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Clinical Manifestations and Therapeutic Strategies in Lumbar Spondylosis: A Comprehensive Review

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Abstract

Low back pain (LBP) represents a highly prevalent musculoskeletal condition, affecting approximately 60–85% of individuals at some point during their lifetime. In the majority of cases, symptoms are mild and self-limiting, with nearly 90% resolving within six weeks. However, chronic low back pain, defined as pain persisting for more than three months, affects an estimated 15–45% of the population and is associated with substantial impairments in quality of life and significant socioeconomic burden. Despite its high prevalence, the diagnostic evaluation and management of LBP remain heterogeneous and often inconsistent, contributing to increased healthcare costs and variability in clinical practice. This variability is largely attributable to the multifactorial nature of LBP, wherein a definitive etiology is difficult to establish, as nociceptive sources may arise from multiple structures within the axial spine. Consequently, LBP has often been described as "a condition in search of a precise diagnosis."

Following the exclusion of serious underlying pathologies, such as malignancy and vertebral fractures, a broad spectrum of potential causes must be considered, particularly degenerative changes of the spinal column. Notably, conventional radiographic findings frequently demonstrate poor correlation with clinical symptoms, and the causal

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relationships remain inadequately defined. This review aims to elucidate the underlying degenerative mechanisms contributing to LBP and their clinical implications. Furthermore, it critically examines current diagnostic approaches and evaluates the efficacy of existing therapeutic strategies, with the objective of providing a comprehensive and evidence-based perspective on the management of low back pain.

Keywords: Spondylosis, Lumbar discomfort; Degenerative Disorders; Intervertebral osteochondrosis; Osteophyte formation.

Low back pain (LBP) is one of the most prevalent musculoskeletal disorders worldwide, affecting a substantial proportion of the population and representing a leading cause of disability. It is estimated that nearly 60–85% of individuals experience LBP at some point in their lifetime, with a global point prevalence of approximately 7–12%[38,88]. In India, LBP is similarly widespread, with reported prevalence rates ranging from 6.2% to 92% across different population groups, reflecting variability in occupational exposure, lifestyle factors, and study methodologies. It is recognized as a significant public health concern contributing to disability and reduced productivity[52,71]. However, chronic LBP, defined as pain persisting for more than three months, affects nearly 15–45% of individuals and is associated with considerable reductions in quality of life and substantial socioeconomic burden[2,3,83].

Despite its widespread occurrence, diagnostic approaches and therapeutic strategies remain heterogeneous and often inconsistent, contributing to increased healthcare costs and variability in clinical management[23]. This inconsistency largely stems from the difficulty in identifying a singular pain generator, as nociceptive sources are distributed across multiple spinal structures, leading to the characterization of LBP as "an illness in search of disease"[94]. After excluding serious conditions such as malignancy and fractures, numerous etiologies remain, particularly degenerative spinal changes that are often poorly correlated with radiographic findings[9,92].

Degenerative alterations of the lumbar spine encompass a spectrum of structural changes involving vertebrae and intervertebral discs, frequently described using terms such as lumbar osteoarthritis, disc degeneration, degenerative disc disease, and spondylosis. Radiographically, spinal osteoarthritis is characterized by joint space narrowing, osteophyte formation, subchondral sclerosis, and cystic changes[62,81]. Osteophytes are broadly classified into two clinical types. Spondylosis deformans involves anterior and lateral osteophyte formation at vertebral end plates, typically associated with annular ligament stress and often remaining asymptomatic without significant disc height reduction[65,81]. In contrast, intervertebral osteochondrosis is associated with excessive osteophyte formation, disc space narrowing, and vertebral body changes, which may lead to neural compression, radiculopathy, or spinal canal stenosis[24,81].

Degenerative disc disease (DDD) refers to symptomatic degeneration of inter-

vertebral discs, characterized by desiccation, fibrosis, annular disruption, and structural compromise, often accompanied by end-plate dysfunction and osteophyte formation. These degenerative processes frequently coexist with osteoarthritic changes, and the terminology is often used interchangeably in the literature to describe overlapping pathological features. The term "lumbar spondylosis" is therefore broadly applied to encompass degenerative conditions affecting intervertebral discs, vertebral bodies, and associated joints. In this review, spondylosis is considered a multifactorial degenerative process involving interconnected alterations in discs, vertebrae, and neural elements, contributing to axial and radicular pain[29,68].

