Osteomyelitis of the Vertebral Body and the Intervertebral Disk: Diskospondylitis
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Review of the Literature

Diskospondylitis is defined as concurrent intervertebral disk infection and vertebral osteomyelitis of contiguous vertebrae. In the dog, the infection has been attributed to Staphylococcus aureus, Brucella Canis, Nocardia-like organisms, Corynebacterium spp, Paecilo-mycetes variotii, and grass-seed foreign body penetration with secondary infection. Other organisms reported in animals and humans include Streptococcus spp, Pseudomonas spp, Escherichia Coli, Pasteurella spp and various mycotic organisms. The similarity of the disease process in humans and animals is interesting, since the comparative value extends into diagnostic, medical, and surgical aspects. Veterinarians need to take advantage of studies and reports in humans that may be beneficial to the animal patient. There are numerous reports in the human literature concerning vertebral osteomyelitis as an entity separate from intervertebral disk infection. Diskitis in children is considered a vertebral osteomyelitis with disk involvement, in contrast to intervertebral disk-space inflammation, which may not involve the endplate. Recently, vertebral osteomyelitis in infants has been distinguished from diskitis because of its rare occurrence and severity. The various manifestations of the disease process recognized in humans occur in animals, but the more subtle and less clinically apparent problems are difficult to diagnose in both.

Pathophysiology

The mechanism behind the resulting pathophysiology in diskospondylitis reflects the pathogenicity of the offending organism or the degree of inflammatory response to a penetrating foreign body and its attendant microorganisms. The nidus invariably arises from hematogenous spread, retrograde spread through the vertebral venous plexus, or directly from migrating foreign bodies, penetrating injury, or previous surgery. Possible sources for a septicemia and hematogenous access include dental extraction, contaminated intravenous injections, penetrating abdominal wounds, bacterial endocarditis, and genitourinary procedures or infection. Susceptibility to a hematogenous source has been implicated in patients with immunosuppression and diabetes. Retrograde spread through the vertebral venous plexus is a plausible consideration but a difficult one to establish definitively. The initial hypothesis of bacterial spread from the urinary system to the vertebral column through the venous pelvic plexus has been modified in humans after more recent studies. Retrograde return of venous blood through the vertebral sinuses after blockage of return through the great vessels has been documented in the dog. Any clinical condition sufficient to provoke a Valsalva maneuver (i.e., prostatitis, urethral obstruction) could divert blood through the vertebral sinuses. Regardless of an
arterial or venous access route, the microorganisms are sequestered by the low-flow venous and arterial systems to the subchondral plate region of the vertebral body. Direct extension of an infective process through foreign body migration from a percutaneous entrance or through the bowel wall has been described. Diskospondylitis (diskitis) has occurred after disk surgery in humans and in dogs after prophylactic disk fenestration.

Locally destructive processes are thought to be the result of lysosomal activity subsequent to leukocyte infiltration. Destruction of bony trabeculae results in penetration of the subchondral end-plate and extension into the intervertebral disk. Reactive change may be seen involving the intervertebral foramen, the vertebral foramen, and the neural arches. Neurologic signs occur as an aftermath to the infection if there is direct extension of the granulation tissue to involve the meninges and occasionally the cord. Compression of nerve roots and inflammation may cause radicular pain and even intermittent claudication depending on lesion location. The basic mechanism for cord damage was proposed by Kulowski in 1936 and remains feasible today. Cord compression may be the result of infiltration of inflammatory cells and meningeal edema or of ischemic changes due to septic thrombosis of spinal vessels. Septic diskitis is of particular concern because of the increased chance for rapid cord involvement and paraplegia.

On occasion, the infective process will extend into adjacent body cavities and may simulate disease of major organ systems. Since the advent of antibiotics, indiscriminant therapy may hold the disease process somewhat in abeyance, but insidious progress may occur, leading to suspicion of neoplasia.

**Clinical Presentation**

Age at presentation is reported to range from 8 months to 10 years. In one study the mean age was 5.1 years for 32 dogs, with a median age of 5 years. Another study involving 27 dogs reported that 30% of the dogs were less than 2 years old, and 14 were 4 years old or less. Large and giant breed dogs predominate for no obvious reason, with a disproportionate number of German shepherds and Great Danes represented.

