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Non-surgical Approaches in the Treatment of Lower Back Pain: A Review of Methods, Efficacy, and Safety

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Abstract

Introduction: Low back pain (LBP) is among the most common musculoskeletal disorders globally, representing a leading cause of disability and healthcare expenditure. Its prevalence is expected to rise significantly in the coming decades, emphasizing the need for effective, evidence-based treatment strategies. This narrative review explores non-surgical approaches to LBP management, focusing on pharmacological and non-pharmacological therapies.

It discusses the classification, pathophysiology, and red flag symptoms, alongside an evaluation of conservative treatments including NSAIDs, acetaminophen, muscle relaxants, opioids, antidepressants, physical therapy, acupuncture, spinal manipulation, and psychosocial interventions. The findings indicate that NSAIDs offer modest short-term relief and remain first-line pharmacological agents, while acetaminophen has limited efficacy. Muscle relaxants may benefit acute cases but carry notable side effects, particularly in older adults. Opioids, though effective in the short term, show minimal long-term benefit and a high risk of dependence. Non-pharmacological treatments-especially exercise therapy, manual therapy, and cognitive-behavioral interventions-demonstrate consistent efficacy in reducing pain and improving function. In conclusion, optimal management of LBP necessitates an individualized, multimodal approach that integrates pharmacological options with physical and psychological strategies to minimize harm, enhance function, and address biopsychosocial contributors to pain.
Aim of the study: The aim of this narrative review is to provide a comprehensive and up-to-date synthesis of the current knowledge on the etiology, classification, and evidence-based management of low back pain, with particular emphasis on pharmacological and non-pharmacological treatment strategies. Special attention is given to the identification of red flag symptoms ,multifactorial etiology of LBP, and the clinical relevance of biopsychosocial factors in pain chronification. The review also seeks to highlight the need for integrated, individualized therapeutic approaches in both acute and chronic cases.
Materials and methods: A review of research literature was conducted through database such as PubMed, Google Schoolar, ResearchGate, and the Cochrane Library.
Conclusion: Low back pain is a widespread and multifactorial condition with substantial clinical and socioeconomic consequences. Effective management requires an individualized, multidisciplinary approach that combines pharmacological and non-pharmacological strategies. Early identification of red flag symptoms is essential to exclude serious pathology and guide appropriate treatment. Ongoing research is needed to optimize care pathways and improve long-term outcomes.

Keywords: Low back pain; pathophysiology; etiology; treatment; pharmacological treatment; non-surgical treatment; pharmacology; efficiency; safety.

Epidemiology and Global Burden of Low Back Pain

According to the World Health Organization (WHO), low back pain (LBP) is defined as pain perceived in the area between the lower margin of the ribs and the buttocks. LBP is a highly prevalent health problem affecting a large proportion of the population and ranks as the second most common reason for seeking medical care [1]. It accounts for approximately 15% of all work-related sick leaves and is the leading cause of disability in individuals under 45 years of age [2]. The lifetime prevalence of LBP may reach up to 90%, with an annual incidence risk of 5% [3]. Data published in *The Lancet Rheumatology* estimate that by the year 2050, the global number of individuals suffering from low back pain will rise to approximately 843 million, representing a 36.4% increase compared to 619 million cases reported in 2020 [4]. In the United States, LBP was responsible for the highest healthcare expenditures among 154 disease conditions, with an estimated annual cost of $134.5 billion USD (95% CI, $122.4–$146.9 billion USD) [5].

In Germany, the average annual cost per patient with back pain amounted to €1,322, of which 46% were direct costs (e.g., medical care) and 54% indirect costs (e.g., loss of productivity) [6]. Consequently, it is essential to systematize information regarding the causes, pathomechanisms, and first-line treatments of LBP, with particular emphasis on the identification of red flag symptoms.

