Journal of Orthopaedic Surgery and Research

REVIEW

Open Access

Diagnostic imaging confusion in infectious spondylitis

Zhihao Xu[1,2†], Weijian Zhu[3†], Sirui Zhou[4], Zhiying Yang[5], Liqiao Xiang[6], Jinming Zhang[3*] and Kangshou Liu[1*]

Abstract

Background Infectious spondylitis is a rare but increasingly recognized condition involving infectious lesions of the spine. It can affect patients of all ages and is typically caused by bacterial, fungal, or other pathogens. The infection commonly targets the vertebral bodies and intervertebral discs. Early symptoms of infectious spondylitis are often nonspecific, making it challenging to differentiate from other spinal disorders such as tumors, degenerative diseases, or other types of infections. As the condition can progress rapidly, early diagnosis and intervention are crucial to improving patient outcomes.

Main body Imaging plays a critical role in the early diagnosis of infectious spondylitis. Magnetic resonance imaging is considered the gold standard due to its superior ability to visualize soft tissue structures and assess the extent of infection. CT imaging and nuclear medicine scans also provide valuable information in certain clinical scenarios, particularly for evaluating bony involvement and detecting occult infection. However, imaging findings in infectious spondylitis can overlap with those of other spinal conditions, making differential diagnosis essential for accurate identification. This study explores the advantages and limitations of different imaging modalities in diagnosing infectious spondylitis, focusing on their ability to suggest the possible etiology, determine the severity of the disease, and guide treatment decisions. Additionally, the relationship between imaging characteristics and clinical symptoms is examined to offer clinicians a systematic approach to improve diagnostic accuracy.

Conclusion Early and accurate diagnosis of infectious spondylitis is critical for preventing long-term complications. Imaging techniques, especially MRI, are invaluable tools for clinicians in identifying this condition, assessing its extent, and guiding treatment. Improved understanding of the imaging features associated with infectious spondylitis is essential for more accurate diagnosis and treatment planning.

Keywords Infectious spondylitis, Imaging characteristics, Spinal tumors, Vertebral hemangiomas, Modic changes type 1, Ankylosing spondylitis, SAPHO syndrome, Spinal neuroarthropathy

†Zhihao Xu and Weijian Zhu contributed equally to this work.

*Correspondence:
Jinming Zhang
zhangjinming1988@163.com
Kangshou Liu
lks1004@jnu.edu.cn
[1]Department of Hepatobiliary Surgery, Huaqiao Hospital, Jinan University, Guangzhou 510630, China
[2]Department of Radiology, Huaqiao Hospital, Jinan University, Guangzhou 510630, China
[3]Department of Orthopedics, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, 1095 Jiefang Street, Wuhan, Hubei 430030, P.R. China
[4]Department of Respiration, Liyuan Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430077, China
[5]Department of Radiology, Zhongshan Hospital of Dalian Medical University, Dalian 116001, China
[6]Qichun Shunkang Nephrology Hospital, Qichun Avenue 127, Huanggang City 435300, China

Introduction

Infectious spondylitis accounts for 2-7% of all cases of osteomyelitis, with a higher prevalence in males (approximately 1:5), and can affect individuals of all age groups [1]. In recent years, the incidence of infectious spondylitis has increased, driven by advancements in medical care and rising risk factors such as aging, diabetes, intravenous drug use, immunosuppressive therapies (including prolonged corticosteroid use, HIV infection, and organ transplantation), and an increase in spinal interventions [2].

The most common symptom of infectious spondylitis is persistent, non-mechanical back pain that is not relieved by rest. Patients may also may also exhibit systemic symptoms, including fever, chills, night sweats, and weight loss. In advanced stages, neurological deficits such as sensory disturbances, motor impairment, and changes in reflexes can occur [3]. The lumbar spine is the most frequently affected region, followed by the cervical spine, thoracic spine, and sacrum, with single vertebral body involvement being the most common [4].

Bacterial pathogens, particularly Staphylococcus aureus, are the leading cause of infectious spondylitis, often via hematogenous spread. Other common pathogens include Streptococcus species and Streptococcus pneumoniae, with Gram-negative bacteria (e.g., Escherichia coli, Pseudomonas aeruginosa, Klebsiella, and Salmonella) being less frequently involved. Non-pyogenic spondylitis, especially granulomatous spondylitis, is most commonly caused by Mycobacterium tuberculosis, a form of extrapulmonary tuberculosis, with other agents including Brucella, fungi (e.g., Aspergillus species), and parasites [5, 6].

Despite its well-established infectious origin, infectious spondylitis can be difficult to distinguish from other common spinal conditions, such as degenerative disc disease, spinal tumors, or inflammatory diseases like rheumatoid arthritis, which may present with similar imaging findings. This overlap in radiographic features often leads to diagnostic uncertainty [7]. Therefore, it is crucial to identify the distinguishing imaging characteristics of infectious spondylitis to improve diagnostic accuracy.

This article aims to review the imaging characteristics of infectious spondylitis and highlight key points for distinguishing it from diseases with similar radiographic presentations. By comparing imaging findings across various diseases, we aim to provide clearer diagnostic guidelines for clinicians, ultimately improving differential diagnosis and patient management.

The main types of infectious spondylitis

Pyogenic spondylitis

Pyogenic spondylitis is a bacterial infection affecting the intervertebral discs, vertebral bodies, and surrounding tissues. Its incidence and mortality have risen significantly over the past decade, often being misdiagnosed due to nonspecific early symptoms. Diagnosis mainly relies on imaging studies, with MRI being the gold standard due to its high specificity (92%) and sensitivity (96%) for detecting infectious spondylitis [8]. Early X-rays have low sensitivity, with typical findings like loss of disc height and endplate changes taking 3–4 weeks to become visible [9]. CT scans offer better resolution for soft tissue and are more sensitive than X-rays, showing features like endplate erosion and intervertebral disc height loss [10].

MRI plays a critical role in early diagnosis by identifying high signal changes in intervertebral discs and adjacent vertebrae on T2-weighted imaging (T2WI), as well as low signal on T1-weighted imaging (T1WI) and enhancement features. The presence of paravertebral inflammation, especially when high signal in the psoas muscle on T2WI, is highly sensitive 92% and specific 92% for pyogenic spondylitis [11], helping to differentiate it from other conditions. As the infection progresses, MRI helps in assessing the involvement of multiple vertebrae, particularly when cervical and thoracic segments are affected [12]. In cases where MRI cannot distinguish late-stage degenerative changes from pyogenic spondylitis, the vacuum phenomenon in the disc strongly indicates degenerative changes [13].

Although MRI provides exceptional sensitivity, it has limitations, such as reduced sensitivity for cortical bone involvement and increasing contraindications in patients with implants [14]. Diffusion-weighted imaging (DWI) can enhance diagnostic accuracy, though artifacts and small canal sizes may impact the results.

Tuberculous spondylitis

Tuberculous spondylitis is commonly seen in immunocompromised individuals and differs from other forms of infectious spondylitis [15]. Tuberculosis commonly affects the thoracic spine and is less frequently seen in the lumbar region, usually beginning from the anterior portion of the spine and soft tissues. Characteristic imaging findings of tuberculous spondylitis include the formation of large paravertebral abscesses, which have thin and smooth walls and involve multiple spinal segments, often extending along the anterior longitudinal ligament, resulting in a phenomenon known as "pseudodiffusion",When multiple adjacent vertebrae are involved, the intervertebral discs are relatively preserved [16]. Skip lesions and large cold paravertebral abscesses also suggest tuberculous spondylitis [17]. Paravertebral calcification identifiable on X-ray and CT is considered a characteristic manifestation of tuberculous spondylitis [18].

Brucellar spondylitis

Brucellar spondylitis is the most common zoonotic disease, with spinal involvement being a major cause of morbidity and mortality. Brucella spondylitis is one of the most severe complications, primarily affecting the lumbar vertebrae and associated with complications such as epidural, paravertebral, and psoas abscesses, which may lead to nerve compression [19].