There is general agreement as to clinical signs noticed initially by the owner, on presentation to the veterinarian, and later as a referral. As in humans, the signs vary in severity and may mimic other diseases especially in large breed, middle-aged to older male dogs. Spinal pain and neurologic deficit are readily recognized, but the subtleties of hyperplasia, depression, loss of appetite, and weight loss may be confusing early in the course of the disease. More profound disease occurring as an extension of the vertebral osteomyelitis may overshadow the initial signs. If the disease process progressively involves the nerve roots and spinal cord, more definitive signs referable to the nervous system are noticeable, primarily as paresis/paralysis. The latter seldom occurs in the absence of pathologic fracture. A history of trauma was noted in 50% of the cases in a third study, but the trauma was considered a contributing factor of susceptibility to disease localization rather than a late complication. Nonsurgical trauma to the back was not a predisposing factor to osteomyelitis in one series of human cases.

General signs of inanition, an arched back, stilted gait, and pyrexia may be associated with a variety of disease processes. In this age and size of dog with no history of previous disk surgery or penetrating foreign body, other disease processes would have higher priority than diskospondylitis, or may be occurring simultaneously. In children with intervertebral disk inflammation, the most common symptoms are back or hip pain and a refusal to walk. Reluctance to walk in a dog with preexisting or documentable musculoskeletal or neurologic problems could be an early premonitory sign. A stilted gait or arched back could be caused by a myriad of conditions from intervertebral disk disease to prostatitis, renal or urethral calculi, to hip dysplasia. Evidence of neurologic disease must be correlated with other hard findings. Older large breed dogs with neoplasia involving the spinal cord and nerve roots, lumbosacral stenosis, lumbosacral malarticulation, and degenerative myelopathy could have similar neurologic presentations.

The duration and severity of signs prior to presentation will vary with the owner's perception and concern, the dog's disposition, the degree of involvement of vital structures, and occurrence of spontaneous stabilization of the lesion. The severity of the lesion does not always correspond to the clinical signs. The diagnosis of diskospondylitis is based on the...
clinical signs and radiographic confirmation of a compatible lesion. Further documentation may be supplied by direct culture of the bony lesion, blood culture, and urine culture. Blood chemistries and hematologic values are usually normal, although a nonspecific leukocytosis may be present. In 50% of the cases at one institution, pyuria or bacteriuria was present; however, cerebrospinal fluid analysis was usually normal.(36)

**Radiology**

Radiographic evaluation is essential to obtaining the diagnosis. The radiographic changes are a reflection of the underlying pathophysiologic processes. The vascular channels in the vertebral end-plates permit passage of bacteria into the subchondral area with secondary penetration of the intervening disk and contiguous vertebral end-plate. The end result is the classic appearance of bony destruction on both sides of the disk with narrowing of the disk space.(18) Varying degrees of vertebral lysis, sclerosis, and spondylolisthesis are seen, but the radiographic changes may not occur until 4 to 6 weeks after the infection is established (36) (Fig. 60-2). Serial radiographic examinations are justified if a high index of suspicion exists. As the lesion progresses, gradual widening of the intervertebral space is seen along with sclerosis and new-bone formation. Finally, there is noticeable narrowing to the point of a collapsed disk space, and occasionally spontaneous fusion occurs.(44) Because of the unpredictability of radiographic signs and the time lag before appearance, considerable attention has been paid to technetium and gallium bone scintiscans.(24,25,43) In one study the use of scintiscans allowed the time required for diagnosis to be reduced from an average of 11 days to 2.7 days.(64) This level of diagnostics is available at some veterinary institutions.

![FIG. 60-2 Lateral radiographic view of a dog with a diskospondylitis lesion atT13-L1. Note the narrowed disk space, sclerotic margins, and central bridging.](image)

**Culture Techniques**

Because of the number of bacteria. actinomycetales, mycobacteria, and fungi capable of causing diskospondylitis, an exact diagnosis of cause is very important.(47,48) Of 56 dogs with diskospondylitis from whom blood was cultured, approximately 75% showed positive results. S. aureus is the most common isolate; B. Canis and Streptococcus spp are occasional isolates. (16)

Good culture results have been obtained using Craig and Ackermann needles for aspiration or biopsy under fluoroscopic control in humans, and this technique should be pursued by veterinarians.(1,18,47,48,56) Histologic study, spinal stains, aerobic and anaerobic cultures, and fungal and mycobacterial cultures are recommended.(56)

Bone and blood isolates have similar or identical sensitivities, which provides reasonable assurance that the organism from the blood culture is the causative agent. Because of ease and reliability, blood cultures should always be taken, preferably prior to antibiotic administration. Although urine culture results are less dependable, with only 25% positive, the procedure is easy and reinforces the diagnosis. A positive culture for an organism other than S. aureus is not considered diagnostic unless the same organism is also isolated from either blood or bone.(37) Urine culture results reported in humans are considered of limited value only, (18) but urinary sepsis has been implicated as providing the majority of examples of vertebral osteomyelitis secondary to infection within the body cavity.(23) One author states "the only definitive laboratory finding of discitis is a positive culture of a biopsy specimen taken from the disc space".(43) However, direct disk cultures often yield negative results characterized by nonspecific inflammatory tissue due to the chronic insidious changes occurring.(43) Although direct biopsy may not contribute to identifying the causative agent, histopathology will be positive for establishing a diagnosis of acute or chronic osteomyelitis.(18)

