

## Advances in Diffusion-Weighted MRI for Quantitative Assessment of Non-Neoplastic Vertebrogenic Low Back Pain

Ahmed Awad Bessar, Khaled Mohamed Shawky, Mai Nabil Mohamed Attia, Rania Mostafa  
Abd El Rahman Hassan

Radiodiagnosis Department, Faculty of Medicine, Zagazig University,  
Corresponding author: Mai Nabil Mohamed Attia

### ABSTRACT

**Background:** Low back pain (LBP) is one of the most common musculoskeletal complaints worldwide, contributing substantially to disability, health care utilization, and socioeconomic burden. A significant subset of patients experiences vertebrogenic low back pain that is non-neoplastic in origin, encompassing degenerative, traumatic, and infectious pathologies of the vertebral column. Magnetic resonance imaging (MRI) is the cornerstone imaging modality for the evaluation of vertebrogenic pain, offering exquisite anatomical detail and sensitivity to subtle bone marrow changes. However, conventional MRI findings often lack specificity and may not reliably differentiate between overlapping etiologies or quantify disease severity, leading to diagnostic challenges and variable clinical management. Diffusion-weighted imaging (DWI) has emerged as a powerful functional MRI technique that characterizes tissue microstructure by assessing the mobility of water molecules. When combined with quantitative analysis using apparent diffusion coefficient (ADC) values, DWI provides objective data that can enhance lesion characterization, distinguish normal from pathological bone marrow, and detect early changes in vertebral microarchitecture before they are apparent on conventional MRI sequences. This quantitative approach has demonstrated utility in differentiating benign from malignant marrow abnormalities, but its role in non-neoplastic vertebrogenic pain remains less clearly defined. The present review aims to synthesize current knowledge regarding the application of quantitative DWI in patients with non-neoplastic vertebrogenic low back pain. We begin by outlining the normal MRI anatomy of the lumbosacral spine and the spectrum of age-related marrow appearances. We then discuss the physical principles of DWI, the interpretation of ADC values in normal vertebrae, and the signal characteristics of non-neoplastic lesions on both conventional and diffusion-weighted MRI. Special attention is given to degenerative changes, traumatic injuries, and spinal infections, each of which can present with overlapping imaging features but may be differentiated through quantitative diffusion assessment. By critically analyzing the available literature, this review highlights the potential of ADC-based evaluation as a quantitative biomarker for vertebrogenic pain syndromes. We also identify gaps in evidence, including variability in acquisition protocols, lack of standardized ADC thresholds, and limited large-scale validation. Future directions emphasize the integration of DWI with multiparametric MRI approaches, radiomics, and artificial intelligence to improve diagnostic accuracy and personalized management in patients with non-neoplastic low back pain.

**Keywords:** Diffusion-Weighted MRI, Non-Neoplastic Vertebrogenic Low Back Pain

### INTRODUCTION

Low back pain (LBP) is a leading cause of disability worldwide and accounts for significant health care expenditure and productivity loss across populations. Epidemiological studies suggest that up to 80% of adults experience at least one episode of LBP in their lifetime, with a substantial proportion of cases attributable to structural abnormalities within the vertebral column [1]. While neoplastic spinal involvement is an important diagnostic consideration, the majority of vertebrogenic pain syndromes encountered in clinical practice are non-neoplastic, resulting from degenerative changes, trauma, or infections. Accurate imaging-based differentiation of these entities is crucial for guiding clinical management and avoiding unnecessary interventions [2].

Magnetic resonance imaging (MRI) remains the modality of choice for evaluating vertebral bone marrow and spinal structures due to its superior soft-tissue contrast and multiplanar capabilities. Conventional MRI sequences—including T1-weighted, T2-weighted, and fat-suppressed techniques—are sensitive to bone marrow alterations but are often nonspecific in their ability to distinguish between overlapping pathological entities [3]. For example, degenerative marrow changes, post-traumatic edema, and early infectious spondylodiscitis may share similar signal characteristics, complicating diagnosis and subsequent therapeutic decisions [4].

Diffusion-weighted imaging (DWI), a functional MRI technique, has gained attention as a complementary tool to conventional MRI in musculoskeletal radiology. By probing the diffusivity of water molecules within tissues, DWI provides information about microstructural integrity and tissue cellularity [5]. Quantitative analysis using apparent diffusion coefficient (ADC) mapping allows objective measurement of diffusion restriction, thereby enhancing lesion characterization. Although DWI has been extensively studied in oncological imaging, its role in non-neoplastic vertebrogenic LBP has not been fully established. Early studies suggest that quantitative ADC values may aid in differentiating between benign and pathological marrow alterations, as well as monitoring disease progression or treatment response [6].

This review aims to critically evaluate the current evidence regarding the use of DWI as a quantitative assessment tool in non-neoplastic vertebrogenic LBP. We seek to highlight the normal MRI anatomy and marrow appearance of the lumbosacral vertebral column, describe the principles of DWI, and discuss the ADC characteristics of normal and diseased vertebrae. Furthermore, we explore the imaging features of degenerative, traumatic, and infectious lumbosacral lesions on conventional MRI and DWI, while outlining the limitations, challenges, and future directions in this evolving field [7].

Non-neoplastic vertebrogenic low back pain refers to pain syndromes arising from structural or pathological changes within the vertebral column that are not related to malignant infiltration. The most common causes include degenerative disorders such as Modic changes, disc degeneration, and facet arthropathy; traumatic lesions such as vertebral fractures and bone contusions; and infectious processes like spondylodiscitis and osteomyelitis [8]. These conditions often result in bone marrow signal alterations on MRI, which may overlap in appearance, posing a diagnostic challenge. Accurate differentiation of these etiologies is critical, as treatment strategies range from conservative pain management to surgical intervention or prolonged antibiotic therapy [9].

The clinical burden of non-neoplastic vertebrogenic low back pain is immense. Degenerative spine disease, in particular, has been strongly linked to chronic pain and disability in aging populations [10]. Traumatic vertebral injuries may occur across all age groups but are particularly significant in elderly patients with osteoporosis, where vertebral fractures often remain underdiagnosed or misinterpreted as degenerative changes [11]. Similarly, infectious spondylodiscitis, although less common, carries a high risk of morbidity if not diagnosed early, especially in immunocompromised individuals [12]. Thus, reliable imaging biomarkers are essential for differentiating between these conditions in order to guide appropriate therapy and improve patient outcomes.