In early X-ray examinations, abnormalities are typically not visible. Bone lysis accompanied by bone sclerosis and loss of the epiphyseal plate may be observed 2–3 weeks after the onset of symptoms. Focal erosion of the upper or lower vertebral body is a characteristic manifestation, mainly occurring at the anterior portion of the endplate, usually in the upper lumbar region. CT can be utilized to identify early manifestations of the disease, with approximately 25-30% of cases showing gas vacuum phenomena in the anterior portion of the intervertebral disc, which may relate to disc ischemia and necrosis or focal instability. Contrast-enhanced CT may reveal the formation of paravertebral abscesses or epidural abscesses [20]. MRI exhibits typical features of infectious spondylitis, with T2WI demonstrating diffuse high signal intensity. Brucella leads to changes in the endplate of the endplate in both acute and chronic stages through phagocytosis, activation of osteoclasts, and suppression of the immune response. Severe erosion by Brucella hampers the potential for bone regeneration, presenting as uneven high signal on T2WI [21]. A minority of patients with brucellar infections of the spine may show mixed signals on T2WI due to therapeutic interventions [22].

Fungal spondylitis

Fungal spondylitis is rare, accounting for only 5% of cases of discitis, with the most common spinal infection being Candida albicans infection [23], typically occurring in immunocompromised individuals. Due to the relatively low virulence of pathogenic microorganisms, abscesses and spinal infections may exhibit signal loss on T2WI. Fungal spondylitis may present with relative preservation of the intervertebral disc, which can be easily confused with tuberculous spondylitis, and may lead to complications such as spinal cord edema and spinal cord cysts [24]. The inherent paramagnetic and ferromagnetic elements of fungi can produce low signal bands on the endplates on T2WI, which is characteristic of fungal spondylitis, whereas other types of spinal infections typically do not exhibit this finding [25, 26].

Diseases to be distinguished

Spinal tumors

Spinal tumors can be classified into benign tumors and malignant tumors, which are further divided into primary tumors (Primary Spinal Tumors, PSTs) and bone metastases. PSTs are relatively rare, whereas the spine is the most common site for bone metastases. In recent years, with the aging population and advancements in medical oncology and radiology, the incidence of bone metastases has continued to rise [27]. Spinal infections and tumors exhibit similar imaging features, such as vertebral destruction, high signal intensity on T2WI, and spinal canal stenosis. These similarities vary according to by the prevalence of diseases in different regions [28]. Therefore, the differential diagnosis between the two is particularly important.

Spinal tumors, especially malignant tumors, can lead to cortical destruction, vertebral collapse, and pathological fractures. X-ray imaging typically shows osteolytic lesions, cortical thinning, and changes in trabecular patterns. Due to aggressive bone remodeling, a periosteal triangle may also form, indicating a periosteal reaction [29]. In contrast, vertebral destruction in infectious spondylitis presents as “fish mouth” or “wedge” deformities [30]. Spinal tumors may also cause vertebral fractures and spinal deformities, whereas include vertebral subluxation and spondylolisthesis. Additionally, tumor-induced fractures or destruction may result in the loss of normal spinal alignment, while deformities associated with spinal infections often involve more gradual collapse or wedging of the vertebrae [31].

On CT imaging, tumors typically demonstrate a larger extent of vertebral destruction compared to infectious spondylitis, often associated with surrounding soft tissue masses. Certain tumors, such as osteosarcoma, may exhibit calcification and bone formation, whereas infectious spondylitis generally shows no significant calcification or bone formation. Tumors are aggressive and have the potential to affect adjacent tissues, intervertebral discs, whereas infectious spondylitis is more likely to involve the intervertebral discs (Table 1), which is particularly evident in the imaging presentation [28] (Fig. 1). Numerous studies have suggested that CT perfusion can aid in distinguishing between spinal tumors and infections. Dynamic contrast-enhanced studies indicate that tumors have a higher capillary density and relative blood volume (rBV). Shankar et al. reported that when the rBV value is 4, the sensitivity for diagnosing inflammatory and neoplastic conditions is 100%. Therefore, an $rBV < 4$ is highly suggestive of tuberculous spondylitis. Furthermore, when the rBV is 3.5, the sensitivity for diagnosing inflammatory diseases is 100%, while for neoplastic diseases, it is 95%. This implies that an $rBV < 3.5$ strongly indicates infectious spondylitis [32]. ^18F-FDG-PET can

Table 1 Imaging distinction between infectious spondylitis and its easily confused diseases

Confusing disease	Confusions	Distinctions
Spinal tumors	Spinal tumors may present with destruction of the vertebral body and enhancement of the soft-tissue mass, which can be confused with an abscess in infectious spondylitis. Both may present with paravertebral soft tissue signal abnormalities	Extent of vertebral destruction: Infectious spondylitis is usually accompanied by disc destruction and involvement of two adjacent vertebrae, whereas tumors often have single-vertebral body involvement and do not involve the disc. Enhancement pattern: The soft tissue lesions of tumors enhance more uniformly, while infectious spondylitis mostly shows irregular enhancement or abscess formation. Bone destruction pattern: Tumors are more inclined to trabecular bone destruction, while infections show osteolytic destruction with blurred edges
Vertebral hemangioma	Vertebral hemangioma showing high signal on imaging, especially on water-sensitive sequences, may be confused with inflammatory edema in infectious spondylitis	Margin definition: Vertebral hemangioma has clear margins while infectious spondylitis has blurred margins. Associated paravertebral lesions: Vertebral hemangiomas do not have paravertebral abscesses or paravertebral soft-tissue lesions, whereas infectious spondylitis is often associated with paravertebral abscesses. Enhancement features: Vertebral hemangiomas show rapid homogeneous enhancement on dynamic enhancement scans, whereas infected lesions show heterogeneous enhancement and may be associated with strengthening of the abscess wall
Modic changes	Modic changes type 1 show T1WI low signal and T2W high signal on MRI, similar to vertebral bone marrow edema in the early stage of infectious spondylitis	Disc erosion: Infectious spondylitis is almost always accompanied by disc destruction and a high degree of collapse, whereas Modic changes usually show no significant change in disc signal. Edge blurring: Infectious lesions have blurred signal edges, whereas Modic changes show relatively clear demarcation lines
Ankylosing spondylitis	Both may present with narrowing of the intervertebral space and inflammatory changes, and advanced ankylosing spondylitis may present with a "bamboo-like spine", which is easily confused with advanced ossification of the infection.	Distribution of lesions: Ankylosing spondylitis usually starts from the sacroiliac joints up the spine, while infectious spondylitis does not have a clear pattern of distribution. Paravertebral abscesses: Infectious spondylitis is often associated with paravertebral abscesses and soft tissue abnormalities, whereas ankylosing spondylitis usually has no abscess formation. Characteristics of vertebral fusion: Vertebral fusion in ankylosing spondylitis is accompanied by the formation of bone bridges, whereas in infectious spondylitis, irregular bone destruction and abnormal ossification are predominant
SAPHO syndrome	SAPHO syndrome may present with bone marrow edema and soft tissue swelling, similar to the inflammatory changes of infectious spondylitis	Distributional features: SAPHO syndrome tends to involve the anterior chest wall and sacroiliac joints, whereas infectious spondylitis tends to involve the lumbar spine. Abscess formation: Infectious spondylitis is often associated with abscesses, whereas SAPHO syndrome usually has no abscess formation
Neuroarthropathy Chemical Discitis	Neuroarthropathy presents with extensive bone destruction and may be associated with loss of joint space, similar to the severe vertebral destruction seen in the later stages of infectious spondylitis. The disc signal typically increases, particularly on T2-weighted images, where the disc and its surrounding areas may show signs of edema.	Bone fragments and gas: Imaging features of neuroarthropathy include bone fragments and gas in the intervertebral space, whereas infectious spondylolisthesis has no gas. Inflammatory manifestations: Infectious spondylolisthesis is often associated with paravertebral inflammatory changes and abscesses, whereas neuroarthropathy does not have a significant inflammatory response. Vertebral architecture: Vertebral destruction is more extensive and irregular in neuroarthropathy, whereas infectious spondylolisthesis usually presents with limited vertebral destruction and soft tissue abscesses. The main distinction between chemical discitis and infectious spondylitis lies in the changes observed in the disc and vertebral body. Chemical discitis typically presents with increased disc signal and minimal bone damage, while infectious spondylitis shows increased disc signal accompanied by significant vertebral bone destruction and paraspinal soft tissue abscesses.

