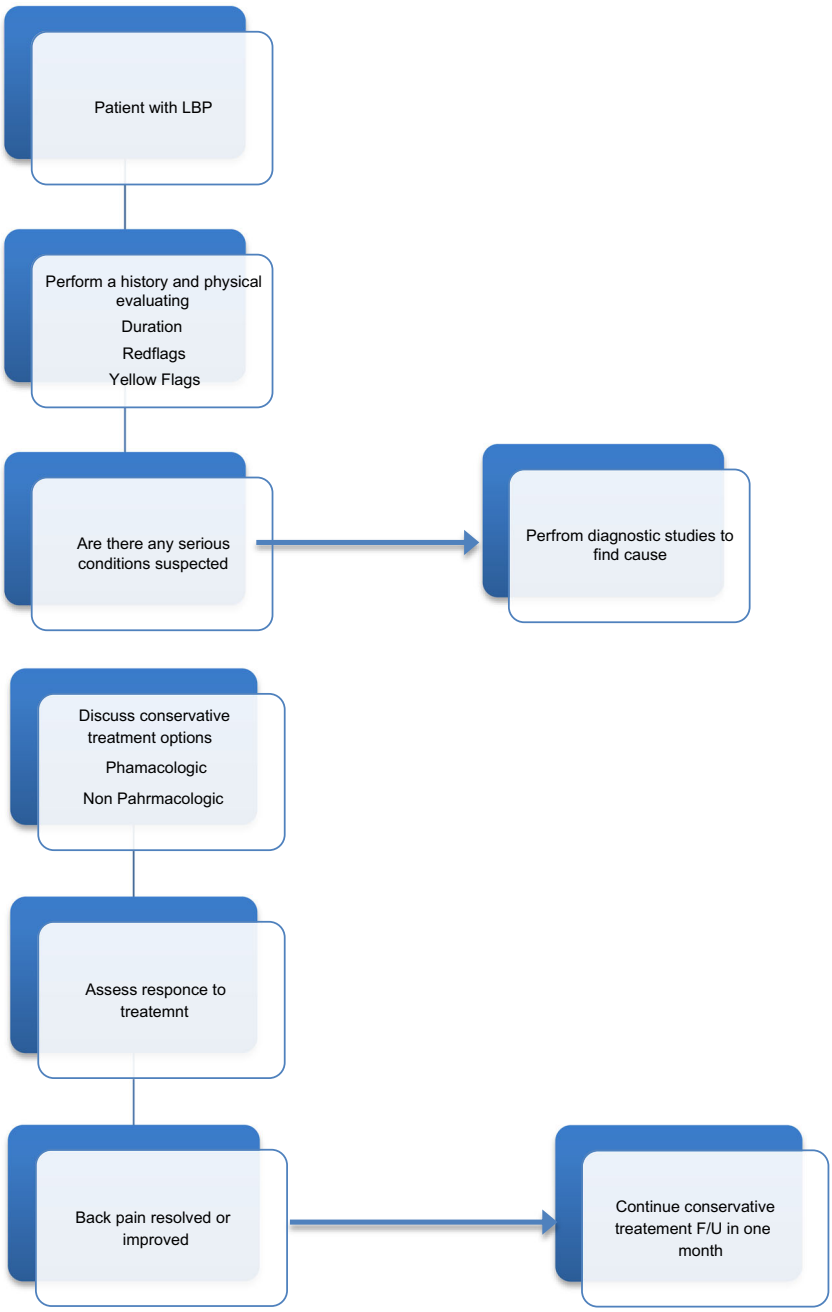

Fig. 1 Diagnostic and therapeutic approach to the patient with low back pain

should be used with caution due to its metabolism to meprobamate, a sedating and potentially addictive barbiturate [19].

