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Brucella canis and Fungal Agents Predominate in Canine Discospondylitis: 5-Year Diagnostic Survey in an Endemic Region

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ABSTRACT

Discospondylitis is a recognized spinal condition in dogs; however, the distribution of its infectious causes and the reliability of diagnostic tools are not yet clearly defined. Over a 5-year span, medical files revealed 117 canine cases diagnosed with discospondylitis at our institution. Among these, 32 were identified incidentally on imaging, and 24 of those also exhibited concurrent neoplastic disease. From the remaining 85 dogs in which blood and urine cultures, Brucella serology, and galactomannan antigen testing were advised, a causative pathogen was confirmed in 45 cases. Brucella canis was identified in 10 dogs, while another 10 showed probable fungal infections. Serologic screening for Brucella suis yielded negative results in all 35 animals tested. Positive blood cultures were obtained in 28 out of 71 dogs (39%), and urine cultures in 12 out of 79 (15%). Bacterial growth was found at the lesion site in 4 of 8 dogs undergoing surgery and in 1 of 5 dogs that had image-guided sampling. Vertebral subluxations caused by discospondylitis were stabilized surgically in four cases. Comparable proportions of favorable outcomes at final evaluation were recorded among dogs with fungal, bacterial, or Brucella-associated disease and those diagnosed solely by imaging, although a few continued antimicrobial therapy or displayed relapsing signs. These findings emphasize the diagnostic usefulness of blood culture and reveal a relatively notable occurrence of Brucella spp. and fungal infections in discospondylitis.

Keywords: Brucella, Aspergillus, Canine, Blood culture, Galactomannan

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Introduction

Discospondylitis is a frequent cause of vertebral pain and, occasionally, neurologic impairment in dogs [1]. The condition arises from an infection—commonly bacterial, sometimes fungal—that begins in the vertebral endplates and progresses to involve adjacent bone and disc tissue. Most cases originate from hematogenous spread, as microorganisms lodge in the endplates where blood flow slows due to arterial looping, facilitating endothelial adhesion and tissue invasion [2–4]. Dogs of any age may be affected; in puppies, infection may occur via the umbilicus, although this route is more typical in livestock [5]. In older dogs, infection sources are often unclear but have been linked in some cases to urinary tract or pelvic infections [6–8].

Despite being well described, discospondylitis remains relatively uncommon [9], with most studies including fewer than 50 subjects. Clinically, it manifests as spinal pain—often during movement, posture change, or jumping. Many dogs display an acute onset with rapid improvement following prolonged antibiotic therapy [1]. Pain can be intense, and if bone destruction advances, pathological fractures and spinal cord compression may

result. Yet, chronic presentations with persistent discomfort and poor therapeutic response are also recognized [2].

A definitive diagnosis is complex, requiring both imaging confirmation [4, 10] and pathogen detection within the affected disc space and adjacent vertebrae. In practice, these criteria are seldom all fulfilled, leading to presumptive diagnoses based primarily on imaging features such as bone destruction and proliferation around an intervertebral disc [10, 11]. Such cases are typically managed empirically with broad-spectrum antimicrobials, even without pathogen identification [12]. This empirical approach often succeeds, though some dogs develop instability or fractures necessitating surgical stabilization [13].

In recent years, clinicians at our hospital observed that discospondylitis often responded poorly, with incomplete recovery despite long-term antibiotics. Given the regional prevalence of Brucella canis in U.S. dogs [14] and its link to chronic disease [15], this pathogen was suspected as a major contributor. Nonetheless, prior evidence suggests Brucella discospondylitis might be relatively uncommon locally [16]. Therefore, this study summarizes findings from a 5-year retrospective review of dogs with discospondylitis that underwent recommended diagnostic testing for multiple infectious causes.

Materials and Methods

To explore the etiology of discospondylitis and evaluate whether B. canis is a significant agent in this endemic area, we retrospectively examined dogs exhibiting imaging signs of the disease that presented to the Neurology Department at the Texas A&M University Small-Animal Hospital. The recommended diagnostics included advanced imaging (MRI, CT, or both), serological testing for B. canis using the indirect fluorescent antibody (IFA) and agar gel immunodiffusion (AGID) assays, B. suis screening with the card agglutination test, plus blood and urine cultures. For blood culture, at least three samples were drawn from separate vessels, spaced ≥1 hour apart, each collected into a 30 mL BD Bactec Plus culture tube (BD 442023) [17]. Ethical approval was unnecessary since this was a retrospective review of naturally occurring cases in privately owned dogs.

At the conclusion of the specified five-year observation period (1 January 2018–31 December 2022), all potential instances of discospondylitis in dogs were identified from the hospital's electronic database using the search terms "dog," "discospondylitis," and "diskospondylitis." Each entry was then manually reviewed to exclude those where diagnostic imaging did not provide sufficient evidence to confirm the condition. Two independent reviewers (NDJ and SCK) evaluated uncertain records against the inclusion standards outlined below; disagreements were resolved through joint discussion and consensus. Notably, a definitive infectious confirmation (e.g., blood culture) was not mandatory for inclusion.

