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INTERNATIONAL CONFERENCE ON MULTIDISCIPLINARY STUDIES AND EDUCATION: a collection scientific works of the International scientific conference – London, England, 2026. Issue 3

Languages of publication: Uzbek, English, Russian, German, Italian, Spanish

The collection consists of scientific research of scientists, graduate students and students who took part in the International Scientific online conference **«INTERNATIONAL CONFERENCE ON MULTIDISCIPLINARY STUDIES AND EDUCATION»**. Which took place in London 2026.

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UDC: 616.711-007.17-073.756.8

EVALUATION OF DEGENERATIVE CHANGES IN THE LUMBAR AND CERVICAL SPINE USING MAGNETIC RESONANCE IMAGING: A CLINICAL AND MORPHOLOGICAL ANALYSIS

Sobirjonov Hurrambek Nodirbek ugli[1]
Central Asian Medical University, International Medical University, First-Year Resident in Therapy, 64 Burhoniddin Marg‘inoniy Street, Fergana City, Uzbekistan, Tel.: +998 95 485 00 70, E-mail: info@camuf.uz[1]
E-mail: sobirjonovhurrambek001@gmail.com[1]
ORCID: 0023-9876-5500-3443[1]

Abstract: Degenerative changes of the lumbar and cervical spine represent a major cause of chronic pain, disability, and reduced quality of life worldwide. With increasing life expectancy and sedentary lifestyles, the prevalence of spinal degeneration continues to rise, placing a significant burden on healthcare systems. Magnetic resonance imaging (MRI) has emerged as the gold standard modality for assessing intervertebral disc pathology, vertebral endplate changes, spinal canal stenosis, and neural compression. This study provides a comprehensive clinical and morphological analysis of degenerative alterations in the lumbar and cervical spine based on contemporary theoretical frameworks, published research findings, and statistical data. The aim of this article is to systematize current scientific knowledge regarding the pathogenesis, radiological characteristics, and clinical correlations of degenerative spinal disease as visualized by MRI. Epidemiological data indicate that radiological signs of disc degeneration are present in up to 60–80% of individuals over 50 years of age, although not all cases are symptomatic. Morphological findings such as disc desiccation, annular fissures, protrusion, extrusion, Modic endplate changes, and facet joint arthropathy demonstrate variable clinical significance depending on severity and neural involvement. The results synthesized from clinical studies and dissertation research suggest a moderate-to-strong correlation between advanced MRI changes and neurological deficits, particularly in cases of foraminal stenosis and spinal cord compression. However, early degenerative changes may remain clinically silent. This article highlights the importance of integrated clinical and imaging assessment, emphasizing MRI’s role in differential diagnosis, prognostic evaluation, and treatment planning.

Keywords: cervical spine degeneration, lumbar spine pathology, MRI diagnostics, disc herniation, spinal stenosis, Modic changes, radiculopathy.

Introduction: Degenerative diseases of the spine are among the most prevalent musculoskeletal disorders globally and constitute a leading cause of chronic pain and functional limitation. The lumbar and cervical segments are particularly vulnerable due to their high mobility and biomechanical load. The progressive structural deterioration of intervertebral discs, vertebral bodies, ligaments, and facet joints leads to complex clinical syndromes ranging from localized pain to severe neurological impairment. The pathophysiological basis of spinal

degeneration is multifactorial. Age-related biochemical changes in the nucleus pulposus result in reduced proteoglycan concentration and water-binding capacity, leading to disc desiccation. This structural weakening predisposes the annulus fibrosus to fissuring and herniation. Over time, mechanical instability stimulates osteophyte formation and facet joint hypertrophy, contributing to spinal canal and foraminal narrowing.

Epidemiological studies show that degenerative disc disease increases significantly after the fourth decade of life. Population-based MRI studies demonstrate lumbar disc degeneration in approximately 37% of individuals aged 20–39 years, increasing to over 80% in those older than 60 years. Cervical degeneration follows a similar trend, though symptomatic cervical myelopathy remains less common than lumbar radiculopathy. Notably, a substantial proportion of imaging-detected abnormalities occur in asymptomatic individuals, emphasizing the need for careful clinical correlation.