differentiate between infectious spondylitis and tumors; the maximum standard uptake value for infectious spondylitis is usually greater than 15, while that for tumors is less than 15 [33]. In CT imaging, spinal tumors typically demonstrate heterogeneous enhancement following contrast injection, indicating angiogenesis. in contrast, infectious spondylitis may exhibit irregular enhancement, typically suggestive of abscesses or granulation tissue [34]. MRI is the preferred diagnostic tool for spinal tumors, as it can accurately assess disease the spread of the disease. Cortical infiltration associated with tumors appears on MRI as well-defined soft tissue masses with variable signal intensity, showing significant enhancement after contrast administration. Spinal tumors can invade structures outside the spine, often involving paravertebral structures, such as paravertebral muscles and neural foramina, and even the spinal canal, eroding cortical bone and infiltrating adjacent structures [10]. On MRI, soft tissue masses, neural foraminal widening, or spinal canal stenosis can be observed. Furthermore, tumor-related extradural compression and spinal cord compression may be indirectly analyzed through spinal cord signal changes or symptoms of spinal cord compression [35]. In infectious spondylitis, MRI can also demonstrate the spread of infection to adjacent soft tissues, typically manifested by the formation of paravertebral or epidural abscesses. These abscesses usually present as

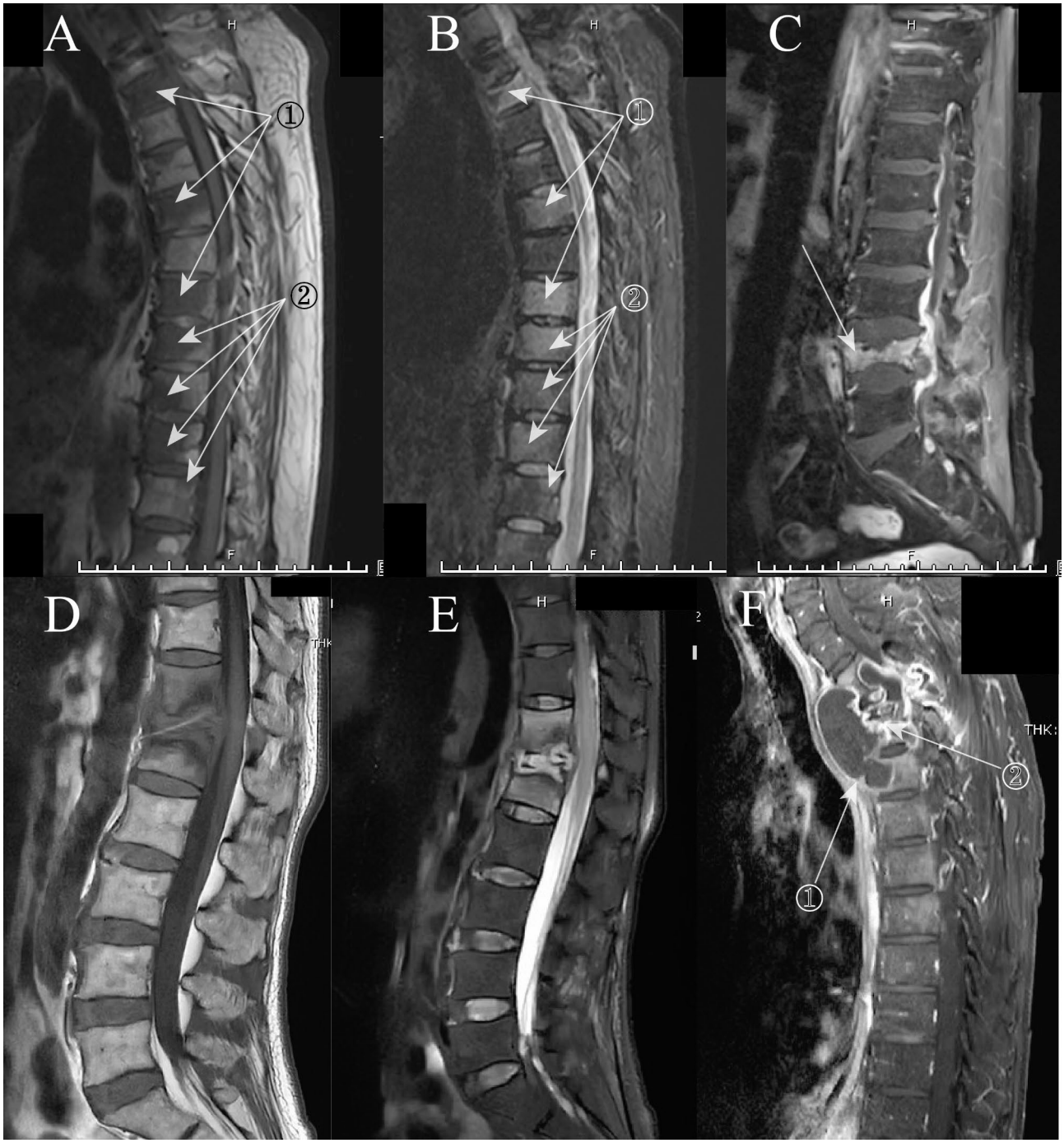

Fig. 1 Figure **A** and **B** show metastatic cancer in the vertebrae. Figure **A** presents the T1-weighted imaging (T1WI) sequence, while Figure **B** displays the short tau inversion recovery (STIR) sequence. The white arrows (① and ②) point to the lesions, which do not involve the intervertebral disc. Figure **C** illustrates the enhancement sequence of a primary malignant vertebral tumor, showing a reduction in the height of the L4 vertebra, while the intervertebral disc remains intact. Figures **D** and **E** show T1WI and STIR sequences, respectively, depicting pyogenic spondylitis with confirmed involvement of the T12-L1 vertebrae and disc destruction. Figure F presents an enhancement sequence of tuberculous spondylitis, ① highlighting the thin, annular enhancement of the paravertebral abscess wall, and ② showing vertebral bone destruction

high signal areas on T2WI, with only linear enhancement observed following contrast administration.

Vertebral hemangiomas

Vertebral hemangiomas (VHs) are the most common benign tumors of the vertebrae, characterized as benign vascular lesions formed by the proliferation of blood vessels in the bone marrow spaces, confined by the trabecular bone [36]. The histopathology of VHs allows them to be classified as typical, atypical, or aggressive. The imaging characteristics are related to the fat content and vascularity of the lesions; lesions with a higher fat content tend to exhibit typical features (Fig. 2), while those with a greater vascular component often lack these