Epidemiology :

Degenerative changes of the lumbar spine are highly prevalent in the general population. Population-based studies have demonstrated that osteophyte formation is present in up to 85.5% of individuals aged 45–64 years[77], while investigations in adults over 50 years report prevalence rates of 84% in men and 74% in women. Increased prevalence has been associated with higher physical activity levels, elevated body mass index (BMI), and the presence of back pain. Notably, men tend to exhibit more severe and extensive degenerative changes compared to women[58].

Radiological abnormalities are frequently observed even in asymptomatic individuals. Magnetic resonance imaging (MRI) studies reveal disc protrusions in approximately 80% of individuals over 60 years and spinal stenosis in nearly 20%[40]. Additionally, similar rates of disc space narrowing and osteophyte formation have been reported among individuals with varying pain intensities, including those without symptoms[26]. Degenerative changes may also occur in younger populations; for instance, 10% of women aged 20–29 exhibit evidence of disc degeneration[47], while lumbar spondylosis has been reported in up to 3% of individuals in the same age group[65]. These findings highlight the limited correlation between imaging abnormalities and clinical symptomatology.

Pathogenesis :

Degenerative processes of the lumbar spine involve a complex and progressive interaction between intervertebral discs, vertebral bodies, and facet joints[68]. The concept of a "degenerative cascade," as described by Kirkaldy-Willis and Bernard[42], outlines three sequential phases. The dysfunction phase is characterized by repetitive microtrauma leading to annular fissures and end-plate separation, impairing nutrient diffusion and promoting disc desiccation and reduced disc height. Neovascularization and nerve ingrowth into fissured regions enhance nociceptive signalling[13].

The instability phase involves mechanical compromise of the disc, resulting in annular disruption, internal derangement, and progressive facet joint degeneration, which may lead to segmental instability. In the stabilization phase, fibrosis, osteophyte formation, and disc space narrowing occur, contributing to reduced mobility and structural rigidity[43]. Secondary biomechanical alterations include narrowing of the intervertebral canal, ligamentum flavum hypertrophy, and facet joint hypertrophy, which may collectively result in neural compression[68].

Osteophyte formation is driven by complex biochemical processes involving

periosteal cartilage proliferation, endochondral ossification, and mechanical stress adaptations [81]. Cellular mechanisms likely involve mesenchymal stem cells, synovial macrophages, and growth factor-mediated pathways, influenced by mechanical loading and local environmental factors[8].

Clinical Presentation :

Clinical manifestations of lumbar spondylosis arise from nociceptive and compressive mechanisms affecting multiple spinal structures, including intervertebral discs, facet joints, sacroiliac joints, and neural elements. Degenerative changes may lead to spinal canal narrowing and subsequent neurogenic claudication, characterized by back pain, lower limb discomfort, paresthesia, and weakness that worsen with standing or walking and improve with rest[73]. Radiculopathy is another common presentation, resulting from mechanical compression of nerve roots by disc protrusions, osteophytes, or hypertrophied facet joints[49,53,93]. The location and pattern of neural compression depend on the direction of disc bulge or osteophyte extension. Cadaveric studies suggest that significant neural compromise occurs when neuroforaminal dimensions are reduced by approximately 70% or when residual space is less than 30%[65]. Additional radiographic thresholds, such as posterior disc protrusion exceeding 4 mm or foraminal height below 15 mm, are associated with clinically significant nerve impingement[31].

Etiology and Risk Factors :

Age remains the most significant risk factor for degenerative spinal changes, with prevalence increasing progressively across the lifespan[18]. Autopsy studies demonstrate a marked rise in spondylotic changes from early adulthood to advanced age[35, 56], although considerable interindividual variability exists [45]. Degenerative changes may also appear early in life, indicating the influence of additional factors[11,56].