Imaging results interpreted as consistent with discospondylitis demonstrated concurrent osseous lysis and bone proliferation adjacent to at least one intervertebral space [10, 11, 18], as seen on radiographs, CT, or MRI. A diagnosis was accepted if the following were observed: (a) for radiographs—erosion of the vertebral endplates on two or more views with bone proliferation [19, 20]; (b) for CT—localized destruction of adjacent vertebral endplates together with sclerosis or bone formation [10, 11, 15]; or (c) for MRI—erosion of neighboring endplates evident in T1-weighted images or enhancement of two or more structures among the disc nucleus, endplates, or adjacent soft tissues. Hyperintensity in T2-weighted (T2W) or STIR sequences, or T1-weighted enhancement of endplates, was not sufficient for confirmation unless accompanied by changes in nearby soft tissue or bony lysis [10, 11, 18].

Lytic foci often appeared as small, bubble-like areas close to the endplates, sometimes resembling Schmorl's nodes [21]. Differentiation from degenerative changes was mainly based on infection indicators and clinical interpretation. Dogs were included if their clinical and imaging characteristics were compatible with discospondylitis and antibiotic therapy had been initiated by the treating veterinarian. T2W or STIR hyperintense signals around discs were not alone diagnostic unless supported by systemic infection evidence (from blood or urine cultures, or serology) or CT-detected osseous lesions, due to possible overlap with noninfectious disorders like degenerative disc disease [21].

Evidence of infection was derived from (i) microbial cultures of blood, urine, or surgically/CT-guided sampled material, (ii) specific antibody testing (Brucella spp.), or (iii) galactomannan antigen assays for fungal infections (mainly Aspergillus spp.). Because serologic and antigen-based tests can produce false positives, fungal infection was confirmed only if culture yielded growth or if galactomannan levels were clearly elevated (>1.5) or consistently exceeded the positive cut-off (≥0.5). A preliminary positive result for Brucella canis by IFA was

followed by confirmatory AGID or tube agglutination tests (or positive culture). Both assays needed to be positive for classification as B. canis-infected [22]. Screening for Brucella suis—prevalent in feral hogs in the area [23]—using a serum card agglutination test (Texas A&M Veterinary Diagnostic Laboratory) was advised for all dogs beginning mid-2020. Blood was also analyzed for galactomannan antigen (MiraVista Veterinary Diagnostics). In certain patients, samples for culture were also obtained from affected discs or vertebral bone using CT-guided or surgical biopsy.

Blood culture samples were sent to the Clinical Microbiology Laboratory (CML), vented with a sterile venting needle (Thermo Scientific™ Remel™ Venting Needle; Thermo Fisher Scientific, Waltham, MA, USA), and incubated at 35 ± 2 °C in 5% CO_2. Anaerobic bottles remained sealed. After 24 h, 48 h, and 7 days, 0.1 mL of the medium was plated on trypticase soy agar with 5% sheep blood (BD 221261), MacConkey agar (BD 221270), and Columbia CNA agar with 5% sheep blood (BD 221353). Blood and CNA plates were incubated under CO_2, while MacConkey plates were placed in room air at 35 ± 2 °C. Growth was assessed daily. Anaerobic samples were plated on Brucella and CNA agars and incubated in sealed jars with anaerobic gas packs (Mitsubishi Gas Chemical Co., Tokyo, Japan) and an anaerobic indicator (BD) to verify oxygen removal; plates were checked daily for growth.

Urine was obtained via cystocentesis and cultured by spreading 1 µL and 10 µL aliquots on separate blood agar plates, and an additional sample was streaked on MacConkey agar. Blood agar plates were kept in a CO_2 incubator, while MacConkey plates were air-incubated at 35 ± 2 °C. Cultures were examined for up to three days, and bacterial counts were determined from colony growth on blood agar, expressed as colony-forming units (CFU) per mL of urine.

Data handling and analysis

Information from all qualified cases was compiled in Excel worksheets to organize clinical manifestations, diagnostic outcomes, lesion locations, pathogen testing, treatments applied, and therapeutic responses. Each case was grouped based on diagnostic confirmation as follows: (1) presumed infectious origin—consistent imaging findings and a positive test for infection; (2) imaging-based diagnosis only—imaging positive but infection tests negative; and (3) incidental finding—lesions discovered during imaging performed for unrelated purposes, such as screening for metastasis in oncology patients.

Clinical sign duration was divided into two categories: less than six weeks or greater than six weeks. This division was applied because (i) owners often could not specify how long signs had been visible; (ii) in certain dogs, symptoms might have been due to other concurrent diseases; and (iii) signs persisting beyond six weeks would typically correspond to visible skeletal alterations on diagnostic imaging [24].

Basic descriptive statistics were produced using Excel, including parameters such as body weight, age, sex, and reproductive status. As many owners did not know their dogs' exact ages, values were approximated to the nearest half-year. To assess the comparative occurrence of discospondylitis in German shepherds versus Labradors (the most frequent breed in the Neurology Clinic), the number of each breed seen during the study was extracted, and a risk ratio for confirmed discospondylitis between these two breeds was computed using Stata 17 (StataCorp, College Station, TX, USA).