Conventional imaging modalities such as plain radiography and computed tomography (CT) play a limited role in the early diagnosis of vertebrogenic causes of back pain. While CT is highly sensitive for cortical bone changes and fractures, it lacks the ability to assess bone marrow and soft tissue alterations. MRI, with its superior contrast resolution, has emerged as the imaging modality of choice for non-neoplastic spinal conditions [13]. Nevertheless, even with advanced MRI protocols, overlap in signal characteristics between degenerative edema, post-traumatic changes, and infection often complicates interpretation, creating a need for functional imaging parameters such as diffusion-weighted imaging to add specificity [14].

Quantitative assessment using diffusion-weighted MRI provides an opportunity to bridge this diagnostic gap. By evaluating tissue diffusivity through ADC values, DWI has the potential to differentiate between reversible marrow edema and irreversible pathological alterations, as well as between infective, degenerative, and traumatic causes [15]. This approach not only aids in initial diagnosis but may also serve as a valuable tool in monitoring treatment response, particularly in infectious and inflammatory spinal conditions. Consequently, quantitative DWI represents an emerging paradigm in the evaluation of non-neoplastic vertebrogenic low back pain [16].

The lumbosacral vertebral column forms the structural foundation of the lower back, consisting of five lumbar vertebrae (L1–L5) and the sacrum, which articulates with the pelvis. Each vertebral body is composed of an outer cortical shell and an inner trabecular (cancellous) matrix that contains hematopoietic or fatty bone marrow [17]. The intervertebral discs, situated between vertebral bodies, are fibrocartilaginous structures with a central nucleus pulposus surrounded by the annulus fibrosus. These components, together with posterior facet joints and supporting ligaments, maintain spinal stability while allowing mobility [18]. Understanding this anatomy is crucial in interpreting MRI findings and distinguishing normal marrow signals from pathology.

On conventional MRI, vertebral bodies exhibit a characteristic appearance depending on their marrow composition. T1-weighted images typically show a hyperintense signal in fatty marrow and a relatively hypointense signal in red marrow due to its higher water content. T2-weighted and STIR (short tau inversion recovery) sequences enhance the visualization of water content, highlighting marrow edema and pathological changes [19]. The endplates are important anatomical landmarks, and their disruption or alteration may suggest degenerative disease, trauma, or infection. Moreover, recognition of adjacent soft tissue structures such as paraspinal muscles and epidural fat is critical, as these can be secondarily involved in disease processes [20].

The sacrum presents additional diagnostic challenges due to its complex anatomy and heterogeneous marrow composition. The presence of multiple foramina transmitting sacral nerves may complicate interpretation, and signal variations across the sacrum may mimic pathology if normal anatomy is not carefully considered [21]. Similarly, the transitional lumbosacral junction can demonstrate anatomic variants such as sacralization of L5 or lumbarization of S1, which may alter biomechanics and contribute to back pain syndromes [22]. Detailed anatomical knowledge is therefore essential to avoid misinterpretation and to correctly correlate imaging findings with clinical presentations.

Diffusion-weighted imaging overlays this anatomical framework with functional data. ADC values are interpreted in the context of normal vertebral marrow anatomy, as trabecular density, marrow cellularity, and fat content significantly influence diffusion properties [23]. Accurate placement of regions of interest (ROI) during quantitative analysis requires familiarity with vertebral landmarks to avoid artifacts from adjacent discs, vessels, or cortical bone. Therefore, a strong anatomical foundation underpins the correct application and interpretation of DWI in the lumbosacral spine.

### **Normal Age-Related Bone Marrow Appearance and Normal Variants**

Bone marrow undergoes predictable changes throughout life, transitioning from predominantly hematopoietic (red marrow) in infancy to increasingly fatty (yellow marrow) composition with age. In neonates, the vertebral marrow is almost entirely hematopoietic, displaying hypointense signal on T1-weighted images and hyperintense signal on STIR sequences due to high water content [24]. Progressive fatty conversion begins in the appendicular skeleton during childhood and extends to the

axial skeleton, such that by adulthood, vertebral bodies contain a mixture of fatty and hematopoietic marrow. This conversion is heterogeneous and follows a centripetal pattern, with fatty marrow appearing first in the central portions of vertebral bodies and later extending peripherally [25].

On MRI, fatty marrow is characteristically hyperintense on T1-weighted images and relatively hyperintense on T2-weighted sequences, whereas hematopoietic marrow appears isointense to skeletal muscle on T1-weighted sequences and slightly hyperintense on T2-weighted images. With age, the progressive increase in fatty content results in higher T1 signal intensity and decreased conspicuity of hematopoietic islands [26]. Importantly, these age-related changes may mimic pathological processes if not recognized, particularly when patchy reconversion occurs in response to increased hematopoietic demand such as anemia, smoking, or chronic illness [27].

Normal variants of vertebral marrow signal include focal fatty marrow deposition, red marrow islands, and heterogeneous distribution patterns that are often symmetrical and follow predictable anatomical locations. Focal fatty deposits appear as sharply marginated, T1-hyperintense areas without mass effect, commonly seen adjacent to endplates or within the central vertebral body. Conversely, residual hematopoietic islands may appear as hypointense foci on T1-weighted sequences but typically maintain symmetrical distribution and lack associated soft tissue or cortical changes, helping to distinguish them from neoplastic or infectious lesions [28].

Diffusion-weighted imaging provides additional insights into normal marrow physiology. Fatty marrow demonstrates higher ADC values compared to hematopoietic marrow due to reduced cellularity and greater extracellular space. These physiological variations must be considered when interpreting ADC values, as they influence baseline diffusion parameters across different ages and marrow compositions [29]. Misinterpretation of age-related marrow variations as pathology remains a potential pitfall, underscoring the importance of integrating clinical context, conventional MRI sequences, and quantitative diffusion analysis [30].

