

Serial radiographic and ultrasonographic evaluation of discospondylitis in a cat: A case report of progressive vertebral lysis despite clinical improvement

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Abstract

A 10-year-old castrated male Korean Shorthair cat was presented with a 5-day history of lethargy and nonambulatory paraparesis. Neurological examination revealed delayed postural reactions in both pelvic limbs. Initial radiography of the lumbar spine revealed narrowing of the L7–S1 intervertebral disc space with ventral bony proliferation. Magnetic resonance imaging revealed collapse of the L7–S1 intervertebral disc space with heterogeneous T2-weighted hyperintensity and ventral contrast enhancement of the affected disc, as well as contrast enhancement of the adjacent paraspinal musculature. These findings suggested the presence of discospondylitis. Coccoid bacteria detected in both urine and splenic fine-needle aspirate indicated hematogenous spread. The cat was treated with broad-spectrum antibiotics and corticosteroids. It showed neurological improvement within 1 month and complete resolution by 6 months. However, serial radiographs revealed progressive L7 vertebral endplate lysis and vertebral body shortening despite clinical improvement. In contrast, serial ultrasonography more clearly delineated the resolution of paravertebral soft tissue changes and the disappearance of perilesional fat edema, which correlated with clinical remission. Serial radiographic follow-up of feline discospondylitis is rarely reported. This case documents progressive vertebral lysis in a cat despite clinical recovery, demonstrating that radiographic severity may not correlate with clinical status in cats with discospondylitis and underscoring the complementary role of ultrasonography in monitoring paraspinal pathology.

Keywords: discospondylitis, feline, follow-up, magnetic resonance imaging, radiography

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Introduction

Discospondylitis is an infectious disease involving one or more intervertebral discs and adjacent vertebral endplates (Moore, 1992; Thomas, 2000; Burkert *et al.*, 2005; Carrera *et al.*, 2011; Gomes *et al.*, 2020, 2022). Although it is typically diagnosed using magnetic resonance imaging (MRI) in conjunction with clinical findings, histopathological confirmation is often limited because of the invasiveness of the procedure and frequency of negative culture results, particularly in feline patients (Malik *et al.*, 1990; Shamir *et al.*, 2001; Gomes *et al.*, 2020, 2022). Hence, clinicians frequently need to rely on a combination of clinical signs and accessible imaging features for both diagnosis and treatment monitoring (Thomas, 2000; Shamir *et al.*, 2001; Gomes *et al.*, 2022). Follow-up imaging is essential for monitoring disease progression or resolution, and radiography is commonly used as the primary tool to assess osseous changes. However, interpreting serial radiographs can be challenging. Studies have reported a “radiographic lag” in dogs, in which osseous lesions continue to progress or appear more severe despite significant clinical improvement (Thomas, 2000; de Freitas *et al.*, 2022). This discordance complicates clinical decision-making, making it difficult to distinguish between ongoing infection and delayed structural remodeling. In this context, ultrasonography is a practical, noninvasive modality for evaluating paravertebral soft tissue changes, such as inflammation or abscessation. It can also provide a more comprehensive assessment of disease status than radiography alone (Gomes *et al.*, 2022).

While this radiographic lag is recognized in canine discospondylitis, the condition is extremely rare in cats, and longitudinal imaging findings remain poorly characterized (Kemelman *et al.*, 2019; Liatis *et al.*, 2020). Thus, a significant gap exists in the literature regarding how feline vertebral lesions change during recovery and whether they mirror the progressive radiographic changes noted in dogs. This case report describes the serial radiographic and ultrasonographic monitoring of discospondylitis in a cat over an extended 6-month period. This case is particularly notable as it demonstrates a clear paradox in which progressive vertebral lysis and shortening occurred simultaneously with marked clinical remission. Frequent rechecks with both modalities confirmed that radiographic osseous changes lagged behind the clinical course, whereas serial ultrasonography provided near real-time evidence of paravertebral soft tissue healing. These findings underscore the crucial clinical utility of ultrasonography for detecting and tracking paravertebral lesions that may be equivocal or unapparent on radiographs. They also indicate that a multimodal imaging approach is essential for accurately assessing therapeutic response in feline discospondylitis.