Mechanical and lifestyle factors, including elevated BMI, repetitive spinal loading, occupational strain, and exposure to whole- body vibration, have been implicated in the development and progression of spondylosis[58,85]. However, longitudinal studies suggest that physical activity alone may not strongly correlate with disease progression[32]. Genetic predisposition is also considered an important contributing factor, influencing both susceptibility and severity of degenerative changes. Interestingly, osteophyte formation may represent an adaptive response to spinal instability rather than purely pathological degeneration. Evidence suggests that intervertebral discs possess dynamic remodeling capabilities, and degenerative changes may occur independently or coexist without necessarily producing symptoms[39,81].

Diagnostic Approach:

The evaluation of low back pain begins with a detailed clinical history and physical examination, supplemented by targeted provocative tests. However, diagnostic accuracy is often limited due to the subjective nature of pain and the difficulty in isolating specific anatomical pain generators. Imaging modalities, including plain radiography, computed tomography (CT), and magnetic resonance imaging (MRI), are frequently used to identify structural abnormalities and neural compression. Despite their utility, imaging

findings often correlate poorly with clinical symptoms, and only a minority of patients without neurological deficits can have a definitive diagnosis established through imaging alone[13]. Discrepancies between radiological severity and symptom intensity further complicate interpretation[29]. Adjunctive diagnostic tools, such as electromyography, can help confirm nerve root involvement, while diagnostic injections (e.g., facet joint blocks, sacroiliac injections, or discography) may assist in localizing pain sources by temporarily anesthetizing suspected structures[46] (Figure 1).

Intervention and Treatment Options:

Management of chronic low back pain associated with lumbar spondylosis remains heterogeneous due to the multifactorial nature of the condition and the lack of a definitive treatment algorithm[23]. Therapeutic approaches can be broadly categorized into conservative, pharmacological, interventional, and surgical modalities.

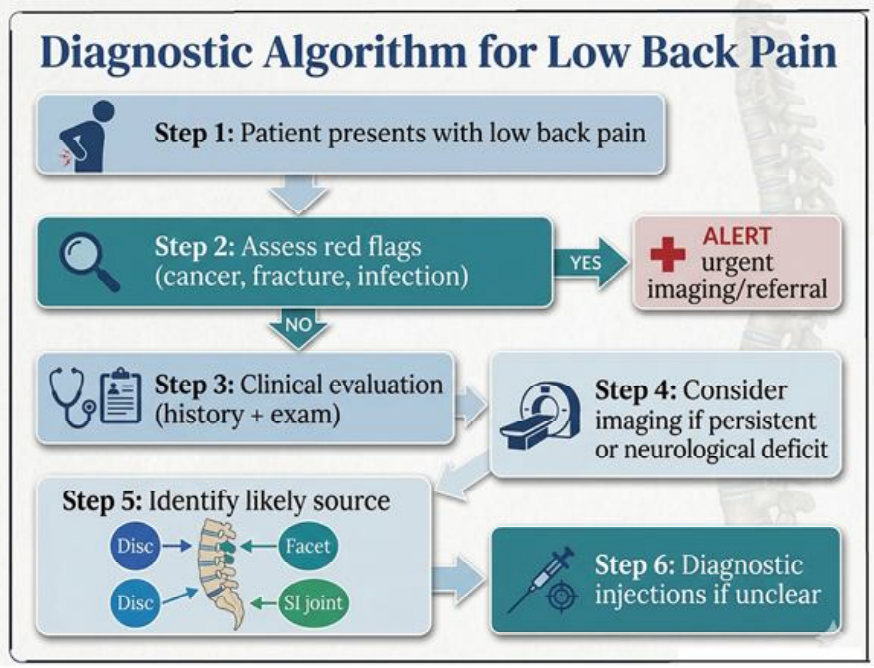

Fig. 1. Diagnosis algorithm for Low Back Pain

Conservative Management:

Exercise therapy is a cornerstone of treatment, incorporating aerobic conditioning, strengthening, and flexibility exercises[34]. Individualized, supervised programs with high adherence demonstrate the greatest benefit, particularly when combined with adjunct therapies such as nonsteroidal anti-inflammatory drugs (NSAIDs) and manual therapy[33]. Other modalities, including transcutaneous electrical nerve stimulation (TENS), lumbar supports, traction, spinal manipulation, and massage therapy, have shown variable and often limited

evidence of efficacy[5,17,22,28,36,57,59,82]. Multidisciplinary approaches integrating psychological and behavioral interventions may provide additional benefit by addressing the biopsychosocial aspects of chronic pain[13,82].