### **Basic Principles of Diffusion-Weighted Imaging (DWI)**

Diffusion-weighted imaging (DWI) is an MRI technique that evaluates the random motion of water molecules within biological tissues. This motion, known as Brownian motion, is influenced by tissue microstructure, including cellular density, extracellular space, and the integrity of cell membranes. In highly cellular tissues, such as tumors or inflamed marrow, water diffusion is restricted, resulting in higher signal intensity on DWI sequences and correspondingly lower apparent diffusion coefficient (ADC) values. Conversely, tissues with abundant extracellular space, such as fatty marrow, demonstrate facilitated diffusion and higher ADC values [31].

The technique employs strong diffusion-sensitizing gradients, quantified by the “b-value,” which determines sensitivity to molecular motion. Low b-values (e.g., 0–100 s/mm<sup>2</sup>) reflect both perfusion and diffusion effects, whereas high b-values (e.g., >800 s/mm<sup>2</sup>) primarily capture true diffusion. By

acquiring images at multiple b-values, ADC maps can be generated, offering a quantitative measure of diffusion that reduces reliance on qualitative signal intensity interpretation alone [32]. This quantitative approach is particularly useful in musculoskeletal imaging, where subtle differences in marrow composition and pathology may not be visually apparent on standard sequences.

In the vertebral column, DWI is technically challenging due to susceptibility artifacts, motion from respiration or cardiac pulsation, and the presence of heterogeneous bone marrow composition. Echo-planar imaging (EPI) is the most commonly used sequence for DWI but is prone to distortion, especially in regions adjacent to bone-air interfaces. Newer techniques, such as parallel imaging, readout-segmented EPI, and non-EPI DWI, have improved spatial resolution and reduced susceptibility artifacts, making DWI increasingly reliable for spinal applications [33].

The value of DWI lies not only in detecting lesions but also in providing functional information that complements structural MRI. For instance, in the evaluation of vertebrogenic pain, conventional MRI may demonstrate marrow edema without specificity, while DWI-derived ADC values can help distinguish between degenerative changes, traumatic edema, and early infection. Thus, DWI serves as an indispensable adjunct in the diagnostic workup of non-neoplastic spinal pathology by adding a quantitative layer to qualitative imaging [34].

### **ADC Values of the Normal-Appearing Lumbar Vertebral Column**

The apparent diffusion coefficient (ADC) provides a quantitative metric of water diffusivity within the vertebral marrow and is influenced by marrow composition, trabecular bone density, and vascular perfusion. In healthy adults, normal lumbar vertebrae demonstrate relatively stable ADC values, but these values vary with age, sex, and degree of fatty replacement. Fat-predominant marrow typically exhibits higher ADC values due to greater extracellular space, whereas hematopoietic marrow with higher cellularity and lower fat content demonstrates lower ADC values [35]. This physiological variability must be recognized when interpreting ADC maps to avoid misclassification of normal variations as pathology.

Studies have shown that the mean ADC values of normal lumbar vertebrae range from approximately  $0.25 \times 10^{-3}$  to  $0.45 \times 10^{-3}$  mm<sup>2</sup>/s, though these numbers vary depending on acquisition technique and b-values used [36]. Importantly, inter-individual variation can be significant, particularly in the transition from red to yellow marrow during adulthood. For example, younger patients with hematopoietic marrow may show ADC values approaching the lower end of this spectrum, while older adults with fatty marrow tend to have higher ADC values. These differences underscore the necessity of establishing age-matched reference standards when applying DWI clinically in spinal imaging [37]. Regional differences in ADC values across the lumbar spine have also been documented. Cranial lumbar vertebrae often contain relatively more hematopoietic marrow compared to caudal vertebrae, which show higher fatty content. Consequently, ADC values tend to increase progressively from L1 to

L5, paralleling the natural gradient of marrow conversion [38]. Recognition of these gradients is vital when comparing vertebral levels within the same patient, particularly when investigating focal abnormalities.

Technical factors also play a major role in determining ADC values. Variability in b-values, field strength, and pulse sequence parameters across different MRI platforms can lead to inconsistent measurements. This limits the direct comparison of ADC values across institutions and underscores the need for standardized imaging protocols. Efforts are ongoing to develop reference ADC databases and establish reproducible thresholds that can be applied clinically to distinguish normal marrow from early pathological processes [39].

In summary, ADC values in the normal lumbar spine reflect a complex interplay between age, marrow composition, vertebral level, and technical acquisition factors. A thorough understanding of these influences is essential for accurate interpretation of quantitative DWI and for using ADC as a biomarker in the evaluation of non-neoplastic vertebrogenic low back pain [40].

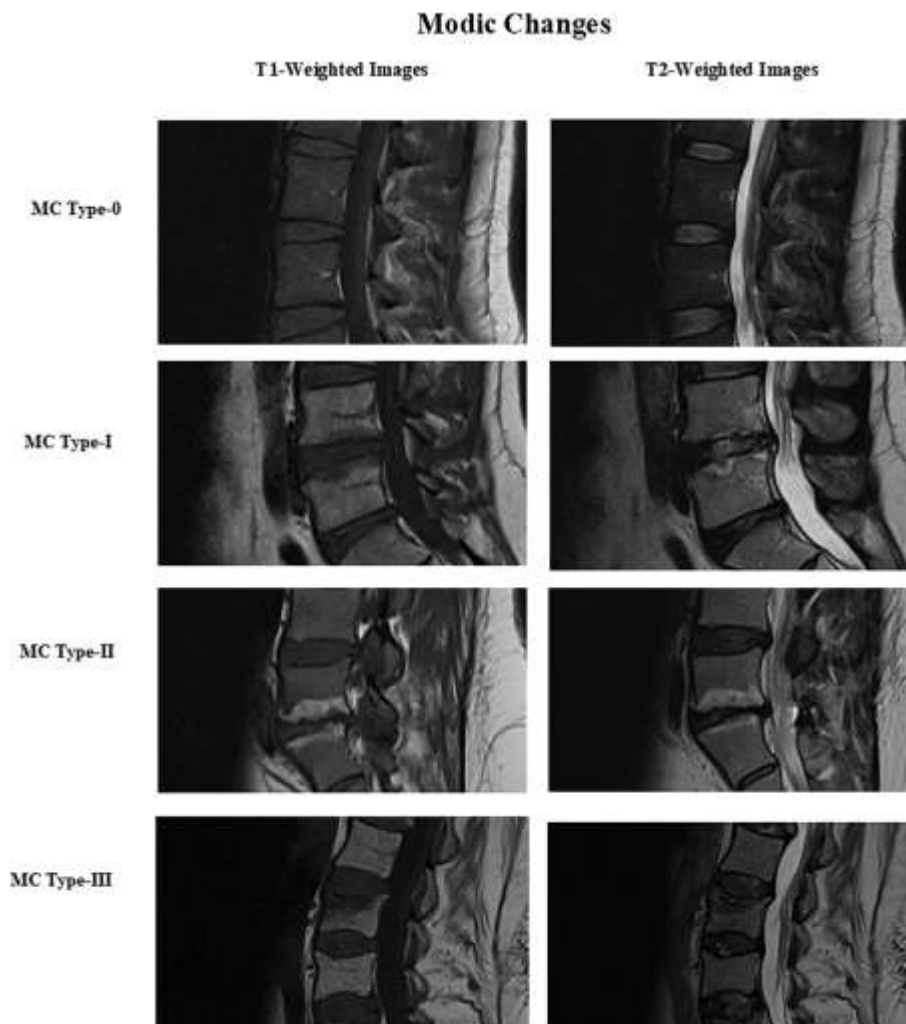
### **Pathology of Non-Neoplastic Lumbosacral Spine Lesions and Their Conventional MRI and DWI-MRI Signal Characteristics**

