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Discospondylitis in the dog

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KEY POINTS

- Discospondylitis is a vertebral infection that mainly involves the intervertebral space and the associated epiphyses.
- Discospondylitis may be the result of a hematogenous infection, a paravertebral infection, or a peri- or postoperative infection.
- The clinical presentation is variable.
- Clinical signs may be mild and difficult to localize. Neurologic deficits are unusual on first presentation.
- Discospondylitis is one of the main differential diagnoses in medium to large breed dogs presenting with spinal pain.

INTRODUCTION

Discospondylitis (DS) is the most frequent presentation of vertebral infection. When the vertebral body is involved, the term spondylitis or vertebral osteomyelitis is preferred. The pathogens frequently isolated are bacteria – fungal causes are most unusual. Middle-aged, large, and very large breeds of dog are particularly affected, although any animal is potentially at risk. Males are affected more frequently than females. The high (C6–C7) cervical, mid (T5–T6) thoracic, thoracolumbar, and lumbosacral regions are most commonly involved (1–4).

The clinical approach to animals presenting with a vertebral infection includes not only the general and neurologic evaluation of the patient but also an investigation of the microbial pathogen responsible for the condition. Treatment and prognosis depend on the severity of the clinical signs and the pathogen involved.

ETIOPATHOGENESIS

Hematogenous infection

Infection via the bloodstream seems to be the most common route of infection in the dog (1, 2, 5–7). The pathogens isolated most frequently are bacteria (particularly coagulase-positive *Staphylococcus* spp.; **Table 1**). Fungi, particularly *Aspergillus* spp., may also be isolated, but less frequently.

The vertebral localization of the pathogens occurs through septic metastasis, which can happen during bacteremia in patients with septic foci in the cardiovascular, urogenital, and skeletal tissues. In addition, some clinical procedures may cause bacteremia (8). Thus, 66% of dogs and 80% of humans undergoing dental surgery have a positive blood culture (9), although the significance of this has been questioned (10). Endoscopy, biopsy, a urinary catheterization (8, 11) may damage the respiratory, digestive, or urogenital mucosa (which harbor a saprophytic microbial flora) and may result in episodes of bacteremia (12). However, the presence of bacteria in bone alone is not enough to cause disease. Many factors, local and systemic, concur in creating the conditions necessary for the bacterial colonization.

Local predisposing conditions

Alterations, especially traumatic, to the vertebral microcirculation are considered to be important cofactors in the development of a vertebral focus of infection. The case history of these patients may reveal previous episodes of trauma, and the high incidence in males of large breeds supports this hypothesis (13–15).

Systemic predisposing conditions

Alterations of the immune response have been identified in various patients with bacterial discospondylitis. However, the role of a deficient immune response in these patients is still being defined. It may be that the immunosuppression is a consequence of the

Table 1
Pathogens isolated in cases of DS in dogs

Bacteria	Fungi
Staphylococcus spp.	*Aspergillus* spp.
Brucella canis	*Paecilomyces varioti*
Streptococcus spp.	*Mucor* spp.
Escherichia coli	*Fusarium* spp.
Pasteurella multocida	
Actinomyces viscosus	
Nocardia spp.	
Mycobacterium avium	
Proteus spp.	
Erysipelothrix rhusiopathiae	
Corynebacterium spp.	

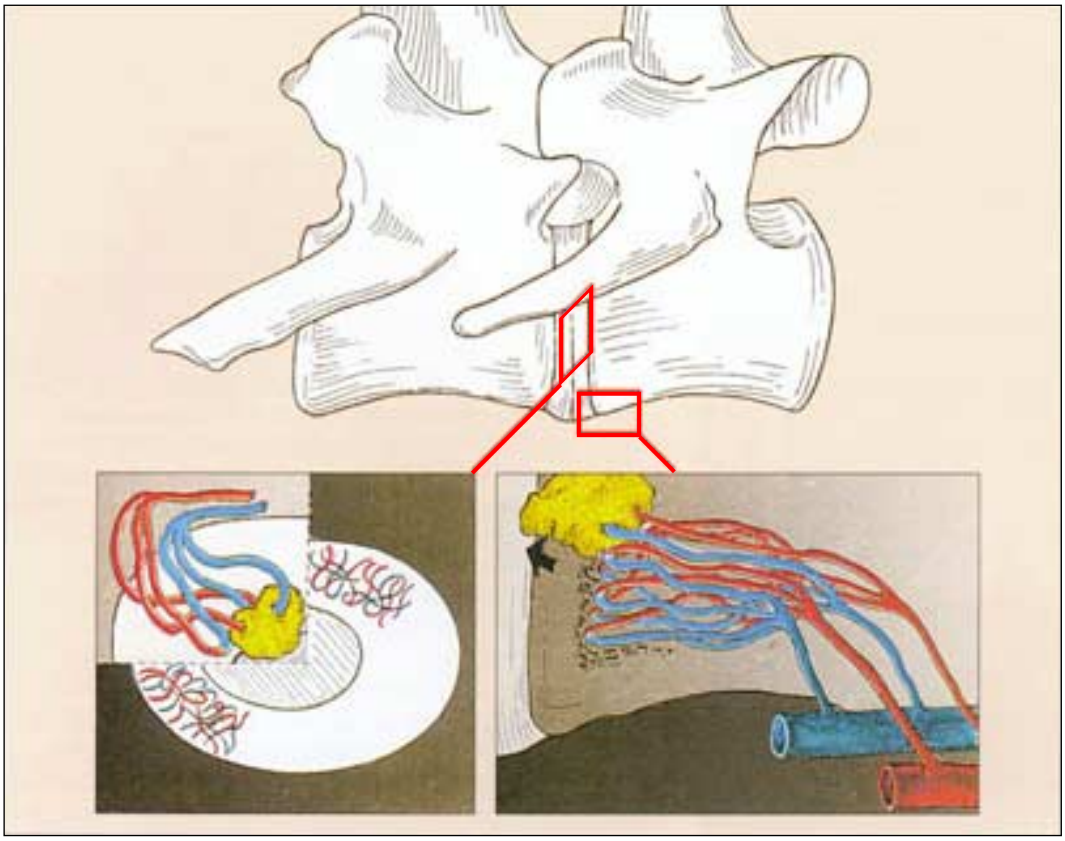
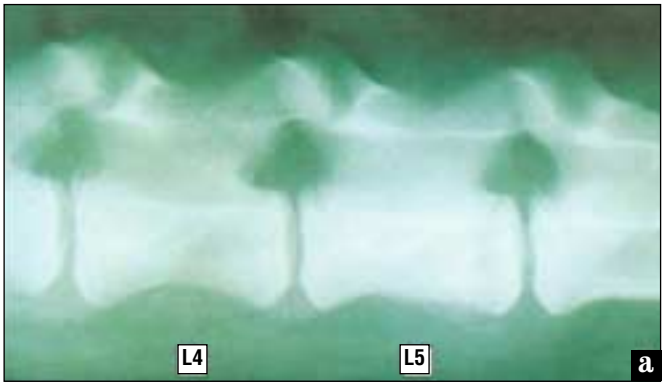