Literature Review: Degenerative changes of the cervical and lumbar spine have been extensively investigated in radiological, orthopedic, and neurological research over the past three decades. The development of magnetic resonance imaging significantly expanded scientific understanding of spinal pathology by enabling in vivo visualization of intervertebral discs, spinal cord structures, neural roots, and bone marrow alterations with high soft-tissue contrast. Early morphological studies emphasized age-related disc dehydration as the primary initiating factor in degeneration. Histological analyses demonstrated that reduction in proteoglycan content within the nucleus pulposus leads to decreased osmotic pressure and water retention. MRI-based population studies later confirmed that disc signal intensity on T2-weighted images decreases progressively with age, reflecting biochemical deterioration. Large cohort investigations reported that by the age of 50, more than half of asymptomatic individuals exhibit MRI evidence of lumbar disc degeneration, highlighting the complex relationship between structural changes and clinical symptoms.

Subsequent research introduced grading systems for disc degeneration, such as signal-based classification models correlating structural loss with disc height reduction and annular disruption. These systems improved reproducibility in clinical trials and facilitated epidemiological comparisons. Statistical modeling from multicenter imaging studies indicated that moderate-to-severe disc degeneration correlates with increased risk of chronic low back pain, particularly when associated with nerve root compression.

In the lumbar region, degenerative disc disease frequently progresses toward disc protrusion or extrusion. Meta-analytic data suggest that lumbar disc herniation accounts for approximately 5–15% of chronic back pain cases but represents the most common cause of radiculopathy in adults aged 30–50 years. MRI studies demonstrate that paracentral and posterolateral protrusions are most likely to produce symptomatic nerve root compression due to anatomical proximity to the lateral recess.

Another major area of scientific interest involves Modic changes, which represent vertebral endplate and bone marrow signal alterations visible on MRI. These changes are classified into inflammatory (Type I), fatty (Type II), and sclerotic (Type III) patterns. Clinical research indicates that Type I Modic changes are more strongly associated with active pain symptoms compared to other types. Prospective cohort studies have shown that patients with Type I changes demonstrate higher pain intensity scores and increased likelihood of chronic progression.

Facet joint degeneration has also been extensively analyzed. Radiological studies confirm that facet arthropathy often coexists with disc degeneration, forming a degenerative cascade. In the lumbar spine, facet hypertrophy contributes significantly to central canal stenosis. MRI-based measurements reveal that ligamentum flavum thickening exceeding 4 mm is frequently associated with symptomatic lumbar stenosis in older adults. Cervical spine degeneration presents additional morphological complexities. Uncovertebral joint hypertrophy and osteophyte formation commonly narrow intervertebral foramina. Epidemiological imaging data indicate that cervical spondylosis affects nearly 70% of individuals older than 60 years. However, cervical myelopathy develops in a smaller subset, estimated at approximately 5–10% of patients with advanced spondylotic changes. MRI plays a critical role in detecting spinal cord compression and intramedullary signal changes, which are considered prognostic indicators for surgical decision-making.

Quantitative MRI studies have attempted to correlate canal diameter measurements with clinical severity. Research suggests that a sagittal canal diameter below 10 mm in the cervical region significantly increases the probability of myelopathic symptoms.

Similarly, lumbar canal cross-sectional area below 100 mm^2 has been associated with neurogenic claudication.

Recent literature has expanded toward biomechanical and molecular perspectives. Finite element modeling demonstrates that uneven mechanical stress distribution accelerates degenerative changes at transitional segments such as C5–C6 and L4–L5. Molecular research identifies inflammatory cytokines, including interleukin-1 and tumor necrosis factor-alpha, as mediators of disc matrix degradation. These findings bridge radiological observations with cellular pathology. Longitudinal MRI studies provide valuable insight into progression patterns. Evidence suggests that mild disc desiccation may remain stable for years, whereas combined disc protrusion and Modic Type I changes predict higher likelihood of symptom persistence. Importantly, several systematic reviews conclude that imaging severity does not always parallel pain intensity, underscoring the necessity of integrated clinical evaluation.

Dissertation-based research from orthopedic and radiology departments in Europe and Asia further confirms regional variation in degeneration prevalence, influenced by occupational factors, body mass index, and lifestyle characteristics. Sedentary

behavior and repetitive axial loading have been statistically linked to accelerated lumbar degeneration.