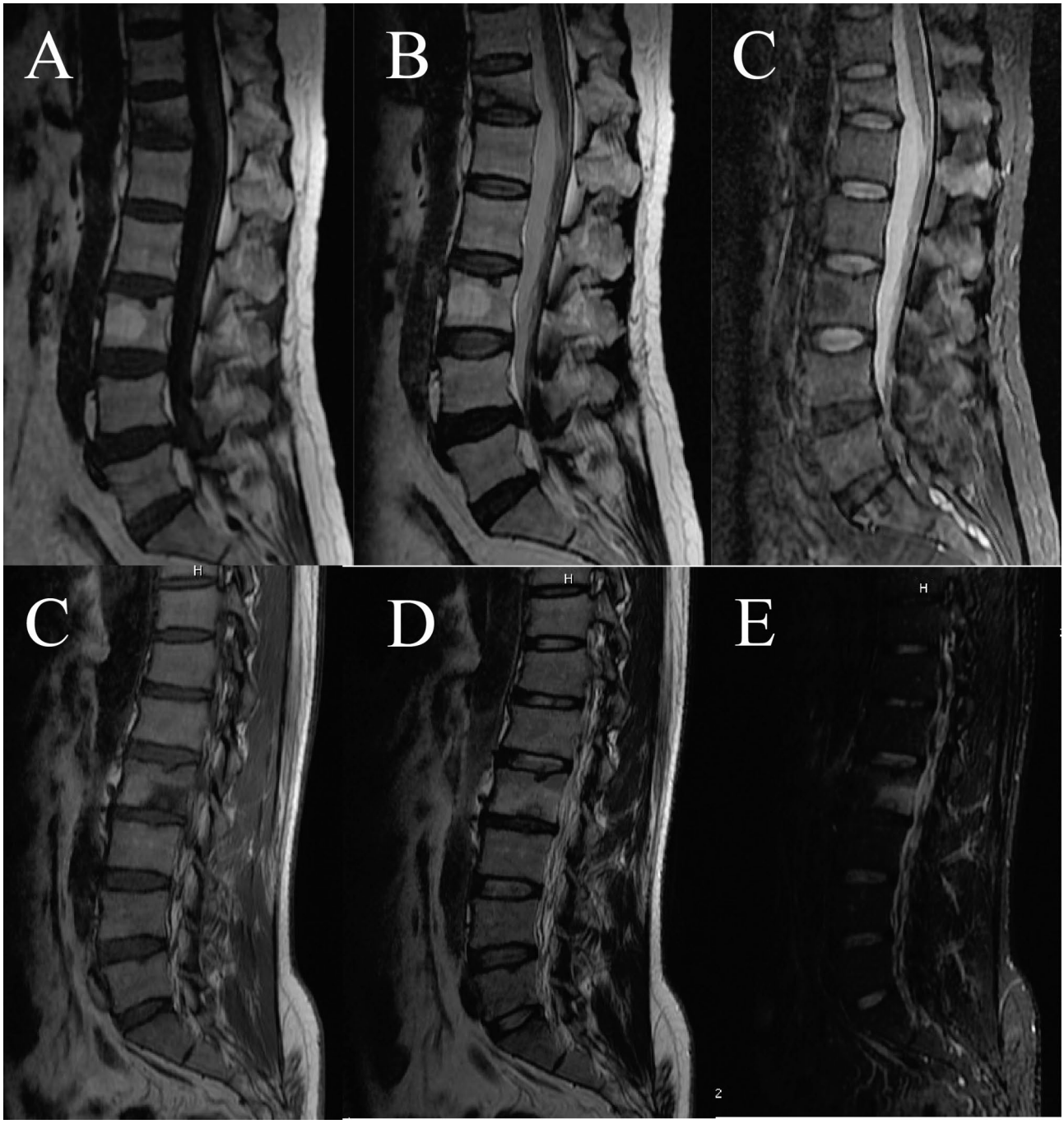

Fig. 2 Figures **A**, **B**, and **C** demonstrate a hemangioma at the L3 vertebra, exhibiting high signal intensity on both T1WI and T2WI, and low signal intensity on the STIR sequence. These findings suggest that the hemangioma has a high fat content. Figures D, E, and F illustrate tuberculous spondylolisthesis, which shows high signal intensity on both T2WI and STIR sequences, and low signal intensity on T1WI

classic manifestations [37]. On X-ray, the appearance resembles a "corduroy cloth" due to the accumulation of thin-walled, blood-filled spaces leading to horizontal trabecular absorption and vertical trabecular strengthening. Typical hemangiomas on CT display the "corduroy sign" and a characteristic honeycomb or dot-like appearance (Fig. 3), generally exhibiting high signal intensity on T1WI and T2WI sequences [38]. Some imaging features seen on CT are also present on MRI; atypical and aggressive lesions tend to have a higher vascular component and may not demonstrate typical imaging findings such as the "corduroy sign" and "dot sign." However, these remain important imaging characteristics for differentiating infectious spondylitis from VHs, with T1WI appearing hypointense or isointense and T2WI showing hyperintensity (due to vascular components) [37, 39]. The cystic or honeycomb lucencies within VHs demonstrate no significant bone destruction, and the vertebral margins are well-defined, which contrasts with infectious spondylitis.

Modic changes type 1

Modic changes include various types, among which Modic changes type 1 is particularly difficult to distinguish from infectious spondylitis. Although Modic changes type 1 shares some imaging features with infectious spondylitis, its clinical significance and

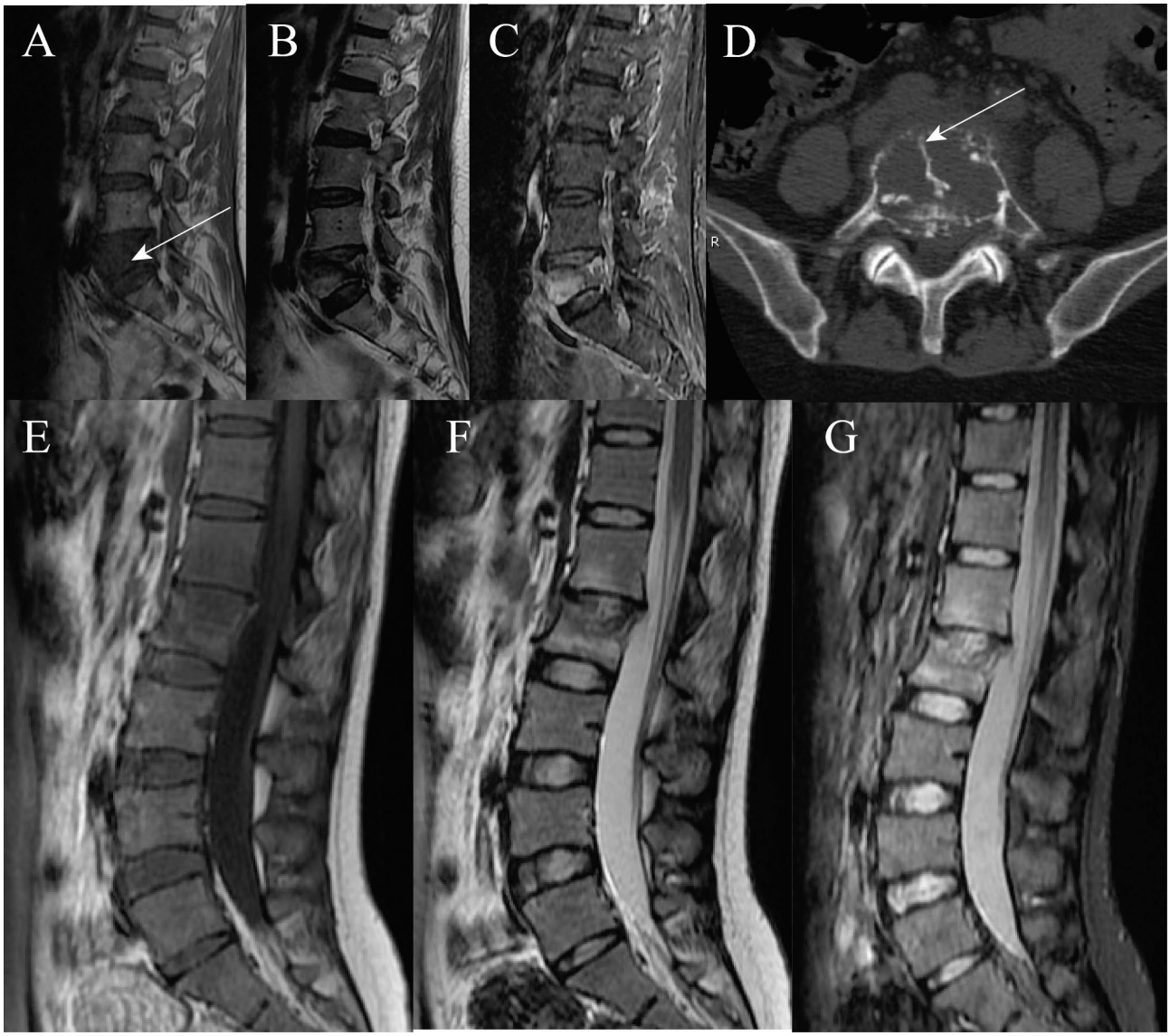

Fig. 3 Figures **A**, **B**, **C**, and **D** show images of an L5 hemangioma, which presents low signal intensity on T1WI and high signal intensity on both T2WI and STIR sequences. The hemangioma exhibits well-defined borders, and Figure D demonstrates its characteristic "corduroy" sign. Figures **E**, **F**, and **G** present T1WI, T2WI, and STIR sequences of tuberculous spondylolisthesis, showing intervertebral disc and endplate destruction, along with vertebral bone presenting mixed signals in water-sensitive sequences

management strategies are markedly different. Neither condition typically shows significant changes on X-rays, particularly in the early stages. Modic changes type 1 generally lacks obvious bone changes, while X-rays of infectious spondylitis may reveal bone destruction, including loss of intervertebral disc height and adjacent destructive endplate changes, bone lysis accompanied by sclerosis, and loss of the epiphyseal plates. However, imaging findings in the early stages of both conditions are often challenging to differentiate [40].