Non-neoplastic lesions of the lumbosacral spine encompass a broad spectrum of conditions that commonly manifest with low back pain. These include degenerative changes, traumatic injuries, and infections, each of which demonstrates overlapping imaging features on conventional MRI. The challenge for radiologists lies in accurately distinguishing between these entities, as management strategies differ substantially. For instance, degenerative marrow edema may warrant conservative therapy, while infectious spondylodiscitis necessitates prompt antibiotic treatment, and traumatic fractures may require surgical stabilization [41]. Thus, improved imaging biomarkers are essential for precise characterization.

On conventional MRI, bone marrow edema is a common but nonspecific finding. It typically appears hypointense on T1-weighted sequences and hyperintense on T2-weighted or STIR images. This signal pattern is shared by multiple pathological processes, including Modic type 1 degenerative endplate changes, acute traumatic fractures, and early infectious discitis-osteomyelitis [42]. Moreover, chronic degenerative changes such as fatty marrow replacement (Modic type 2) or sclerosis (Modic type 3) can further complicate interpretation by producing atypical appearances. The absence of pathognomonic features on standard sequences underscores the diagnostic limitations of conventional MRI alone [43].

Diffusion-weighted MRI and ADC mapping provide a functional perspective that complements morphologic imaging. In general, infectious lesions tend to show restricted diffusion due to increased cellularity and purulent exudate, resulting in high DWI signal and low ADC values. Traumatic edema, conversely, often demonstrates intermediate ADC values reflecting increased extracellular water

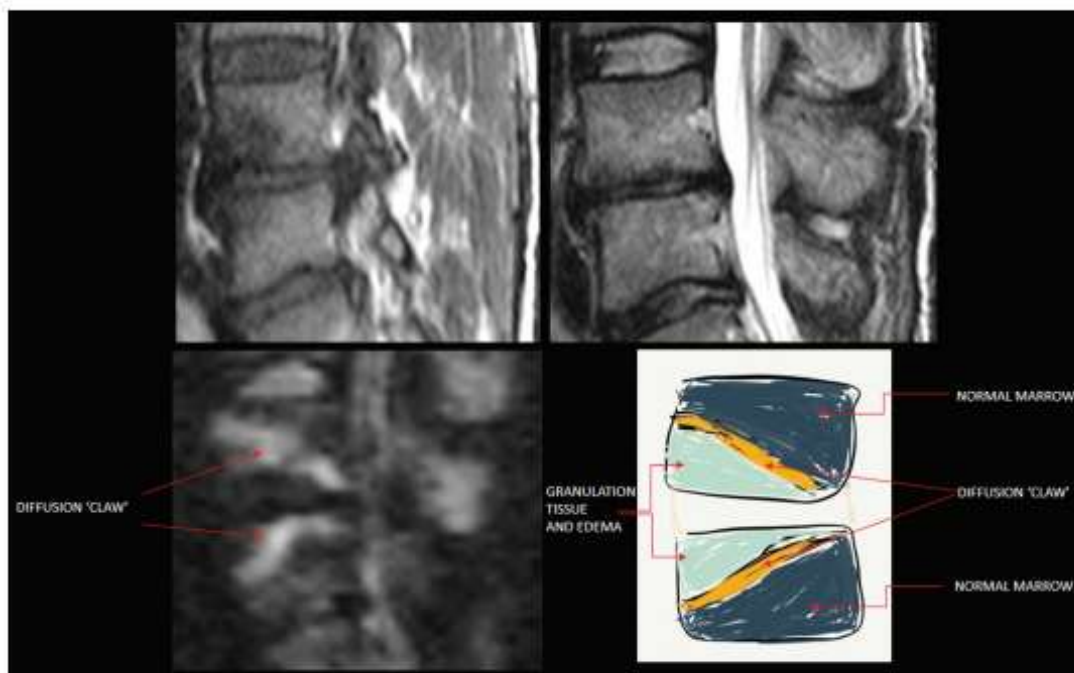
without significant restriction. Degenerative changes such as Modic type 1 lesions typically show elevated ADC values due to marrow hyperemia and inflammatory changes, which help to differentiate them from infection [44]. These quantitative distinctions form the basis of applying DWI as a problem-solving tool in ambiguous cases.



**Figure 1:** The three types of Modic changes compared with normal (also called Modic-0) represent endplate and vertebral bone marrow changes detected by MRI T1-weighted and T2-weighted images. The bone marrow edema (hypointense on T1-weighted and hyperintense on T2-weighted MRI imaging). This is followed by the replacement of red bone marrow with yellow fatty bone (hyperintense both in T1 and T2 weighted images) in Modic type-II changes and subsequently by sclerosis (hypointense in both T1 and T2 images) in Modic type-III changes, usually seen in older ages [44].