Figure 1
The particular structure of the capillary loops in the epiphyseal area is a predisposing factor to the development of vertebral infections. In human medicine it is postulated that similar vascularization also exists in the disc in some subjects. (Source: Corlazzoli, D.S. et al.: La discospondilite nel cane: aspetti eziopatogenetici, clinici, terapeutici e prime esperienze personali (Parte 1). *Veterinaria, 1994 (September); 8(3): 5–20. Reproduced with the Editor's permission).*



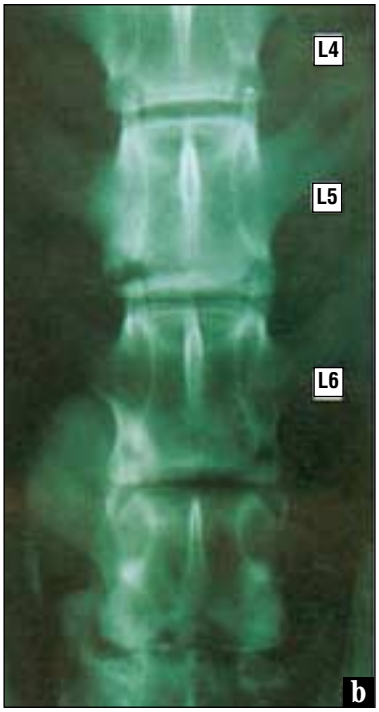


Figure 2
Male Rottweiler, 7 months old. Posterior limb ataxia, kyphosis, pain on lumbar palpation.
(a) *Lumbar spine, lateral projection: The L5 caudal physis is more radiolucent than in neighboring vertebral bodies, and the L5 body appears more radiopaque.*
(b) *Lumbar spine, ventro-dorsal projection: The L5 caudal physis shows an erosion in the right lateral part. The radiographic image is attributable to vertebral physitis.*

condition and not a predisposing factor (16).

Systemic mycoses are often associated with various forms of immunosuppression in humans and in domesticated animals (17). In the dog there may be breed or familial tendencies to various degrees of immunosuppression. Thus, the majority of cases of mycotic DS reported in the literature have been in German Shepherd Dogs, although whether this reflects a breed-related immunosuppression is unclear (14, 18–23).

Cause of the vertebral localization

In the metaphysis of the young animal, and in the epiphysis of the adult animal, the arterial capillaries form a narrow loop with the concavity directed to the feeding artery **(Figure 1)**. This narrow loop, and the sudden change in diameter from a fine arterial capillary to a large venous sinus, causes a slowing of blood flow and an increase in turbulence. As a result, microorganisms tend to accumulate in the efferent loop, aided by the reduced concentration of phagocytic cells here. This is followed by an initial inflammatory reaction leading to the formation of a microthrombus.

The capillary loops arise as nonanastomotic branches of the feeding artery. Their occlusion causes the formation of small areas of vascular necrosis and thus creates optimal conditions for bacterial multiplication. Alternatively, infection may spread from the epiphyseal area of a vertebra into the disc, and may even spread subsequently to the vertebra on the opposite side of the disc (4, 24) **(Figure 2)**.

It has been generally considered that the intervertebral disc becomes an avascular structure in adult life, both in humans and dogs. It receives its nutrient supply by diffusion across the cartilaginous part of the vertebral body and the periphery of the fibrous ring of the intervertebral disc (*annulus fibrosus*). However, some human studies contradict this hypothesis (25, 26). In effect, in humans there is a progressive decrease in perfusion, especially in the *nucleus pulposus*, but vascularization may be maintained, being derived from the fibrous ring and perhaps reaching the junction between this ring and the *nucleus pulposus*. Histologic studies of

dogs with fibrocartilaginous embolism demonstrated an inflammatory vascularization and reactive fibroplasia in the *annulus fibrosus*, close to the *nucleus pulposus* (24). This vascularization may explain why the microorganisms sometimes exhibit an initially discal localization. If this hypothesis was confirmed, it would explain those clinical cases in which the first detectable radiographic alterations are exclusively discal **(Figure 3)**.

Progression of the septic process

Once the infection is established, the tissue necrosis and bone destruction are perpetuated by multiplication of the pathogen, by the lytic nature that the exudate acquires due to elevated local lysosomal activity, and by the ischemic damage that follows as a consequence of the accumulation of an exudate in a rigid structure. A subarachnoid abscess may develop in some cases (27).

Subsequent development depends on the balance between the host defenses and the virulence of the organism of the lesion. If the defenses prevail, the devitalized tissue becomes surrounded by granulation tissue; if not, the infection progresses with invasion of the subchondral bony tissue and, eventually, the intervertebral disc.

The exudate that accumulates in the vertebrae may then diffuse to the vertebral canal or to the paravertebral soft tissues, destroying osteocytes and vascular structures and passing through the Haversian canals and Volkmann's canals **(Figure 4)**.

Propagation of the infection may affect the spinal cord, both by the action of the exudate and by the growth of granulation tissue, leading to spinal compression or, more rarely, meningitis.

The damage to the vertebral architecture is sometimes so great that there is complete disappearance of the intervertebral space, vertebral collapse, or, more rarely, vertebral fracture **(Figure 5)**.

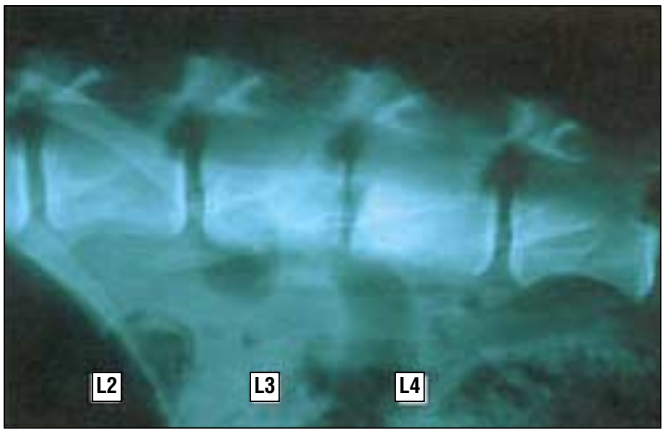


Figure 3 *Male Rottweiler, 4 years old, with paraparesis. The L3–L4 intervertebral space is reduced, and the vertebral epiphyses appear eroded.*

Paravertebral infection

Paravertebral infections may result from penetration by any type of foreign body – for example, inoculation of foreign material by bite wounds, injections, or gunshot wounds. However, the cases reported in the literature exclusively involve vertebral infections consequent to the migration of vegetable foreign bodies. These foreign bodies are most commonly the seeds of certain grasses, particularly *Bromus sterilis*, *Avena fatua*, and *Hordeum silvestre*, which, because of their shape, tend to move along fascial planes, aided by active muscle contractions. The most common penetration route is respiratory, although gastrointestinal and cutaneous penetration may occur. The final localization is typically in the ventral lumbar region, and they particularly come to rest at the insertion of the diaphragm on the ventral L2, L3, or L4 vertebral bodies **(Figure 6)**. These plant-derived foreign bodies carry a predominantly anaerobic microbial flora, although mixed infections are not uncommon **(Table 2)**.