As a routine precautionary and diagnostic measure, the tube agglutination test for B. canis should be done in conjunction with blood culture.(26,30,35,36) Results of tube agglutination test are positive in about 10% of cases, and B. Canis will be isolated from most blood cultures in seropositive dogs. Titers from 1:250 to 1:500 are indicative of bacteremia; titers between 1:50 and 1:100 are indicative of previous exposure that may progress to bacteremia; and a titer of 1:25 may occur from cross agglutination with another organism.(36) B. Canis has also been isolated from bone biopsies of dogs with diskospondylitis. (26)
Treatment

As early as 1960 the optimum treatment was considered an accurate bacteriologic diagnosis followed by appropriate antibiotic therapy, confinement, and immobilization. Excellent results occurred in 7 of 9 dogs with cervical disease and in all 21 dogs with thoracolumbar involvement when treated aggressively by a combination of spinal cord decompression, spinal column immobilization (thoracolumbar only), and systemic antibiotic therapy. Kornegay has recently suggested a therapeutic regimen based on the degree of neurologic dysfunction; the results or B. Canis titer and blood culture; the multiplicity of lesions; and the surgical accessibility of the lesion.

Antibiotic therapy plus confinement is reserved for dogs with minimal or no neurologic dysfunction. Choice of antibiotic is based on serology and blood culture sensitivities. If test results are negative, the dog is treated empirically for S. aureus (Table 60-1). Lack of clinical improvement within 5 days justifies reassessment. Dogs with solitary, readily accessible lesions are curetted and cultured. A different antibiotic is tried for dogs with multiple lesions or lesions less accessible surgically. Similar regimens are advocated in human patients who have primarily back pain. Bed rest and even body casts are used to enforce immobilization.

Surgical intervention is advised for dogs with moderate to marked neurologic dysfunction to relieve spinal cord compression. The disk is thoroughly debrided. Decompression is afforded by dorsal laminectomy or hemilaminectomy if dorsal spinal plating is contemplated. Spinal stability is subjectively assessed after debridement and curettage to determine the necessity of vertebral stabilization.

| Table 60-1 Results of Antibiotic Sensitivities of Bacteria Isolated from Blood or Bone of Dogs with Diskospondylosis Evaluated at the Georgia Veterinary Medical Teaching Hospital. |

In both dogs and humans, antibiotic therapy for at least 4 to 6 weeks is strongly encouraged for medical as well as surgical cases. Recently, the role of antibiotics in chronic vertebral osteomyelitis in adults, postoperative disk-space infection, and disk infections in young children has been challenged. The treatment of choice in these studies was immobilization in a double hip spica for 6 weeks, which is not a rational choice for the veterinary patient. A viral etiology has been proposed for disk infection in young children because of the low morbidity, control by host defense mechanisms, and minimal tissue damage. This has not been documented, however, and veterinarians have not reported a similar manifestation in young dogs.

Initially, dogs should be treated parenterally to halt acutely progressive clinical signs and then with oral antibiotic therapy for at least 4 to 6 weeks. Bactericidal antibiotics, especially the cephalosporins and semisynthetic penicillins, are the drugs of choice for S. aureus, whereas penicillin and ampicillin are usually ineffective. In human studies, failure rates increased when tetracycline, erythromycin, and chloramphenicol were widely used prior to the advent of penicillinase-resistant penicillins. An appropriate dosage administered conscientiously is essential for adequate treatment over the prolonged periods of therapy. A recent study emphasized that S. aureus may become tolerant and merely be suppressed or inhibited by bactericidal antibiotics. Symptoms disappear for short periods of time but surface again. Cure was achieved by combining an aminoglycoside with the beta-lactam antibiotics.

Trimethoprim combined with rifampin, both given orally, has been shown to be effective in treatment of experimental staphylococcal osteomyelitis. Trimethoprim was also effective in a clinical case of cervical diskospondylitis treated conservatively in a dog.