Case Description

A 10-year-old castrated male Korean Shorthair cat weighing 3.4 kg was presented with a 5-day history of lethargy and nonambulatory paraparesis. Neurological examination revealed delayed postural reactions in both pelvic limbs and marked spinal

hyperesthesia over the T13–L1 region. The patellar and gastrocnemius reflexes were increased in the left pelvic limb; however, the right side appeared normal. These findings were initially interpreted as being consistent with a thoracolumbar myelopathy. Hematological and biochemical analyses revealed elevated feline serum amyloid A levels (8.7 µg/mL; reference interval [RI], 0–5 µg/mL), hyperlactatemia (10.5 mg/dL; RI, 0.6–2.5 mg/dL), and mild hyperglycemia (152 mg/dL; RI, 71–148 mg/dL).

Thoracolumbar radiographs were initially obtained according to the suspected neuroanatomical localization. Moreover, lumbar spinal radiographs were obtained for a more comprehensive assessment. Thoracolumbar spinal radiographs were unremarkable. Lumbar spinal radiography revealed mild narrowing of the L7–S1 intervertebral disc space with ventral bony proliferation and a round soft tissue opacity ventral to the L7–S1 level (Fig. 1). Based on the clinical signs and radiographic findings, differential diagnoses included vertebral or spinal cord neoplasia and inflammatory or infectious diseases, such as discospondylitis. Given the incongruence between the neuroanatomical localization (T13–L1) and radiographic findings (L7–S1), MRI was performed to assess the thoracolumbar spinal cord parenchyma and to further characterize the ventral soft tissue lesion in the lumbosacral region.

Preanesthetic evaluation, including thoracic radiography, performed before the MRI procedure revealed no remarkable abnormalities. MRI scans were performed using a 1.5 T system (SIGNA Explorer; GE Healthcare) with a commercial 16-channel flex coil. MRI of the thoracic spine showed no evidence of lesions at the T13–L1 level despite the presence of clinical hyperesthesia. However, MRI of the lumbosacral region revealed a collapsed L7–S1 intervertebral disc space with dorsoventral extension of disc material, resulting in mild dorsal spinal cord compression (Fig. 2A). The affected disc and adjacent endplates exhibited signal changes consistent with inflammation, characterized by T2 hyperintensity and corresponding contrast enhancement on T1-weighted contrast-enhanced (T1CE) images (Figs. 2A and 2D). The paraspinal soft tissues exhibited ill-defined hyperintensity on transverse short tau inversion recovery (STIR) images. They also exhibited marked contrast enhancement on T1CE images (Figs. 2C and 2D). Neural involvement was also noted, characterized by mild thickening and enhancement of the bilateral sciatic nerve roots and the left L7 spinal nerve root (Figs. 2E, 2F, 2G, 2H). Moreover, the right sublumbar lymph node was mildly enlarged on T2-weighted images (Fig. 2B). Based on these comprehensive imaging findings, discospondylitis was considered the top differential diagnosis.

Subsequently, abdominal ultrasonography was performed using an ultrasound unit (ProSound F75; Hitachi Aloka Medical, Tokyo, Japan) with a 3.4–13 MHz linear transducer (UST-5415) to identify a potential primary focus of infection. A small amount of sludge was detected in the urinary bladder. Moreover, a hypoechoic splenic nodule, presumed to represent lymphoid hyperplasia, was identified. Evaluation of the L7–S1 region revealed a hypoechoic structure

extending ventrally from the intervertebral disc space, accompanied by irregular adjacent bone proliferation and marked perilesional fat edema (Fig. 3A). Urinalysis was performed due to the presence of bladder sludge and clinical suspicion, revealing coccoid bacteria. To assess potential hematogenous dissemination, ultrasound-guided fine-needle aspiration (FNA) of hepatic and splenic parenchyma was subsequently performed. While the hepatic aspirate revealed no remarkable findings, coccoid bacteria were detected in the splenic aspirate, consistent with the urinalysis results. These results, together with the imaging data, supported a diagnosis of infectious discospondylitis secondary to hematogenous dissemination.