The paravertebral infection may give rise to discospondylitis or spondylitis. Chronic sinus formation in the lateral fossa is frequently encountered **(Figures 7a, 7b** and **8)**.

Once the infection is established, the subsequent developments are exactly the same as those described above for the hematogenous form, with the sole difference of a marked tendency toward sinus formation and the reactive and productive involvement of the periosteum of the vertebral body.

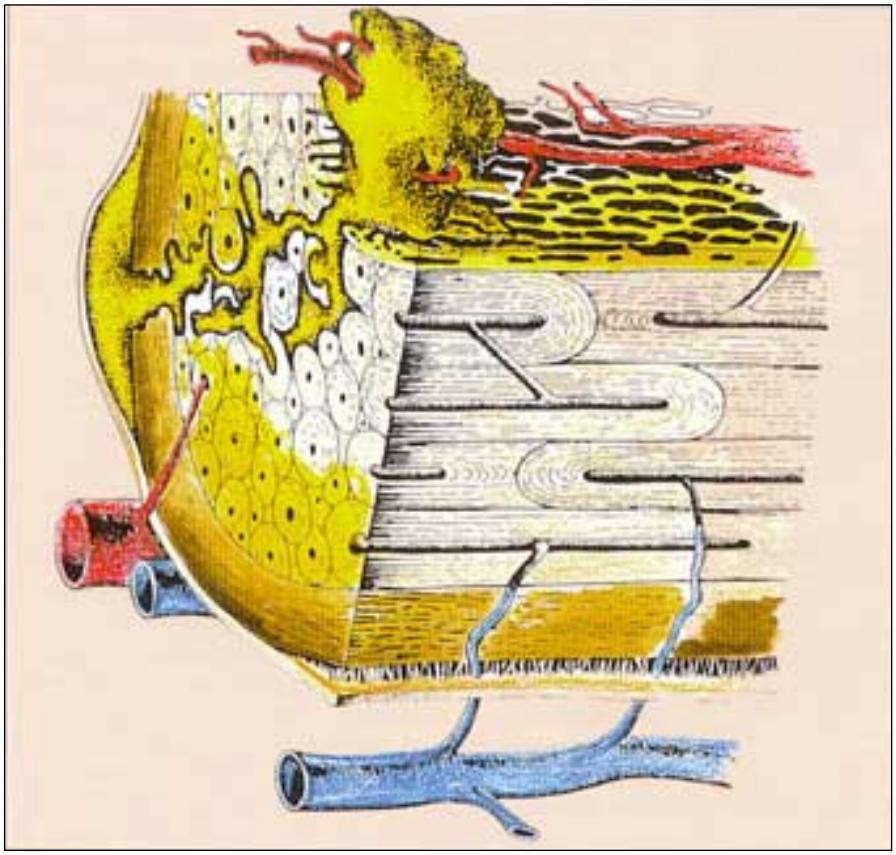


Figure 4
Exudate accumulating around the vertebrae may then diffuse into the vertebral canal or the paravertebral soft tissues, destroying osteocytes and vascular structures (modified from Resnick, D. and Niwayama, G.: Osteomyelitis, septic arthritis and soft tissue infection: The mechanism and situations. *In: Resnick, D. and Niwayama, G. eds.:* Diagnosis of bone and joint disorders. *W.B. Saunders Co., Philadelphia, 1988. Reproduced with the Editor's permission).*

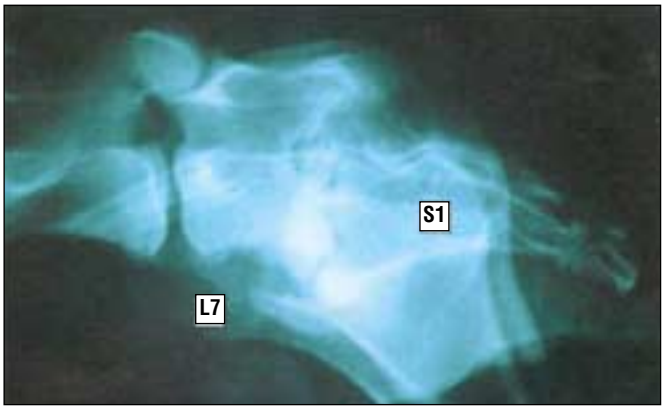


Figure 5 *Male mongrel, 10 years old, acute paraparesis. The L7 and S1 vertebral epiphyses are eroded, the intervertebral space has disappeared, and the sacral body is ventrally dislocated.*

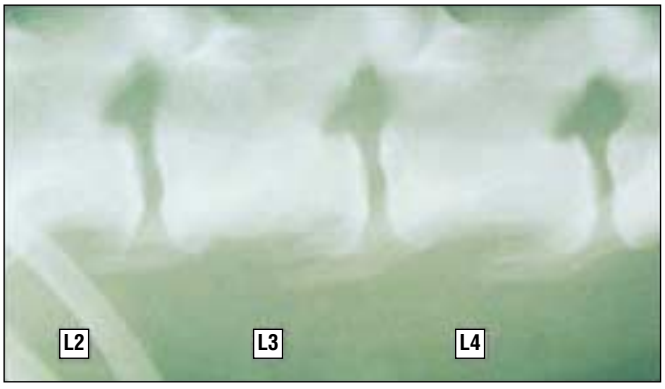


Figure 6 *Male English Setter, 5 years old, lumbar pain and kyphosis. Lumbar spine in lateral projection. The ventral L3 profile is irregular, with signs of osteolysis and bone production. The radiographic image led to a suspicion of a spondylitis due to plant-derived foreign body.*

