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Chronic Low Back Pain and Radiculopathy: An Overview, Key Points, and Treatment Approaches

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Abstract

Low back pain (LBP) is a major health, social, and economic concern worldwide. It was once believed to be mainly caused by nerve compression from a degenerated intervertebral disc (DID), but it's now seen as a complex condition involving mechanical, inflammatory, compressive, immune, and degenerative factors. Even with medical progress, treatment remains tough, as many options offer limited relief or cause unwanted side effects. When standard methods don't work, surgery is often considered, though it has its own risks and limits. This review examines the pathophysiology and the role of inflammation in the DID and nerve root damage, and presents a minimally invasive, outpatient treatment aimed at targeting both the disc and affected nerves to reverse the problem rather than just easing compression. In a study of 88 patients followed over one and two years, results showed significant improvements in pain, neuropathic symptoms, and disability, with no reported side effects.

Keywords: Chronic low back pain, Radiculopathy, Intervertebral disc, intervertebral disc degeneration, Failed back surgery syndrome.

1. Introduction and Background

LBP is one of the most common and impactful health issues worldwide, carrying significant clinical, social, economic, and public health burdens (1). People with chronic low back pain and radiculopathy (CLBP-R) often face ongoing pain, reduced mobility, and a lower quality of life, which come with substantial personal and societal costs (2). The World Health Organization (WHO) defines LBP as pain or discomfort between the lower ribs and the gluteal folds, sometimes radiating to the legs (3). According to the 2025 Global Burden of Disease Study, LBP is the leading cause of years lived with disability globally (4), with its growing prevalence partly tied to increased life expectancy. In 2013, spinal disorders like neck and back pain ranked as the

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third most expensive health conditions in the U.S., after diabetes and heart disease. By 2016, about one in ten Americans—roughly 31.6 million people—were living with CLBP, with both cases and related costs continuing to climb (5). The National Institutes of Health Research Task Force defines CLBP as pain lasting at least three months and present on at least half the days in the past six months (6), This highlights that CLBP is more than just a symptom-it's a disease with identifiable pathological mechanisms. Radiculopathy (7) often occurs alongside CLBP, caused by impaired conduction along a spinal nerve or its roots, leading to sensory issues like numbness or tingling and motor problems such as weakness, usually in a myotomal pattern. Since Mixter and Barr (8) first linked CLBP and neurological problems to ruptured intervertebral discs in 1934, understanding of the condition has expanded, now recognizing it as a complex disorder shaped by mechanical, inflammatory, compressive, immune, and degenerative factors.

2. Anatomy and Physiology of the Intervertebral Disc

The intervertebral disc (IVD) is an important cartilaginous joint in the spine, designed to handle compressive forces while providing both strength and flexibility. Each disc has three main parts: the nucleus pulposus (NP), a soft, gel-like center; the annulus fibrosus (AF), a tough, fibrous ring; and the cartilaginous endplate (CEP), which covers the top and bottom surfaces of the disc (9). Blood supply only reaches the CEP, while nerve supply mainly comes from the sinuvertebral nerve. The main biochemical components include collagen fibers, elastin fibers, and aggrecan (10).

The NP is a gel-like core made of type II collagen fibers, proteoglycans (PGs), and a mucoprotein matrix containing 66–86% water. PGs such as aggrecan and versican attach to hyaluronan and glycosaminoglycans (GAGs) like chondroitin sulfate and keratan sulfate, allowing the NP to draw in and retain water. This creates osmotic pressure that keeps the disc hydrated and expanded, enabling it to absorb and distribute loads evenly across the AF. The NP also contains a small number of chondrocyte-like cells that produce and maintain the extracellular matrix (ECM) (11).

The AF is the tough outer ring made up of 15 to 25 concentric layers of collagen fibers, arranged in alternating directions to provide both strength and flexibility. The outer layers contain Sharpey type I collagen fibers that anchor to nearby ligaments and vertebrae, while the inner layers are rich in type II collagen and chondrocytes. Trans lamellar bridges connect these layers, adding stability and helping the AF resist stress (12).