Medical treatment was initiated with amoxicillin-clavulanic acid (18.3 mg/kg, BID), metronidazole (15 mg/kg, BID), prednisolone (1 mg/kg, BID), gabapentin (10 mg/kg, BID), and omeprazole (1 mg/kg, BID); the medications were gradually tapered over 10 weeks. Neurological signs began to improve within 1 week of treatment, as evidenced by increased activity and improved gait scores, and had completely resolved by the 6-month follow-up, when neurological examination was normal.

Despite the favorable clinical response, serial radiographic and ultrasonographic follow-up over 6 months revealed divergent changes in the L7–S1 region. During the initial 2 months, radiographs revealed narrowing of the L7–S1 intervertebral disc space accompanied by new bone formation along the caudal endplate of L7 (Figs. 4A and 4B). By 3–4 months, a clear discrepancy was noted between the two imaging modalities. Radiography revealed advanced osseous alterations, including pronounced endplate lysis and progressive shortening of the L7 vertebral body (Figs. 4C and 4D). In contrast, ultrasonography indicated a reduction in the perilesional fat edema, despite the continued presence of the ventral hypoechoic structure and presumed new bone (Fig. 3B). At the final 5–6-month follow-up, the radiographic abnormalities appeared to have stabilized, with persistent ventral bridging and sclerosis noted (Figs. 4E and 4F). During this period, ultrasonography confirmed complete resolution of the previously noted perilesional fat edema, with no residual soft tissue abnormalities detected (Fig. 3C). Overall, while the serial radiographs revealed ongoing osseous remodeling, the ultrasonographic findings demonstrated resolution of the paravertebral soft tissue changes, which corresponded more closely with the patient's clinical recovery.

Discussion

This case highlights a clinically significant dissociation between recovery and imaging findings during follow-up in cats with discospondylitis. Although clinical improvement was evident under medical management, radiographic changes failed to parallel the clinical course. On the other hand, ultrasonography consistently demonstrated gradual resolution of paravertebral soft tissue abnormalities.

Based on our review of the literature, this represents the first feline case in which persistent radiographic lag was documented despite complete clinical recovery, and disease evolution was longitudinally characterized using ultrasonography. These findings emphasize that reliance on radiography alone may be misleading when evaluating therapeutic response in cats and reinforce the importance of a multimodal imaging approach for comprehensive assessment.

In this case, a distinct discrepancy was noted between the initial neuroanatomical localization (T13–L1) and the definitive lesion site identified using MRI (L7–S1). This apparent “pseudolocalization” can be explained by a combination of referred pain and pseudohyperreflexia. The perceived hyperesthesia over the cranial lumbar region was most likely attributable to referred pain, or a phenomenon known as “nerve root signature” caused by spinal nerve or nerve root entrapment (Garosi, 2012). Furthermore, the exaggerated patellar reflex observed in the left pelvic limb was interpreted as pseudohyperreflexia rather than a true upper motor neuron sign. In feline patients, lesions affecting the L6–S1 spinal segments or the sciatic nerve can result in apparent hyperreflexia due to decreased tone in the stifle flexor muscles, which normally have an inhibitory effect on the reflex response (Garosi, 2009). These findings indicate that clinical localization in cats may be misleading because of complex pain referral patterns and reflex alterations, reinforcing the necessity of comprehensive imaging when discospondylitis is suspected.