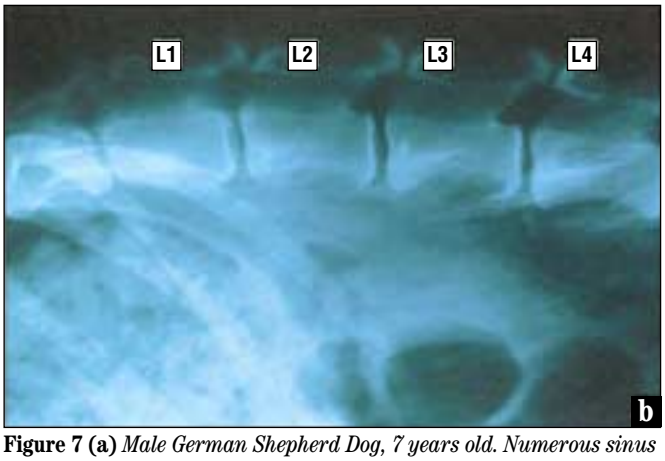


Figure 7 (a) *Male German Shepherd Dog, 7 years old. Numerous sinus formations in the left flank. The radiograph reveals vertebral infection.* **(b)** *Radiograph of the case in* Figure 7 (a). *Irregularity of the ventral margin of the L3 body. Spondylitis due to plant-derived foreign body.*

Table 2
Pathogens isolated during foreign body infections

Actinomyces spp.	*Proteus mirabilis*
Staphylococcus spp.	*Pasteurella* spp.
Streptococcus spp.	*Pseudomonas* spp.
Bacteroides spp.	*Nocardia* spp.

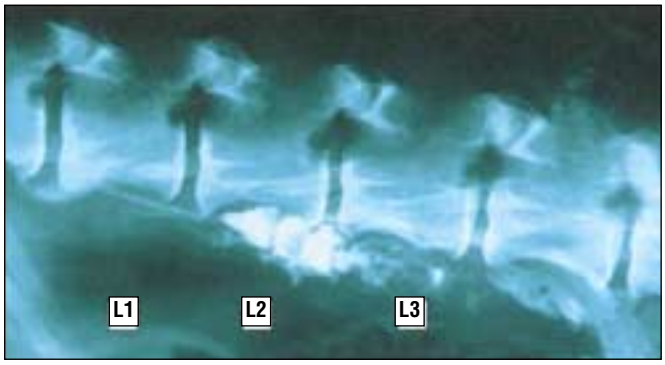


Figure 8 *Male mongrel, 7 years old. Numerous fistulae on the flank, and pain on lumbar palpation. Fistulography: the contrast medium reaches the paravertebral space as far as the L2–L3 articulation.*

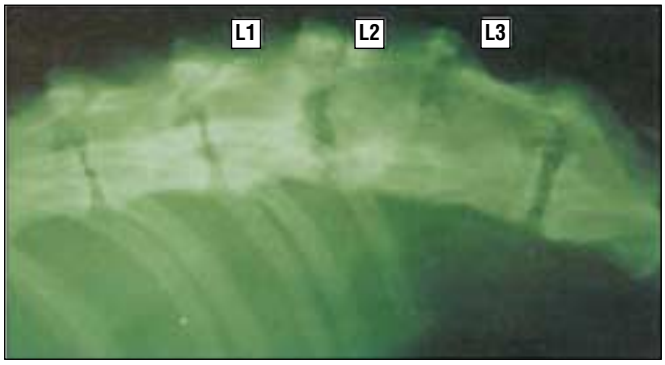
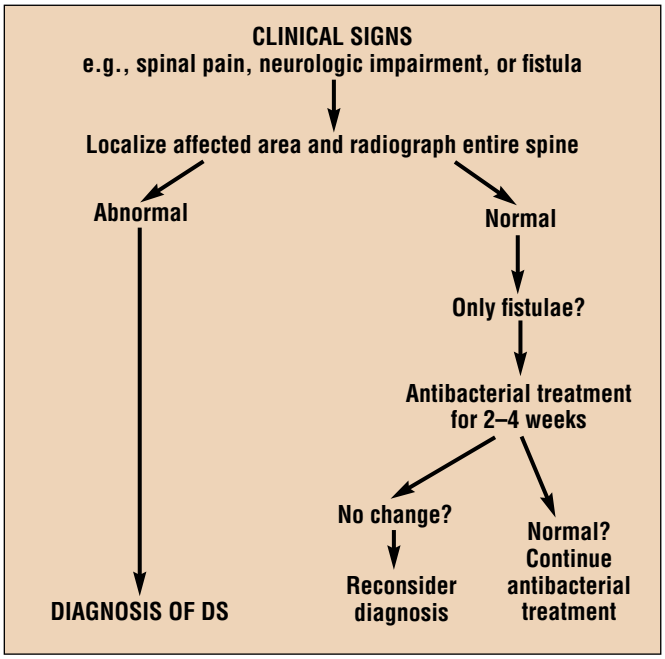


Figure 9 *Female Dachshund, 9 years old, spinal pain, kyphosis, right flank fistula. The patient underwent decompression surgery for acute disc extrusion 3 years previously. The radiographs suggest that extensive dorsal laminectomy had been performed. The L2–L3 space is fused. The L1–L2 intervertebral space is irregular and exhibits signs of lysis. It is unclear whether this is postsurgical DS or DS as a result of altered vertebral biomechanics.*

Post-operative infection

Post-operative infections, especially discal, are frequently reported in human medicine. In veterinary medicine they are a rare consequence of vertebral surgery. One of the authors (Daniele Corlazzoli) has observed a case of postoperative DS in a Dachshund that had undergone a dorsal laminectomy 3 years previously **(Figure 9)**. This patient was referred because of a right flank sinus and back pain. *Staphylococcus aureus* was isolated from the sinus. In this case the infection may have developed as a result of contamination of the surgical site, as a result of altered vertebral vascularization following the operation, or as a result of altered vertebral biomechanics. The extensive removal of the dorsal compartment of the vertebral column can potentially result in an increase of mechanical stress to the ventral compartment. Mechanical stress is a contributing factor to the development of DS.



Algorithm 1 *Management of DS: from clinical signs to diagnosis.*

MANAGEMENT OF DISCOSPONDYLITIS

The clinical management of a patient with DS may be divided into three phases:

- Diagnosis of vertebral infection **(Algorithm 1)**.
- Identification of the etiologic agent.
- Treatment **(Algorithm 2)**.

Diagnosis

History

The development of clinical signs and the clinical presentation of the patient may be extremely variable. The clinical signs may occur suddenly, sometimes accompanied by a neurologic deficit, or they may take a chronic, slowly progressive course. The owner often notes that their pet experiences difficulties climbing stairs or jumping into a car. There may be reduced activity, kyphosis, weakness of the hind limbs, a decline in general condition, weight loss, pain, and depression. Ataxia, paresis, and paralysis are the consequences of infections left untreated for a long time or treated with immunosuppressive drugs, such as glucocorticoids. Important factors in the case history include previous treatment with immunosuppressive drugs, injuries, urinary or respiratory infections, sinus formation, and surgical interventions.

Physical examination

The physical examination rarely reveals any specific features. Pyrexia is not a consistent sign, nor is deterioration in general condition. Alterations of gait, rigidity, kyphosis, and lameness are found in the majority of patients. Foreign body infections are frequently accompanied by sinus formation in the lateral fossa or by asymmetry of the lumbar musculature.