The CEP is made up of upper and lower cartilage layers, each about 0.6 mm thick, covering the top and bottom of the disc, with calcified cartilage next to the bone. These structures help nourish the disc, distribute mechanical loads, and prevent bulging into the vertebral body. Their size, shape, and composition are essential for proper function, and when they deteriorate, can cause early disc degeneration (13)

Together, the NP, AF, and CEP create a specialized structure that supports spinal stability, mobility, and resilience. Working in harmony is essential for disc health, and their breakdown plays a major role in CLBP-R.

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3. Pathomechanism of Intervertebral Disc Degeneration

Intervertebral disc degeneration (IDD) is multifactorial process due to various risk factors like genetics, mechanical stress, trauma, and smoking (14). These biological and environmental influences reduce cell numbers and alter disc cells through different molecular processes, leading to less ECM production because of higher breakdown activity and lower building activity. Over time, the disc's structural integrity is compromised, which speeds up the degeneration process. Nutritional factors play a big role in how degenerative disc disease (DDD) develops. Since the disc is the body's largest organ without its own blood supply, it depends on nutrients diffusing in through the cartilaginous endplates. When these endplates calcify, blood flow is restricted, oxygen levels drop, pH decreases, and metabolism is thrown off. This weakens the disc's structure, making it less able to handle stress and fueling the cycle of degeneration (15).

The intervertebral disc (IVD) is also an immune-privileged organ, which is both a strength and a weakness. Its design keeps NP hidden from the immune system, with certain molecular factors blocking immune cells and cytokines to maintain health and keep inflammation at bay. The AF and CEP, along with these molecular defenses, create the blood–NP barrier (BNB), a strong line of protection. If this barrier is breached, the NP can trigger an autoimmune response, leading to problems like disc degeneration, herniation, sciatica, or even the spontaneous shrinkage of herniated material (16). Understanding this immune privilege could open doors to new ways of treating disc problems.

4. Inflammatory Mechanisms Related to Intervertebral Disc Degeneration

Inflammation plays a key role in the development of IDD. Pro-inflammatory cytokines, especially tumor necrosis factor-alpha (TNF-α), upset the ECM balance by triggering disc cells to release mediators like interleukin-6 (IL-6) and interleukin-8 (IL-8), which fuel the inflammatory process. Substance P, a neuropeptide known for its link to pain perception, also acts as an inflammatory regulator in disc tissue, worsening degeneration (17). Oxidative stress is another major factor in IDD, as reactive oxygen species (ROS) disrupt disc stability by causing apoptosis, cell aging, and metabolic issues, while shifting the microenvironment toward catabolic activity and reducing ECM levels. Together, these changes lead to structural damage, disc degeneration, and chronic pain (18).

Zhou et al. (19) proposed that herniated disc material can release cytokines and chemokines that spark a localized inflammatory response, even without mechanical compression, and that this reaction may persist even after the herniation subsides. This reaction may continue even after the herniation reduces on its own. IL-6 works as both an inflammatory signal and a pain sensitivity modulator, boosting nociceptor activation and adding to neuropathic pain. IL-8 draws in neutrophils and other immune cells, ramping up inflammation. TNF-α not only keeps cytokine production going but also directly activates nociceptors, fueling a cycle of ongoing inflammation and pain. These mediators interact with nerve tissue in complex ways—sensitizing nociceptors, driving neuropathic pain, and contributing to tissue damage, which sustains chronic inflammation. These insights highlight how inflammation plays a major role in the condition's

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development, going beyond the physical effects of disc compression and pointing to possible targets for new treatments (19).

