

Discospondylitis associated with three unreported bacteria in the dog

Three cases of discospondylitis in the dog, caused respectively by *Pseudomonas aeruginosa*, *Enterococcus faecalis* and *Staphylococcus epidermidis*, are reported. These bacteria have not previously been documented as being the cause of the condition in this species.

The neurological involvement was severe in all three cases. Two dogs were paraplegic and one was tetraplegic, and one of the paraplegic dogs had no deep pain on the extremities of both pelvic limbs. All dogs were treated surgically with a satisfactory neurological recovery.

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INTRODUCTION

Discospondylitis is an infection/inflammation of the intervertebral disc which extends to the adjacent end-plates and vertebral body (Kornegay and Barber 1980). The resulting erosion at the intervertebral disc and at the end-plates may cause vertebral body instability, intervertebral space collapse and pathological vertebral fractures. The secondary granulation tissue and bone proliferation within the vertebral canal may result in extradural spinal cord or nerve root compression.

Untreated discospondylitis may lead to diffuse or focal meningitis and myelitis (Braund and others 1987, LeCouteur and Child 1989, Kornegay 1993). Any vertebral space can be affected, although caudocervical, thoracolumbar and lumbosacral spaces are more often involved (Gage 1975, Kornegay 1983, Gilmore 1987). Bacterial infection is the most common cause of discospondylitis in the dog. Fungal infection is also reported, but is less common (Moore 1992).

This report documents three clinical cases of discospondylitis in the dog caused by one of three different bacterial species not previously associated with the condition.

CASE HISTORY

Case 1

A seven-month-old male rottweiler was referred for paraparesis progressing to para-

plegia. The history revealed that the dog had had gastroenteritis two months previously, from which it had completely recovered. Two weeks later the dog had been presented to the local veterinarian for ataxia progressing to paraparesis. Non-steroidal anti-inflammatory drugs (NSAIDs) and antibiotic therapy were instituted for two weeks with little improvement observed. The dog's neurological status slowly worsened and one day before the referral the dog was paraplegic.

On physical examination at referral, the dog was alert, and temperature, pulse and respiration were normal. The neurological examination showed paraplegia, spinal hyperpathia in the lumbar region, increased spinal reflexes in both pelvic limbs, hyper-tonia in the right pelvic leg and absent deep pain in both pelvic limb extremities. The neuroanatomical localisation was between the third thoracic (T3) and third lumbar (L3) spinal cord segments. The initial differential diagnosis included vertebral or spinal cord developmental abnormalities, infection/inflammation and neoplasia.

Blood cell count, serum biochemical analysis and urinalysis were all within normal limits. The plain radiographic study showed lysis of the L2-L3 end-plates with narrowing of the L2-L3 intervertebral space. Myelography showed a ventral extradural spinal cord compression at the L2-L3 intervertebral space and an additional dorsal extradural compression on the right side at the L1-L2 intervertebral space (Fig 1A,B).

A right L2-L3 dorsolateral hemilaminectomy was performed and a large amount of pus surrounded by a membrane was removed from the vertebral canal. Curettage of the intervertebral space was performed. The bacteriological study isolated *Pseudomonas aeruginosa* sensitive to ceftazidime, piperacillin and ciprofloxacin.

The dog was treated with ciprofloxacin (15 mg/kg twice daily) for eight weeks, B-complex vitamins and carprofen (2 mg/kg twice daily for the first week). Two days after the surgery the dog regained deep pain sensation and during the following



FIG 1. (A) Lateral view. Narrowing of the L2-L3 intervertebral space with lysis of the L2-L3 end-plates is evident. Myelography shows a ventral extradural spinal cord compression at the level of the L2-L3 intervertebral space and an additional dorsal extradural spinal cord compression from the L1-L2 vertebrae. (B) Ventrodorsal view. This shows the right-side extradural compression at the level of the L2-L3 intervertebral space



FIG 2. Follow-up at eight months. A complete L2-L3 vertebral bone fusion is evident. In addition, a ventral bone bridge can also be seen between the L1 and L2-L3 vertebral bodies

few days its neurological condition progressively improved.

After one month, the dog remained paraparetic. Radiography showed no further progression of the vertebral lysis.

After two months, the dog was able to walk, with a slight ataxia and mild proprioceptive deficits in the right leg. Radiography showed the beginning of L2-L3 vertebral fusion and the antibiotic therapy was discontinued. The follow-up at eight months revealed only a slight ataxia and a complete L2-L3 vertebral fusion (Fig 2).

Case 2

A 19-month-old female rottweiler was referred for paraparesis progressing to para-

plegia. The owner reported that the dog had started decreasing its physical activity two months previously. One month before referral, a gastrointestinal barium study was performed because of acute abdominal pain and this was normal. However, an L2-L3 intervertebral space narrowing was not noticed. Two days before the referral, the dog was ataxic and progressively became paraplegic.