Neurologic examination

In the earliest phase the only identifiable clinical sign is vertebral pain. This may be revealed by deep spinal palpation but sometimes may give rise only to kyphosis, stiffness, lameness, or a reluctance to move. It is important to note that although pain is the clinical sign most frequently observed, it may not always be induced by spinal palpation and manipulation. Sometimes, especially in patients with multiple lesions, only a radiographic examination can reveal infectious foci in apparently asymptomatic areas.

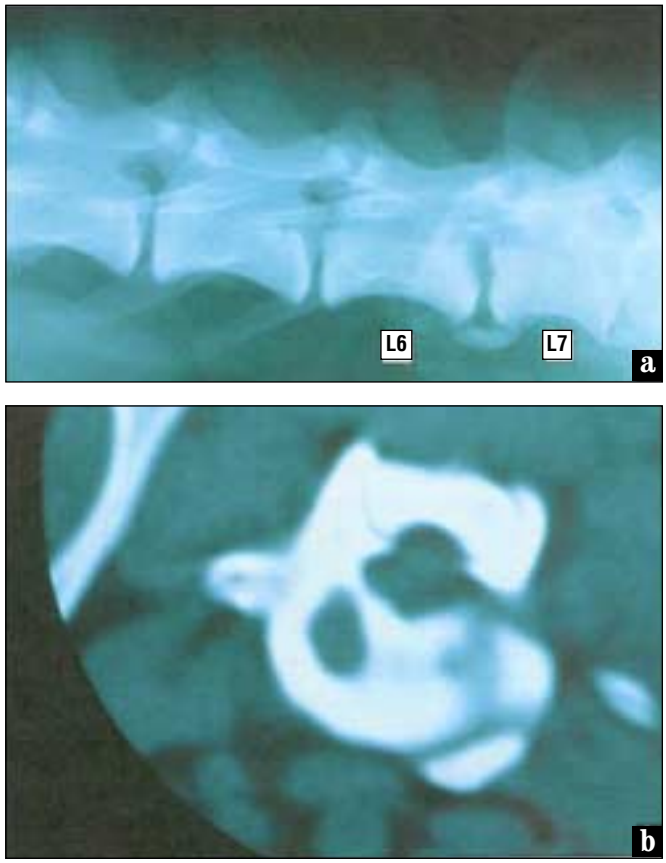


Figure 10 *Male Cocker Spaniel, 7 years old, pain on caudal lumbar palpation, modest proprioceptive deficit of the hind limbs.*
(a) *Lumbar spine, lateral projection. The dorsal part of the L6 caudal epiphysis is eroded. A 'bridging' osteophyte is present ventral to the L6–L7 articulation.*
(b) *Computer-assisted axial tomography of the lumbar spine, transverse scan: The L6 body shows an area of lysis on the right side.*

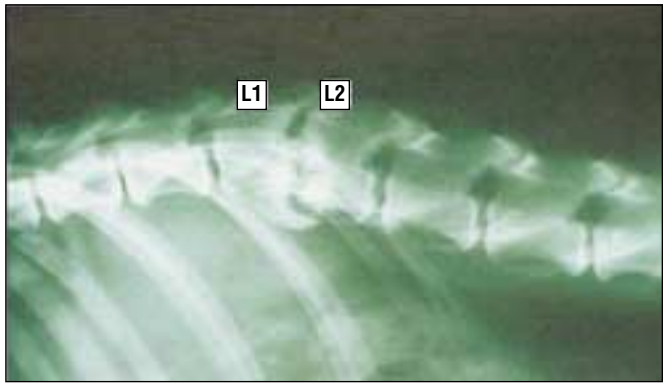


Figure 11 *Male mongrel, 9 years old: reduced L1–L2 intervertebral space and vertebral epiphyseal erosions. A large osteophytic lesion is present ventrally.*

If the infection is not stopped in its initial phase, signs attributable to involvement of the central nervous system (CNS) may become apparent. CNS involvement is typically a chronic process in which granulation tissue extends dorsally to compress the spinal cord or spinal nerve roots, leading to a deficit that depends on the degree of compression and the region involved (3, 27). This course may more rarely be interrupted by an acute complication, including vertebral collapse or pathologic fracture, diffusion of the infection into the vertebral canal with consequent local or diffuse meningitis, or, more rarely, myelitis.

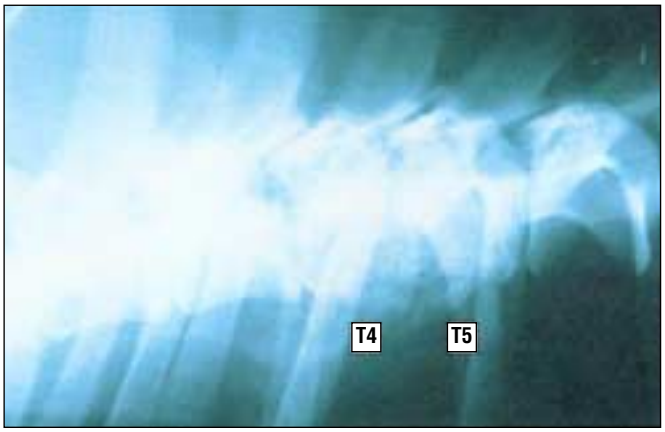


Figure 12 *Female German Shepherd Dog, 7 years old, acute paraplegia. Disappearance of the T4–T5 intervertebral space, altered radiopacity of the vertebral bodies, and ventral periosteal reaction.*

Figure 13
Male Boxer, 8 years old, paraparesis, pain on lumbosacral palpation. Area of erosion on the left caudal part of the L7 vertebral body and of the epiphysis of the sacral vertebra on the corresponding side.

Radiographic examination

A presumptive diagnosis is confirmed radiographically. The radiographic examination should be performed under general anesthesia. The entire spinal column should be examined **(Table 3)** to enable detection of clinically silent lesions.

In the initial phase DS is characterized by a reduction in the intervertebral space and lytic alterations of the associated vertebral epiphyses **(***see* **Figure 3)**. These alterations may sometimes be missing in the first 2–6 weeks (6, 23), so it is necessary to repeat the radiographic examination at a later date if the suspicion of vertebral infection remains.

In early phases, or dubious cases, computer-assisted axial tomography or magnetic resonance imaging may be helpful in revealing lytic phenomena that are less clearly imaged with conventional radiographic methods (28) **(Figure 10)**. However, this technology is rarely available to veterinary practitioners.

As the lesions progress, the destruction of the joint surfaces increases and progressive bone proliferation appears ventral and lateral to the intervertebral space **(Figures 11** and **12)**. The intervertebral space is usually reduced but may sometimes appear increased if there is marked joint surface lysis **(Figures 13** and **14a)**.