Tramadol and more potent opioids should be considered judiciously and only for severe, disabling pain that cannot be controlled with the aforementioned options [19]. These medications should be used in a time-limited course with re-evaluation of analgesic efficacy, improved activity, adverse effects, and aberrant behavior (4A's) [19]. Additional caution should be exercised in patients at risk for addiction or aberrant behavior (personal or family history of addiction, poorly controlled psychological comorbidity, sexual abuse history, young age < 45) [18•]. While tramadol has shown limited analgesia with mild functional improvement for chronic low back pain, potent opioids have shown significant analgesia and improved function at 3 and 6 months duration in randomized trials [56].

Tricyclic antidepressant (TCA) use has shown beneficial effects for lower back pain treatment [16, 18•, 19]. RCT efficacy has been established for chronic low back pain. TCAs function by exerting analgesia primarily through serotonin and norepinephrine reuptake inhibition, sodium channel blockade, and NMDA antagonism [18•]. The side effects

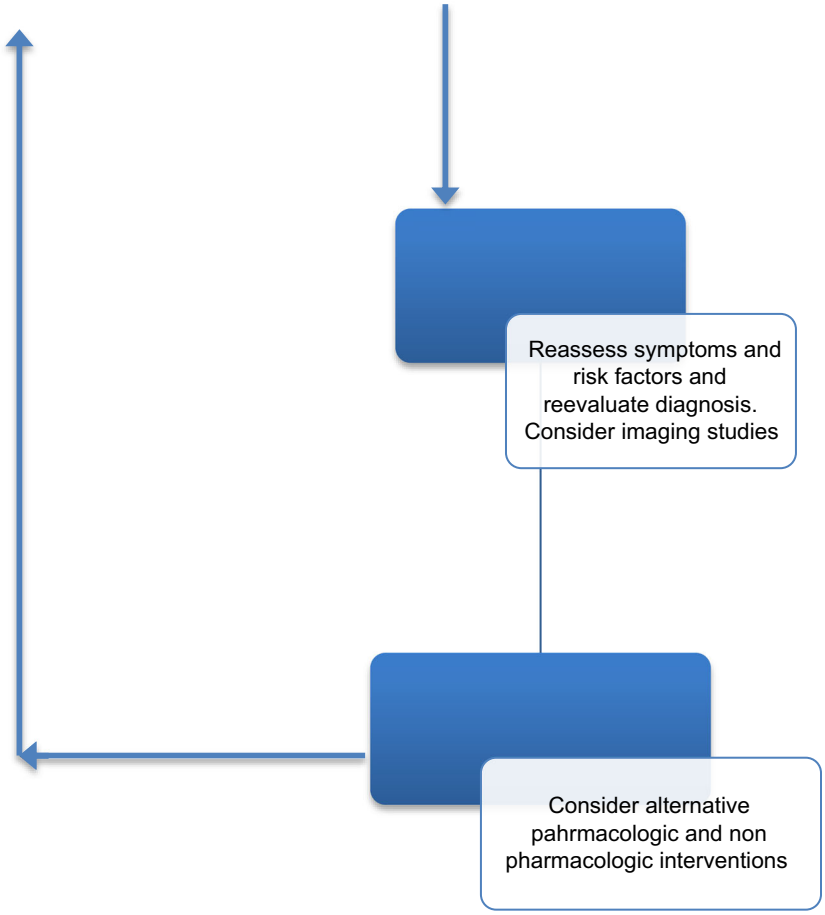

Fig. 1 continued.

commonly displayed include dry mouth/constipation (anticholinergic) and dizziness/drowsiness (antihistaminergic).

Additionally, serotonin norepinephrine reuptake inhibitors (SNRIs) are also pharmacologic treatment for chronic low back pain [18•]. RCT efficacy has been established for duloxetine and venlafaxine, with the former being better tolerated [18•]. SNRIs function by exerting analgesia through serotonin and norepinephrine reuptake inhibition, which is important for descending pain inhibition. The most common side effects include dry mouth, self-limited nausea, dizziness, headache, and insomnia.