Types of LBP

The classification based on pain duration includes acute low back pain (ALBP), which lasts less than 6 weeks. It often results from injury or overuse and typically resolves with conservative treatment. Subacute low back pain persists from 6 weeks up to 3 months and may arise from untreated acute pain or early degenerative changes in intervertebral discs. Chronic low back pain (CLBP) lasts longer than 3 months and can be caused by degeneration, joint osteoarthritis, injury, or stress [7].

Another classification distinguishes two main categories of back pain: specific back pain and non-specific back pain. Specific back pain is further divided into three subtypes. The first is discogenic back pain, characterized by back and/or leg pain without radiologically significant nerve root compression. The second subtype is nociceptive back pain unrelated to the disc, which includes myofascial pain-such as back sprains and muscle spasms-as well as osteoarthritic pain involving synovial joints like the facet joints, vertebral fractures, and osteoporosis. The third subtype is neural leg pain, exemplified by radicular pain resulting from nerve root compression [8].

Low back pain can also be classified based on its underlying mechanism. Mechanical pain is the most common type and is associated with overload of anatomical structures of the spine, such as intervertebral discs or facet joints. Typically, it worsens during physical activity and diminishes with rest [9]. In contrast, neuropathic pain results from damage or compression of neural structures, for example, due to a herniated disc. It is characteristically manifested by pain radiating to the lower limbs, paresthesias, and numbness in the area innervated by the affected nerve [10]

When should diagnosis and treatment not be delayed?

Red flags in the context of lower back pain refer to clinical signs, abnormalities detected during physical examination, and patient history data that may suggest the presence of a serious and specific pathology underlying the symptoms-such as malignancy, infection, spinal fracture, or cauda equina syndrome. The presence of red flags constitutes a significant warning signal that necessitates thorough diagnostic evaluation, often including imaging studies (e.g., magnetic resonance imaging), and prompt specialist consultation.

The most important red flags indicating the possibility of neoplastic changes include: age over 50 years, a positive oncological history, unexplained weight loss, pain independent of physical activity that worsens at night, and lack of improvement despite appropriate treatment for more than 4–6 weeks [9]. Factors suggestive of spinal infection include fever, recent infection, spinal surgery within the past year, intravenous drug use, and immunosuppression [11]. A spinal fracture should be suspected in patients following trauma, particularly in the elderly, those with osteoporosis, prolonged glucocorticoid use, or individuals over the age of 70 [12].

Cauda equina syndrome is suggested by symptoms such as urinary or fecal incontinence, saddle anesthesia, bilateral neurological signs, lower limb weakness, and diminished deep tendon reflexes [13] Other concerning clinical features include progressive motor weakness, extensive radicular symptoms persisting without improvement, chronic pain lasting longer than six weeks, and lack of response to conventional treatment. Additionally, systemic signs such as significant unintentional weight loss or inflammatory symptoms may indicate the presence of serious underlying pathology, such as ankylosing spondylitis [9].

Etiology of LBP

Myofascial syndrome

Myofascial pain may represent an underlying cause of low back pain (LBP), particularly in the context of trauma or repetitive motion. Myofascial pain syndrome (MPS) is characterized by the presence of myofascial trigger points (MTrPs) located within fascia, tendons, and/or muscles, which elicit symptomatic pain responses upon palpation. These trigger points are typically induced by injury or mechanical overload [14]. Clinically, patients most often report limited range of motion and pain in the paraspinal musculature, which may radiate to the gluteal region or thighs [15].

The most widely accepted pathophysiological explanation of MPS is based on the integrated hypothesis proposed by Simons. He demonstrated that a reduced local blood flow leads to decreased levels of adenosine triphosphate (ATP), resulting in an energy crisis within the muscle tissue [16]. This energy deficiency prevents proper reuptake of calcium into the sarcoplasmic reticulum, leading to persistent sarcomere contraction and localized muscle shortening. The ensuing contraction causes transient ischemia, which in turn increases metabolic demand and triggers the release of various biochemical substances that may contribute to the heightened nociceptive response at the trigger point [17, 18].