Results and Discussion

A review of hospital records from the five-year period yielded 117 dogs that met the imaging inclusion criteria. Of these, 85 were evaluated by the Neurology Service and underwent a full diagnostic workup, while the remaining 32 were diagnosed incidentally—i.e., discospondylitis was identified during imaging for unrelated clinical reasons **(Table 1)**. Among the 85 dogs with complete diagnostic testing, 45 had a confirmed infectious origin and were placed in the presumed etiologic agent group, whereas 40 showed no infectious confirmation and were classified under imaging diagnosis only.

Table 1. Demographic characteristics of 117 dogs diagnosed with discospondylitis.

	Diagnostic Category					
	Presumed Etiologic Agent			Presumed Etiologic Agent	Imaging Diagnosis Only	Incidental
	Brucella spp.	Fungus	Other Bacteria			

Male castrated	6	5	10	21	18	18
Male intact	1	1	5	7	7	3
Female spayed	2	4	9	15	15	11
Female intact	1	0	1	2	0	0
Mean age (year)	3	5.5	6.3	5.4	8.0	11.6
Mean weight (kg)	29.3	29.8	29.5	29.6	23.8	25.6
CT	5	7	18	30	29	24
MRI	2	2	13	17	15	1

Cases classified as presumed etiologic agent (n = 45, **(Table 2)***)*

The subset of dogs in which an infectious cause was confirmed had a mean age of 5.4 years (SD 3.5) and an average body weight of 29.6 kg (SD 14.1). The sample included various breeds, but only six (13%) individuals weighed below 10 kg. The breeds most frequently represented were German shepherds (n = 11) and Labradors (n = 6).

Among these 45 dogs, 30 underwent CT scanning, 17 underwent MRI, and 8 were examined with both modalities. Multifocal lesions were noted in 25 dogs (56%), while the most prevalent site was the L7/S1 disc, affected in 21/45 (47%) cases. Clinical signs lasting over six weeks were reported in 20 dogs.

Table 2. Diagnostic procedures in the presumed etiologic agent group (infectious cause confirmed).

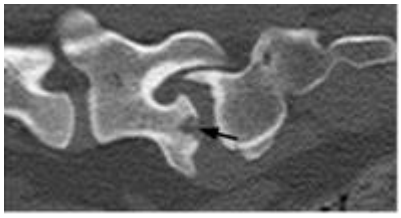
Test	Number Tested	Positive
Blood culture	37	28
Urine culture	39	12
Lesion culture	6	4
Brucella canis serology	39	10
Brucella suis serology	18	0
Galactomannan antigen	34	9 *

* One case verified via blood culture.

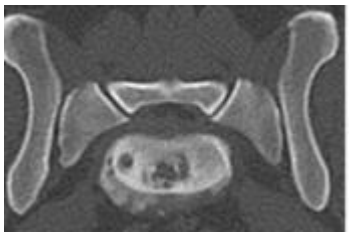
Of the 37 dogs tested by blood culture, 28 (76%) yielded positive results **(Table 1)**. Some individuals lacked blood culture data because infection was identified by other means: urine culture (n = 1), positive Brucella canis serology (n = 3), or direct lesion sampling (n = 4). Urine culture produced positive growth in 12 of 39 (30%) samples.

Antibody testing for Brucella canis was carried out in 39 dogs within this group; infection confirmation—through positive culture, a secondary positive test following an initial IFA, or both—was achieved in 10, primarily (7/10) from blood cultures. Another seven dogs belonging to the imaging-only group showed an initial IFA-positive result but lacked confirmatory positivity. None of the 18 dogs screened for Brucella suis/abortus tested positive.

In those dogs infected with Brucella canis, CT scans commonly revealed small, smoothly contoured lytic areas producing a “flask-like” erosion encircled by a thin sclerotic margin **(Figure 1)**.



a)