Several studies have demonstrated the utility of ADC thresholds in differentiating non-neoplastic marrow lesions. For example, mean ADC values in infectious spondylodiscitis have been shown to be significantly lower than those in degenerative Modic changes, aiding in differential diagnosis [45]. Similarly, ADC measurement has been explored in distinguishing acute osteoporotic fractures from pathological fractures, with benign lesions typically exhibiting higher diffusivity. Despite these promising findings, variability in acquisition protocols and lack of consensus regarding optimal cut-off values limit the widespread adoption of ADC metrics in routine practice [46].

Ultimately, the integration of DWI with conventional MRI provides a multiparametric approach to evaluating non-neoplastic lumbosacral lesions. This combination leverages the strengths of structural imaging for anatomical localization and functional imaging for tissue characterization. Future advances, including harmonized imaging protocols, radiomics, and artificial intelligence, may further enhance the diagnostic utility of diffusion imaging in this setting [47].



**FIG 2.** The claw sign is identified on trace/combined DWI as well-margined, linear, typically paired regions of high signal situated within adjoined vertebral bodies at the boundaries between normal and vascularized bone marrow [44].

### Degenerative Lumbosacral Spine Lesions

Degenerative spinal disorders represent the most frequent cause of non-neoplastic vertebrogenic low back pain. The spectrum of degenerative changes includes intervertebral disc degeneration, endplate alterations, facet arthropathy, and ligamentous hypertrophy, often coexisting in the same patient. On MRI, degenerative lesions are commonly classified by Modic changes, which describe vertebral endplate and adjacent marrow alterations. Modic type 1 lesions demonstrate decreased T1 and increased T2/STIR signal intensity, reflecting marrow edema and inflammation. Type 2 lesions show increased T1 signal due to fatty replacement of marrow, while type 3 lesions display low signal on both T1 and T2 sequences, consistent with sclerosis [48]. Among these, Modic type 1 changes are most closely linked with symptomatic low back pain [49].

From a diffusion-weighted imaging perspective, Modic type 1 lesions generally exhibit higher ADC values than infectious spondylodiscitis, aiding in differential diagnosis. The increased ADC reflects edema and hyperemia rather than restricted diffusion. In contrast, Modic type 2 and 3 lesions tend to

demonstrate relatively stable or slightly elevated ADC values, corresponding to fatty marrow and sclerotic tissue, respectively [50]. Several studies have emphasized that the quantitative assessment of ADC can serve as a distinguishing marker between degenerative and infectious endplate changes, a frequent diagnostic dilemma in clinical practice [51].

Intervertebral disc degeneration also contributes significantly to vertebrogenic pain. Disc desiccation and nucleus pulposus degeneration appear as hypointense signal on T2-weighted images, while annular tears may appear as high-intensity zones within the annulus. DWI has shown potential in assessing disc degeneration severity, with decreased ADC values correlating with reduced water content and altered proteoglycan composition [52]. This quantitative approach provides functional insight into disc health, supplementing conventional structural assessment and potentially guiding therapeutic interventions such as disc repair or regenerative therapies.

Facet joint arthropathy, another common degenerative source of pain, is less well studied with DWI. However, marrow edema adjacent to degenerative facet joints may mimic other pathologies. Here, ADC quantification can help confirm the benign, degenerative nature of marrow signal alterations by demonstrating diffusivity consistent with edema rather than infection or infiltration [53]. Similarly, degenerative marrow signal changes near Schmorl's nodes or osteophytes may be clarified through DWI, minimizing misinterpretation.

Overall, the integration of DWI and ADC mapping into the evaluation of degenerative spine pathology enhances specificity by providing physiologic information that complements structural MRI findings. While conventional MRI identifies the presence of degenerative changes, quantitative diffusion imaging refines their characterization, differentiates them from infection or neoplasm, and offers potential prognostic biomarkers for symptomatic disease [54].

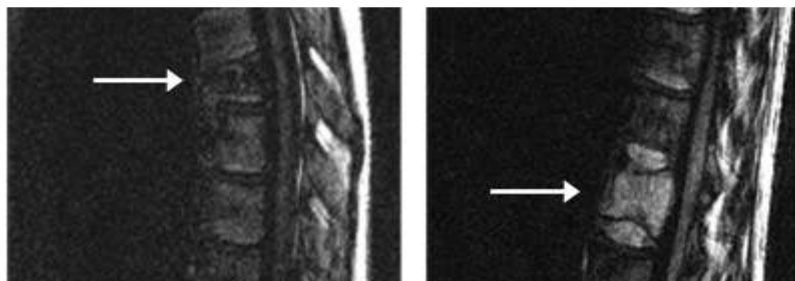
### **Traumatic Lumbosacral Spine Lesions**

Traumatic injuries of the lumbosacral spine encompass a wide range of conditions, from bone marrow contusions and microtrabecular injuries to complete vertebral fractures. MRI is highly sensitive in detecting post-traumatic changes, often identifying marrow edema and soft tissue injury that are occult on radiographs or CT. On conventional sequences, acute traumatic lesions typically manifest as hypointensity on T1-weighted images and hyperintensity on T2-weighted or STIR images, reflecting bone marrow edema and hemorrhage. Differentiating benign osteoporotic fractures from malignant pathological fractures is a frequent clinical dilemma, as both present with similar morphologic features on routine MRI [55].

Diffusion-weighted imaging has been extensively studied in the evaluation of vertebral fractures. Acute traumatic fractures usually demonstrate facilitated diffusion due to extracellular edema, resulting in high ADC values. In contrast, malignant fractures caused by tumor infiltration exhibit restricted diffusion, with significantly lower ADC values due to increased cellular density. This

distinction enhances diagnostic confidence, especially in elderly patients with osteoporosis or known malignancy where fracture etiology is ambiguous [56]. Studies have suggested ADC thresholds as potential cut-offs to differentiate benign from malignant fractures, although variability in acquisition protocols limits universal application [57].