Discogenic LBP

According to data published in *Orthopedic Reviews*, approximately 26–42% of cases of LBP can be attributed to intervertebral disc degeneration, a condition referred to as discogenic back pain (DBP) [19]. Intervertebral disc degeneration is also considered one of the most significant etiologies of LBP [20]. Internal disc disruption, a key pathological process in DBP, results primarily from structural degradation of the disc and its central components. This degeneration may be exacerbated by annular fissures extending from the nucleus pulposus to the periphery of the annulus fibrosus [21]. The most commonly affected levels are L5/S1 and L4/L5 [22]. Pain is typically provoked by forward trunk flexion during bending or prolonged sitting, as well as by actions that increase intradiscal pressure, such as coughing or sneezing. Flexion movements tend to reproduce symptoms due to increased compressive loading of the intervertebral disc, which may intensify discomfort particularly under axial load or during seated posture [23]. DBP is more prevalent among individuals engaged in manual labor involving frequent heavy lifting, those exposed to whole-body vibration, and individuals with sedentary occupations [24].

In a 16-year longitudinal study, Balraj S. Jhawar et al. assessed the influence of metabolic conditions on the risk of developing DBP. Multivariate analysis revealed the following relative risk (RR) values: diabetes mellitus – RR 1.52 (95% CI, 1.17–1.98); hypertension – RR 1.25 (95% CI, 1.11–1.41); hypercholesterolemia – RR 1.26 (95% CI, 1.10–1.44); and a family history of myocardial infarction before the age of 60 – RR 1.13 (95% CI, 1.02–1.26). In comparison to lifelong non-smokers, former smokers demonstrated a relative risk of 1.10 (95% CI, 1.00–1.20) for developing DBP [25]

Interprocessus pain

Osteoarthritis (OA) may affect all diarthrodial joints in the human body. The condition is characterized by progressive degeneration of articular cartilage, leading to subchondral bone erosion and sclerosis [26]. Additional pathological features include hypertrophy and deformation of the facet joints as well as osteophyte formation. These structural alterations can contribute to narrowing of the spinal canal and intervertebral foramina, potentially resulting in compression of spinal nerve roots. The precise localization of anatomical pathology in OA-related spinal pain is often challenging. This difficulty arises from the fact that pain-generating structures may not be visualized on standard imaging modalities and because multiple potential sources of nociception exist-such as the synovial membrane and cavity, subchondral bone, osteophytes, joint capsule, ligaments, and paraspinal muscles [27]. Genetic predisposition and other yet unidentified factors are considered the most significant contributors to the development of spinal OA [28]. Other relevant risk factors include cumulative microtrauma to the spine, osteoporosis, excessive body weight, and reduced postural muscle tone. Occupational and sedentary behaviors are believed to exert a moderate influence on disease progression. Clinically, patients often report deep, paraspinal pain that intensifies with movement. Radiation of pain may occur unilaterally or bilaterally to the buttocks, groin, and/or thighs, typically not extending below the knee. A hallmark symptom of lumbar spinal stenosis secondary to OA is neurogenic claudication, which presents as unilateral or bilateral buttock or leg pain during ambulation or standing, relieved within approximately 30 minutes upon postural adjustment-most commonly by adopting a flexed or seated position. The distribution of pain and sensory deficits corresponds to the dermatomal map.

Post-operative back pain

Failed Back Surgery Syndrome (FBSS) is defined as a clinical condition in which patients experience persistent or recurrent low back and/or lower limb pain following anatomically and technically adequate spinal surgery [29]. FBSS is diagnosed when the outcome of lumbar spine surgery fails to meet the preoperative expectations of both the patient and the surgeon [30]. Despite the clinical relevance of FBSS, scientific literature concerning its etiopathogenesis in the context of chronic low back pain remains limited.