A notable feature of this case was the marked dissociation between clinical resolution and progressive deterioration of radiographic findings. Although substantial improvement was noted in neurological function and overall condition within 1 week of initiating antibiotic therapy, serial radiographs continued to demonstrate osteolysis and vertebral body shortening for up to 3–4 months. This temporal discrepancy, commonly referred to as radiographic lag, likely reflects the physiological delay between osteoclastic bone resorption and subsequent osteoblastic remodeling (Shamir *et al.*, 2001; Kobayashi *et al.*, 2022). As infection control is achieved, osteoclastic activity initially predominates to remove the necrotic bone, paradoxically accentuating the appearance of lysis during the early phase of treatment. With disease resolution, osteoblastic activity gradually becomes dominant, resulting in sclerosis and bony bridging, which are radiographic features consistent with repair. These radiographic features were evident in our case at the 5–6-month follow-up. Although this phenomenon has been well-characterized in canine discospondylitis, longitudinal documentation of similar temporal patterns in feline patients remains limited (Shamir *et al.*, 2001; Gomes *et al.*, 2022). Therefore, the continuous radiographic progression of osteolysis and subsequent definitive stabilization observed in this case represents a previously unreported pattern in cats and supports the applicability of the concept of radiographic lag to feline discospondylitis.

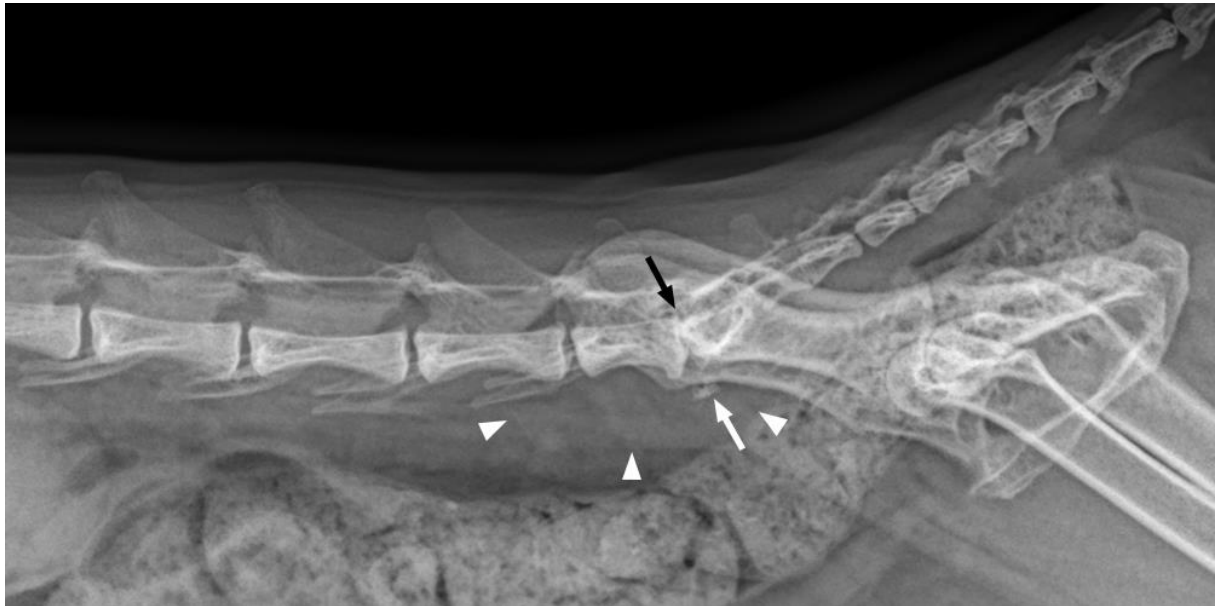


Figure 1 Right lateral radiograph of the L7-S1 region. Mild narrowing of the L7-S1 intervertebral disc space (black arrow) was noted. A ventral bone opacity (white arrow) at the L7-S1 level and a ventral faintly mineralized soft tissue opacity (arrowheads) with enthesophyte formation at the vertebral level were observed.