Lesions undergoing resolution are characterized by osseous

Table 3
A complete study normally comprises at least six radiographs in lateral projection

- Cervical (C1–C6)
- Cervicothoracic (C5–T3)
- Thoracic (T1–T12)
- Thoracolumbar (T10–L4)
- Lumbar (L1–L6)
- Lumbosacral

VD views should be taken in cases where a lesion has been identified, particularly if surgery is planned. In some rare cases the VD view can be diagnostic in the face of a 'negative' lateral view.

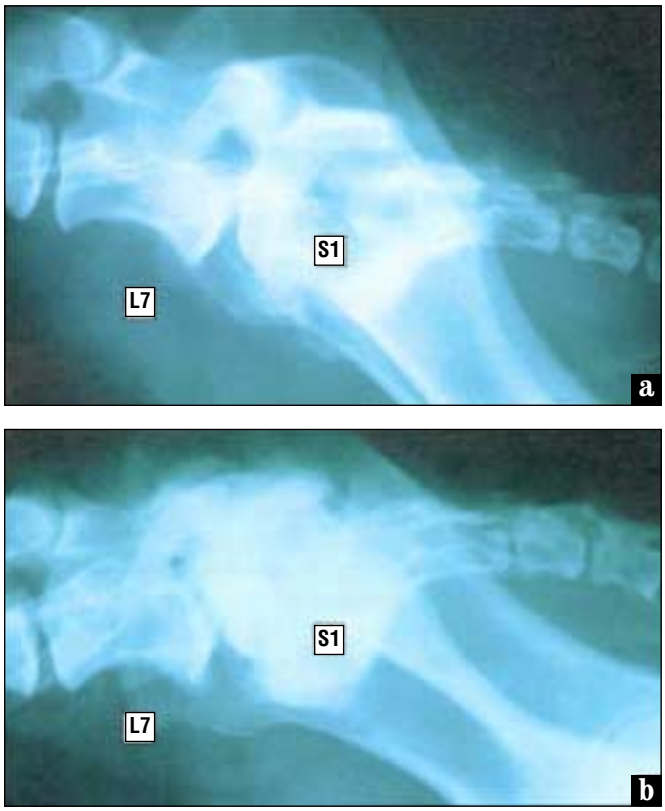


Figure 14 *Male Breton, 7 years old, rapidly progressive paraparesis, marked pain on lumbo-sacral palpation.*
(a) *Lumbar spine, lateral projection. The vertebral epiphyses and parts of the vertebral bodies of the L7–S1 intersomatic space are eroded.*
(b) *Same subject after 8 weeks, following antibiotic therapy. The clinical condition has improved greatly. Radiographically there is an increase in density in the previously eroded area, indicating cessation of lysis and attempted bone fusion.*

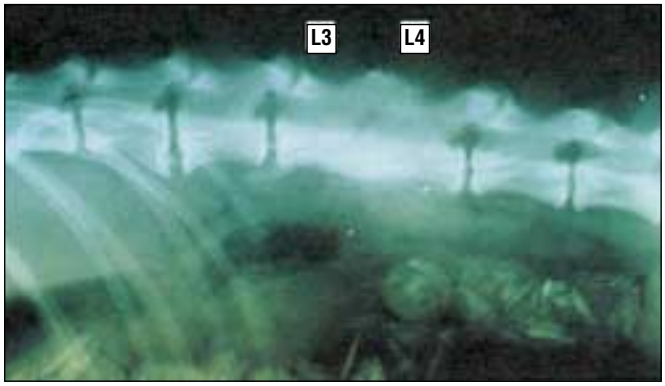


Figure 15 *Male Italian Bloodhound, 6 years old. Outcome of discospondylitis, the L3–L4 intervertebral space appears fused.*

sclerosis of the vertebral joint surfaces and arrest of the osteolytic process **(Figure 14b)**. It is sometimes possible to observe a tendency toward fusion of the intervertebral space **(Figure 15)**.

There is no correlation between the severity of the clinical signs and the severity of the radiographic signs, and it is not possible to differentiate radiographically between bacterial and mycotic DS.

Radiographic differentiation between spondylosis deformans (SD) and DS is important. SD is a degenerative condition characterized radiographically by the presence of vertebral osteophytes corresponding to the intervertebral spaces. In SD, the joint surfaces are regular and have a sharp profile, whereas in DS they are irregular and with a more or less extensive area of lysis. In addition, the osteophytes are regular and smooth in SD.

SD is a common finding, especially in breeds affected by DS, so that it is possible to find images attributable to both DS and SD co-existing in the same animal. Although differential diagnosis is sometimes difficult, it is aided by the radiographic differences between the two diseases, especially if the radiographic examination is repeated at a later time. Only in DS may there be a rapid change in the radiographic state **(Figure 16)**. The outcome of vertebral infection may present characteristics similar to those of DS, but it is generally possible to detect traces of previous infection sufficient to allow correct diagnosis.

The forms of DS caused by plant-derived foreign bodies present characteristic radiographic images. The lumbar spine is affected, typically L2–L4. At first there is osseous proliferation along the ventral surfaces of the vertebral bodies, which then extends to the lateral parts **(***see* **Figure 7b)**. The infection may be limited to the vertebral bodies (spondylitis) or extend to and involve the intervertebral spaces (discospondylitis).

Laboratory tests

Laboratory tests comprise blood counts, blood chemistry parameters, urine examination and cultural, and serologic tests. The hematologic results are rarely altered. It is sometimes possible to detect leukocytosis with a left-shift, but this is an inconstant finding of little indicative value. The blood chemistry tests and the urine examination are required to exclude the coinvolvement of other organs.

IDENTIFICATION OF THE ETIOLOGICAL AGENT

Serologic tests

Serologic tests to detect *Brucella canis* are extremely important in countries where this infection is present. The tests available at present are the rapid agglutination test on glass slides and the agglutination test in tubes. False-negative results are rare, but false-positive findings are frequent (29). It is appropriate to combine the two tests and, in doubtful cases, to seek confirmation by blood culture.

Serologic tests are also available for aspergillosis. However, the *Aspergillus* spp. antibody titer test indicates only exposure, not active infection. In addition, only 15% of patients with systemic aspergillosis give positive results.

Culture

Blood culture and urine culture must be included among the initial tests in all patients (29). The probability of isolating the pathogen is greatly reduced by previous antibiotic treatment and culture and sensitivity testing are mandatory. If the patient is undergoing antibiotic therapy, this must be suspended at least 24–48 hours before the first samples are taken for culture.