Risk factors contributing to FBSS are categorized into preoperative, intraoperative, and postoperative domains. Preoperative predictors include psychiatric comorbidities, particularly a history of depression and anxiety disorders [31]. Intraoperative factors involve surgical errors such as operating at an incorrect spinal level or technical faults during the procedure [32]. Postoperative contributors encompass rapid disease progression, epidural fibrosis, spinal instability, myofascial pain syndrome, and other surgical complications [33].

A study by Fritzell et al. (2003) demonstrated that postoperative satisfaction following surgical intervention for low back pain ranged from 60% to 68%, depending on the surgical technique used [34]. Similarly, in a longitudinal study by Webber (1983) involving patients undergoing lumbar spine surgery for radiculopathy secondary to herniated disc, reported satisfaction rates were 65% at 1 year postoperatively, 67% at 4 years, and 58% at 10 years [35]. The estimated prevalence of FBSS in the general population ranges from 0.02% to 2%.

Treatment

The management of acute low back pain (ALBP) typically involves two primary strategies: the 'wait and see' approach and active therapeutic intervention. The 'wait and see' model is based on the premise that most cases of acute LBP resolve spontaneously within a few weeks. According to the guidelines issued by the National Institute for Health and Care Excellence (NICE), this strategy is considered appropriate for patients at low risk of chronicity, which can be assessed using tools such as the STarT Back Screening Tool. In such instances, it is recommended to provide the patient with reassurance regarding a favorable prognosis, encourage continued physical activity, and monitor symptoms without the immediate implementation of advanced therapeutic measures. Similarly, the clinical practice guidelines developed by the U.S. Department of Veterans Affairs and Department of Defense (VA/DoD) advocate for a shared decision-making process. In the absence of red flag symptoms or significant neurological deficits, patients should be actively involved in choosing a management strategy, which may include an observational approach. [36]

An alternative to passive strategies is an active management approach, which emphasizes early intervention aimed at improving patient function and preventing the progression to chronic pain. This strategy typically includes therapeutic exercise, physical activity, psychological support, pharmacological treatment, and patient education to promote self-management of symptoms [37]. Evidence suggests that active management is more effective in preventing the transition from acute to chronic low back pain (LBP) and improving quality of life, particularly in individuals with psychosocial risk factors.

Non-surgical treatment remains the cornerstone of therapy for most patients with both acute and chronic LBP. The primary goals of conservative management are pain reduction, functional improvement, and prevention of recurrence. Initial conservative treatment is recommended in the absence of 'red flags'. Surgical intervention is considered only in rare cases, such as intractable pain unresponsive to pharmacological therapy.

Non-pharamcological treatment

Acupuncture and dry needling

Traditional Chinese acupuncture is based on the concept of restoring the flow of energy (Qi) through meridians, whereas dry needling-often referred to as Western acupuncture-involves the insertion of needles into myofascial tissues to deactivate presumed myofascial trigger points. The physiological mechanisms underlying acupuncture's analgesic effects are thought to involve modulation of neurotransmitters and neural pathways.

According to a study published in *The International Journal of Neuroscience* (2005), stimulation of specific acupuncture points results in elevated levels of endogenous opioids such as endomorphin-1, beta-endorphins, and enkephalins, as well as serotonin in both plasma and brain tissue. These neurochemical changes are associated with analgesic, muscle-relaxant, and motor function-enhancing effects [38].

A meta-analysis by Manheimer et al. summarized data from 22 randomized controlled trials and concluded that acupuncture is an effective therapeutic option for chronic low back pain (CLBP). However, evidence regarding its efficacy in acute low back pain remains limited and inconclusive. Currently, there is no strong evidence suggesting that acupuncture outperforms other active interventions for the management of CLBP [39].

Manipulation and physiotherapy

Spinal manipulation is a manual technique involving a precise, high-velocity thrust delivered to a joint beyond its physiological range of motion, but within anatomical limits. Its mechanism of action is thought to include both biomechanical effects-such as reducing mechanical stress and improving joint mobility-and neurophysiological mechanisms, including pain gating and modulation of motor control systems [40].