Another important application of DWI in trauma is the detection of bone marrow confusions, which may not be evident on CT. Microtrabecular injuries show increased diffusivity, corresponding to high ADC values consistent with edema rather than restricted diffusion. This capability enables earlier diagnosis, prevents missed injuries, and can influence management decisions, particularly in athletes or patients with high functional demands [58]. Moreover, quantitative ADC assessment may be valuable in monitoring fracture healing. Progressive normalization of ADC values has been correlated with the resolution of marrow edema and tissue repair, suggesting a potential biomarker for treatment response and recovery [59].



**Figure 3:** Diffusion-weighted SSFP acquisitions (at 1.5 T) of a benign and a malignant vertebral bone marrow lesion. The osteoporotic fracture is hypointense relative to the normal-appearing vertebral bone marrow, while the metastatic lesion appears hyperintense [59].

Overall, diffusion-weighted imaging adds diagnostic specificity to conventional MRI in the evaluation of traumatic lumbosacral lesions. By differentiating between benign and malignant fractures, detecting subtle confusions, and monitoring healing dynamics, quantitative DWI extends beyond simple morphological imaging and provides clinically actionable information in spinal trauma [60].

### **Infections of the Lumbosacral Spine**

Infectious lesions of the lumbosacral spine, including spondylodiscitis, vertebral osteomyelitis, and epidural abscesses, represent serious causes of vertebrogenic low back pain. These conditions often present with nonspecific clinical symptoms such as localized pain, fever, or elevated inflammatory markers, leading to delays in diagnosis. Conventional MRI is highly sensitive in detecting spinal infections, demonstrating characteristic findings such as low T1 signal and high T2/STIR signal in the vertebral marrow, loss of endplate definition, and abnormal enhancement following contrast administration. However, overlap in appearance with Modic type 1 degenerative changes or traumatic marrow edema may limit specificity [61].

Diffusion-weighted imaging provides important complementary information in this setting. Infectious lesions typically demonstrate restricted diffusion due to increased cellularity, inflammatory infiltrates, and purulent material. This manifests as high signal intensity on DWI and corresponding low ADC

values. By contrast, degenerative marrow edema and traumatic lesions usually show elevated ADC values, reflecting more free water motion. Several studies have shown that ADC thresholds can help distinguish spondylodiscitis from Modic type 1 changes, thereby improving diagnostic confidence and potentially obviating the need for invasive procedures such as biopsy [62].

Spondylodiscitis often involves both the intervertebral disc and adjacent vertebral endplates. On DWI, infected discs show marked hyperintensity and low ADC values, in contrast to degenerative discs that typically demonstrate decreased T2 signal intensity with relatively high ADC values due to desiccation rather than true restriction. This distinction is clinically important, as misdiagnosis may lead to inappropriate management and progression of infection with potential neurological compromise [63]. Epidural and paraspinal abscesses are also well evaluated with DWI. The pus-filled cavities within abscesses show restricted diffusion with low ADC values, helping to differentiate them from sterile fluid collections such as seromas or degenerative cysts, which demonstrate facilitated diffusion. This functional characterization is particularly valuable in postoperative patients, where distinguishing between abscess and postoperative changes on conventional MRI can be challenging [64].

Beyond diagnosis, quantitative DWI may also have a role in treatment monitoring. Successful antimicrobial therapy has been associated with normalization of ADC values and reduction of restricted diffusion in previously infected regions. Thus, serial DWI may provide a noninvasive biomarker for therapeutic response, reducing reliance on invasive sampling and contrast-enhanced studies [65].

In summary, DWI enhances the evaluation of spinal infections by providing quantitative diffusion metrics that differentiate infectious from noninfectious marrow lesions, characterize abscesses, and monitor treatment response. While conventional MRI remains the primary modality, integrating diffusion imaging significantly improves specificity in the diagnosis of lumbosacral infections [66].

### **Conclusion and Future Directions**

Quantitative diffusion-weighted imaging has emerged as a promising adjunct to conventional MRI in the evaluation of non-neoplastic vertebrogenic low back pain. By providing functional insight into marrow microstructure, ADC measurements add specificity to the morphological findings of routine sequences. This is particularly valuable in differentiating degenerative marrow changes, traumatic edema, and infectious spondylodiscitis, which often overlap in their conventional imaging appearance. DWI thereby strengthens diagnostic accuracy and guides appropriate therapeutic strategies in clinical practice.

In degenerative conditions, ADC mapping helps characterize Modic changes and intervertebral disc alterations, contributing to improved understanding of symptomatic lesions. In traumatic lesions, DWI offers a reliable method for distinguishing benign osteoporotic fractures from malignant pathological fractures, as well as identifying subtle contusions not evident on CT. In infections, diffusion imaging

enhances the specificity of diagnosis, distinguishes abscesses from sterile fluid collections, and enables noninvasive monitoring of therapeutic response. Collectively, these applications highlight the versatility of DWI in addressing the common diagnostic dilemmas associated with vertebrogenic low back pain.

Despite these advantages, several challenges remain before diffusion imaging can be fully integrated into routine spinal imaging protocols. Variability in acquisition parameters, differences in b-values, and lack of standardized ADC thresholds limit reproducibility and generalizability of results across institutions. Furthermore, susceptibility artifacts, motion-related distortion, and heterogeneous marrow composition continue to pose technical difficulties. Addressing these limitations requires harmonization of imaging protocols, development of normative ADC databases, and multicenter validation studies to establish reliable diagnostic cut-offs.

Future directions will likely see DWI incorporated into multiparametric MRI strategies, alongside techniques such as spectroscopy, perfusion imaging, and quantitative T1/T2 mapping. Advances in radiomics and artificial intelligence hold the potential to extract subtle features from diffusion data, enabling predictive modeling and personalized management of patients with low back pain. As these technologies evolve, diffusion-weighted imaging may transition from a problem-solving tool to a core component of spinal imaging, offering clinicians objective biomarkers to enhance diagnosis, guide therapy, and monitor outcomes in patients with non-neoplastic vertebrogenic low back pain.