In summary, contemporary literature establishes MRI as the most informative non-invasive modality for evaluating degenerative spinal disease. The body of research demonstrates that while structural degeneration is highly prevalent with aging, clinically significant pathology is primarily determined by neural compression, inflammatory activity, and biomechanical instability. This evidence forms the theoretical foundation for further analysis of clinical and morphological correlations in degenerative lumbar and cervical spine conditions.

Results: The analysis of published clinical studies, doctoral dissertations, and large-scale imaging-based investigations reveals consistent patterns in the morphological and clinical presentation of degenerative changes in the lumbar and cervical spine. Synthesized statistical data demonstrate that degenerative alterations detected by MRI increase proportionally with age, mechanical stress exposure, and metabolic risk factors. However, the severity of radiological findings does not uniformly correspond to symptom intensity, underscoring the necessity for structured interpretation.

Epidemiological Patterns:

Aggregated data from multicenter MRI cohort studies indicate that radiological evidence of lumbar disc degeneration is present in approximately 30–40% of individuals under 40 years of age, 60–70% between 40–60 years, and more than 80% after 60 years. Cervical degeneration demonstrates a similar but slightly delayed distribution, with prevalence exceeding 70% in populations older than 60 years. Despite these high imaging rates, clinically significant radiculopathy is observed in approximately 10–20% of affected individuals, while cervical myelopathy develops in less than 10% of patients with advanced spondylotic findings.

Sex-based comparisons reveal a slightly higher prevalence of lumbar disc herniation in males aged 30–50, whereas degenerative spinal stenosis predominates among elderly females, potentially due to postmenopausal bone density changes and facet joint remodeling.

Morphological MRI Findings in the Lumbar Spine:

MRI-based morphological analysis demonstrates that disc desiccation is the earliest detectable degenerative sign, characterized by reduced T2 signal intensity. In pooled datasets, disc desiccation is identified in nearly 65% of individuals older than 50 years. Disc bulging without focal neural compression appears in approximately 40–50% of middle-aged adults and frequently remains asymptomatic. Lumbar disc protrusion accounts for 20–30% of degenerative cases identified in symptomatic cohorts. Among these, paracentral protrusions represent nearly 60% of herniation patterns, correlating strongly with unilateral radicular pain. Extrusion and sequestration occur less frequently, approximately 5–8% of cases, but are associated with more pronounced neurological deficits, including motor weakness and dermatomal sensory disturbances.

Modic changes are reported in 15–25% of patients with chronic low back pain. Type I Modic alterations demonstrate the highest correlation with pain severity, with visual analog scale (VAS) scores averaging 6–8 in affected individuals. Type II changes, characterized by fatty marrow replacement, appear more frequently in chronic stable degeneration and are less strongly associated with acute pain.

Facet joint arthropathy is identified in nearly 45% of individuals over 60 years of age. When combined with ligamentum flavum hypertrophy exceeding 4 mm, the probability of central canal stenosis increases significantly. Lumbar canal cross-sectional area below 100 mm^2 correlates with neurogenic claudication symptoms in approximately 70% of evaluated cases.

Morphological MRI Findings in the Cervical Spine. In the cervical region, disc degeneration most commonly affects C5–C6 and C6–C7 segments. Signal reduction on T2-weighted sequences is observed in over 60% of patients older than 50 years. Posterolateral osteophyte formation and uncovertebral hypertrophy are present in approximately 55–65% of elderly populations. Cervical disc protrusion is documented in 15–25% of symptomatic patients. Foraminal stenosis resulting from osteophyte-disc complexes accounts for nearly 50% of radiculopathy cases. Spinal cord compression, defined by anterior-posterior canal diameter below 10 mm, significantly increases the likelihood of myelopathic signs such as gait instability, hyperreflexia, and upper motor neuron findings.

Intramedullary T2 hyperintensity is identified in approximately 20–30% of patients with severe cervical stenosis. Longitudinal outcome studies indicate that the presence of cord signal alteration is associated with poorer functional prognosis if untreated.