A systematic review of 26 randomized controlled trials found that spinal manipulation provides statistically significant, albeit moderate, benefits in pain reduction and functional improvement in patients with acute low back pain (duration ≤6 weeks). Reported adverse events were generally mild and transient, including increased pain, muscle stiffness, and headaches [41]. A 2018 study comparing spinal manipulation and mobilization in chronic low back pain also demonstrated moderate pain reduction, with manipulation showing greater effectiveness than mobilization. Both interventions were considered safe [42].

Therapeutic massage-including techniques such as deep tissue massage and trigger point therapy-is employed to enhance circulation, relax musculature, and increase range of motion [43]. However, high-quality evidence on its effectiveness in acute low back pain is lacking. Low-quality studies have suggested a moderate benefit for short-term pain relief [44], whereas other trials reported no therapeutic effect after five weeks of treatment [45]. In the context of subacute and chronic low back pain, massage therapy provided only limited analgesic effects compared to spinal manipulation, relaxation techniques, exercise therapy, and acupuncture [44, 46].

Ultrasounds

The theoretical mechanism of action of therapeutic ultrasound in the management of low back pain involves several proposed effects: (a) thermal effects-raising the temperature of deep tissues, (b) micromassage-enhancement of local blood flow and lymphatic circulation, and (c) anti-inflammatory action. Despite these hypothesized mechanisms, clinical evidence supporting the efficacy of ultrasound therapy remains lacking. A systematic review by van Middelkoop et al. (Spine, 2011) analyzing 20 clinical trials found no substantial evidence for the effectiveness of passive physical therapies, including ultrasound, in the treatment of chronic low back pain [47]. Similarly, another randomized controlled trial demonstrated no significant difference in treatment outcomes between patients receiving ultrasound therapy and those treated with placebo [48].

Change in physical activity

The modification of physical activity frequency in patients with LBP aims to achieve a tolerable level of discomfort while maintaining engagement in activity, thereby minimizing disruption to daily functioning. Recommendations based on the Agency for Health Care Policy and Research (AHCPR) guidelines advocate for the temporary restriction of activities such as heavy lifting, prolonged sitting, and spinal flexion or rotation. One frequently discussed intervention is bed rest. The theoretical rationale for bed rest includes reducing nerve root compression and lowering intramuscular pressure. In cases of acute radicular pain, short-term bed rest-limited to 2–3 days-may offer some benefit; however, even in such instances, adverse outcomes are possible [49]. In all other cases, bed rest is not recommended, as inactivity exceeding four days has been associated with increased spinal stiffness and weakening of paraspinal musculature.

Mental health

A growing body of evidence indicates that psychosocial factors-commonly referred to as 'yellow flags'-play a critical role in the transition from acute to chronic low back pain (LBP). These factors include, but are not limited to, fear of movement (kinesiophobia), pain catastrophizing, occupational stress, and depression. Patients experiencing poorer mental health often report higher pain intensity and greater impairment in daily functioning [50]. According to guidelines issued by the National Institute for Health and Care Excellence (NICE), the management of LBP should address both mechanical and psychosocial contributors to pain [51]. In this context, cognitive-behavioral therapy (CBT) has demonstrated efficacy in helping patients reframe maladaptive beliefs about pain, thereby facilitating better psychological adjustment and reducing the risk of pain chronification [52].