On physical examination at referral, the dog was alert, its temperature was increased (39.8 °C), the pulse and respiration were normal, and abdominal pain and diarrhoea were present. The neurological examination showed paraplegia with increased spinal reflexes in both pelvic

limbs. Deep pain perception was maintained in the pelvic limb extremities. The neuroanatomical localisation was between the T3-L3 spinal cord segments. The initial differential diagnosis was the same as for case 1.

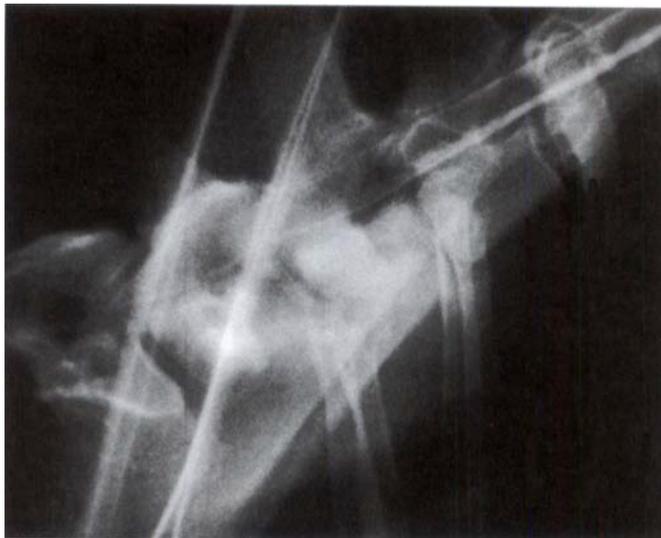
The blood cell count and the serum biochemical analysis results were consistent with previous corticosteroid administration, and urinalysis was normal. The plain radiographic examination showed lysis of the L2-L3 end-plates and narrowing of the L2-L3 intervertebral disc space. Myelography showed a slight extradural deviation of the contrast medium above the L2-L3 intervertebral space and a dorsal extradural compression of the spinal cord at the level of the L3-L4 intervertebral space (Fig 3A,B).



FIG 3. (A) Lateral view. Myelography shows a ventral extradural compression at the level of the L2-L3 intervertebral space and a dorsal extradural compression from the L3-L4 vertebrae. (B) Ventrodorsal view. This shows right-side extradural compression at the level of the L2-L3 intervertebral space



FIG 4. Narrowing of the C7-T1 intervertebral space, ventrocranial sclerosis of the T1 vertebral body and a slight dorsal deviation of the contrast medium at the level of the C7-T1 intervertebral space are evident



A dorsolateral right hemilaminectomy was performed and a large amount of purulent material was removed from the vertebral canal. The intervertebral space of L2-L3 was curettaged. Bacterial culture isolated *Enterococcus faecalis* sensitive to ampicillin, amoxicillin/clavulanate, piperacillin and ceftriaxone. The dog was treated with amoxicillin/clavulanate (25 mg/kg twice daily) for eight weeks, B-complex vitamins and carprofen (2 mg/kg twice daily for the first week).

After one month, the dog was ambulatory with a slight ataxia. Radiography showed no further progression of the vertebral lysis. After two months, the dog was still ataxic but without detectable proprioceptive deficits. Radiography showed the beginning of vertebral L2-L3 fusion, and antibiotic therapy was discontinued. On the follow-up at four months, the dog had completely regained its neurological functions.

Case 3

A 15-month-old female German shepherd dog was referred for tetraplegia. The history revealed pain and stiffness in the thoracic limbs three months before the referral. The dog was treated by the local veterinarian with NSAIDs, which had resulted in some improvement. One month later, the dog became suddenly tetraparetic and three days before the referral it was tetraplegic and anorectic.

On physical examination at referral, the dog was alert, and temperature, pulse and respiration were normal. Dehydration of approximately 5 per cent and a skin sore at the thoracic inlet were noted. The neurological examination showed tetraplegia,

severe cervical pain, and spinal reflexes increased in both pelvic limbs and reduced in both thoracic limbs, with deep pain perception maintained in all limb extremities. The neuroanatomical localisation was between the sixth cervical (C6)-T2 spinal cord segments. The initial differential diagnosis was the same as for cases 1 and 2, but also included caudal cervical spondylomyelopathy.

The blood cell count revealed mature neutrophilia, while serum biochemical analysis and urinalysis were normal. The plain radiographic examination showed lysis of the C7-T1 end-plates and narrowing of the C7-T1 intervertebral space. Myelography showed a ventral extradural spinal cord compression at C7-T1 (Fig 4). A fistula from the skin sore to the C7-T1 intervertebral space was noted. A ventral slot with deep curettage of the C7-T1 intervertebral space was performed and a grass awn was found within the T1 vertebral body. The bacterial culture isolated *Staphylococcus epidermidis* sensitive to amoxicillin/clavulanate, ceftriaxone, cephalexin and cefamandole. The dog was treated with cephalexin (25 mg/kg twice daily) for eight weeks, B-complex vitamins and carprofen (2 mg/kg twice daily for the first week).