With regard to the blood culture, the best results are obtained with 3–4 samples taken no less than 1 hour apart over a 24-hour period (12, 29, 30). Surgical preparation of the skin is advisable. Urine cultures should be undertaken with urine obtained by cystocentesis. The blood and urine should be cultured aerobically and anaerobically, and the media used should include one for isolating fungi. Urine culture is considered to be the examination of choice for an early diagnosis of aspergillosis, and because of this, we include it routinely in the panel of tests for all patients with DS.

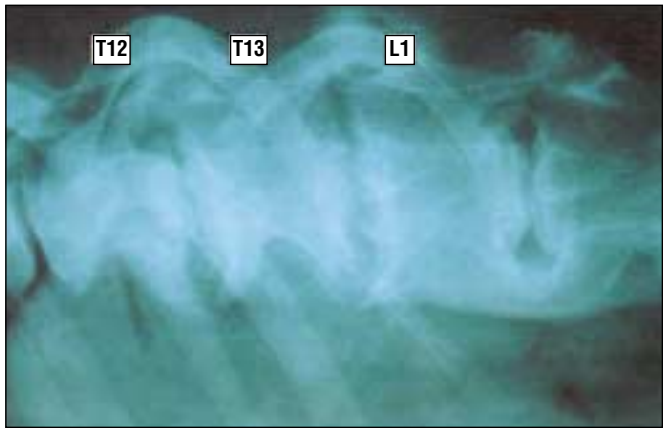


Figure 16 *Male German Shepherd Dog, 11 years old. Simultaneous presence of DS and spondylosis deformans. The T12–T13 and T13–L1 intervertebral spaces are narrowed and irregular, and the vertebral osteophytes have irregular margins (discospondylitis). The next space, L1–L2, exhibits a vertebral osteophyte with vertebral fusion, the intervertebral space does not exhibit lysis, and the vertebral epiphyses are regular, as are the osteophyte margins.*

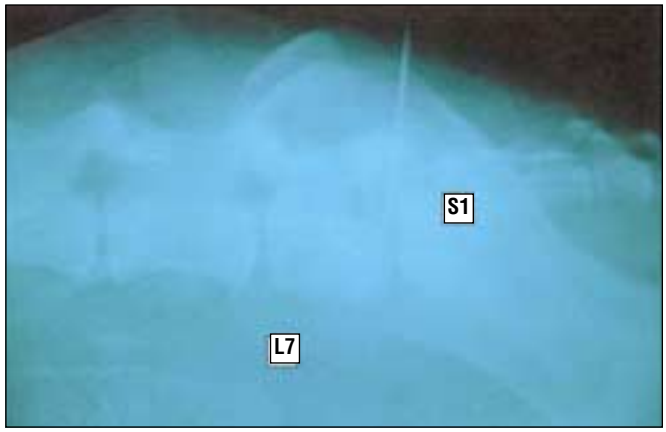


Figure 17 *Male German Shepherd Dog, 7 years old, DS of L7–S1. Percutaneous aspiration with spinal needle; radiographic control allows correct positioning of the needle to be evaluated.*

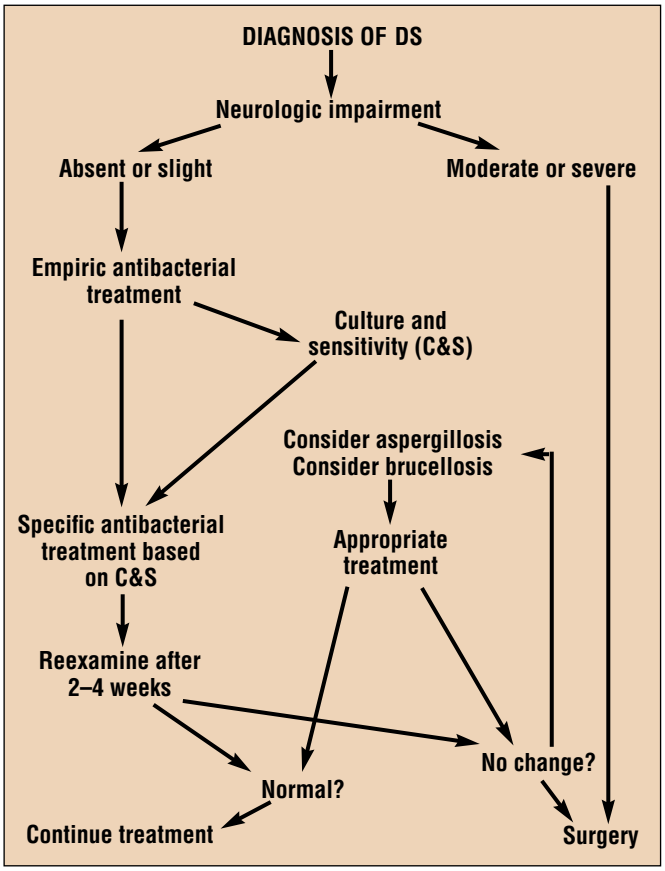
Material for cultural examination is obtained in all cases in which surgical decompression or curettage of the lesion is undertaken. The transcutaneous biopsy/aspiration of vertebral lesions to obtain material for culture is still a rare practice in veterinary medicine **(Figure 17)**, although it is commonly performed under fluoroscopic or radiographic guidance in referral institutions. It is advisable in those patients with a confirmed negative culture not responding to conservative therapy.

Urine sediment examination

The detection of fungal hyphae on examination of the sediment is considered highly significant during the course of systemic aspergillosis.

TREATMENT

Patients with DS can be divided into a number of categories (*see* (31) for example):



Algorithm 2 *Management of DS: from diagnosis to treatment.*

- Those without or with only mild neurologic symptoms.
- Those with moderate or severe neurologic dysfunction and that are not responding to medical therapy.

Patients in the first group are candidates for medical therapy, with good prospects of success, whereas surgery should also be considered for those in the second group **(Algorithm 2)**.

Medical treatment

The medical treatment comprises the use of antibiotics, analgesics (when necessary), and absolute rest. Steroidal anti-inflammatory drugs should not be used, as they may affect the immune response. Their use for other reasons or in incorrectly diagnosed cases sometimes masks the neurologic and radiographic signs of DS. Antibiotic therapy should be based on the culture results. In theory, the orthodox approach is to undertake blood cultures or at least urine cultures for confirmation whenever a diagnosis of DS is reached. Thorough examination of the heart, teeth, mouth, and (especially in males) the urogenital tract is also advisable. The early identification of patients suffering from aspergillosis is a fundamental requirement both because of the grave prognosis and in order to start specific therapy in good time.

Since *Staphylococcus* spp. are the pathogen encountered most frequently, it is advisable to start treatment immediately with β-lactamase–resistant antibiotics, such as cephradine and cloxacillin, while waiting for the culture results. Those two antibiotics achieve concentrations that exceed the minimum inhibitory concentrations in bone and pus in infected humans and have been effective *in vitro* in isolates from dogs with discospondylitis (31–33). If aspergillosis is not suspected, it is advisable to use cephradine (20 mg/kg t.i.d.).