On CT scans, changes in vertebral bone structure can be observed in both conditions, particularly during the chronic stages. CT imaging does not show significant bone marrow edema in Modic changes type 1, whereas it can reveal bone lysis and possible abscess formation in infectious spondylitis. According to the definition of Modic changes type 1, it presents as low signal intensity on T1WI and high signal intensity on T2WI, which is essentially consistent with the early signal intensities seen in infectious spondylitis [25] (Fig. 4). However, in the early stages of infection, changes to the endplates may be subtle. Prior to the appearance of abnormal signals in the intervertebral discs and endplates on T2WI, T1WI may demonstrate discontinuity or blurring of the bone cortex. In contrast, Modic1, despite having irregular signal abnormalities, typically maintains a complete low-signal profile of the endplate on T1WI [14] (Fig. 4). DWI can also effectively differentiate pyogenic spondylitis from Modic changes type 1. A clear linear high signal area at the boundary between normal and abnormal bone marrow strongly suggests degenerative changes [41]. Studies indicate that Modic1 exhibits higher gray-level values on DWI compared to pyogenic infectious spondylitis, which shows diffuse high signal enhancement on DWI but lacks a definitive linear high signal area. Additionally, an apparent diffusion coefficient value of $<1250\times10\text{^-}6$ mm^2/s has been utilized as a critical threshold for infection, with reported sensitivity of 66% and specificity of 88%[38].

Ankylosing spondylitis

Ankylosing spondylitis (AS) is a chronic inflammatory disease characterized by fibrosis, ossification, and stiffness of the intervertebral discs and adjacent connective tissues. Through imaging studies, AS can be classified into early AS and late AS [42]. Andersson lesions (AL) are a rare complication of AS, typically presenting as vertebral fractures, endplate inflammation, discitis, spinal kyphosis, and even pseudoarthrosis and irreversible nerve damage [43, 44].

Early manifestations of AS typically involve inflammation of the sacroiliac joints, which can be observed as blurring and bone destruction of the sacroiliac joints on X-ray and CT. As the disease progresses, bridging osteophytes (i.e., "joint fusion") may occur. Over time, patients with AS may experience osteophyte formation and calcification of the spine, resulting in a "bamboo spine" appearance, indicating spinal ankylosis [45]. The hallmark of spinal disease in AS is the development of characteristic osteophytes, which usually grow vertically from areas of thin bone, typically beginning at the corners of the vertebrae [46]. CT imaging provides clearer visualization of bone destruction and osteophyte formation in the sacroiliac joints, with typical findings including osteophytes and bone fusion. The classic X-ray features of Andersson lesions include narrowing of the disc space, erosion of the vertebral endplates, and sclerosis of adjacent bone, which resemble the manifestations seen in infectious spondylitis [47].

Generally, the bone destruction associated with infectious spondylitis is more severe than that in AS, often leading to significant narrowing of the joint space [6]. Infectious spondylitis, compared to AS, infectious spondylitis may not exhibit typical osteophyte formation but typically shows marked bone destruction and abscess formation. MRI can reveal early inflammation, including bone marrow edema and periosteitis. These features are more sensitive in the early stages of AS than X-ray or CT, making MRI the most sensitive imaging modality for detecting early AS. MRI can show active disease in the sacroiliac joints (sacroiliitis), including bone marrow edema adjacent to the joints and enhancement of the bone marrow and joint spaces following contrast administration [46]. Furthermore, Andersson lesions predominantly appear as isointense signals on T1WI, while T2WI shows complex signal characteristics, with surrounding vertebrae exhibiting mild to severe edema but no distinctive findings. Literature also indicates that Andersson lesions present as high signal intensity on fat-saturated T2-weighted MRI and as low signal intensity on T1-weighted imaging, typically appearing semi-spherical [42]. These features can effectively differentiate Andersson lesions from infectious spondylitis.

SAPHO syndrome

SAPHO syndrome refers to synovitis, acne, pustulosis, hyperostosis, and osteitis. The anterior chest wall and spine are the most commonly affected skeletal sites [48]. Some features of SAPHO Overlap with infectious spondylitis. Early lesions may exhibit lytic changes on X-ray and CT imaging. However, these imaging modalities are not effective in assessing early or active inflammatory lesions [49]. In the later stages of SAPHO, conventional X-rays can show evidence of bone proliferation and osteitis, with bone proliferation potentially manifesting as sclerosis and cortical thickening due to chronic inflammation, resembling the findings in infectious spondylitis.

CT imaging provides clearer visualization of bony and joint manifestations, such as the formation of bone

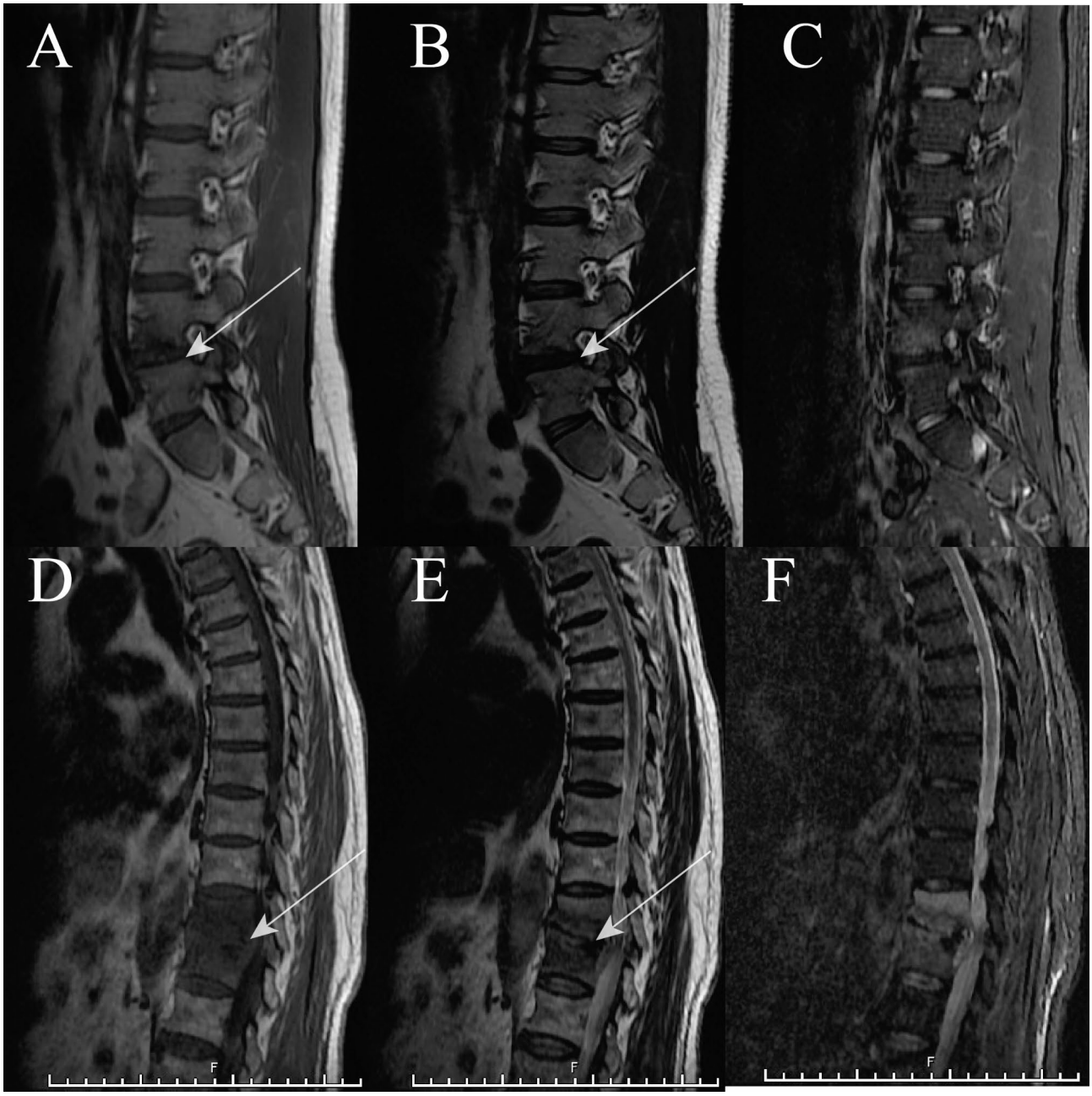

Fig. 4 Figures **A**, **B**, and **C** show Modic changes, which exhibit inflammatory signals. On T1WI, these changes present low signal intensity while maintaining an intact low-signal profile of the endplate. Water-sensitive sequences, such as T2WI and STIR, show high signal intensity. Figures **D**, **E**, and **F** demonstrate pyogenic spondylitis, also showing inflammatory signals. However, the disc destruction is more severe, and T1WI reveals discontinuity or blurring of the bone cortex

bridges, subchondral sclerosis, paravertebral ossification, joint space narrowing, periosteal bone formation, and ligamentous ossification [48]. MRI can clearly display early soft tissue changes, such as soft tissue swelling and synovial reaction, which other imaging modalities may not consistently identify. The incidence of enhancement in the intervertebral disc spaces on MRI is lower than that seen in infectious spondylitis. Furthermore, one of the characteristic findings of SAPHO is erosion of the anterior vertebral body, with involvement of the sacroiliac joints (with a high probability of bilateral involvement), and a possible semicircular distribution of lesions in adjacent vertebral corners, which distinguishes it from the typical presentation of infectious spondylitis [22, 50, 51] (Fig. 5).