After one month, the dog was able to walk with a moderate ataxia, and radiography showed no further progression of the vertebral lysis. After two months, the ataxia was still present. Radiography showed the beginning of vertebral C7-T1 bone fusion, and antibiotic therapy was discontinued. On the follow-up at five months, only mild ataxia was present.

DISCUSSION

The most common bacterium identified in discospondylitis in the dog is *Staphylococcus intermedius*. Other bacteria frequently isolated are *Staphylococcus aureus*, *Streptococcus* species, *Escherichia coli*, and *Brucella canis*. Less frequently isolated bacteria are *Pasteurella* species, *Proteus* species, *Corynebacterium* species, *Mycobacterium* species, *Nocardia* species, *Actinomyces* species and *Bacteroides* species (Braund and others 1987, LeCouteur and Child 1989, Kornegay and Anson 1990, Kornegay 1993). In rare cases, *Aspergillus* species, *Mucor* species, *Fusarium* species and *Paecilomyces variotii* have been isolated (Hurov and others 1978, Johnson and Prata 1982).

In the pathogenesis of discospondylitis in the present cases, the vertebrae probably become infected secondary to haematogenous dissemination of bacteria from foci elsewhere in the body or by direct inoculation from tracking foreign bodies. The most commonly incriminated sources of infection in such cases are the urogenital tract, skin infections, dental diseases and endocarditis. In many cases, the source of the infection is not detected.

The pathophysiology of discospondylitis is not completely clear. One theory is that the presence of subchondral vascular loops in the vertebral epiphysis slows circulation, allowing colonisation of blood-borne bacteria, which then diffuse through the cartilaginous end-plate of the vertebral body to reach the disc (Trueta 1959). Infection is further disseminated to adjacent vertebrae through freely communicating venous sinuses (Kornegay and Anson 1990). Predisposing factors have included immune suppression (Barta and others 1985, Kornegay 1985), previous trauma (Gage 1975), surgery (Kornegay 1993), osteomyelitis in other sites, such as the femur and sternum (LeCouteur and Child 1989), wounds of paravertebral structures (Moore 1992) and biomechanical stress (Kornegay 1993). Foreign bodies, including grass awns, have also

been associated with discospondylitis (Case 1983).

Some grass awns, such as wheat, barley and cheat grass, have barbed ends which, once lodged in the tissues, allow them to advance forward but not backward. The exact portal for entry for the grass in 'grass awn-associated discospondylitis' is not known. Several theories have been proposed to explain how the grass awns enter the body, their migration and how they reach the vertebral body. The grass may migrate to paravertebral structures after penetrating skin over the paravertebral structures or abdomen. Swallowing the grass awn and penetration through the bowel wall has been suggested as another portal of entry. In such cases, the awn would have to migrate along the mesentery, to the attachment of the mesenteric root on the paravertebral structures and ultimately localise to the vertebral column. Inhalation of the grass awn, and migration through the lung or mediastinum, then along the crura of the diaphragm, has also been suggested. The muscular insertion of the crura of the diaphragm is the ventral lumbar body of L2-L4. This could explain why grass awn-associated discospondylitis occurs most frequently in the L2-L4 vertebral bodies (LeCouteur and Child 1989).

Detailed reviews of the epidemiology, diagnosis, treatment and prognosis for discospondylitis have been well described previously (Kornegay 1979, 1983, 1993, Kornegay and Barber 1980, LeCouteur and Child 1989, Moore 1992, Budsberg 1998, LeCouteur and Grandy 1999).

Delayed diagnosis of discospondylitis can be detrimental to the prognosis, especially where severe neurological abnormalities are present. The dogs in the present study were referred to the authors' clinic a long time after the appearance of the clinical and radiographic signs of discospondylitis. The consequence of the initial misdiagnosis was a worsening of the vertebral lesion and of the neurological status. In cases 1 and 2, if the radiographic signs of discospondylitis had been recognised

then medical therapy could have been undertaken and surgery could perhaps have been avoided.

Conclusions

Surgery on the three cases in this report allowed a complete removal of the spinal cord compression and made the bacteriological study of the infected material possible. The combined surgical and long-term antibiotic therapy achieved a satisfactory neurological recovery despite the initial guarded prognosis.

To the authors' knowledge, there have been no previous reported cases of discospondylitis in association with *Pseudomonas aeruginosa*, *Enterococcus faecalis* or *Staphylococcus epidermidis*.

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ABSTRACTS

Pericardial effusion and pericardiocentesis in a guinea pig (*Cavia porcellus*)

A three-year-old female guinea pig was presented with tachypnoea, dyspnoea and pale mucous membranes. Auscultation revealed tachycardia and markedly increased lung sounds. Femoral pulses were weak. Bilateral pleural effusion was demonstrated on radiography and echocardiography confirmed pericardial effusion and heart failure, secondary to cardiac tamponade. Pericardiocentesis was undertaken, under ultrasonographic guidance. Clinical signs quickly resolved. The animal was monitored using echocardiography and radiographs over the next six months and cardiac function remained normal. This is the first recorded case of pericardial effusion in a guinea pig. Its cause is unknown.

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