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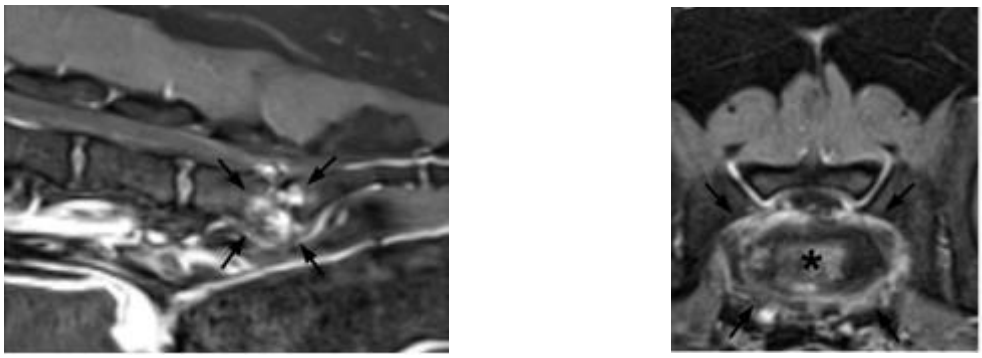
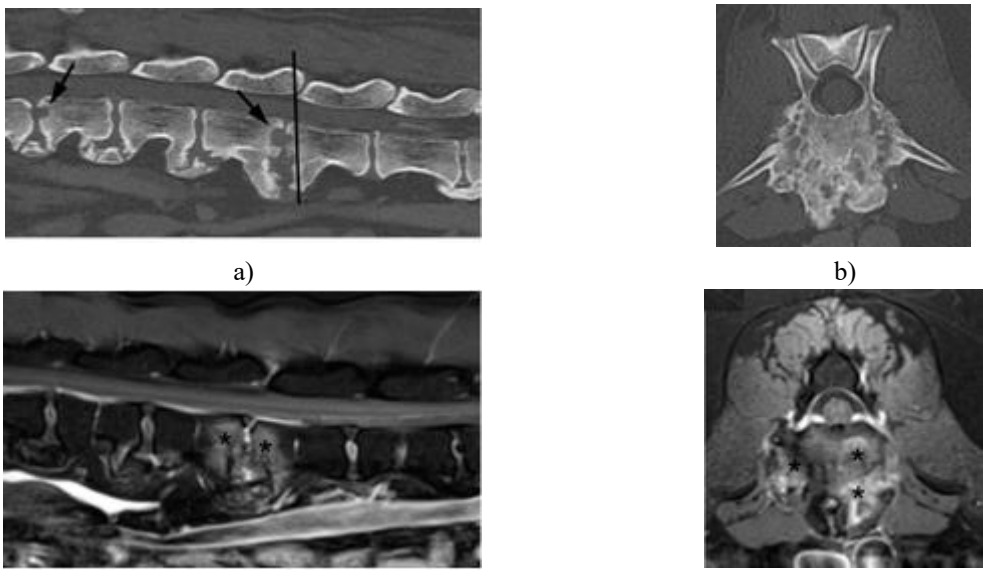


c) d)

Figure 1. CT and MRI examples from a dog diagnosed with Brucella canis-associated discospondylitis.
(a) Parasagittal CT at the lumbosacral junction showing a flask-shaped erosion in the L7 vertebral body (arrow).
(b) Transverse CT through caudal L7, showing bone loss with marginal sclerosis and ventral spondylosis.
(c) Mid-sagittal and (d) transverse T1-weighted post-contrast MR images of the same area demonstrate widespread enhancement within (*) and surrounding (arrows) the intervertebral disc.

A diverse set of bacterial species was isolated from blood, urine, and other sampling sites. Among these, E. coli (n = 7), Staphylococcus pseudintermedius (n = 6), and Streptococcus canis (n = 5) were most frequently identified. Ten dogs were determined to have probable fungal infections. In nine of these animals, the diagnosis was established through antigen detection assays (a total of 34 tests, comprising 32 serum, 2 urine, and 2 with both sample types), and in one case, through blood culture. The majority, seven dogs, were German shepherds, while the others were a Golden Retriever, a mixed-breed, and a Labrador.
Dogs showing fungal discospondylitis typically exhibited larger osteolytic zones on CT, sometimes with irregular, coarse margins, limited sclerosis, and increased adjacent bone proliferation **(Figure 2)**.

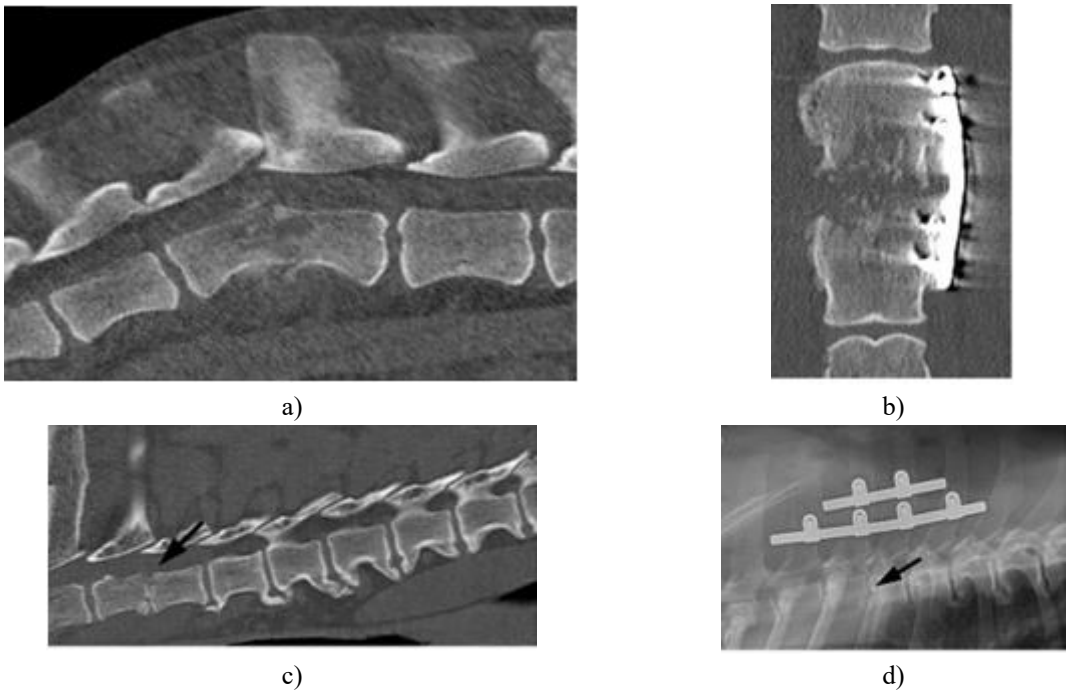


a) b)

c) d)