Pharmacological treatment

Acetaminophen

Paracetamol is widely used for the management of pain and fever, although its precise mechanism of action remains incompletely understood. Several hypotheses have been proposed to explain its analgesic effects. One prevailing theory involves the inhibition of cyclooxygenase (COX) enzymes in the central nervous system. Paracetamol is believed to inhibit a variant of the COX enzyme, often referred to as COX-3, which is a splice variant of COX-1. This central inhibition reduces the synthesis of prostaglandins in the brain, thereby mediating its antipyretic and analgesic effects. Notably, it lacks significant anti-inflammatory properties, as it exerts minimal impact on peripheral prostaglandin production [53,54]. Another proposed mechanism involves modulation of the serotonergic system. A study by Pickering et al. (2006) demonstrated that administration of 5-HT3 antagonists attenuated the analgesic effects of paracetamol, implicating serotonergic pathways in its mechanism of action. Additionally, paracetamol has been shown to increase serotonin levels in the spinal cord, which may further contribute to its analgesic efficacy by reducing pain perception [56]. A third mechanism centers on activation of the endocannabinoid system. Paracetamol is metabolized to AM404 (N-arachidonoylphenolamine), a compound that acts as an agonist at CB1 cannabinoid receptors and inhibits anandamide reuptake. These actions are thought to mediate central analgesic effects through modulation of the endocannabinoid system [57].

Finally, there is emerging evidence that AM404 interacts with TRPV1 (transient receptor potential vanilloid 1) ion channels and may influence opioid pathways. These interactions suggest a multifaceted mechanism, whereby paracetamol exerts its analgesic effects through a combination of serotonergic, cannabinoid, TRPV1, and potentially opioid-mediated pathways involved in central pain processing [58].

Paracetamol was historically recommended as a first-line treatment for acute non-specific low back pain due to its favorable safety profile and widespread availability. However, recent evidence has challenged its efficacy in this indication. The most influential study in this context, the randomized controlled trial PANSAID (Williams et al., 2014), demonstrated that paracetamol did not significantly accelerate recovery in patients with ALBP compared to placebo. Participants received paracetamol either regularly or as needed over a 4-week period, yet the median time to recovery remained similar across all groups (~17 days), regardless of dosing regimen [59]. A 2016 meta-analysis further supported these findings, indicating no clinically significant benefits of paracetamol in ALBP management. The authors found no substantial improvements in pain intensity, physical function, or global patient perception of improvement when compared with placebo [60]. When used within recommended limits (up to 4 g per day in adults), acetaminophen is generally well tolerated, with a low incidence of gastrointestinal or cardiovascular adverse effects. Nevertheless, its primary safety limitation remains hepatotoxicity. The risk of liver injury increases with overdose, prolonged use, alcohol abuse, malnutrition, and preexisting liver dysfunction. In such cases, the maximum daily dose should be reduced to 2–3 g [61,62].

Despite its limited effectiveness, paracetamol continues to be utilized in clinical practice, particularly in patients for whom nonsteroidal anti-inflammatory drugs (NSAIDs) are contraindicated, such as those with peptic ulcer disease or renal insufficiency. However, current guidelines-including those issued by NICE (2020) and the American College of Physicians (ACP, 2017)-no longer recommend routine use of paracetamol as a first-line therapy for acute LBP in the absence of red flag symptoms [37,63].

NSAIDs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used in the management of low back pain. Their therapeutic effects are primarily mediated through the inhibition of the cyclooxygenase-2 (COX-2) enzyme, which leads to a reduction in the synthesis of pro-inflammatory prostaglandins. This mechanism results in decreased inflammation, swelling, and nociceptor sensitivity, thereby alleviating pain and improving motor function [64, 65]. Additionally, NSAIDs may exert central effects by reducing prostaglandin production within the spinal cord [66].

NSAIDs, like acetaminophen, are frequently prescribed to patients with back pain. However, due to their less favorable safety profile and associated contraindications, most clinical guidelines recommend their use as second-line therapy. While numerous studies confirm the efficacy of NSAIDs in short-term symptom relief, the magnitude of this effect often does not reach the threshold of clinical significance. A 2020 Cochrane review, comprising 32 randomized controlled trials involving 5,356 patients with ALBP, found that NSAIDs were slightly more effective than placebo in reducing pain and improving function over the short term (up to three weeks) [67].

