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# Modic Changes in Lumbar Disc Herniation: Pathophysiology, MRI Classification and Implications for Microdiscectomy; A Narrative Review

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**Abstract**

**Background:** Modic changes are magnetic resonance imaging signal alterations in the vertebral marrow adjacent to the endplates. They reflect interactions between disc degeneration, mechanical injury, inflammation and bone remodeling. Their association with low back pain and potential influence on outcomes after lumbar microdiscectomy remain clinically relevant.

**Objective:** To review the pathophysiology and classification of Modic changes, their diagnostic and clinical implications in lumbar disc herniation, and the considerations relevant to surgical treatment and postoperative outcomes.

**Conclusion:** Modic changes represent a dynamic pathological process rather than isolated imaging findings. Type I is associated with active inflammatory and marrow edema changes, type II with fatty replacement, and type III with sclerosis. Magnetic resonance imaging provides the basis for classification and assessment of coexisting disc herniation. Microdiscectomy addresses neural compression and radicular symptoms, but the contribution of Modic subtype to persistent axial back pain and surgical outcomes remains debated. Further studies stratified by Modic type are needed.

**Keywords:** Modic changes; Lumbar disc herniation; Vertebral endplate; Microdiscectomy

## Introduction

Modic changes (MCs) are MRI signal intensity alterations in the vertebral bone marrow adjacent to the endplates, first described by Modic et al. in 1988. They correlate with specific histopathological changes involving the vertebral endplate and subchondral bone marrow and are considered important imaging markers of intervertebral disc degeneration and chronic low back pain. Their pathogenesis is multifactorial, involving mechanical, inflammatory, vascular, immunological, and genetic factors. [(1)]

The vertebral endplate forms the interface between the intervertebral disc and vertebral body, providing structural support, transmitting axial loads, and facilitating nutrient diffusion to the avascular disc. Degeneration of the intervertebral disc is regarded as the initiating event in Modic changes. Age-related loss of proteoglycans and water from the nucleus pulposus reduces its ability to distribute compressive forces, resulting in abnormal stress concentrations, endplate microfractures, and disruption of the cartilaginous endplate. [(1)]

Overall, Modic changes represent a dynamic pathological process rather than isolated MRI findings. They arise from complex interactions between disc degeneration, vertebral endplate injury, inflammatory activation, and bone remodeling, which collectively determine their progression, clinical manifestations, and potential influence on the outcome of lumbar spine surgery [(1)]

**Pathophysiology and Endplate Degeneration**

Endplate injury increases its permeability, allowing inflammatory mediators and disc matrix components to enter the adjacent vertebral marrow. This stimulates the release of pro-inflammatory cytokines, including TNF-α, IL-1β, and IL-6, leading to bone marrow edema, angiogenesis, osteoclastic activation, and progressive vertebral remodeling, which characterize Modic changes on MRI. [2]

Mechanical loading remains the principal pathogenic mechanism. Repetitive axial loading, obesity, heavy physical activity, spinal instability, and hypermobility increase stress on degenerated discs and vertebral endplates, promoting microfractures and subchondral bone remodeling. Inflammation further accelerates this process through increased expression of cytokines, prostaglandin E2 (PGE2), vascular endothelial growth factor (VEGF), and nerve growth factor (NGF), which promote angiogenesis, neurovascular ingrowth, and pain sensitization. [2]

Additional theories include low-grade bacterial infection by Cutibacterium acnes and autoimmune reactions following exposure of nucleus pulposus antigens after annular or endplate disruption. Although both mechanisms may contribute to persistent inflammation in selected patients, their exact roles remain controversial. [2]

Recent molecular studies have shown that Modic changes result from an imbalance between bone resorption and bone formation, accompanied by increased angiogenesis and neurovascular invasion mediated by VEGF, substance P, and calcitonin gene-related peptide (CGRP). These alterations provide the biological basis for vertebrogenic pain, particularly in active Modic lesions. [2]

The vertebral endplate is a specialized interface located between the intervertebral disc and the vertebral body. It consists of a cartilaginous endplate and an underlying osseous endplate, which together provide mechanical support, distribute axial loads, and regulate the exchange of nutrients and metabolic waste between the vertebral body and the avascular intervertebral disc. Therefore, preservation of endplate integrity is essential for maintaining normal disc metabolism and biomechanics. [2]

Following the loss of the disc's direct vascular supply during early childhood, the intervertebral disc becomes dependent mainly on passive diffusion through the vertebral endplate for nutritional support. Consequently, any alteration in endplate structure or permeability may impair nutrient transport, resulting in reduced disc cell viability, extracellular matrix degradation, and progressive disc degeneration. [2]

**MRI Classification of Modic Changes**

Modic changes are classified into three main types according to their MRI signal characteristics and the corresponding histopathological alterations within the vertebral endplate and adjacent bone marrow. This classification, originally described by Modic et al. in 1988, remains the most widely accepted system for evaluating vertebral endplate marrow changes associated with lumbar disc degeneration. Although the three types represent distinct pathological entities, they are considered different stages of a dynamic remodeling process rather than completely separate diseases. Furthermore, mixed patterns are frequently encountered, reflecting the transition between different stages of degeneration. [3]

Modic type I represents the earliest and most biologically active stage of vertebral endplate degeneration. On MRI, it appears as low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, corresponding histologically to vascularized granulation tissue replacing the normal fatty bone marrow. [3]

This stage is characterized by disruption of the vertebral endplate, bone marrow edema, increased capillary density, inflammatory cell infiltration, and active bone remodeling. [3]