5. Lumbar Radicular Pain

Lumbar radicular pain (LRP) is a nerve-related condition caused by problems in the sensory lumbar nerve roots. It's marked by pain that follows a dermatomal distribution, often accompanied by paresthesia, sensory deficits, muscle weakness, and reduced reflexes. Radiculopathy covers all the symptoms from nerve root issues, while sciatica refers specifically to pain along the sciatic nerve, sometimes with motor symptoms. In both medical and everyday language, "sciatica," "radiculopathy," and "radicular pain" are often used interchangeably (20). LRP happens when changes in the vertebrae, joints, ligaments, or discs—such as degeneration or displacement—narrow spaces in the spine, compressing or irritating nerve roots. This can be due to mechanical pressure, which raises nerve pressure, limits blood flow, and causes ischemia, or biochemical factors, where inflammation from disc material or tissue damage releases substances that worsen nerve injury (21).

Nerve roots are especially vulnerable due to their structure. Unlike peripheral nerves, they don't have a perineurium, giving them less tensile strength and weaker diffusion barriers. Their epineurium is also thinner, offering little protection from outside pressure. Plus, poor lymphatic drainage slows the removal of inflammatory substances, raising the risk of fibroblast invasion and intraneural fibrosis (20). Lumbar radiculopathy can result from mechanical issues, chemical irritation, or both. Mechanical injury boosts pressure in the dorsal roots and dorsal root ganglia (DRG), leading to ischemia and neuropathic pain, while chemical injury stems from inflammation around the dorsal root or DRG, with cytokines and other mediators sparking inflammatory pain (21). Peripheral sensory neurons, especially nociceptors, react to mechanical, inflammatory, and thermal signals. These signals travel from nerve endings through the DRGs to the spinal cord, brainstem, thalamus, and cortex, creating the sensation of pain. In disc degeneration, new pain-sensitive fibers grow into the inner AF and CEP toward the NP, broadening the area where pain can start. Both mechanical and chemical factors can change nociceptor function, increasing sensitivity and fueling CLBP (22).

6. Treatment Interventions:

A. **Symptomatic:** Management of CLBP-R usually starts with conservative, symptom-focused care lasting about six weeks. This often includes medications like NSAIDs, acetaminophen, muscle relaxants, and, in some cases, short courses of opioids, paired with non-drug approaches such as physical therapy (23).

B. **Anticonvulsants:** Gabapentin and pregabalin have become more common in recent years, though evidence for their effectiveness is limited. A systematic review and meta-analysis found them ineffective for LBP or lumbar radicular pain, and strong evidence links gabapentinoids to a higher risk of side effects (24).

C. **Antidepressants:** Around 75% of clinical guidelines recommend antidepressants for LBP, with duloxetine (a serotonin-noradrenaline reuptake inhibitor, SNRI) specifically supported by the American College of Physicians. Tricyclic antidepressants (TCAs) and SNRIs may

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help with sciatica, but the certainty of this evidence is low. A recent meta-analysis showed SNRIs offer only modest improvements in pain and disability—changes that aren't clinically significant. While TCAs and SNRIs might have some benefit, their overall effectiveness is uncertain. SNRIs also carry a slightly higher risk of side effects, though not serious ones (25).

7. Surgical Procedures: Facts, Implications, and Socioeconomic Burden

Back in 1934, Mixter and Barr (8) identified a ruptured intervertebral disc as the cause of back pain and related neurological issues, recommending spinal surgery as the solution. Since then, surgery has been the go-to option when conservative care doesn't work (26), though later research showed this approach is more complex than once believed. These days, there's a wide range of surgical options, from traditional open discectomy to minimally invasive procedures like percutaneous endoscopic lumbar discectomy (PELD), percutaneous endoscopic transforaminal discectomy (PETD), and percutaneous endoscopic interlaminar discectomy (PEID). Full-endoscopic discectomy has recently gained popularity for its ability to offer continuous visualization of the spinal canal helping reduce tissue damage and speeding recovery (27). With aging populations and medical advances, these surgeries are becoming more common worldwide. Data from the American Spine Registry shows spinal fusions are especially on the rise in the U.S., with interbody fusions leading the way. Between January 2020 and June 2023, 184,682 spinal surgeries were recorded, including transforaminal/posterior lumbar interbody fusion (32.3%), posterior disc laminectomy (29.5%), and posterolateral fusion (29.2%). Reoperations occurred in 10.4% of cases, often due to infection, wound problems, ongoing pain, or adjacent segment disease (28). While these procedures have benefited some patients, it's fair to wonder why so many different surgical techniques are used to treat this condition. On top of that, they add heavily to the economic strain of CLBP, and the strong reliance on surgery and specialized care drives healthcare costs even higher in the United States. In 2016, ambulatory visits made up 61% of the $22.9 billion spent on treatment. Though less common than other interventions, surgery is the priciest option, averaging $51,500 per admission and topping $10 billion in total costs in 2015 (5).