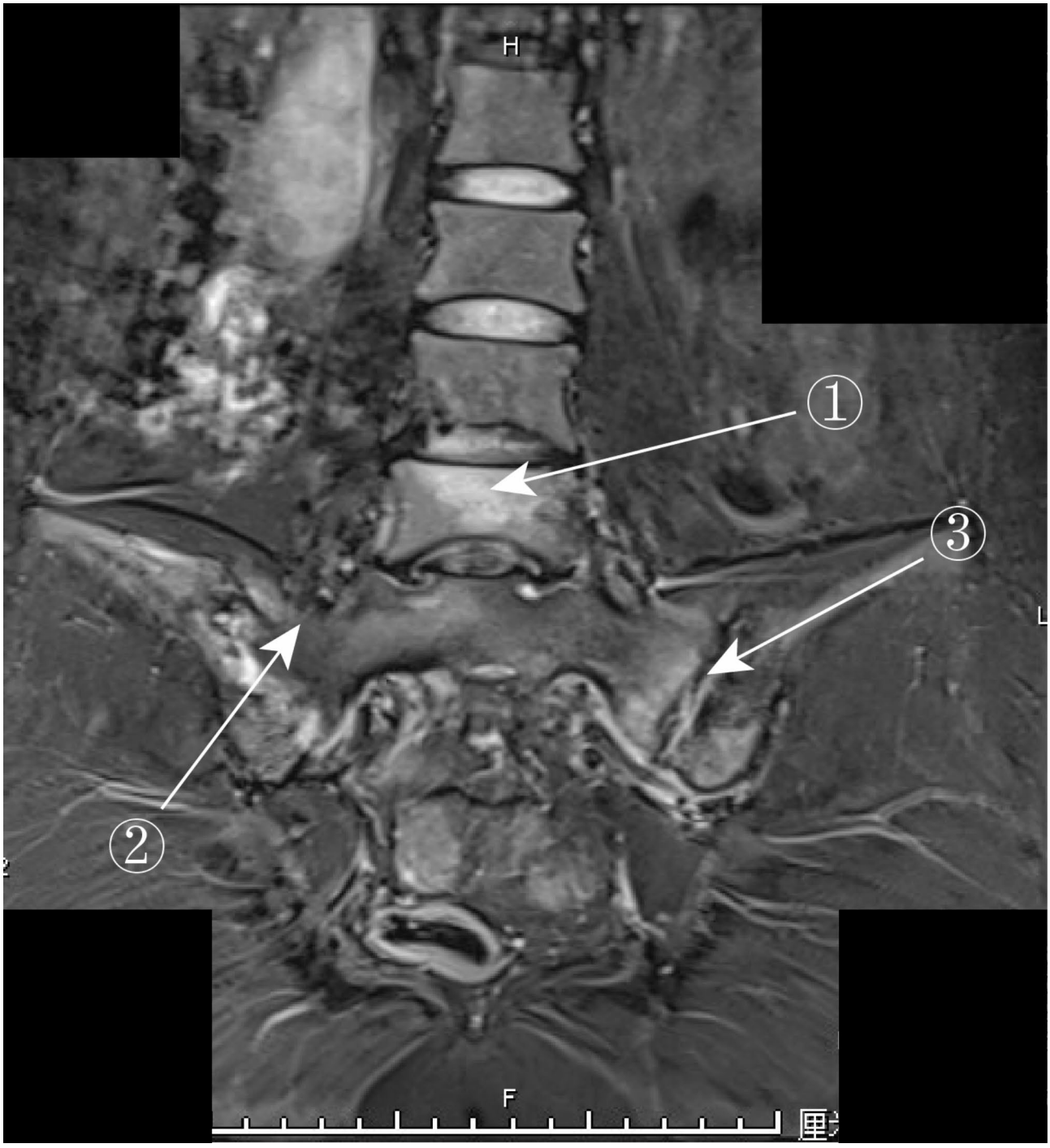

Fig. 5 The anterior horn of L5 shows high signal intensity at ①, while ② and ③ demonstrate severe bilateral damage to the sacroiliac joints

Spinal neuroarthropathy

Spinal neuropathic arthropathy (SNA), also known as Charcot spine or spinal neuropathic joint, is a rare progressive condition characterized by vertebral and ligamentous damage secondary to the loss of deep sensation and proprioception. The radiological manifestations of SNA are commonly described using the "6D" framework: Distension (soft tissue masses), Debris (bony fragments), Density (retained bone density and sclerosis), Disorganization (alterations in joint contours), Dislocation (vertebral slippage), and Destruction (erosion of endplates and facet joints) [52]. While SNA shares similarities with many diseases, the differential diagnosis from infectious spondylitis is particularly critical [53].

SNA most commonly affects the thoracolumbar and lumbosacral junction, with involvement in other spinal

regions being relatively rare [54]. Its classic radiological features encompass both anterior structures (vertebral bodies and intervertebral discs) and posterior elements (such as laminae and ligaments), whereas infectious spondylitis typically predominantly affects the anterior structures [18]. Early SNA may be difficult to detect on X-rays, which usually reveal late-stage manifestations. The typical radiographic features include severe bone destruction and disorganization near the joints, with large, loose marginal osteophytes, bony debris, and possibly dense new bone formation that may give the appearance of enlarged vertebrae [55].

SNA exhibits highly specific radiological findings, including gas, debris, and disorganization within the intervertebral disc space. On CT imaging, the presentation of SNA resembles degenerative changes in the intervertebral discs and small joints, with narrowing of the joint spaces. In later stages, soft tissues and bony fragments replace the normal intervertebral disc space, leading to destruction of the endplates and small joints, accompanied by disorganized soft tissues, often presenting as large paravertebral masses containing bony debris. Compared to X-rays, CT is more effective in detecting the vacuum phenomenon in the intervertebral discs. The presence of disc vacuum phenomenon, debris, and disorganization are critical imaging features for distinguishing SNA from infectious spondylitis. Another important imaging characteristic is the involvement of the entire intervertebral joints, including the anterior intervertebral joints and the posterior facet joints. Progressive joint destruction can lead to spinal dislocation and slippage [52].

Tuberculous spondylitis typically begins in the anterior portion of the spine and soft tissues, with rare involvement of the posterior vertebral bodies [16]. Its characteristic manifestation is kyphotic deformity of the vertebral bodies, commonly seen in the lower thoracic and upper lumbar regions, due to involvement of the intervertebral disc space and adjacent vertebrae, resulting in vertebral collapse and spinal instability [56]. Brucellar spondylitis primarily presents with localized erosions, usually located at the anterior part of the endplate, particularly in the upper lumbar region [20]. It may also involve the entire vertebral endplate, potentially leading to osteomalacia and destruction, allowing infection to spread to surrounding soft tissues and the spinal canal. This diffuse change resembles SNA; however, the vertebral morphology in brucellar spondylitis is typically preserved, and the absence of paravertebral masses is a notable distinguishing feature [9].

MRI, due to its high resolution for soft tissues, is generally more effective than CT in detecting early disease changes. MRI typically shows alterations in the intervertebral disc space, vertebral bodies, facet joints, and paravertebral soft tissues. When the intervertebral disc space is involved, it leads to a decrease in disc height, with the vertebrae often displaying diffuse edema and enhancement, and potentially resulting in vertebral dislocation and compression deformity. Affected facet joints present with edema, bone destruction, and disorganization. The appearance of paravertebral soft tissues on MRI is similar to that of typical infectious spondylitis, often complicated by fluid accumulation due to inflammation, micro-hemorrhages, and micro-fractures, typically occurring in the intervertebral disc space, small joints, and at sites of pseudoarthrosis. The most significant distinguishing features between SNA and infectious spondylitis include disorganization of the entire intervertebral joints, destruction of the endplates and facet joint surfaces, vertebral slippage, and uniform high signal intensity on T2WI with enhancement [57].