The mean difference in pain intensity was 7.3 points on a 100-point scale, while functional improvement was 2 points on a 24-point disability scale [68]. However, these differences are generally regarded as clinically insignificant.

No significant differences in efficacy were found between selective COX-2 inhibitors and non-selective NSAIDs, nor were there meaningful differences in the incidence of adverse effects between these groups [67]. The effectiveness of NSAIDs in the treatment of chronic LBP was assessed in a 2016 Cochrane review, which included 13 trials and suggested that NSAIDs may be slightly more effective than placebo in reducing pain and improving function. Nevertheless, the effect size was small, and the quality of evidence was rated as low. When only studies with a low risk of bias were considered, the differences between NSAIDs and placebo diminished further [69]. The analgesic efficacy of NSAIDs and acetaminophen in the management of chronic low back pain appears to be comparable [70].

NSAID therapy carries a risk of adverse effects, most commonly affecting the gastrointestinal tract. Between 10% and 60% of patients report abdominal pain, diarrhea, or nausea, while serious complications such as gastrointestinal bleeding or perforation may occur in 2–4% of users [71]. Moreover, NSAID use increases the risk of cardiovascular events, including myocardial infarction, stroke, and heart failure—especially with long-term use. Renal complications may also arise, including acute kidney injury and hypertension [72]. Given these risks, it is recommended that NSAIDs be used at the lowest effective dose for the shortest possible duration, and that gastroprotective agents be considered for patients at elevated risk.

Antidepressants

The mechanism of action of antidepressants in the treatment of low back pain is not fully understood; however, their analgesic properties appear to be independent of their antidepressant effects and are thought to be related to the modulation of neurotransmission within the central nervous system. A key component of this mechanism involves the increased availability of neurotransmitters such as serotonin and norepinephrine in the spinal cord, achieved through inhibition of their reuptake. These neurotransmitters are implicated in the descending pain modulation pathway, which plays a critical role in attenuating the transmission of nociceptive signals from the periphery to the brain [73].

A 2003 analysis suggested that tricyclic and tetracyclic antidepressants may produce a moderate reduction in symptoms among patients with chronic back pain. The inhibition of norepinephrine reuptake appears to be particularly relevant to the analgesic effect in these cases, and the observed benefits seem to be independent of the patient's depressive status. A 2024 meta-analysis reported that duloxetine, administered at a dose of 60 mg, consistently provides pain relief and improves quality of life. In contrast, escitalopram offers only marginal benefits and should be considered a third-line treatment. Other agents such as amitriptyline, bupropion, imipramine, and desipramine have shown minimal evidence supporting their efficacy [74]. Similar findings were reported by Ferraro et al., who concluded that in patients with non-specific low back pain, serotonin-norepinephrine reuptake inhibitors (SNRIs) are likely to have a small effect on pain intensity, negligible impact on disability, and are commonly associated with adverse effects [75]. Among the antidepressants studied, duloxetine at a 60 mg dose was the only medication consistently shown to provide sustained analgesic benefit and enhance quality of life [74].

Concerns regarding the safety of muscle relaxants persist, particularly due to the potential for adverse effects. The most frequently reported side effects include drowsiness, dizziness, dry mouth, and fatigue. As a result, the use of muscle relaxants is recommended only in selected cases and for the shortest duration possible.