8. A Proposal for a Creative and Minimally Invasive Outpatient Approach

CLBP-R is a complicated condition involving mechanical, inflammatory, degenerative, and immune-related factors, mainly affecting IVD and sciatic nerve. Despite advances in treatment, it's still challenging to manage, as many current options provide only partial relief and may carry risks or side effects. New insights into the structural and molecular aspects IDD highlight the importance of therapies that address the root causes rather than just the symptoms. Ideally, these treatments should aim to reverse, as much as possible, the structural, biochemical, and immune changes in both the disc and surrounding nerve tissues. Based on this, our recent publication (33) reports the results of a treatment protocol that combines two complementary therapies applied at the same time to both affected regions—the lumbar spine and sciatic nerve—designed to reverse the histopathological damage in degenerated discs and impacted nerve structures.

This protocol consists of 12 therapy sessions, each lasting 20 minutes, one every three days.

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A. **Paravertebral Ozone Therapy (POT):** Involves injecting 10 ml of an oxygen-ozone mixture (27 µg/ml concentration) into the paravertebral lumbar muscles on each side. This aims to reverse the abnormal production of inflammatory molecules from IDDs, modulate the immune response, reduce reactive oxygen species (ROS), and promote contraction of the affected discs.
B. **High-Frequency Low -Level Transcutaneous Electrical Nerve Stimulation (HLF-TENS):** Using four electrodes placed on the skin of each affected limb—one on the lower back and three along the sciatic nerve pathway (thigh, calf, and sole of the foot)—this method delivers non-invasive electrical stimulation to help regulate nerve conduction and lessen pain signals. The goal is to ease discomfort, reduce nerve-related symptoms, and enhance the function of the affected nerves.

This approach works to ease discomfort, improve nerve function, and lessen related symptoms. In a study of 88 patients following this protocol, 80% experienced significant improvements in pain, neuropathic symptoms, and functional ability, with no reported side effects.

This new protocol brings several key benefits. It works through a well-rounded mechanism:

- POT helps fight inflammation and oxidative stress, which drive disc degeneration, while HLF-TENS eases pain signaling and supports nerve recovery.
- It's minimally invasive and done on an outpatient basis, lowering risks, costs, and recovery time.
- It has a strong safety record, with no side effects seen in the treated group.
- Plus, it can be easily rolled out across outpatient clinics, making it available to more patients.

These treatments work together to tackle disc issues, inflammation, and nerve problems, all at once, which main explain the positive results. Being a low-risk outpatient procedure, it's more accessible, costs less than surgery, and is great choice for those who can't or shouldn't have surgery or rely on long term medication.

9. Conclusion

CLBP-R is a complex condition influenced by a mix of mechanical, inflammatory, degenerative, and immune-related factors, mainly affecting the intervertebral discs and sciatic nerve. Treating it effectively means getting to the root of disc and nerve damage. This is important because standard treatments often just ease symptoms, can cause side effects, and don't offer much long-term relief. Surgery can work in some cases, but it's expensive, carries risks, and can sometimes make CLBP harder to handle. The proposed treatment plan focuses on the underlying causes, which might explain the positive outcomes see. It's an outpatient procedure that's both low-risk and affordable. However, larger multicenter studies are still needed to confirm these results.

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