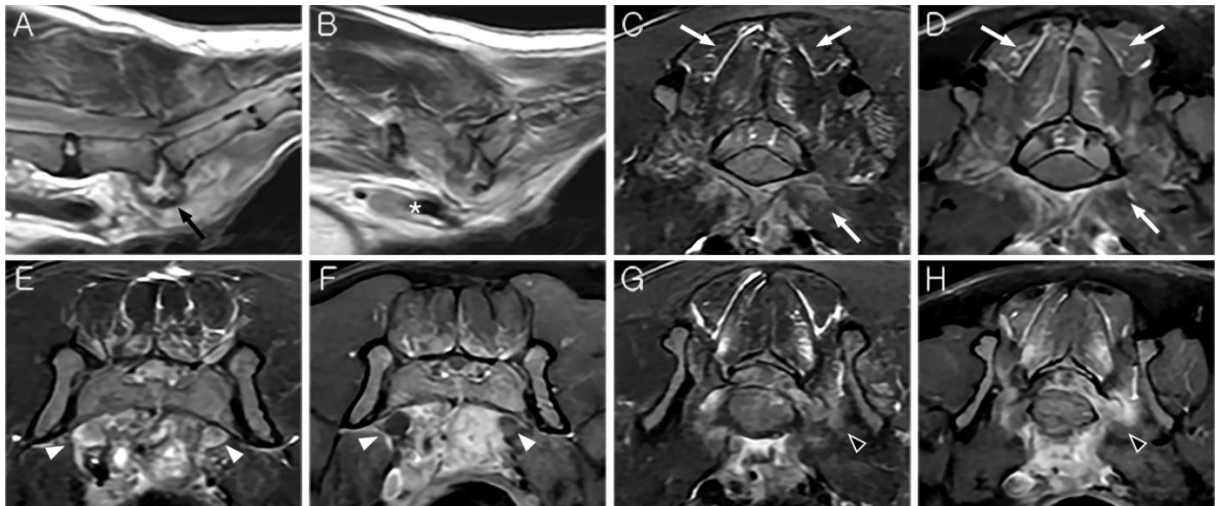


Figure 2 Magnetic resonance imaging findings indicative of discospondylitis in the L7-S1 region. T2-weighted (T2W) (A, B) sagittal, short tau inversion recovery (STIR) (C, E, G), and T1-weighted contrast-enhanced (T1CE) (D, F, H) transverse planes at the L7-S1 level. Collapse of the L7-S1 intervertebral disc space with dorsoventral extension of T2 hyperintense disc material (black arrow), resulting in mild dorsal spinal cord compression (A) and mild right sublumbar lymph node (asterisk) enlargement (B). Ill-defined paraspinal STIR hyperintensity and contrast enhancement (white arrows) (C, D). Mild thickening and enhancement of bilateral sciatic nerve roots (white arrowheads) (E, F) and the left L7 spinal nerve root (black arrowheads) (G, H).



Figure 3 Serial ultrasonographic changes indicative of discospondylitis in the L7-S1 region over 6 months. Ultrasonographic images of the lumbosacral spine at the initial examination (A), 3-month follow-up (B), and 6-month follow-up (C). At the initial examination (A), a hypoechoic structure (arrows) extending ventrally and a hyperechoic area (asterisk) suspected to represent new bone formation were identified; associated perilesional fat edema was also noted. At 3 months (B), the perilesional fat edema had improved, while the hypoechoic structure and new bone formation (asterisk) remained visible. At 6 months (C), the ventral lesion and caudal new bone proliferation (asterisk) at L7 persisted; however, no definite soft tissue lesion or perilesional fat edema was observed.