Figure 2. CT and MR scans of a dog diagnosed with fungal discospondylitis based on galactomannan antigen testing.
(a) Mid-sagittal CT of the mid-lumbar area and
(b) Transverse CT at the cranial L4 level (as shown in A) reveals multifocal lesions (arrows) with combined bone lysis and proliferation.
(c, d) Corresponding T1-weighted post-contrast MR images demonstrate marked enhancement (*) within L3–L4 vertebral bodies and endplates.

Cases involving bacterial agents other than Brucella canis displayed highly variable radiological patterns **(Figure 3)**. Some presented extensive bone destruction, while others had milder erosions with less proliferation. No clear correlation was observed between infecting organism and lesion type.



a) b) c) d)

Figure 3. Surgically managed discospondylitis due to **non-Brucella bacterial infection**.
(a) Mid-sagittal CT showing **L2/L3 subluxation** in a **Great Dane** with **Pseudomonas discospondylitis**.
(b) Stabilization with a **6-hole 3.5 mm DCP** and **pin–wire tension band** [25].
(c) Mid-sagittal CT showing **T3/T4 subluxation** (arrow) associated with **S. canis** infection.
(d) Site fixed using a **stacked De Puy CRIF implant** [26] with **5 mm clamps and 3.5 mm screws** (arrow).

Dogs positive for Brucella canis received doxycycline with enrofloxacin or cephalexin, while those infected with other bacteria were treated according to antibiotic susceptibility tests. Animals with fungal discospondylitis were managed using itraconazole, voriconazole, or terbinafine, sometimes in combination with antibiotics. One dog with repeatedly positive galactomannan tests was treated only with cephalexin due to diagnostic uncertainty.

The duration of clinical signs did not clearly distinguish Brucella canis cases—4/10 (40%) dogs had signs persisting over 6 weeks—whereas fungal infections were more prolonged (8/10; 80% with symptoms beyond 6 weeks). In contrast, dogs with other bacterial infections more often had shorter disease courses, with 18/25 showing signs for under 6 weeks, and 7 (28%) exceeding that duration.

At data collection, 8 of 10 dogs with Brucella canis infection were alive 1–22 months post-diagnosis, but continued antibiotic and NSAID therapy. One was euthanized due to zoonotic concerns.

Outcomes for other bacterial infections varied: 22 dogs received antibiotics for over 3 months; 19 were doing well between 1 and 30 weeks before loss to follow-up. Four later showed recurrent spinal pain (20–30 weeks post-diagnosis), though the cause was unclear. Two had persistent pain at 1–2 weeks, managed locally without additional data. Four dogs were euthanized between 2 and 82 weeks due to unrelated conditions, mostly neoplasia.

Among ten fungal cases, one was euthanized immediately, one died 16 days post-imaging, and the rest survived 1 month to 4 years (see Supplementary Material). One galactomannan-positive dog improved significantly in pain and mobility for 4 months under cephalexin monotherapy.

Six dogs underwent surgery: three for stabilization, two for biopsy, and one for foreign body (broken drill bit) removal from the affected disc. Cultures from these yielded four bacterial isolates. Of the stabilized dogs, two recovered well and were pain-free and ambulatory at 3–4 months post-op. Another, with L3/L4 stabilization, developed multicentric lymphoma at 4 weeks and was lost to follow-up.

Three dogs also had CT-guided aspirations; one confirmed S. pseudintermedius infection.

Comparative breed prevalence

During the study, 576 Labradors and 128 German Shepherds were admitted. Among them, 6 Labradors and 11 German Shepherds were definitively diagnosed with discospondylitis, yielding a risk ratio of 7.7 (95% CI: 2.9–20.4) for the latter breed.
Of the 10 fungal cases, 7 were German Shepherds and 1 was a Labrador, resulting in a risk ratio of 3.5 (95% CI: 0.96–12.9) for fungal discospondylitis in German Shepherds.