Lastly, pharmacologic treatment of low back pain includes antiepileptics. While gabapentin has shown analgesic efficacy for chronic low back pain with radiculopathy [19], only topiramate has been studied for chronic axial low back pain with evidence of analgesia and improved quality of life [57]. Topiramate has the advantageous side effect of weight loss, but is also associated with dizziness, somnolence, and rare nephrolithiasis [57].

Moreover, psychological treatments are an imperative aspect of low back pain treatment and should be evaluated for implementation in the course of patient management. Psychological interventions have been most studied for *chronic* low back pain, though application to *acute* low back pain patients with multiple yellow flags (see section I.D) is prudent to prevent chronicity of pain [18•]. Addressing psychosocial and motivational factors is also important within a multidisciplinary framework for analgesic efficacy and reducing disability. Types of psychological treatments include cognitive behavioral therapy (CBT), progressive relaxation, and Biofeedback.

CBT is a goal-oriented approach that targets maladaptive thinking and coping strategies to change behavior and improve mood. RCT evidence points to short-term improvement in pain intensity and disability [18•]. In addition, progressive relaxation consists of muscle tension-reducing technique involving systematic flexing and relaxing of specific muscles with the aim of achieving profound relaxation [19]. This provides short-term improvement in pain and function. Thirdly, Biofeedback is a relaxation approach that utilizes auditory and visual feedback from muscle activity to reduce muscle tension. Yet, studies show mixed data for pain intensity reduction [58].

Physical and rehabilitation treatments are methods to increase functionality and pain management and may be

coupled with other methods of care for low back pain. Exercise therapy is defined as a series of specific movements with the goal of training the body to promote good physical health [16, 19]. Short-term reduction in pain intensity and disability has been shown in *chronic* low back pain relative to usual care [18•]. Within this type of therapy, stretching exercises are most associated with pain reduction, while strengthening yields greatest functional gains. Multidisciplinary functional rehabilitation programs have also been effective for pain relief, disability reduction, and improved mood [20]. This is multidisciplinary biopsychosocial rehabilitation with at least one physical dimension (exercise, physical modalities) and one other dimension (psychological or social or occupational) [20]. Other physical therapy or rehab modalities require further research and assessment prior to implementation. These programs lack RCT support for chronic low back pain, including effectiveness of lumbar support, inadequate data to support massage therapy, back schools, traction, and superficial heat or cold application, and for transcutaneous electrical nerve stimulation (TENS).

Another treatment approach for low back pain is complementary and alternative medicine therapies. Acupuncture is a type of intervention that utilizes specific anatomical points along classic meridians typically with the use of small needles that are either manipulated or electrically stimulated to achieve effect. Meta-analysis of randomized control trials focusing on chronic low back pain has revealed reduced pain intensity and improved function immediately post-intervention compared to sham, NSAIDs, or muscle relaxants [18•]. Additionally, through osteopathic or chiropractic treatment, manipulation of the spine involves the goal of restoring spinal alignment and optimal range of motion. Meta-analysis reveals equal effectiveness to general practitioner care, analgesics, physical therapy, and exercise therapy [18•]. Lastly, sleep support through medium-firm mattresses reduces pain levels during the day, night, and with rising from bed relative to firm mattress [19].

In certain instances, interventional minimally invasive percutaneous approaches can be used for axial low back pain. In cases when pain remains refractory to conservative multidisciplinary treatment, minimally invasive interventional approaches are rationally considered with the goal of improving function, relieving pain, and reducing side effects from medical management. The predictors of poor outcomes of interventional procedures include poorly controlled psychiatric disorder, catastrophization and fear avoidance behavior, co-existing chronic pain complaints, high baseline pain scores and disability, previous treatment failures, chronic escalating opioid use, secondary gain, and previous spine surgery.