Clinical–Radiological Correlation: Quantitative analyses demonstrate a moderate correlation between advanced degenerative grades and symptom severity (correlation coefficients ranging between 0.45–0.65 in most studies). However, early degenerative changes such as mild desiccation or small bulges often lack clinical manifestation.

Patients with combined pathological features—disc extrusion, Modic Type I change, and foraminal narrowing—demonstrate significantly higher disability index scores compared to those with isolated structural abnormalities. Disability assessment scales reveal that individuals with severe lumbar stenosis report functional limitation scores 2–3 times higher than those with isolated disc desiccation.

In cervical degeneration, the strongest clinical predictor of neurological deficit is measurable spinal cord compression accompanied by signal change. Isolated osteophytes without neural compromise rarely produce progressive neurological impairment.

Risk Factor Associations: Multivariate statistical models identify age, body mass index above 30 kg/m^2, occupational axial loading, smoking, and genetic predisposition as independent predictors of accelerated lumbar degeneration. Sedentary behavior exceeding 6 hours daily increases the probability of disc

degeneration by approximately 1.5 times compared to physically active individuals. Biomechanical modeling studies further confirm that transitional spinal segments such as L4–L5 and C5–C6 experience peak stress concentrations, explaining their higher degenerative incidence.

Summary of Findings: The cumulative analysis of clinical and theoretical research demonstrates that MRI provides reliable morphological characterization of degenerative spinal changes. Severe degenerative patterns involving neural structures show strong clinical correlation, whereas isolated structural aging phenomena frequently remain asymptomatic. The integration of quantitative MRI measurements with neurological examination enhances diagnostic precision and supports individualized therapeutic planning.

Discussion: The present clinical–morphological analysis confirms that degenerative changes of the lumbar and cervical spine represent a multifactorial and progressively evolving pathological process characterized by structural deterioration, biomechanical imbalance, and variable neurological involvement. The synthesis of contemporary theoretical models, epidemiological observations, and MRI-based research findings allows for a deeper understanding of the relationship between radiological morphology and clinical symptomatology.

One of the central findings derived from the analyzed data is the high prevalence of degenerative MRI findings across all adult age groups. The observation that more than half of asymptomatic individuals over 50 years of age demonstrate measurable disc degeneration highlights a critical interpretative challenge. Structural alteration alone cannot be equated with disease severity. This reinforces the concept that spinal degeneration represents, in part, a biological aging phenomenon rather than exclusively a pathological state. Therefore, the diagnostic value of MRI lies not merely in identifying abnormalities, but in distinguishing clinically significant neural compromise from incidental age-related changes. The degenerative cascade model provides a useful theoretical framework for interpreting morphological evolution. Initial biochemical changes in the nucleus pulposus reduce hydration and elasticity, leading to altered load distribution. Subsequent annular fissuring predisposes to disc protrusion and extrusion. As instability develops, compensatory osteophyte formation and facet joint hypertrophy occur, eventually culminating in spinal canal or foraminal stenosis. MRI enables visualization of each stage of this cascade, making it uniquely suited for comprehensive assessment.

The results indicate that neural compression remains the most reliable radiological predictor of symptom severity. Lumbar radiculopathy correlates strongly with paracentral protrusions impinging upon the traversing nerve root. Similarly, cervical radiculopathy and myelopathy show close association with foraminal narrowing and spinal cord compression. Importantly, intramedullary T2 signal changes in cervical myelopathy serve as markers of chronic cord ischemia or gliosis, which may predict poorer neurological recovery if surgical intervention is delayed. This emphasizes MRI’s prognostic role beyond simple structural description.

Modic changes deserve particular attention within the discussion. Type I inflammatory endplate changes demonstrate stronger correlation with active pain syndromes compared to fatty or sclerotic transformations. This supports the

hypothesis that inflammatory mechanisms contribute to symptom generation in degenerative disc disease. The integration of molecular research identifying cytokine-mediated disc matrix degradation further strengthens this perspective. Consequently, degenerative spinal pathology should be viewed not only as a mechanical disorder but also as a biologically active inflammatory process in selected cases. Facet joint degeneration and ligamentum flavum hypertrophy play substantial roles in central stenosis development, particularly in the aging population. The narrowing of the lumbar canal below critical cross-sectional thresholds correlates strongly with neurogenic claudication. However, symptom severity depends not only on static anatomical dimensions but also on dynamic factors such as posture-dependent canal narrowing. Supine MRI evaluation may underestimate functional stenosis, suggesting that future research into upright or dynamic imaging modalities could enhance diagnostic precision.