As degeneration progresses, inflammatory marrow changes gradually evolve into Modic type II, the most frequently encountered subtype in clinical practice. On MRI, Modic type II demonstrates high signal intensity on T1-weighted images and isointense or mildly hyperintense signal on T2-weighted images. [3]

Histologically, this stage is characterized by replacement of normal hematopoietic marrow with fatty marrow following resolution of active inflammation. Although inflammatory activity decreases considerably, degenerative changes within the disc and vertebral endplate continue to progress. (3)

The final stage of vertebral marrow remodeling is represented by Modic type III, which is relatively uncommon compared with the other two subtypes. MRI demonstrates low signal intensity on both T1- and T2-weighted images, reflecting dense subchondral bony sclerosis. (3)

Histopathological examination reveals marked trabecular thickening, increased bone formation, extensive mineralization, and replacement of normal marrow by dense sclerotic bone. Vascularity is markedly reduced, inflammatory activity is minimal, and the vertebral endplate becomes mechanically rigid. These changes indicate completion of the remodeling process and restoration of structural stability. (3)

**Table 1. MRI classification of Modic changes (adapted from the classification described in the thesis Review of Literature).**

| Type | T1-weighted MRI | T2-weighted MRI |
|---|---|---|
| **I** | Low signal | High signal |
| **II** | High signal | Isointense or high signal |
| **III** | Low signal | Low signal |

**Modic Changes and Vertebrogenic Low Back Pain**

Chronic low back pain was traditionally attributed to intervertebral disc degeneration; however, increasing evidence indicates that the vertebral endplate and adjacent bone marrow also play a major role in pain generation. Modic changes (MCs) are recognized as imaging markers of pathological alterations at the vertebral endplate–bone marrow interface and are strongly associated with vertebrogenic low back pain. Modic type I shows the strongest correlation with pain because it represents an active inflammatory stage with bone marrow edema and ongoing tissue remodeling. (4)

The vertebral endplate is richly innervated by the basivertebral nerve, which becomes sensitized when endplate degeneration allows inflammatory mediators from the degenerated disc to enter the vertebral marrow. Mechanical disruption also permits diffusion of cytokines and matrix degradation products, stimulating inflammatory cells to release TNF-α, IL-1β, and IL-6, thereby promoting inflammation, extracellular matrix degradation, and bone resorption. (4)

Pain-related mediators including NGF, substance P, CGRP, and VEGF are markedly increased in Modic changes. NGF sensitizes nociceptive fibers, substance P and CGRP enhance pain transmission, while VEGF promotes angiogenesis and neurovascular ingrowth into the damaged endplate, significantly contributing to chronic vertebrogenic pain. (4)

Bone remodeling progresses from inflammatory Modic type I with bone marrow edema to fatty replacement in type II and subchondral sclerosis in type III, explaining the differences in symptom severity among Modic subtypes. Nevertheless, Modic changes may also occur in asymptomatic individuals, indicating that pain depends on factors such as the extent of endplate damage, inflammatory activity, mechanical instability, and individual pain sensitivity. (4)

The concept of vertebrogenic pain distinguishes pain arising from the vertebral endplate and subchondral bone from traditional discogenic pain originating in the annulus fibrosus. The basivertebral nerve is the principal pain pathway, providing the rationale for basivertebral nerve radiofrequency ablation, which has shown significant benefit in selected patients with Modic type I and II changes. (4)

Overall, Modic changes represent active pathological processes involving mechanical injury, inflammation, neurovascular ingrowth, and bone remodeling, rather than simple MRI findings, explaining their close association with vertebrogenic low back pain. [4]

**Clinical Assessment and Magnetic Resonance Imaging**

MRI has evolved into the gold standard neuroimaging study for patients with LDH. CT-myelography was the neuroimaging study of choice through the 1980s. A transition from CT-myelography to MRI occurred in the 1990s. [5]

Magnetic resonance imaging (MRI) is excellent in demonstrating potential causes of nerve root compression, including LDH,Modic Changes , spinal stenosis. Compared to computed tomography (CT), MRI has a significant advantage because of its better soft tissue resolution. (Fig 44 )Encroachment on the spinal canal with inward bulging of discs and yellow ligaments usually plays a significant role in narrowing of the bony spinal canal and also demonstrate the changes that occurs at the level of body of vertebra and end plates which confirm the diagnosis of modic changes and classify them into the three types 1,2,3 and can be depicted excellently by MRI.MRI studies usually encompass a T1- and T2-weighted sagittal and a T2-weighted axial scan [5]

**Management and Microdiscectomy**

Treatment of lumbar disc herniation with Modic changes may be conservative or surgical, depending on symptom severity, neurological deficits, and response to treatment. [6]

Although microdiscectomy is highly effective for radicular pain, its efficacy in patients with different types of Modic changes remains debated. [6]

Therefore, further evaluation of surgical outcomes in LDH patients with various Modic changes is warranted. [6]

Surgical treatment is indicated in patients with persistent radicular pain despite adequate conservative treatment or progressive motor weakness. Surgical options described in the thesis include open discectomy, microdiscectomy, endoscopic discectomy and minimally invasive tubular discectomy. [6]

**Conclusion**

Modic changes reflect complex interactions between disc degeneration, endplate injury, inflammation and bone remodeling. Their MRI classification provides a framework for understanding different vertebral marrow phenotypes. Although microdiscectomy is an established treatment for selected patients with lumbar disc herniation and radiculopathy, the clinical significance of individual Modic types for postoperative back pain and functional outcomes requires further evaluation.

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