Lumbar facet (zygapophyseal) joint interventions is one type of percutaneous approach. Lumbar facet joints are innervated by the medial branches of the dorsal rami, with anatomical studies documenting nerve endings within the facet joints [25]. Facet joint pain has been targeted by intra-articular injections, medial branch nerve blocks, and radiofrequency neurotomy of the medial branch nerves [25]. Intra-articular facet joint steroid injections have shown limited or negative RCT evidence for benefit and are not recommended [35]. If diagnostic lumbar medial branch nerve blocks (single or preferably comparative to decrease placebo response) provide substantial temporary relief, neuroablation with radiofrequency neurotomy is considered for longer-term benefit [59]. Likewise, lumbar medial branch radiofrequency neurotomy has positive RCT evidence for improved pain and function lasting 6–12 months [25]. Facet joint intervention complications are rare and are primarily limited to pain or swelling at needle insertion site and temporary flare of pain post-neurotomy [25].

Moreover, sacroiliac joint interventions are other minimally invasive procedures for low back pain. Sacroiliac joints are known to be a significant source of pain in patients with spondyloarthropathies as well as with advanced age and post-lumbar fusion. Intra-articular sacroiliac joint steroid injections have been studied in one small RCT in patients with ankylosing spondyloarthropathy with analgesia and decreased NSAID use [28••]. However, no RCT's explore this intervention for non-rheumatologic sacroiliac joint pain. Additionally, the sacroiliac joints are innervated by both ventral and dorsal sacral rami and the posterior sacroiliac joint and ligaments (posterior sacroiliac complex) are innervated by the lateral branches of the sacral dorsal rami [38••, 53]. Sacral lateral branch radiofrequency neurotomy targeting the posterior sacroiliac complex has been studied in two small RCTs utilizing cooled as well as unipolar radiofrequency lesioning techniques [40]. Diagnostic blocks were performed either with sacroiliac joint intra-articular injections or L5 dorsal ramus plus S1-S3 lateral branch blocks. Analgesia post-neurotomy was significant for approximately 3–6 months with improved function [54].

Lumbar radicular pain and spinal stenosis treatment with epidural steroid injections (ESI) is an alternative treatment option. Epidural injections of steroid provide significant, though temporary (< 3 month), analgesia with best supportive RCT evidence in patients with acute radicular pain concordant with site of lumbar disc herniation rather than axial lumbar pain [49]. Epidural steroid injection for neuroclaudication secondary to spinal stenosis has been studied with RCT evidence revealing equivalent reduction in pain and improved function for both the steroid and local anesthetic groups, but no sham injection group was studied [49]. Local anesthetic alone can provide analgesia by increasing blood flow to ischemic nerve roots, suppressing nociceptive transmission, and washing out inflammatory mediators.

Lastly, lumbar post-laminectomy syndrome and spinal cord stimulation (SCS) are minimally invasive procedures for low back pain. Spinal cord stimulation delivers electrical pulses via epidural electrodes at vertebral levels associated with pain, either overlapping the pain with masking

paresthesia (traditional low frequency SCS devices) or through the use of high frequency (10 kHz) non-paresthesia neuromodulation. The latter is thought to provide better analgesia for axial low back pain [60]. Furthermore, RCT studies have shown significantly improved analgesia, function, and patient satisfaction with use of SCS compared to conventional medical management or repeat spine surgery in patients with lumbar post-laminectomy syndrome up to 2 years post-implantation [60]. High frequency neuromodulation has also shown RCT superiority to low frequency SCS for both axial and radicular analgesia at 2 years follow-up.

