Discospondylitis is most commonly seen in young to middle-aged medium- to large-breed dogs. Predisposed breeds include Great Danes, boxers, Rottweilers, English bull dogs, German shepherd dogs, Doberman pinschers, and Rhodesian ridgebacks. Courtesy of shutterstock.com/Best Dog Photo
Discospondylitis
An Overview

**INFECTION CONTROL**

Discospondylitis is an infection of the vertebral endplates and associated intervertebral disk. The infection typically starts in the vertebral end plate, then spreads to the adjacent intervertebral disc. The blood supply within the vertebral endplates consists of capillary beds with reduced blood flow velocity. Pores in the end plate that normally allow for distribution of nutrients also provide a route for organisms to enter the intervertebral disc. The minimal vascular supply of the intervertebral disc further enables infection within the disk.1

**PATHOPHYSIOLOGY**

Infectious organisms can gain access to the disc space in many ways. Hematogenous spread of bacteria or fungi from the urogenital tract, oral cavity, heart valves, and skin is believed to be the most common method. Foreign-body migration, such as a plant awn, may introduce bacteria as migration occurs throughout tissues. Less commonly, discospondylitis occurs as an iatrogenic infection following spinal surgery or paravertebral injections.2 In cats, abscesses from bite wounds can cause discospondylitis by direct inoculation of bacterial organisms.1

**MEET THE AUTHOR**

Heather Anderson, RVT, VTS (Neurology)
The Ohio State University Veterinary Medical CenterColumbus, Ohio

Heather Anderson works at The Ohio State University Veterinary Medical Center as a neurology/neurosurgery technician. She graduated with an associate’s degree in Veterinary Technology from Stautzenberger College in Ohio in 2003 and became a credentialed Veterinary Technician Specialist in 2016. She is a member of the Academy of Internal Medicine Veterinary Technicians and the National Association of Veterinary Technicians in America and enjoys teaching neurology to her 4th-year veterinary students.
The most common causes of discospondylitis are coagulase-positive *Staphylococcus pseudintermedius* or *Staphylococcus aureus*. Less commonly, *Streptococcus* species, *Escherichia coli*, and fungal organisms (such as *Aspergillus* species) can be causes.  

*Brucella canis* can also cause discospondylitis and is an important infectious agent to consider in all suspected cases because of its zoonotic potential. *B. canis* is transmitted by direct exposure to bodily fluids or transplacental transmission from mother to puppy. Precautions, such as routine hand washing, minimization of exposure to urine, and the wearing of gloves, should be taken until *Brucella* has been ruled out as a cause.

**SIGNALMENT/PRESENTATION**

Discospondylitis is most commonly seen in young to middle-aged medium- to large-breed dogs. Less commonly, it has been reported in small-breed dogs and cats. Predisposed breeds include Great Danes, boxers, Rottweilers, English bull dogs, German shepherd dogs, Doberman pinschers, and Rhodesian ridgebacks. Most dogs will display progressive clinical signs over several weeks, but vertebral pathologic fractures can cause acute deterioration. Clinical signs may be nonspecific and include systemic illness, such as depression, anorexia, pyrexia, and lethargy.

Paraspinal hyperesthesia is the most common finding on neurologic examination. Gait abnormalities, such as ataxia, paresis, or paralysis, may be present if secondary spinal cord or nerve root compression occurs. When neurologic signs are present, they correlate to the location of the discospondylitis lesion; cervical vertebral column lesions may cause tetraparesis and neck pain; thoracolumbar lesions may cause pelvic limb paresis, proprioceptive ataxia, and back pain; and lumbosacral lesions may cause a stiff, stilted pelvic limb gait. Discospondylitis can affect any area of the vertebral column, but the most commonly affected sites are L7 to S1, caudal cervical, mid-thoracic, and the thoracolumbar spine. Multifocal lesions occur in 30% to 40% of cases.

**DIAGNOSTIC IMAGING**

Definitive diagnosis of discospondylitis is based on characteristic findings on spinal radiography in a patient with supportive clinical features. Typical radiographic findings include collapse of the affected disc space, lysis of the adjacent vertebral endplates, and bony proliferation of adjacent vertebral bodies. Because multiple lesions are common, radiography of the entire spine should be performed to properly assess the extent and severity of disease. The patient should be sedated for spinal radiography to ensure proper positioning and an accurate assessment of the area of interest. It is important that the veterinary technician use caution when transporting and positioning a sedated patient with potential spinal lesions. Care should be taken to avoid patient manipulation and to prevent further injury. The main limitation of standard radiography is that radiographic changes may lag behind the onset of clinical signs in the first 2 to 4 weeks of infection. Patients with suspected discospondylitis and normal radiographs may still have the disease; radiography should be repeated every 1 to 2 weeks or more advanced imaging should be considered.