Cases diagnosed solely through imaging (n = 40)
A total of forty dogs were tentatively diagnosed with discospondylitis based on imaging outcomes alone. Although radiologic findings were highly indicative of the disease, no causative microorganism could be confirmed. The group's mean age was 8.0 years (SD = 3.6), and average body mass was 23.8 kg (SD = 12.1). Similar to the category with a suspected infectious cause, these dogs represented multiple breeds; the most frequent were Labrador retrievers (n = 8), German shepherds (n = 4), and mixed breeds (n = 3). Eight dogs (18%) weighed under 10 kg. More than half, specifically twenty-one (53%), had clinical manifestations consistent with discospondylitis persisting beyond six weeks.
CT imaging was carried out on 29 of these animals, while MRI was performed on 15. Multifocal lesions were observed in 23 cases (58%), and the L7–S1 intervertebral space was involved in 18 (45%). Radiologic abnormalities were comparable to those documented in dogs with presumed etiologic confirmation. Twelve dogs exhibited intervertebral collapse, subluxation, or both, while others presented mixed lytic and sclerotic patterns typical of milder discospondylitis.
All animals underwent infectious disease screening similar to the protocol for the other cohort, with all findings being negative (per inclusion requirements; **(Table 3)**). In total, 33 blood cultures, 36 urine cultures, and 33 serological assessments for Brucella canis were performed. Five dogs initially yielded positive IFA results for B. canis but tested negative upon repeat examination, while two additional dogs tested positive initially and lacked confirmatory testing. Seventeen dogs were analyzed for B. suis, all producing negative results. Galactomannan antigen detection was conducted in 26 dogs (25 from serum, 1 from urine, and 1 from both sources); all were below the diagnostic cut-off value of 0.5 (MiraVista Diagnostics), though two showed borderline increases not reproducible on retesting. Twelve dogs in this group developed additional non-orthopedic diseases either at presentation or within eight months after the discospondylitis diagnosis.

Table 3. Diagnostic investigations in the 40-dog cohort with imaging-based diagnosis (no infectious organism identified).

Test	Number Tested
Blood culture	33
Urine culture	36
Lesion culture	4
Brucella canis serology	33
Brucella suis serology	17
Galactomannan antigen	26

Two dogs underwent targeted image-guided sampling at lesion sites, but cultures yielded no bacterial growth. Surgical management was required in two other dogs—an 8.5-year-old neutered male Rottweiler and a 10-month-old Labrador retriever. The Rottweiler received decompression at T6–T7, with removal of epidural soft tissue presumed to be related to discospondylitis. The Labrador exhibited vertebral subluxation associated with the lesion and was stabilized via an internal fixation construct. Cultures from both surgeries were sterile. Following 3 months of cephalexin therapy, both dogs showed satisfactory clinical recovery.
Most dogs were treated with antibiotics—primarily cephalexin—combined with analgesic therapy (NSAIDs and amantadine). None were prescribed antifungal drugs. Twenty-six received antibiotics for periods exceeding three months. Of the total forty, twelve were lost to follow-up within three months, limiting assessment of therapeutic success. Sixteen animals recovered fully after 4–9 months of treatment, eight remained symptomatic after 5–9 months of antibiotics, and one failed to improve after a three-month course. Three dogs relapsed, showing recurrent pain at 6, 10, and 11 months after completing an initial three-month antibiotic regimen.

Incidentally detected discospondylitis (n = 32)

Discospondylitis was incidentally identified in thirty-two dogs during diagnostic imaging performed for unrelated health issues, mainly neoplastic disorders. The mean age in this subset was 11.6 years (SD = 2.7), with a mean weight of 25.6 kg (SD = 12.8). Twenty-four dogs underwent CT scans, one had an MRI, and seven were identified using standard radiographs. Multifocal involvement occurred in 21 cases (67%); eighteen dogs displayed mid-to-caudal thoracic lesions, and seven had changes at L7/S1. In general, the lesions appeared mild, showing rounded lucent regions surrounded by reactive sclerotic bone.

The predominant breeds were Labrador retrievers (n = 5) and mixed-breed dogs (n = 4). Twenty-four (77%) had concurrent neoplastic lesions **(Figure 4)**. Two dogs had identifiable bacterial sources—one postoperative wound infection and one case of septic peritonitis. Five dogs had unrelated systemic illnesses possibly linked to infection sources, such as stage-C valvular disease, acute renal failure, pituitary neoplasm, tibial plateau-leveling osteotomy implant, and prostate enlargement. The final case involved discospondylitis at T5/6 along with an acute herniated disc at T13/L1; this dog did not receive any specific treatment for the spinal infection.

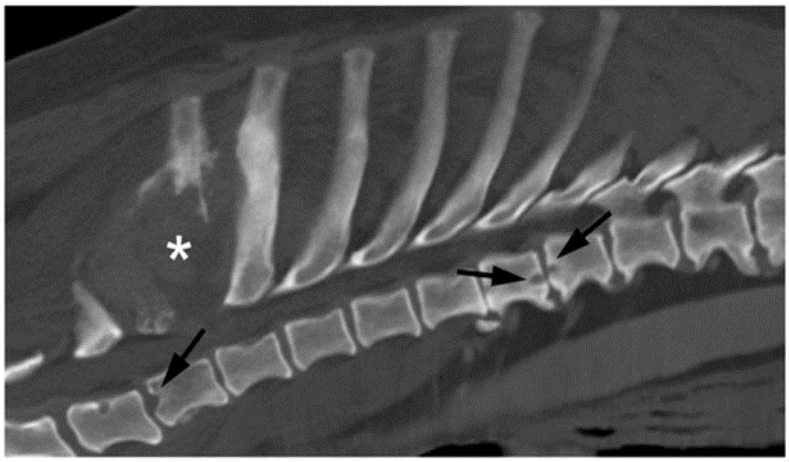


Figure 4. Multiple discospondylitis lesions (arrows) were incidentally discovered in a dog undergoing CT evaluation for a spinal neoplastic mass (*). No infectious organism was confirmed in this animal.