Pharmacotherapy:

Pharmacological management aims to reduce pain and improve function. NSAIDs are commonly used as first-line agents, demonstrating efficacy in chronic low back pain, although gastrointestinal side effects may limit their use[21,37]. COX-2 inhibitors offer similar benefits with reduced gastrointestinal risk but are associated with cardiovascular concerns[82]. Opioids may provide short-term pain relief in selected patients; however, their use is limited by risks of tolerance, dependence, and inconsistent long-term efficacy [25,51,69,78]. Antidepressants and muscle relaxants may also offer modest benefits, particularly in improving pain and functional outcomes[66,67,75] (Figure 2).

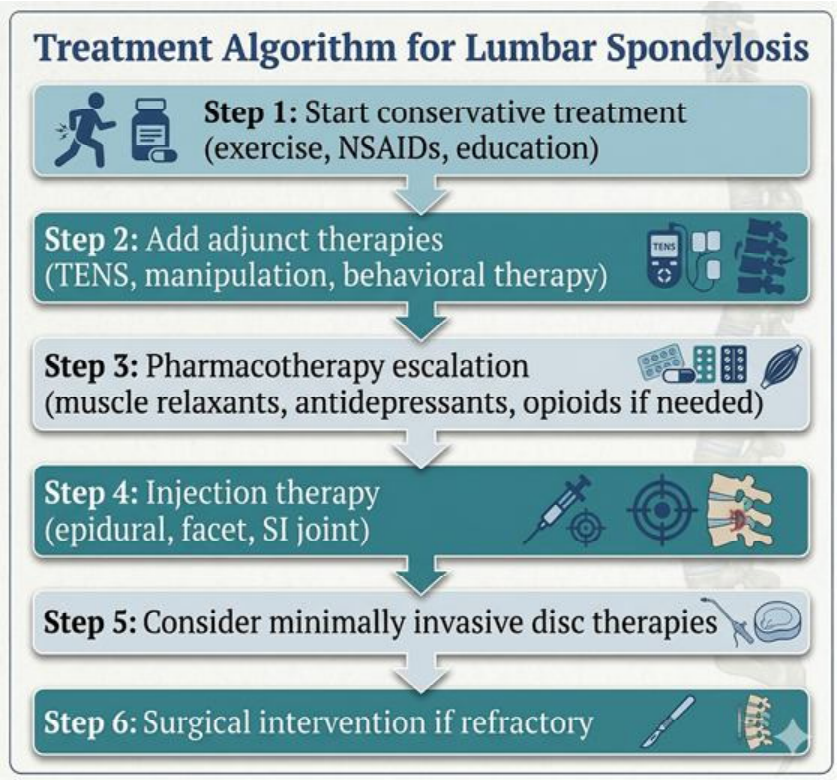

Fig. 2. Treatment algorithm for Lumbar Spondylosis

Injection Therapies:

Epidural steroid injections (ESIs) are widely used for radicular pain and may provide short-term symptom relief, although long-term benefits remain uncertain[1,44,76]. Transforaminal approaches have demonstrated more favorable outcomes in selected cases[16,48,63,64,79,95]. Facet

joint and medial branch block injections are utilized both diagnostically and therapeutically, with moderate evidence supporting short- and long-term pain relief[14,20,27]. Sacroiliac joint injections may also provide symptomatic benefit, although evidence remains limited [30,54,61]. Intradiscal therapies, including intradiscal electrothermal therapy (IDET) and radiofrequency annuloplasty, have shown moderate to limited evidence for discogenic pain management, with associated procedural risks[13, 91].