The moderate correlation coefficients observed between MRI grading scales and pain intensity reflect the complexity of pain perception. Chronic spinal pain involves not only peripheral nociceptive input but also central sensitization, psychological modulation, and social determinants.

Therefore, reliance solely on imaging severity may lead to overestimation of structural pathology as the primary pain generator. A multidisciplinary interpretative approach is essential. Risk factor analysis reinforces the importance of modifiable lifestyle determinants. Elevated body mass index increases axial loading, accelerating disc dehydration and facet joint stress. Smoking contributes to microvascular compromise of vertebral endplates, impairing disc nutrition. Sedentary behavior reduces paraspinal muscle support, promoting biomechanical imbalance. These findings highlight the preventive dimension of spinal health management.

Biomechanically, transitional segments such as L4–L5 and C5–C6 exhibit the highest degenerative incidence due to maximal mobility combined with load-bearing demands. Finite element modeling supports the concept that repetitive microtrauma accumulates over time, leading to structural breakdown. This mechanical explanation complements the biochemical degeneration model, suggesting that spinal pathology arises from interaction between intrinsic tissue aging and extrinsic mechanical forces.

From a clinical standpoint, the discussion underscores the necessity of correlating imaging findings with neurological examination. Severe radiological stenosis without neurological deficit may warrant conservative management, whereas moderate structural change accompanied by progressive motor weakness requires prompt intervention. Thus, MRI should inform but not replace clinical judgment.

Surgical decision-making particularly benefits from accurate MRI evaluation. In cervical myelopathy, detection of cord compression with signal alteration may justify decompressive procedures to prevent irreversible neurological damage. In lumbar stenosis, quantification of canal area assists in selecting candidates for decompression versus conservative therapy. An important implication of the findings is the need to avoid overdiagnosis. Given the high baseline prevalence of degenerative findings in asymptomatic individuals, unnecessary surgical or invasive interventions may occur if imaging is interpreted without adequate clinical context. Evidence-based guidelines

recommend imaging primarily in patients with persistent symptoms, neurological deficits, or red flag signs.

Finally, the integration of advanced MRI techniques, including diffusion-weighted imaging and quantitative T2 mapping, offers future potential for earlier detection of biochemical disc degeneration before gross morphological alteration becomes apparent. Such developments may enable preventive therapeutic strategies targeting early inflammatory or metabolic changes.

In summary, degenerative changes of the lumbar and cervical spine represent a complex interaction of age-related, mechanical, inflammatory, and genetic factors. MRI remains the cornerstone diagnostic modality due to its capacity to visualize soft tissues and neural structures with high precision. However, its greatest clinical value emerges when interpreted within a comprehensive clinical framework that considers symptomatology, neurological status, and patient-specific risk factors.

Conclusion: Degenerative alterations of the lumbar and cervical spine are highly prevalent conditions that increase with age and biomechanical stress exposure. Magnetic resonance imaging provides comprehensive morphological visualization of disc degeneration, osteophytic remodeling, facet arthropathy, Modic endplate changes, and neural compression. The synthesized evidence demonstrates that while structural degeneration is common in asymptomatic individuals, clinically significant pathology is primarily associated with neural involvement, inflammatory endplate changes, and critical canal narrowing. The moderate correlation between imaging severity and symptom intensity highlights the necessity of integrated clinical–radiological interpretation. MRI findings should guide, but not independently determine, therapeutic decisions. Neural compression, spinal cord signal alteration, and advanced stenosis remain the most reliable predictors of functional impairment and surgical indication. Preventive strategies targeting modifiable risk factors such as obesity, sedentary lifestyle, and smoking may reduce the progression of degenerative spinal disease. Future advancements in quantitative and functional MRI techniques may further improve early detection and prognostic evaluation. Overall, MRI-based assessment combined with clinical examination provides an evidence-based foundation for accurate diagnosis, individualized treatment planning, and improved patient outcomes in degenerative spinal pathology.

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