Compliance with Ethical Standards

Conflict of Interest Ivan Urits, Aaron Burshtein, Medha Sharma, Lauren Testa, Peter A. Gold, Vwaire Orhurhu, Omar Viswanath, Mark R. Jones, Moises A. Sidransky, and Boris Spektor declare no conflict of interest. Dr. Kaye is a speaker for Depomed, Inc. and Merck, Inc.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1.•• Bogduk N. On the definitions and physiology of back pain, referred pain, and radicular pain. Pain. 2009;147:17–9. **A delineation of low back pain diagnosis and definition.**
2. Atlas SJ, Deyo RA. Evaluating and managing acute low back pain in the primary care setting. J Gen Intern Med, Springer. 2001;16:120–31.
3. Petering RC, Webb C. Treatment options for low back pain in athletes. Sports Health, SAGE Publications. 2011;3:550–5.
4. Longo UG, Loppini M, Denaro L, Maffulli N, Denaro V. Rating scales for low back pain. Br Med Bull, Oxford University Press. 2010;94:81–144.
5. Lennard T, Vivian D, Walkowski S, Singla A. Pain procedures in clinical practice. Third Edition. Elsevier/Saunders. 2011.
6.•• Hoy D, March L, Brooks P, Blyth F, Woolf A, Bain C, et al. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis, BMJ Publishing Group Ltd. 2014;73:968–74. **An epidemiological study of global low back pain burden.**
7. Heuch I, Foss IS. Acute low back usually resolves quickly but persistent low back pain often persists. J Physiother. 2013;59:127.
8. American Academy of Family Physicians. TH, Randolph DC. American family physician. Am. Fam. Physician. Leawood: American Academy of Family Physicians; 1970.
9. French SD, Cameron M, Walker BF, Reggars JW, Esterman AJ. Superficial heat or cold for low back pain. Cochrane database Syst Rev, John Wiley & Sons, Ltd. 2006:CD004750.
10. Frerick H, Keitel W, Kuhn U, Schmidt S, Bredehorst A, Kuhlmann M. Topical treatment of chronic low back pain with a capsicum plaster. Pain. 2003;106:59–64.
11. Witenko C, Moorman-Li R, Motycka C, Duane K, Hincapie-Castillo J, Leonard P, et al. Considerations for the appropriate use of skeletal muscle relaxants for the management of acute low back pain. P T, MediMedia, USA. 2014;39:427–35.
12. American Academy of Family Physicians. AT, Ogle AA. American family physician. Am. Fam. Physician. Leawood: American Academy of Family Physicians; 1970.
13. Samanta J, Kendall J, Samanta A. 10-minute consultation: chronic low back pain. BMJ, British Medical Journal Publishing Group. 2003;326:535.
14. Pincus T, Vlaeyen JWS, Kendall NAS, Von Korff MR, Kalauokalani DA, Reis S. Cognitive-behavioral therapy and psychosocial factors in low back pain: directions for the future. Spine (Phila Pa 1976). 2002;27:E133–8.
15. Elkayam O, Ben Itzhak S, Avrahami E, Meidan Y, Doron N, Eldar I, et al. Multidisciplinary approach to chronic back pain: prognostic elements of the outcome. Clin Exp Rheumatol. 14:281–8.
16. Deyo RA, Weinstein JN. Low Back Pain. N Engl J Med. 2001;344:363–70.
17. Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? JAMA J Am Med Assoc, American Medical Association. 1992;268:760.
18.• Chou R. Low back pain. Ann Intern Med, American College of Physicians. 2014;160:ITC6–1. **An overview of low back pain.**
19. Chou R, Qaseem A, Snow V, Casey D, Cross JT, Shekelle P, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med, American College of Physicians. 2007;147:478.
20. van Tulder M, Becker A, Bekkering T, Breen A, Gil del Real MT, Hutchinson A, et al. Chapter 3 European guidelines for the management of acute nonspecific low back pain in primary care. Eur Spine J. 2006;15:s169–91.
21. Deyo RA, Diehl AK. Cancer as a cause of back pain: frequency, clinical presentation, and diagnostic strategies. J Gen Intern Med. 3:230–8.
22. Rubinstein SM, van Tulder M. A best-evidence review of diagnostic procedures for neck and low-back pain. Best Pract Res Clin Rheumatol. 2008;22:471–82.
23. Partanen JV, Ojala TA, Arokoski JPA. Myofascial syndrome and pain: a neurophysiological approach. Pathophysiology. 2010;17:19–28.
24. Todd AJ, Vaccaro AR. Physical Examination of the Spine. Second Edition. New York: Thieme Medical Publishers, Inc; 2016.
25. Kalichman L, Hunter DJ. Lumbar facet joint osteoarthritis: a review. Semin Arthritis Rheum. 2007;37:69–80.
26. Park WM, Kim K, Kim YH. Effects of degenerated intervertebral discs on intersegmental rotations, intradiscal pressures, and facet joint forces of the whole lumbar spine. Comput Biol Med. 2013;43:1234–40.
27.•• Bagwell JJ, Bauer L, Gradoz M, Grindstaff TL. The reliability of FABER test hip range of motion measurements. Int J Sports Phys Ther, The Sports Physical Therapy Section of the American Physical Therapy Association. 2016;11:1101–5. **Use of the FABER test for diagnosis of sacroiliac joint related pain.**
28.•• Zelle BA, Gruen GS, Brown S, George S. Sacroiliac joint dysfunction: evaluation and management. Clin J Pain. 21:446–55. **An overview of sacroiliac joint related pain.**
29. Chou R, Qaseem A, Owens DK, Shekelle P. Clinical Guidelines Committee of the American College of Physicians. Diagnostic imaging for low back pain: advice for high-value health care from the American College of Physicians. Ann Intern Med. 2011;154:181.