Chemical discitis

Chemical discitis is an inflammatory response typically triggered by chemical substances, such as the release of nucleus pulposus material, within the intervertebral disc. It is commonly seen in patients following trauma or with degenerative disc disease [58]. This condition generally does not exhibit significant loss of disc height or substantial bone changes, with only occasional mild alterations in bone density. In contrast, infectious spondylitis is characterized by notable loss of disc height [9], and vertebral destruction is commonly observed, with irregular vertebral endplates or bone resorption. Surrounding bone marrow edema may be present, manifesting as decreased vertebral density. On CT, chemical discitis presents with low-density areas in the disc due to soft tissue edema, without significant bone erosion between the vertebral endplates and disc. In contrast, CT of infectious spondylitis typically shows vertebral edge erosion or resorption and may even reveal vertebral collapse or spinal deformity [10]. MRI is the preferred imaging method for early differentiation of these two conditions. In chemical discitis, MRI typically shows enhanced disc signal, especially on T2-weighted images, with possible edema around the disc. However, there are usually no obvious spinal fractures or structural damage. In contrast, MRI of infectious spondylitis demonstrates significantly increased disc signal, particularly on T2-weighted images, often accompanied by vertebral bone marrow edema. Enhanced disc signal is common, with associated vertebral bone destruction presenting as lytic changes at the vertebral endplates [59].

The main distinction between chemical discitis and infectious spondylitis lies in the changes observed in the disc and vertebral body. Chemical discitis typically presents with increased disc signal and minimal bone damage, while infectious spondylitis shows increased disc

signal accompanied by significant vertebral bone destruction and paraspinal soft tissue abscesses.

Limitations of imaging and future prospects

Spinal imaging plays a critical role in the differential diagnosis of infectious spondylitis. Modern imaging techniques, such as X-rays, CT scans, and MRI, have become essential tools for evaluating spinal structure and pathological changes. Each imaging modality has its unique advantages and limitations in diagnosis. Relying solely on imaging findings to differentiate infectious spondylitis presents certain limitations.

Firstly, the imaging characteristics of infectious spondylitis can resemble those of other pathological conditions, such as tumors or degenerative diseases, including vertebral destruction, joint space narrowing, and soft tissue edema, which may lead to misdiagnosis. Secondly, in the early stages of the disease, imaging changes may be subtle or even normal, making accurate assessment challenging. This article aims to enhance awareness of the imaging manifestations of infectious spondylitis and to emphasize the distinctions between these and the similar imaging features of other diseases.

Furthermore, imaging results cannot provide clinical manifestations and historical information of the patient, such as fever, local tenderness, and functional impairment. Laboratory investigations play a crucial role in the differentiation and diagnosis of spinal infections, offering several detailed advantages. Firstly, they provide objective biomarkers (elevated white blood cell count, CRP, and ESR), which can indicate systemic inflammation or infection. When imaging results are inconclusive, these biomarkers help confirm the presence of an infectious process. Secondly, these laboratory investigations (including blood and tissue cultures) can identify specific pathogenic microorganisms. Therefore, a comprehensive consideration of imaging findings, clinical symptoms, and laboratory test results is essential for improving diagnostic accuracy and avoiding missed or incorrect diagnoses in the differential diagnosis of infectious spondylitis.

Future directions in imaging development primarily include the application of Artificial Intelligence (AI) and machine learning. AI can enhance diagnostic accuracy and efficiency by analyzing large volumes of imaging data to automatically identify and classify lesions. Deep learning algorithms can extract subtle pathological features from images to assist clinicians in early diagnosis. Additionally, AI can provide decision support during the diagnostic process, reducing human errors and diagnostic delays. Another significant trend is the integration of imaging with genomics and biomarkers. By combining imaging data with patients' genomic information and biomarkers, clinicians can develop personalized treatment plans. This integrated approach is expected to improve early detection rates and treatment outcomes for infectious spondylitis, thereby reducing patient suffering and economic burden.

Conclusion

The differential diagnosis in spinal imaging should advance toward higher resolution and greater intelligence. With technological advancements and enhanced data analysis capabilities, future imaging techniques will more accurately support the diagnosis and differential diagnosis of infectious spondylitis, ultimately improving patients' quality of life.

Abbreviations

MRI	Magnetic resonance imaging
CT	Computed tomography
PSTs	Primary Spinal Tumors
rBV	relative blood volume
VHs	Vertebral hemangiomas
AS	Ankylosing spondylitis
AL	Andersson lesions
SNA	Spinal neuropathic arthropathy
AI	Artificial Intelligence

Acknowledgements

Not applicable.

Author contributions

Conception of the work: JMZ and KSL. Acquisition and analysis of images: SRZ, ZYY, and LQX. The work drafted: WJZ, XZH.

Funding

No funding.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology (TJ-IRB202402127).

Consent for publication

Patients have submitted written informed consent for the release of their personal or clinical details and any recognizable images in this study.

Competing interests

The authors declare no competing interests.

Received: 18 January 2025 / Accepted: 2 April 2025
Published online: 21 May 2025

References

1. Smith B. Infectious and inflammatory processes of the spine. Radiol Clin North Am. 1991;29(4):809–27.
2. Fantoni T, Rossi, Mazzotta D, Giacomo, et al. Epidemiological and clinical features of pyogenic spondylodiscitis. Eur Rev Med Pharmacol Sci. 2012;16(Suppl 2):1128–3602.
3. Mylona S. Kakalou, Fanourgiakis, Skoutelis: pyogenic vertebral osteomyelitis: a systematic review of clinical characteristics. Semin Arthritis Rheum. 2009;39(1):10–7.
4. Zimmerli. Clinical practice. Vertebral osteomyelitis. N Engl J Med. 2010;362(11):1022–9.