Testing for infectious causes of discospondylitis in this subset was limited, largely because the concurrent diseases were severe and often the primary clinical concern. Eleven dogs in this category underwent partial infectious disease screening (comprising one blood culture, four urine cultures, eleven Brucella canis IFA assays, and nine galactomannan antigen assessments—eight from serum and one from urine). All results were negative except for one case, in which E. coli was isolated from the urine sample. Seven dogs received antimicrobial therapy for discospondylitis—six treated with cephalexin and one with enrofloxacin.

Over the five-year observation period, 117 dogs were identified with imaging findings compatible with discospondylitis. Owners of 85 of these dogs consented to a standardized diagnostic workup aimed at determining etiology. The overall profile of our study population resembled that reported in a recent multi-regional study, though our dogs were slightly younger on average and showed a greater frequency of lesions at L7/S1 [27]. Across all subgroups, approximately 55–65% exhibited multifocal lesions on imaging.

Among the 85 dogs recommended for diagnostic testing, a definitive infectious cause was detected in 45 (53%), designated as the presumed etiologic agent group. Within this subset, Brucella canis—a pathogen with zoonotic potential—was confirmed in 10 dogs. Another 10 dogs had findings consistent with fungal infection, which remains relatively uncommon in discospondylitis cases [28–30]. The remaining cases involved other bacteria historically recognized as causal agents of the disease.

Dogs in the imaging-only diagnosis cohort had comparable breed and body-size characteristics to those with confirmed infections, though they were slightly older (8.0 vs. 5.4 years). Their imaging patterns were also similar but generally displayed milder osseous destruction. Taken together, these results imply that the imaging-only cases likely shared similar infectious origins with the confirmed group, but the organisms were undetected through available diagnostics. This inference provides useful guidance for clinical management when an infectious source cannot be identified.

The detection of Brucella canis as a cause of discospondylitis warrants particular attention due to its zoonotic implications. Experimental studies indicate that while most dogs clear B. canis infection spontaneously, those that remain chronically infected rarely respond to antibiotic therapy [14, 31]. Consequently, dogs testing positive for Brucella species should be regarded as persistent potential reservoirs for human transmission. Despite relatively high canine infection rates in parts of the United States, laboratory-confirmed human cases remain infrequent [32]. However, serological studies suggest higher exposure rates among individuals with occupational contact with dogs, even though overt illness remains rare [33]. Brucella suis, by contrast, represents a more significant zoonotic hazard and is known to be transmissible from dogs to humans [34].

Comparing the frequency of B. canis in this cohort to previous reports is challenging due to varying study durations. For instance, Long *et al*. (2022) [15] described 33 cases of B. canis-associated discospondylitis across four veterinary centers in Arizona and Colorado, though their sampling timeframe was unspecified. In our study, a definitive diagnosis of Brucella infection required both a positive initial IFA and a confirmatory test, or a positive blood culture. If the seven dogs in our imaging-only group with single unconfirmed positive IFAs were included, our positive rate would be substantially higher.

Serological testing for B. suis was completed in 35 dogs, all of which were negative, suggesting minimal exposure risk from wild hogs or their secretions. Hence, Brucella infections in this region likely stem from direct contact with infected dogs or contaminated reproductive material. Texas may represent an area of elevated risk compared to some regions in Europe, as infected animals are not legally required to be euthanized, potentially contributing to greater environmental persistence of the pathogen. Nonetheless, as noted previously [15], no clear age, breed, or sex predisposition was evident for B. canis discospondylitis.

Clinically, dogs infected with B. canis often relapse once antimicrobial therapy is discontinued, implying that bacteremia may subside during treatment but reemerge later. However, clinical improvement during therapy could also result from concurrent analgesic use, complicating assessment of antibiotic efficacy. It is generally advised that antibiotic regimens for discospondylitis not be excessively prolonged, due both to the low likelihood of complete eradication [14] and the risks of antimicrobial resistance arising from extended or indiscriminate use [35]. Findings from this series suggest that B. canis is responsible for a chronic, slow-progressing, and often refractory form of discospondylitis characterized radiographically by multiple, small, smoothly demarcated zones of vertebral endplate lysis—a pattern frequently observed in this part of the United States.

The incidental group and its implications for pathogenesis

The incidentally diagnosed cohort offered valuable clues regarding the mechanisms underlying discospondylitis in dogs. Animals in this group were notably older than those in the other categories (11.6 vs. 8.1 and 5.4 years) and exhibited spinal involvement at distinct anatomical regions, most often within the thoracic vertebrae rather than the lumbosacral (L7) area. Although a small subset displayed evident distant sources of infection, the majority were affected by neoplastic processes. This observation points to immunosuppression as a potentially important predisposing factor. It is well recognized that malignancies—and often their therapeutic management, particularly chemotherapy—are closely linked to reduced immune function [36].