Oral antibiotic therapy may be sufficient for mild symptoms and is continued for at least 8–10 weeks, although in some cases courses of treatment as long as 6 months may be necessary. Whenever possible, it is best to continue the therapy for at least 2–4 weeks after the resolution of clinical symptoms in order to prevent relapse (4, 33). Patients should be monitored clinically and radiographically every 2–4 weeks (31–33). Patients not responding rapidly to medical treatment and eventually worsening while on treatment need more frequent rechecks. A decrease in osteolytic processes and a tendency to vertebral fusion can be seen radiographically. The fusion state is often not reached, especially in young animals.

There is usually a rapid improvement in general condition and pain symptoms. If the patient has not shown a marked improvement in 7–10 days, it becomes necessary to review the treatment. If cultural confirmation has not already been undertaken, it is now imperative. Otherwise the therapy should be reviewed in light of the antibiotic sensitivity findings. Patients not improving despite medical therapy are candidates for surgery (31, 32).

Treatment of brucellar forms of DS includes two courses, at an interval of 3 weeks, of tetracycline hydrochloride (20 mg/kg, t.i.d., p.o. for 3 weeks) and streptomycin sulfate (20 mg/kg, b.i.d., i.m.). Streptomycin sulfate is administered only during the first 5 days of tetracycline therapy. To restrict the spread of the infection, entire males should be neutered. Patients require serologic and bacteriologic follow-up for at least 6 months. Brucellosis presents a zoonotic risk, and owners, as well as clinical staff, should be made aware of this.

In the case of systemic aspergillosis, medical therapy is administered if diagnosed early. Despite its nephrotoxicity, amphotericin B has been considered the treatment of choice, especially in severe infections (34). It is administered by slow intravenous infusion at a rate of 0.5 mg/kg in 500–1,000 ml of 5% dextrose solution on alternate days until a cumulative dose of 9–12 mg/kg is reached. Due to the nephrotoxicity and the route of administration, itraconazole (5 mg/kg, b.i.d., p.o.) is generally preferred for long-term treatment. If a good response to therapy is not obtained, the dose may be increased by 50%. Signs of toxicity include anorexia, increased serum ALT activity, ulcerative dermatitis, and vasculitis. In the event of side-effects, the treatment may be interrupted until appetite is restored, and the drug is then resumed at half-dose. In human medicine, removal of the infected tissue in invasive aspergillosis is recommended whenever possible in addition to the drug therapy (34).

The forms of DS caused by plant-derived foreign bodies require special treatment. The microbial flora carried by these foreign bodies is mainly anaerobic, and the pathogen isolated most frequently is *Actinomyces* spp. The treatment of choice for actinomycosis in humans and dogs is considered to be high-dose penicillin (65,000 IU/kg, t.i.d., s.c. or i.m.) for long periods (at least 4 months beyond clinical and radiographic resolution). Metronidazole is bactericidal to all clinically important anaerobes and is able to reach therapeutically effective concentrations even in bone tissue.

Surgical treatment

Surgery is reserved for patients with moderate or severe neurologic dysfunction and those not responding to medical therapy. Sometimes when the patient's general condition is good, the disease has been present for only a short time, and the patient has not yet received adequate medical treatment, it may still be possible to consider conservative treatment, even in the face of a moderate neurologic deficit.

The objectives of surgery are:

- Decompression.
- Curettage.
- Stabilization where necessary.
- Sampling of material for cultural examination.

In these patients it is preferable to administer treatments parenterally. Myelography is an obligatory step in these patients to define the position and extent of compression. However, if the cerebrospinal fluid analysis reveals an inflammatory process, myelography is contraindicated (35).

Isolated and readily accessible lesions are treated with curettage and decompression and are stabilized where necessary. In patients with multiple lesions it is necessary to identify the most significant of the lesions from the clinical point of view. Sometimes, especially in patients with multiple and poorly accessible lesions, surgical biopsy may be indicated if culture and antibiotic sensitivity tests have not been performed.

Curettage of lesions must be extensive. The necrotic bone tissue and disc material are removed completely. If the resulting defect is large, the possibility of a cancellous bone graft may be considered (3, 5, 31–33, 36, 37).

The decompression technique most widely indicated in the thoracolumbar region is hemilaminectomy, as the compression is particularly ventral. Dorsal laminectomy not only leads to greater instability of the spinal column but also involves removal of the spinous processes that may be useful if stabilization is required.

In the cervical region, a ventral approach allows adequate curettage of the lesions and better stabilization but not good decompression.

Discospondylitic foci are usually stable, but focal stabilization becomes necessary if more than one intersomatic space is decompressed or when vertebral instability is encountered radiographically or intraoperatively.

Patients with persistent sinus are also considered to be surgical patients, even in the absence of a neurologic deficit. The objectives of surgical treatment are:

- Exploration of the sinus track.
- Extensive curettage.
- Sampling of material for cultural examination.
- Removal of the foreign body/bodies.

The surgical approach may be lateral or transabdominal (36–38). The sinus may be revealed by injecting with methylene blue (a vital dye) 12–24 hours before the intervention (35, 38, 40). The dye reveals avascular tissues. Vascularized tissues do not retain the dye, which is then eliminated via the kidneys and liver.

The treatment of infections due to anaerobes requires extensive curettage down to the underlying vital bone tissue and the removal of hematoma and sequestra. It is extremely difficult to find any plant-derived foreign bodies that may be present, and the removal of one foreign body does not exclude the possibility that other fragments may be present. Remission of symptoms is possible only by the complete removal of all foreign material. Lateral drainage of these lesions is advisable.

Prognosis

The prognosis is principally influenced by:

- Early diagnosis and treatment.
- The pathogen involved and its sensitivity to antibiotic therapy.
- The degree of neurologic involvement.

The mortality rate associated with DS varies from 9–31% according to different authors (3, 4, 37, 39, 40), and many animals are put down because they do not respond to treatment.

The prognosis for patients with brucellosis is moderately poor, and the treatment may be long with frequent relapses.

Mycotic infections have a poor prognosis. The mortality rate associated with systemic aspergillosis in humans is 80%, but reports of cases that have survived are rare in the veterinary literature (41). Consequently, we consider it essential to exclude the presence of *Aspergillus* spp. before starting any treatment. The incidence of mycotic DS in dogs varies from 4–11% in retrospective studies (1, 2, 42, 43)

Discospondylitis due to plant-derived foreign bodies has a moderately poor prognosis if it does not respond to conservative therapy. Surgical removal of the foreign body is extremely difficult and relapses are very common.

Patients with mild or no neurologic deficit generally have a good prognosis, although some may deteriorate despite treatment. The prognosis for subjects with marked or severe neurologic deficits is less favorable (3, 4, 31, 37).

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