5. Acosta G, Aryan A. Recent advances: infections of the spine. Curr Infect Dis Rep. 2006;8(5):390–3.
6. Scharrenberg Y, Brinkmann, Brune S, et al. The diagnostic value of soluble urokinase-type plasminogen activator receptor (suPAR) for the discrimination of vertebral osteomyelitis and degenerative diseases of the spine. J Orthop Surg Res. 2019;14(1):367.
7. Dayer D, Marco, Vazquez, Tabard-Fougere C, et al. Laboratory diagnostics for primary spinal infections in pediatric and adult populations: a narrative review. North Am Spine Soc J. 2023;16:100270.
8. Berbari K, Kowalski, Darouiche W et al. 2015 Infectious Diseases Society of America (IDSA) Clinical Practice Guidelines for the Diagnosis and Treatment of Native Vertebral Osteomyelitis in Adults. 2015;(1537–6591 (Electronic)).
9. Diehn. Imaging of spine infection. Radiol Clin North Am. 2012;50(4):777–98.
10. Herren. Jung, Pishnamaz, Breuninger, Siewe, Sobottke: spondylodiscitis: diagnosis and treatment options. Dtsch Arztebl Int. 2017;114(51–52):875–82.
11. Kim B, Kim, Kim K, et al. Comparison of pyogenic postoperative and native vertebral osteomyelitis. Spine J. 2019;19(5):880–7.
12. Ledermann, Schweitzer, Morrison, Carrino. MR imaging findings in spinal infections: rules or Myths?? Radiology. 2003;228(2):506–14.
13. Balcescu O, Rosinski, Nudelman S, et al. Pyogenic spinal infections warrant a total spine MRI. J Bone Jt Infect. 2023;8(1):1–9.
14. Prodromou ZP. Fau - Poulou, Poulou Ls Fau - Karsaliakos, Karsaliakos P Fau - Thanos, Thanos L Fau - Mylonakis, Mylonakis: FDG PET is a robust tool for the diagnosis of spondylodiscitis: a meta-analysis of diagnostic data. (1536–0229 (Electronic)).
15. Abid L. Chidambaranathan, Peh, vanhoenacker: imaging of musculoskeletal tuberculosis. Skeletal Radiol. 2024;53(10):2081–97.
16. Hong CJF-, Lee LJF-, Kim. Kim Nr Fau - Choi, Choi Ja Fau - Kang, Kang: MR imaging assessment of the spine: infection or an imitation? (1527–1323 (Electronic)).
17. Spernovasilis K, Markaki, Konsoula N, et al. *Brucella Spondylitis*: current knowledge and recent advances. J Clin Med. 2024;13(2):595. https://doi.org/10.3390/jcm13020595.
18. Shahed al, S, Haddad, A, Sammak, M. Imaging features of musculoskeletal brucellosis. Radiographics. 1994;14(2):333–48.
19. Huy TX, Nguyen TT, Kim H, et al. *Brucella* phagocytosis mediated by pathogen-host interactions and their intracellular survival. Microorganisms. 2022;10(10):2003.https://doi.org/10.3390/microorganisms10102003.
20. Kanna B. Kannan, Shetty, Rajasekaran: Diagnostic accuracy of whole spine magnetic resonance imaging in spinal tuberculosis validated through tissue studies. (1432–0932 (Electronic)).
21. Cevolani L, Facchini G, Pasini S, Bianchi G. Fungal spondylodiscitis: imaging findings and brief review of the literature. BMJ Case Rep CP. 2021;14(9):e242515. https://doi.org/10.1136/bcr-2021-242515.
22. Crete G, Karis R. Spinal coccidioidomycosis: MR imaging findings in 41 patients. AJNR Am J Neuroradiol. 2018;39(11):2148–53.
23. Lee L, Chung, Kim, Seo S. Candida spondylitis: comparison of MRI findings with bacterial and tuberculous causes. AJR Am J Roentgenol. 2013;201(4):872–7.
24. Furlan W, Massicotte, Sahgal F. Recent advances and new discoveries in the pipeline of the treatment of primary spinal tumors and spinal metastases: a scoping review of registered clinical studies from 2000 to 2020. Neuro Oncol. 2022;24(1):1–13.
25. Morales. Infectious Spondylitis Mimics: Mechanisms of Disease and Imaging Findings. (1558–5034 (Electronic)).
26. Kaya H, Karahan G, Sabah D. Is hydatid cyst with musculoskeletal involvement a problem that causes morbidity? Long-term follow-up and functional results. Indian J Orthop. 2021;56:680–8. https://doi.org/10.1007/s43465-021-00556-6.
27. Compagnone D, Cecchinato R, Pezzi A, et al. Diagnostic approach and differences between spinal infections and tumors. Diagnostics. 2023;13(17):2737. https://doi.org/10.3390/diagnostics13172737.
28. Rasouli. Mirkoohi, Vaccaro, Yarandi, Rahimi-Movaghar: spinal tuberculosis: diagnosis and management. Asian Spine J. 2012;6(4):294–308.
29. Theodorou T. Sartoris: an imaging overview of primary tumors of the spine: part 1. Benign tumors. Clin Imaging. 2008;32(3):196–203.
30. Shankar JP, Fau - Vasudev, Vasudev M, Fau - Ravishankar, Ravishankar S, Fau -, Sinha. Sinha: The usefulness of CT perfusion in differentiation between neoplastic and tuberculous disease of the spine. (1552–6569 (Electronic)).
31. Page MK, Bossuyt, Boutron H, et al. Mapping of reporting guidance for systematic reviews and meta-analyses generated a comprehensive item bank for future reporting guidelines. J Clin Epidemiol. 2020;118(Electronic):1878–5921.
32. Rajasekaran. Soundararajan, Shetty, Kanna: spinal tuberculosis: current concepts. Global Spine J. 2018;8(4 Suppl):S96–108.
33. Schwarz-Nemec U, Friedrich KM, Stihsen C, et al. VertebralVertebral bone marrow and endplate assessment on MR imaging for the differentiation of Modic type 1 endplate changes and infectious spondylodiscitis. J Clin Med. 2020;9(3):826. https://doi.org/10.3390/jcm9030826.
34. Teferi C, Challa M. Eschbacher: Surgical management of symptomatic vertebral hemangiomas: a single institution experience and literature review. (1878–1632 (Electronic)).
35. Cross A, Laing X. Imaging of compressive vertebral haemangiomas. Eur Radiol. 2000;10(6):997–1002.
36. Hanrahan, Christensen, Crim. Current concepts in the evaluation of multiple myeloma with MR imaging and FDG PET/CT. Radiographics. 2010;30(1):127–42.
37. Gaudino M, Colantonio, Lozupone V, et al. A systematic approach to vertebral hemangioma. Skeletal Radiol. 2015;44(1):25–36.
38. Patel KB, Poplawski MM, Pawha PS, Naidich TP, Tanenbaum LN. Diffusion-weighted MRI "claw sign" improves differentiation of infectious from degenerative modic type 1 signal changes of the spine. Am J Neuroradiol. 2014;35(8):1647–52.
39. Oztekin C, Kitis, Adibelli E, et al. Reliability of diffusion weighted MR imaging in differentiating degenerative and infectious end plate changes. Radiol Oncol. 2010;44(2):97–102.
40. Dumont K, Bloomer, Schwartz T, et al. Clinical utility of Diffusion-Weighted imaging in spinal infections. Clin Neuroradiol. 2019;29(3):515–22.
41. de Bron S, van der Horst-Bruinsma, van Royen. Discovertebral (Andersson) lesions of the spine in ankylosing spondylitis revisited. Clin Rheumatol. 2009;28(8):883–92.
42. Cawley Mi Fau - Chalmers. Chalmers Tm Fau - Kellgren, Kellgren Jh Fau - Ball, Ball: Destructive lesions of vertebral bodies in ankylosing spondylitis. (0003-4967 (Print)).
43. Berens. Roentgen features of ankylosing spondylitis. Clin Orthop Relat Res. 1971;74:0009–921. X.
44. Ostergaard L. Imaging in ankylosing spondylitis. Ther Adv Musculoskelet Dis. 2012;4(4):301–11.
45. Kim S, Song, Lee K. Andersson lesions of whole spine magnetic resonance imaging compared with plain radiography in ankylosing spondylitis. Rheumatol Int. 2016;36(12):1663–70.
46. McGauvran K. Diehn, Wald, Carr, Morris: SAPHO syndrome: imaging findings of vertebral involvement. AJNR Am J Neuroradiol. 2016;37(8):1567–72.
47. Franquet G. Alegret, Sanchis, Rivas: imaging findings of sternal abnormalities. Eur Radiol. 1997;7(4):492–7.
48. Depasquale KN, Fau -, Lalam. Lalam Rk Fau - Tins, Tins Bj Fau - Tyrrell, Tyrrell Pn Fau - Singh: SAPHO: What radiologists should know. (1365-229X (Electronic)).
49. Guglielmi C. Scalzo, Salaffi, grassi: imaging of sternocostoclavicular joint in spondyloarthropaties and other rheumatic conditions. Clin Exp Rheumatol. 2009;27(3):402–8.
50. Laredo V-B. Boutry, Cotten, Parlier-Cuau: SAPHO syndrome: MR appearance of vertebral involvement. Radiology. 2007;242(3):825–31.
51. Cao, Li, Xu, Wu S, et al. Spinal and sacroiliac involvement in SAPHO syndrome: A single center study of a cohort of 354 patients. Semin Arthritis Rheum. 2019;48(6):990–6.
52. Wagner SC, Schweitzer ME, Morrison WB, Przybylski GJ, Parker L. Can imaging findings help differentiate spinal neuropathic arthropathy from disk space infection? Initial experience. Radiology. 2000;214(3):693–9.
53. Ledbetter S, Sanders S. Spinal neuroarthropathy: pathophysiology, clinical and imaging features, and differential diagnosis. Radiographics. 2016;36(3):783–99.
54. Barrey M. Cotton, Perrin, rode: Charcot spine: two new case reports and a systematic review of 109 clinical cases from the literature. Ann Phys Rehabil Med. 2010;53(3):200–20.
55. Park. Taylor, szollar, Resnick: imaging findings in spinal neuroarthropathy. Spine (Phila Pa 1976). 1994;19(13):1499–504.
56. Ridley, Shaikh. Remedios, Mitchell: radiology of skeletal tuberculosis. Orthopedics. 1998;21(11):1213–20.
57. Sharif A, Clark, Madkour A et al. Brucellar and tuberculous spondylitis: comparative imaging features. Radiology: 1989;171(2):419–25.
58. Lyu FJ, Cui H, Pan H, et al. Painful intervertebral disc degeneration and inflammation: from laboratory evidence to clinical interventions. Bone Res. 2021;9(1):7.

59. Salaffi F, Ceccarelli L, Carotti M, et al. Differentiation between infectious spondylodiscitis versus inflammatory or degenerative spinal changes: How can magnetic resonance imaging help the clinician? Radiol Med. 2021;126(6):843–59.

Publisher's note