30. Wilkinson LS, Elson E, Saifuddin A, Ransford AO. Defining the use of gadolinium enhanced MRI in the assessment of the postoperative lumbar spine. Clin Radiol, Elsevier. 1997;52:530–4.
31. Simons DG. New views of myofascial trigger points: etiology and diagnosis. Arch Phys Med Rehabil. 2008;89:157–9.
32. Lucas N, Macaskill P, Irwig L, Moran R, Bogduk N. Reliability of physical examination for diagnosis of myofascial trigger points. Clin J Pain. 2009;25:80–9.
33. Giamberardino MA, Affaitati G, Fabrizio A, Costantini R. Myofascial pain syndromes and their evaluation. Best Pract Res Clin Rheumatol. 2011;25:185–98.
34. Donnelly JM, Fernández-de-Las-Peñas C, Finnegan M, Freeman JL. Travell, Simons & Simons' Myofascial Pain and Dysfunction. Third Edition. Philadelphia: Wolters Kluwer Health; 2018.
35. Bogduk N. Evidence-informed management of chronic low back pain with facet injections and radiofrequency neurotomy. Spine J. 2008;8:56–64.
36. Comer C, Conaghan PG. Tackling persistent low back pain in primary care. Practitioner. 2009;253:32–4 3.
37. Pneumaticos SG, Chatziioannou SN, Hipp JA, Moore WH, Esses SI. Low back pain: prediction of short-term outcome of facet joint injection with bone scintigraphy. Radiology. 2006;238:693–8.
38.•• Manchikanti L, Staats PS, Singh V, Schultz DM, Vilims BD, Jasper JF, et al. Evidence-based practice guidelines for interventional techniques in the management of chronic spinal pain. Pain Physician. 2003;6:3–81. **Guidelines for an interventional approach to the treatment of low back pain.**
39. Aprill C, Bogduk N. High-intensity zone: a diagnostic sign of painful lumbar disc on magnetic resonance imaging. Br J Radiol. 1992;65:361–9.
40. Bogduk N. Degenerative joint disease of the spine. Radiol Clin N Am. 2012;50:613–28.
41. Jhawar BS, Fuchs CS, Colditz GA, Stampfer MJ. Cardiovascular risk factors for physician-diagnosed lumbar disc herniation. Spine J. 2006;6:684–91.
42. Zawilla NH, Darweesh H, Mansour N, Helal S, Taha FM, Awadallah M, et al. Matrix metalloproteinase-3, vitamin D receptor gene polymorphisms, and occupational risk factors in lumbar disc degeneration. J Occup Rehabil. 2014;24:370–81.
43. Bogduk N, Bogduk N. Clinical and radiological anatomy of the lumbar spine. Elsevier/Churchill Livingstone; 2012.
44. Taylor RS, Taylor RJ. The economic impact of failed back surgery syndrome. Br J Pain. 2012;6:174–81.
45. Waguespack A, Schofferman J, Slosar P, Reynolds J. Etiology of long-term failures of lumbar spine surgery. Pain Med. 2002;3:18–22.