Dicloxacillin attains serum levels four times those of oxacillin and twice those of cloxacillin when given in equivalent doses orally. In recent studies in a rabbit model and in children with acute staphylococcal osteomyelitis, dicloxacillin penetrated purulent marrow and joint cavities in adequate concentrations. With the advantage of oral administration, dicloxacillin would be a reasonable alternative to the cephalosporins if they are ineffective. Carefully monitored parenteral-oral therapy has been effective in treating acute staphylococcal osteomyelitis.

The penetrability of bone by antibiotics in measurable therapeutic levels has received considerable attention. Although penetrance of bone by lincomycin is 8 to 10 times that of penicillin, it could theoretically require 53 times more...
lincomycin than penicillin to produce the same bactericidal effect. This emphasizes the fact that the efficiency of an antibiotic depends on the sensitivity of the organism to it as well as on the amount of drug penetrating the area and its duration of effect. (17) An interesting study comparing lincomycin and cephalothin in normal and osteomyelitis bone of rabbits established that untreated rabbits had uniformly severe disease with positive bone cultures. Treatment with either antibiotic decreased the severity of the osteomyelitis, but lincomycin-treated rabbits had significantly less severe bone disease than rabbits receiving cephalothin. Finally, 2 weeks of lincomycin therapy was equivalent to 4 weeks of cephalothin in sterilizing bone. (50, 51) Cephalosporins are still the drug of choice in most clinical reports. The penicillin-resistant penicillins are a close second. (see Table 60-1.)

Clindamycin phosphate and methicillin have been shown to exceed the minimal inhibitory concentration (MIC) for common gram-positive cocci. (57) Clindamycin concentration levels in bone exceeded the MIC by a larger factor than those of methicillin, but both antibiotics produced levels in bone/serum concentrations in excess of the MIC when evaluated in human patients during total hip replacement. Clindamycin is the 7-chloro substituted analogue of lincomycin. It is four times as active in vitro as lincomycin against staphylococci and penetrates well into bone and synovial fluid. (21) Good to excellent results have been obtained in clinical and experimental cases of osteomyelitis in dogs. * Clindamycin is effective given orally and should be considered along with dicloxacillin as a suitable antibiotic for treatment of vertebral osteomyelitis. Antibiotics proven effective against S. aureus in dogs are the cephalosporins (cephradine) and the penicillinase-resistant penicillins (cloxacillin) (Table 60-2). Unfortunately, chloramphenicol and trimethoprim are not as effective, although less expensive, and penicillin and ampicillin are usually ineffective. (36)


B. canis infections are treated with tetracycline and streptomycin as outlined in Table 60-2, and intact dogs are neutered. (26, 36, 42) The reader is referred to article references in the text for specific treatment of mycotic (58) and bacterial infections other than S. aureus, B. Canis, and streptococcus.

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Dosages Recommended for Treatment of Diskospondylosis in Dogs</th>
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<tbody>
<tr>
<td>Cephalosporins</td>
<td>Cephradine: 25 mg/kg/12 h for IV and/or 50 mg/kg q24h p.o.</td>
</tr>
<tr>
<td>Penicillinase-resistant penicillins</td>
<td>Cloxacillin: 25 mg/kg/12 h for IV and/or 50 mg/kg q24h p.o.</td>
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<tr>
<td>Chloramphenicol</td>
<td>25 mg/kg/12 h for IV and/or 50 mg/kg q24h p.o.</td>
</tr>
<tr>
<td>Trimethoprim</td>
<td>5 mg/kg/12 h for IV and/or 10 mg/kg q12h p.o.</td>
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The prognosis varies with the causative agent and the degree of neurologic involvement. Lesions caused by S. aureus often progress rapidly and necessitate extensive treatment. The prognosis becomes less favorable with advancing neurologic dysfunction, even with medical and surgical intervention. Dogs with minimal neurologic involvement have a good prognosis with selective antibiotic therapy alone or with concurrent vertebral curettage. Owners should be warned of occasional poor response in these cases. Recurrence is infrequent in dogs with S. aureus. The need for repeated therapy is to be expected, however, when treating dogs with a B. Canis infection. The potential for the owners to become infected with B. Canis must be explained, although cross infection is rare usually mild and responsive to tetracycline therapy. Fortunately, Brucella lesions seldom need decompression and progress more slowly than the ones caused by S. aureus. (36)

In all cases, a successful outcome is most likely with early diagnosis and appropriate antibiotic therapy for prolonged periods of time in conjunction with strict confinement. Surgery is indicated in select cases for diagnostic and therapeutic effect, but the prognosis is heavily influenced by the degree of neurologic dysfunction with or without surgery. A successful endpoint is the elimination of infection, relief of pain, and return of function followed by spontaneous osseous fusion or stable fibrous bridging. (32, 33)

References

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