Among the forty-five dogs classified as having an identifiable etiologic agent, ten (22%) were suspected of harboring fungal infections. Fungal discospondylitis in dogs has been infrequently reported in the literature [28–30], yet anecdotal evidence suggests it may occur relatively often in this geographic area. The comparatively high detection rate of fungal disease in our data could also reflect that a large proportion (62 of 85, or 73%) underwent galactomannan antigen testing—substantially more than in other large-scale reports on discospondylitis [27]. This condition is strongly linked with the German Shepherd breed, which is known to possess deficits in cell-mediated immunity, rendering them more vulnerable to fungal pathogens [37]. Although German shepherds were not commonly represented among referrals to our Neurology Service overall, they appeared disproportionately among those with suspected fungal infections.

In the current series, the suspected fungal form of discospondylitis appeared to behave as a chronic infection that often responded favorably to antifungal treatments such as voriconazole or terbinafine. While this region of the United States is generally considered endemic for certain fungal infections, it is improbable that the local climate alone predisposes to infection, since Aspergillus—the organism most often implicated in canine discospondylitis—has an almost global distribution [38]. Thus, exposure rates should not differ greatly from those in other regions or institutions.

Treatment response and surgical considerations

Some dogs in the present investigation exhibited persistent pain or recurrence of clinical symptoms typical of discospondylitis despite administration of suitable antimicrobial therapy. This outcome may indicate that surgical stabilization warrants more frequent consideration in canine discospondylitis to alleviate instability-related discomfort and neurological compromise. Comparable to human medicine [39], similar surgical principles may be applicable. Stabilization aids recovery by restricting motion, relieving compression on neural structures, and facilitating revascularization across the affected zone, thereby promoting bone fusion—similar to outcomes observed in long-bone osteomyelitis [40].

Given that bacterial adhesion is increased with polymethylmethacrylate compared to metallic implants [41], this material should be avoided for fixation in infected vertebrae, even though successful outcomes have been reported when used in combination with screws or pins [42]. Metallic implants can also serve as bacterial substrates, yet evidence from human spondylodiscitis suggests that the mechanical stability they provide outweighs this drawback [38, 43, 44]. Within this case series, internal stabilization procedures demonstrated comparable success in canine discospondylitis.

Diagnostic limitations and imaging interpretation

The diagnosis of discospondylitis raises significant interpretive challenges. A definitive diagnosis would ideally require histopathologic confirmation of inflammation and, when present, infectious organisms. However, inflammatory reactions in or near intervertebral discs may occasionally occur without an infectious cause [45, 46], explaining why a microbial agent is not always identified. Nevertheless, it is more reasonable to attribute the absence of detection to low diagnostic sensitivity rather than true pathogen absence.

Imaging alone can be imprecise: certain dogs may harbor infections without detectable bone abnormalities (possibly due to early-stage or low-virulence infections), while others present clear radiographic bone lesions without confirmed microbial isolation. Diagnostic interpretation also varies between observers, and different studies may use inconsistent inclusion standards—particularly older ones based solely on radiographs compared with modern MRI-based investigations. Currently, MRI is considered the most sensitive imaging technique for discospondylitis [10, 47], though differentiation from other endplate disorders can be difficult [22].

In this study, discrepancies between reviewers occurred most frequently in MRI interpretations, particularly regarding areas of contrast enhancement near vertebral endplates. Although such enhancement is abnormal, it is nonspecific and may accompany degenerative processes [18, 21]. Moreover, MRI provides limited resolution of bone endplates, making it difficult to assess sclerosis or bone loss, which are more clearly visualized with CT. While MRI can illustrate vascular patterns and bone marrow changes, it remains less informative regarding cortical destruction, traditionally key to diagnosis. Consequently, in this dataset, MRI findings showing soft tissue or marrow changes without concurrent evidence of bone lysis or proliferation in any modality were interpreted as noninfectious lesions. These corresponded mainly to dogs with prolonged, nonprogressive symptoms and no prior antimicrobial treatment.

Study limitations

While we suggested a defined set of diagnostic tests to the owners, the lack of financial support meant that not every test could be performed for all subjects. This limits the strength of our conclusions about the diagnostic efficiency of individual tests. However, this also improves the external validity of our findings [48], since the data likely reflect the actual detection rate expected in routine clinical settings, where, as in our study, many owners may not be able to afford all recommended diagnostic procedures.

Conclusion

Our findings indicate that the diagnostic approach used for identifying the causes of discospondylitis in dogs—through a recommended set of owner-approved tests—successfully detected B. canis and suspected fungal infections, both of which have significant clinical implications for case management. Among 71 dogs that underwent blood culture testing, 28 (39%) yielded positive results, whereas only 12 (15%) out of 79 urine cultures were positive. Earlier studies have advocated urine culture as the preferred diagnostic method for discospondylitis, recommending it over blood culture [49]. In contrast, while considering the noted limitations, our results suggest that blood cultures should be given priority. Evidence also indicates that suspected fungal discospondylitis can

often be effectively managed with extended antifungal therapy. Additionally, our data support that, when practical—particularly in cases involving single vertebral unit instability such as the lumbosacral junction—surgical stabilization may offer notable benefits and should be considered more frequently.

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