Surgical Management:

Surgical intervention is reserved for patients with persistent symptoms refractory to conservative treatment. Procedures generally aim at spinal fusion or decompression, depending on the underlying pathology. Fusion is indicated in cases of instability or deformity, while decompression is preferred in conditions involving neural compression, such as spinal stenosis or disc herniation[29]. Although surgical techniques have advanced significantly, their long-term efficacy in managing chronic low back pain remains a subject of ongoing debate (Figure 3).

Controversy surrounding surgical management persists largely due to challenges in comparing existing evidence. Systematic reviews emphasize substantial heterogeneity among studies, including variations in surgical techniques, comparator groups, and limited follow-up durations, often with insufficient emphasis on patient-centered or pain-related outcomes[29]. While certain case series report favorable results[41], high-quality evidence remains inconclusive.

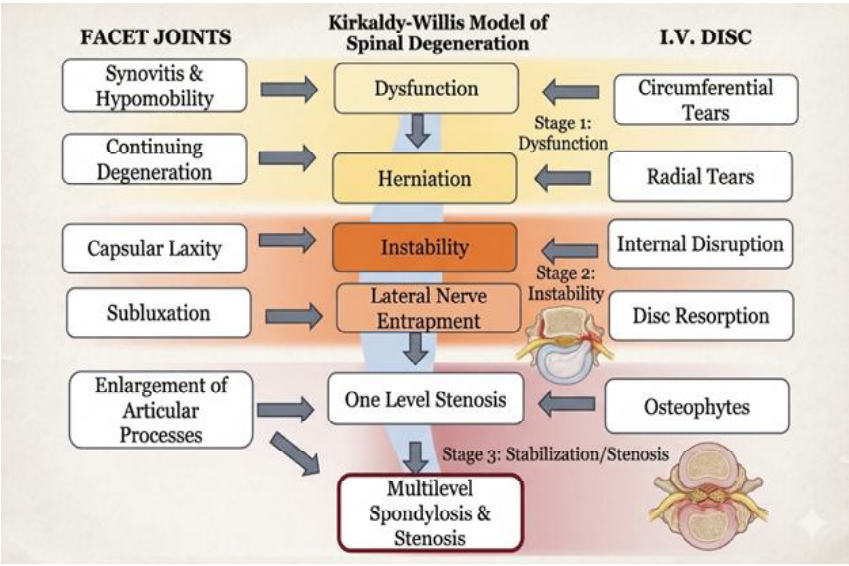

Fig. 3. Spectrum of pathological alterations involving the facet joints and intervertebral disc, along with their dynamic interrelationship. The upper light horizontal bar denotes the dysfunction phase, the middle darker bar represents instability, and the lower dark bar indicates the stabilization phase.

A recent meta-analysis of 31 randomized controlled trials found no clear superiority of any specific decompression technique for spinal stenosis, nor consensus regarding the optimal extent of decompression. Furthermore, current evidence does not strongly support the addition of spinal fusion to decompression in degenerative spondylolisthesis, as it does not consistently improve clinical outcomes or reduce disease progression compared to decompression alone. Similarly, no significant benefit has been demonstrated for fusion over non-surgical management in chronic low back pain, leading to recommendations for cautious and selective use of fusion procedures. Additional long-term studies are required to establish more definitive conclusions regarding optimal surgical strategies[89].

Lumbar spondylosis represents a complex and multifactorial clinical entity, with considerable variability in its definition across the literature. Although radiological identification is relatively straightforward, the high prevalence of degenerative changes across both symptomatic and asymptomatic populations complicates the accurate diagnosis of clinically relevant cases.

Despite extensive investigation, no universally accepted gold-standard treatment has been established, reflecting the heterogeneity of patient presentations and underlying pathophysiological mechanisms. Both conservative and invasive interventions aim to alleviate symptoms and limit disease progression; however, optimal management strategies remain uncertain.

Given the substantial prevalence of low back pain and its significant socioeconomic impact, lumbar spondylosis will continue to be an important focus of research. Advances in genetic studies, identification of risk factors, and development of novel therapeutic approaches are expected to improve disease understanding and contribute to more targeted strategies for prevention, progression control, and symptom management.

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