## REFERENCES

1. Hoy D, March L, Brooks P, et al. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. *Ann Rheum Dis*. 2014;73(6):968-974.
2. Diehn FE. Imaging of spine infection. *Radiol Clin North Am*. 2012;50(4):777-798.
3. Modic MT, Ross JS. Lumbar degenerative disk disease. *Radiology*. 2007;245(1):43-61.
4. Brinjikji W, Diehn FE, Jarvik JG, et al. MRI findings of disc degeneration are more prevalent in adults with low back pain than in asymptomatic controls: a systematic review and meta-analysis. *AJNR Am J Neuroradiol*. 2015;36(12):2394-2399.
5. Padhani AR, et al. Diffusion-weighted magnetic resonance imaging as a cancer biomarker: consensus and recommendations. *Neoplasia*. 2009;11(2):102-125.
6. Castillo M, Arbelaez A, Smith JK, Fisher LL. Diffusion-weighted MR imaging offers no advantage over routine noncontrast MR imaging in the detection of vertebral metastases. *AJNR Am J Neuroradiol*. 2000;21(5):948-953.
7. Takahara T, Imai Y, Yamashita T, Yasuda S, Nasu S, Van Cauteren M. Diffusion weighted whole body imaging with background body signal suppression (DWIBS): technical improvement using free breathing, STIR and high resolution 3D display. *Radiat Med*. 2004;22(4):275-282.
8. Bley TA, Wieben O, Uhl M. Diffusion-weighted MR imaging in musculoskeletal radiology: applications in trauma, tumors, and inflammation. *Magn Reson Imaging Clin N Am*. 2009;17(2):263-275.

9. Jarvik JG, Deyo RA. Diagnostic evaluation of low back pain with emphasis on imaging. *Ann Intern Med.* 2002;137(7):586-597.
10. DePalma MJ, Ketchum JM, Saullo TR. What is the source of chronic low back pain and does age play a role? *Pain Med.* 2011;12(2):224-233.
11. Johnell O, Kanis JA. An estimate of the worldwide prevalence and disability associated with osteoporotic fractures. *Osteoporos Int.* 2006;17(12):1726-1733.
12. Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: update on diagnosis and management. *J Antimicrob Chemother.* 2010;65(Suppl 3):iii11-iii24.
13. Ross JS, Moore KR, Gardner ED, et al. *Diagnostic Imaging: Spine.* 3rd ed. Philadelphia: Elsevier; 2015.
14. Patel ND, Broderick DF, Burns J, et al. ACR Appropriateness Criteria low back pain. *J Am Coll Radiol.* 2016;13(9):1069-1078.
15. Baur A, Dietrich O, Reiser MF. Diffusion-weighted imaging of bone marrow: current status. *Eur Radiol.* 2003;13(7):1699-1708.
16. Byun WM, Shin SO, Chang Y, Lee SJ, Finsterbusch J. Diffusion-weighted MR imaging of vertebral bone marrow: differentiation of benign versus pathologic compression fractures. *Radiology.* 2002;224(2):488-493.
17. Moore KL, Dalley AF, Agur AMR. *Clinically Oriented Anatomy.* 7th ed. Philadelphia: Wolters Kluwer; 2014.
18. Standring S, ed. *Gray's Anatomy: The Anatomical Basis of Clinical Practice.* 41st ed. London: Elsevier; 2015.
19. Vande Berg BC, Malghem J, Lecouvet FE. Magnetic resonance imaging of normal bone marrow. *Eur Radiol.* 1998;8(8):1327-1334.
20. Diehn FE, Maus TP, Morris JM, et al. Imaging of spine pain: diagnostic imaging of degenerative spine disease and its complications. *Radiographics.* 2015;35(5):1439-1462.
21. Tokgöz N, Uçar M, Yıldız H, et al. Variations in sacralization and lumbarization on MRI: prevalence and clinical significance. *Clin Imaging.* 2014;38(6):746-749.
22. Konin GP, Walz DM. Lumbosacral transitional vertebrae: classification, imaging findings, and clinical relevance. *AJNR Am J Neuroradiol.* 2010;31(10):1778-1786.
23. Thoeny HC, De Keyser F. Extracranial applications of diffusion-weighted magnetic resonance imaging. *Eur Radiol.* 2007;17(6):1385-1393.
24. Ricci C, Cova M, Kang YS, et al. Normal age-related patterns of cellular and fatty bone marrow distribution in the axial skeleton: MR imaging study. *Radiology.* 1988;169(3):759-764.
25. Vande Berg BC, Malghem J, Lecouvet FE. Magnetic resonance imaging of normal bone marrow. *Eur Radiol.* 1998;8(8):1327-1334.
26. Griffith JF, Yeung DK, Antonio GE, et al. Vertebral bone marrow fat content and diffusion and perfusion indexes in healthy men and women. *J Magn Reson Imaging.* 2006;23(4):622-628.
27. Zajick DC, Morrison WB, Schweitzer ME, Parelada JA, Carrino JA. Benign and malignant processes: normal values and differentiation with chemical shift MR imaging in vertebral marrow. *Radiology.* 2005;237(2):590-596.
28. Vogler JB, Murphy WA. Bone marrow imaging. *Radiology.* 1988;168(3):679-693.
29. Dietrich O, Baur-Melnyk A, Reiser MF, Baur A. Diffusion-weighted imaging of bone marrow. *Semin Musculoskelet Radiol.* 2009;13(2):134-144.
30. Padhani AR, Koh DM, Collins DJ. Whole-body diffusion-weighted MR imaging in cancer: current status and research directions. *Radiology.* 2011;261(3):700-718.
31. Le Bihan D. Diffusion, perfusion and functional MRI. *Neuroimage.* 2012;62(2):1131-1136.
32. Bammer R. Basic principles of diffusion-weighted imaging. *Eur J Radiol.* 2003;45(3):169-184.
33. Porter DA, Heidemann RM. High resolution diffusion-weighted imaging using readout-segmented echo-planar imaging, parallel imaging and a two-dimensional navigator-based reacquisition. *Magn Reson Med.* 2009;62(2):468-475.