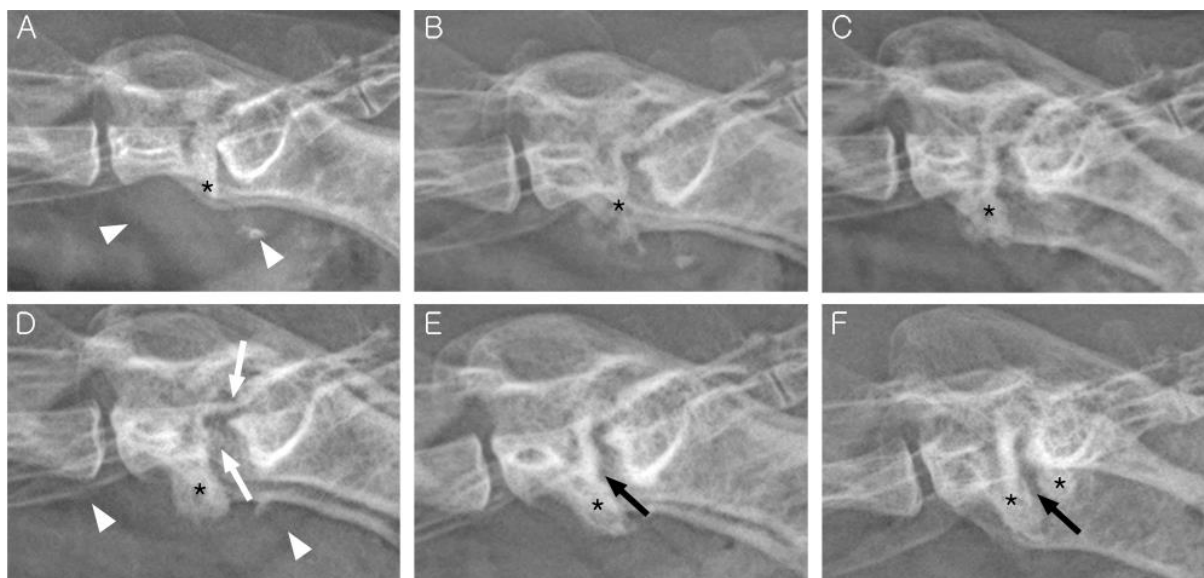


Figure 4 Serial radiographic changes indicative of discospondylitis in the L7–S1 region over 6 months. Right lateral radiographs of the lumbosacral spine at 1 (A), 2 (B), 3 (C), 4 (D), 5 (E), and 6 months (F). At 1–2 months (A, B), new bone formation at the caudal L7 endplate (asterisk) and ventral soft tissue opacity (arrowheads) were noted. At 3–4 months (C, D), L7 shortening, marked endplate lysis (white arrows), ventral bridging (asterisk), increased sclerosis (black arrow), and intervertebral space narrowing were observed. At 5–6 months (E, F), changes remained stable; indistinct soft tissue opacity and progression of bridging and sclerosis were noted.

Furthermore, this report highlights the distinct diagnostic advantages of ultrasonography for evaluating paraspinal soft tissue involvement during follow-up. Radiographic evaluation of the L7–S1 region is frequently limited by the superimposition of pelvic structures. In contrast, ultrasonography provides superior spatial resolution for detecting paraspinal soft tissue changes, including muscle enlargement, inflammatory swelling, and abscess formation (Gomes *et al.*, 2022). Prior veterinary studies have demonstrated that ultrasonography enables the direct visualization of inflammatory soft tissue abnormalities and perispinal or pelvic fluid accumulations that may be poorly defined or obscured on radiographs, thereby providing clinically relevant information beyond osseous assessment alone (Garosi, 2012; Kerwin, 2015). In this case, serial ultrasonographic examinations revealed the resolution of perilesional fat edema and associated hypoechoic soft tissue changes, supporting the interpretation that persistent radiographic abnormalities represented reparative remodeling rather than ongoing active infection.

In conclusion, this case documents feline discospondylitis at the L7–S1 junction confirmed by MRI. Serial follow-up revealed a pronounced radiographic lag, indicating that radiographic findings may not reliably reflect the therapeutic response. Adjunct ultrasonography corroborated clinical recovery by demonstrating progressive improvement of paraspinal soft tissue changes, thereby emphasizing the value of multimodal imaging during follow-up evaluation.

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Conflicts of interest: The authors declare no conflicts of interest.

Ethics statement: The owner provided consent for the use of the information.

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