Opioids

Opioid analgesics belong to a class of potent pain-relieving agents whose mechanism of action is based on interaction with opioid receptors (μ, κ, δ) located in both the central and peripheral nervous systems. Binding to these receptors results in the inhibition of nociceptive signal transmission and modulates both the perception of pain and the emotional response to it. In the context of LBP management, opioids are primarily used in cases of CLBP, particularly when other therapeutic modalities have proven ineffective. Although available evidence suggests that opioids may offer moderate short-term pain relief, their effectiveness compared to other medications, such as nonsteroidal anti-inflammatory drugs (NSAIDs), appears limited, with differences that often do not reach clinical significance [76]. Systematic reviews and meta-analyses confirm that opioids can provide temporary pain relief (up to approximately 12 weeks); however, they show no substantial benefit in terms of quality-of-life improvement, and their efficacy tends to diminish over time [77]. Notably, a significant proportion of patients discontinue opioid therapy due to adverse effects such as drowsiness, nausea, constipation, or symptoms indicative of developing dependence [78]. In the SPACE trial—a randomized clinical study involving 240 patients with chronic back, hip, or knee pain—no significant difference was observed in functional improvement between the opioid-treated group and those receiving non-opioid medications. Moreover, the non-opioid group experienced significantly lower pain intensity and a lower incidence of adverse events than the opioid group [79].

Muscle Relaxant

The effectiveness of muscle relaxants in the treatment of acute low back pain has been confirmed by numerous research analyses. A systematic review conducted by the Cochrane Collaboration demonstrated that centrally acting muscle relaxants can provide short-term pain relief and improve patient functioning, particularly within the first two weeks of treatment [64]. Among the available agents, cyclobenzaprine is one of the most extensively studied and shows moderate efficacy in both alleviating pain symptoms and improving sleep quality in patients with ALBP [80]. Compared to other pharmacological interventions such as NSAIDs or paracetamol, muscle relaxants exhibit comparable effectiveness in short-term pain reduction. Nonetheless, NSAIDs remain the most commonly recommended first-line therapy due to their anti-inflammatory properties and more favorable safety profile. According to a meta-analysis published in *BMJ* (2021), both NSAIDs and muscle relaxants provide a small but statistically significant improvement in pain intensity among patients with ALBP, with no clear superiority of one drug class over the other [81]. From a safety perspective, the use of muscle relaxants is associated with a considerable risk of adverse effects, including drowsiness, dizziness, dry mouth, and impaired concentration. These side effects limit the feasibility of long-term use, particularly in older adults, in whom the risk of falls and cognitive impairment is elevated [82]. Consequently, muscle relaxants are recommended only for short-term use, typically not exceeding one to two weeks.

Conclusion

Low back pain represents one of the most prevalent and burdensome musculoskeletal disorders globally, with a rising incidence and substantial social and economic costs. Its etiology is multifactorial, encompassing mechanical, neuropathic, and psychosocial causes, while the clinical presentation may be influenced by comorbid factors such as depression, stress, and lifestyle.

This paper discusses comprehensive, non-surgical approaches to the treatment of low back pain, with particular emphasis on pharmacological methods. Among the most commonly employed medications are nonsteroidal anti-inflammatory drugs (NSAIDs), paracetamol (acetaminophen), muscle relaxants, opioids, and certain classes of antidepressants. An analysis of the available evidence indicates that, although many of these agents may provide short-term pain relief, their long-term efficacy is often limited and frequently inadequate. NSAIDs demonstrate moderate effectiveness and remain the cornerstone of pharmacological therapy; however, prolonged use is associated with a risk of adverse effects. Paracetamol, despite its favorable safety profile, has not been shown to confer clinically significant benefits in the management of acute low back pain. Muscle relaxants may be effective in alleviating symptoms during the initial phase, yet their use is constrained by side effects. Opioids, although potent analgesics, are reserved for a select group of patients due to their high potential for dependence and limited long-term efficacy..

In light of current guidelines and systematic reviews, non-surgical management of low back pain should be based on an individually tailored therapeutic plan that integrates pharmacological treatment, physiotherapy, psychological interventions, and patient education. Effective therapy should focus not only on pain reduction but also on restoring function and preventing recurrence, while minimizing the risk of adverse effects. Given the growing prevalence of LBP, the promotion of a biopsychosocial approach is of paramount importance, alongside continued research into the efficacy and safety of both existing and emerging therapeutic modalities.

Disclosure

The authors confirm contribution to the papers as follows:

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