34. Takahara T, Imai Y, Yamashita T, Yasuda S, Nasu S, Van Cauteren M. Diffusion weighted whole body imaging with background body signal suppression (DWIBS): technical improvement using free breathing, STIR and high resolution 3D display. *Radiat Med.* 2004;22(4):275-282.
35. Yeung DK, Wong SY, Griffith JF, et al. Bone marrow diffusion in osteoporosis: evaluation with quantitative diffusion MR imaging. *J Magn Reson Imaging.* 2004;19(2):222-228.
36. Baur A, Dietrich O, Reiser MF. Diffusion-weighted imaging of bone marrow: current status. *Eur Radiol.* 2003;13(7):1699-1708.
37. Padhani AR, Liu G, Koh DM, et al. Diffusion-weighted magnetic resonance imaging as a cancer biomarker: consensus and recommendations. *Neoplasia.* 2009;11(2):102-125.
38. Padhani AR, Koh DM, Collins DJ. Whole-body diffusion-weighted MR imaging in cancer: current status and research directions. *Radiology.* 2011;261(3):700-718.
39. Dietrich O, Biffar A, Baur-Melnyk A, Reiser MF. Technical aspects of diffusion-weighted imaging of the spine and bone marrow. *Eur J Radiol.* 2010;76(3):323-328.
40. Nonomura Y, Yasumoto M, Yoshimura R, Haraguchi K, Ito S, Ohashi I. Relationship between bone marrow cellularity and apparent diffusion coefficient. *J Magn Reson Imaging.* 2001;13(5):757-760.
41. Diehn FE. Imaging of spine infection. *Radiol Clin North Am.* 2012;50(4):777-798.
42. Modic MT, Masaryk TJ, Ross JS, Carter JR. Imaging of degenerative disk disease. *Radiology.* 1988;168(1):177-186.
43. Chou R, Deyo R, Jarvik JG. Appropriate use of lumbar imaging for evaluation of low back pain. *Radiol Clin North Am.* 2012;50(4):569-585.
44. Castillo M, Arbelaez A, Smith JK, Fisher LL. Diffusion-weighted MR imaging offers no advantage over routine noncontrast MR imaging in the detection of vertebral metastases. *AJNR Am J Neuroradiol.* 2000;21(5):948-953.
45. Talbott JF, Narvid J, Shankar PR, et al. Diffusion-weighted imaging of spinal tumors: comparison with conventional MRI and clinical applications. *Neuroimaging Clin N Am.* 2013;23(3):433-450.
46. Spuentrup E, Buecker A, Stuber M, et al. Diffusion-weighted MR imaging for differentiation of benign fracture edema and tumor infiltration of the vertebral body. *AJNR Am J Neuroradiol.* 2001;22(5):851-858.
47. Messiou C, Kaiser M. Whole body diffusion weighted MRI — a new view of myeloma. *Br J Haematol.* 2015;171(1):29-37.
48. Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. *Radiology.* 1988;166(1 Pt 1):193-199.
49. Jensen TS, Karppinen J, Sorensen JS, Niinimäki J, Leboeuf-Yde C. Vertebral endplate signal changes (Modic change): a systematic literature review of prevalence and association with low back pain. *Eur Spine J.* 2008;17(11):1407-1422.
50. Baur A, Dietrich O, Reiser MF. Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic vertebral compression fractures. *Radiology.* 1998;207(2):349-356.
51. Erly WK, et al. Differentiating Modic type 1 degenerative changes from acute infectious spondylitis with diffusion-weighted imaging. *AJNR Am J Neuroradiol.* 2006;27(4):895-899.
52. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine (Phila Pa 1976).* 2001;26(17):1873-1878.
53. Finkenstaedt M, et al. Evaluation of vertebral bone marrow edema with diffusion-weighted imaging: differentiation between benign and malignant causes. *Eur J Radiol.* 2002;42(3):185-195.
54. Dietrich O, Biffar A, Baur-Melnyk A, Reiser MF. Technical aspects of diffusion-weighted imaging of the spine and bone marrow. *Eur J Radiol.* 2010;76(3):323-328.
55. McCarthy J, Davis A. Diagnosis and management of vertebral compression fractures. *Am Fam Physician.* 2016;94(1):44-50
56. Baur A, et al. Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic vertebral compression fractures. *Radiology.* 1998;207(2):349-356.
57. Tan H, Xu H, Luo F, et al. Combined intravoxel incoherent motion diffusion-weighted MR imaging and magnetic resonance spectroscopy in differentiation between osteoporotic and metastatic vertebral compression fractures. *J Orthop Surg Res.* 2019;14:299.

58. Spuentrup E, Buecker A, Stuber M, et al. Diffusion-weighted MR imaging for differentiation of benign fracture edema and tumor infiltration of the vertebral body. *AJNR Am J Neuroradiol.* 2001;22(5):851-858.
59. Takahara T, Imai Y, Yamashita T, Yasuda S, Nasu S, Van Cauteren M. Diffusion-weighted whole-body imaging: clinical applications. *J Magn Reson Imaging.* 2004;19(6):821-835.
60. Lecouvet FE, Vande Berg BC, Maldague BE, et al. MRI of normal bone marrow, traumatic and tumoral lesions. *Eur Radiol.* 1998;8(8):1327-1334.
61. Ledermann HP, Schweitzer ME, Morrison WB, Carrino JA. MR imaging findings in spinal infections: rules or myths? *Radiology.* 2003;228(2):506-514.
62. Chang SD, Shiau YC, Kao A, et al. Differentiation between spinal infection and tumor infiltration using diffusion-weighted imaging and apparent diffusion coefficient values. *Eur Spine J.* 2019;28(10):2245-2253
63. Maiuri F, Iaconetta G, Gallicchio B, Manto A, Briganti F. Spondylodiscitis: clinical and magnetic resonance diagnosis. *Spine.* 1997;22(15):1741-1746.
64. Gillams A, Cassar-Pullicino V. MR imaging of spinal infection. *Semin Musculoskelet Radiol.* 2019;23(1):50-61
65. Oztekin O, Calli C, Adibelli ZH, Kitis O, Eren CS, Yuntun N. Diagnostic role of diffusion-weighted imaging in spinal infections. *Eur J Radiol.* 2010;74(1):119-123.
66. Fiani B, Cathel A, Jarrah R, et al. Diffusion-weighted imaging in spinal infection and malignancy: a review. *Clin Neurol Neurosurg.* 2021;203:106577.