46. Vwaire Orhurhu, Ivan Urits, Mayowa Olusunmade, Khurram Owais, Mark Jones, Annemarie Galasso, et al. Trends of Co-Morbid Depression in Hospitalized Patients with Failed Back Surgery Syndrome: An Analysis of the Nationwide Inpatient Sample. Pain Ther. 2018;7:217–26.
47. Chan C. Failed back surgery syndrome - review article. Pain Med. 2011;12:577–606.
48. Weinstein JN, Lurie JD, Tosteson TD, Hanscom B, Tosteson ANA, Blood EA, et al. Surgical versus nonsurgical treatment for lumbar degenerative spondylolisthesis. N Engl J Med. 2007;356:2257–70.
49. Koc Z, Ozcakir S, Sivrioglu K, Gurbet A, Kucukoglu S. Effectiveness of physical therapy and epidural steroid injections in lumbar spinal stenosis. Spine (Phila Pa 1976). 2009;34:985–9.
50. Bischoff RJ, Rodriguez RP, Gupta K, Righi A, Dalton JE, Whitecloud TS. A comparison of computed tomography-myelography, magnetic resonance imaging, and myelography in the diagnosis of herniated nucleus pulposus and spinal stenosis. J Spinal Disord. 1993;6:289–95.
51. Foley BS, Buschbacher RM. Sacroiliac joint pain. Am J Phys Med Rehabil. 2006;85:997–1006.
52. Slipman CW, Jackson HB, Lipetz JS, Chan KT, Lenrow D, Vresilovic EJ. Sacroiliac joint pain referral zones. Arch Phys Med Rehabil. 2000;81:334–8.
53. McKenzie-Brown AM, Shah RV, Sehgal N, Everett CR. A systematic review of sacroiliac joint interventions. Pain Physician. 2005;8:115–25.
54. King W, Ahmed SU, Baisden J, Patel N, Kennedy DJ, MacVicar J, et al. Diagnosis and treatment of posterior sacroiliac complex pain: a systematic review with comprehensive analysis of the published data. Pain Med. 2015;16:257–65.
55. Roelofs PD, Deyo RA, Koes BW, Scholten RJ, van Tulder MW. Non-steroidal anti-inflammatory drugs for low back pain. Cochrane Database Syst Rev. 2008:CD000396.
56. Koes BW, Backes D, Bindels PJE. Pharmacotherapy for chronic non-specific low back pain: current and future options. Expert Opin Pharmacother. 2018;19:537–45.
57. Muehlbacher M, Nickel MK, Kettler C, Tritt K, Lahmann C, Leiberich PK, et al. Topiramate in treatment of patients with chronic low back pain. Clin J Pain. 2006;22:526–31.
58. Vitoula K, Venneri A, Varrassi G, Paladini A, Sykioti P, Adewusi J, et al. Behavioral therapy approaches for the management of low back pain: an up-to-date systematic review. Pain Ther, Springer. 2018;7:1.
59. Gofeld M, Jitendra J, Faclier G. Radiofrequency denervation of the lumbar zygapophysial joints: 10-year prospective clinical audit. Pain Physician. 2007;10:291–300.
60. Kumar K, Taylor RS, Jacques L, Eldabe S, Meglio M, Molet J, et al. Spinal cord stimulation versus conventional medical management for neuropathic pain: a multicentre randomised controlled trial in patients with failed back surgery syndrome. Pain. 2007;132:179–88.