Magnetic resonance imaging (MRI) and computed tomography (CT) have an increased sensitivity for...
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the diagnosis of discospondylitis and can be useful in revealing early lesions. MRI is superior for evaluating soft tissue structures, and its advantages over standard radiography include its visualization of the spinal cord and nerve roots, its high contrast resolution, and the ability to use contrast agents. The use of MRI in a patient with discospondylitis may reveal T2-weighted increased signal intensity and T1-weighted decreased signal intensity in the intervertebral disc, vertebral end plates, and vertebral bodies (Figures 3 and 4). Contrast enhancement is often observed in the endplates of the affected vertebrae, and inflammation in the surrounding muscles may also be highlighted. Compressive lesions that can potentially be surgically corrected, such as disc protrusion/extrusion, vertebral subluxations, fractures, and concurrent epidural abscess, can be easily identified.

**HEMATOLOGIC EVALUATION AND MICROBIOLOGY**

Veterinary technicians play a vital role in the collection of laboratory tests and interpretation of the results. Blood values are often unremarkable, but leukocytosis characterized by neutrophilia and monocytosis can occasionally be seen on complete blood count. Leukocytosis is more common in dogs with associated endocarditis. Mild hypoalbuminemia and mild to moderate hyperglobulinemia are the most common abnormalities noted on the serum biochemistry panel.

Ideally, treatment for discospondylitis is guided by culture and antibiotic sensitivity. A urine sample should be collected aseptically via cystocentesis and urinalysis, and culture/susceptibility may reveal a urinary tract infection as a possible source of infection. Blood cultures should be collected by using meticulous sterile technique (Box 1). Blood cultures in dogs yield positive results in about 45% to 75% of cases, and urine cultures are positive in about 25% to 50% of cases. Serologic testing for *Brucella* antibody and *Aspergillus* antigen may be performed.

When urine, blood cultures, and serology have not identified a cause, CT or fluoroscopy-guided fine-needle aspiration of the infected disc space may be performed under general anesthesia, or a surgical biopsy specimen from the lesion can be obtained.

*Brucella canis* can also cause discospondylitis and is an important infectious agent to consider in all suspected cases because of its zoonotic potential.
Henry was a 1.5-year-old male castrated great Pyrenees. He was presented to the Ohio State University Veterinary Medical Center for a progressive history of pain and difficulty rising. The owners reported that for the past 3 months, Henry would cry out when touched near his hind end and was no longer willing to climb stairs. Henry was taken to his general practitioner, who obtained radiographs of his pelvis and lumbar spine. The radiographs were found to be unremarkable. Henry was prescribed tramadol, 2.4 mg/kg (100 mg) PO q8-12h as needed for pain; deracoxib, 0.89 mg/kg (37.5 mg) PO q12h for pain; and gabapentin, 2.4 mg/kg (100 mg) PO q8-12h for pain. His owners noticed a slight improvement, but he remained painful most days.

Upon arrival, Henry weighed 42 kg. Vital signs were temperature of 100.2°F, pulse of 120 beats/min, and respirations of 20 breaths/min. No murmurs or arrhythmias were auscultated; pulse quality was good and without pulse deficits. Thoracic auscultation revealed normal lung sounds. Mental status was appropriate. On gait analysis, Henry displayed difficulty rising from a lying position and had ambulatory paraparesis with no ataxia. His postural reactions were normal in the thoracic limbs and minimally decreased in the pelvic limbs. Henry exhibited pain upon spinal palpation of the lumbar and lumbosacral regions and pain with dorsal pressure on rectal exam. All other aspects of the neurologic and general physical exam were normal.

Initial diagnostic testing included a complete blood count, serum biochemical profile, and spinal radiography. The complete blood count and serum biochemical profile were within normal limits. Radiography of the thoracic and lumbar spine revealed vertebral body endplates that were irregular, with permeative lysis centered at the disc space of T10 to T11, T13 to L1, L1 to L2, and the lumbosacral junction. Enlarged sublumbar lymph nodes were also noted. On the basis of these findings, discospondylitis was the primary differential diagnosis; MRI was recommended and scheduled for the following day. This was to determine the extent of the damage and to help direct further treatment of Henry’s condition.

The following day, Henry was placed under general anesthesia and was positioned in dorsal recumbency in the MRI machine. T2-weighted, T1-weighted, short tau inversion recovery, and gradient echo images were obtained in 3 planes (sagittal, transverse, and dorsal). After administration of the contrast agent gadodiamide (0.2 mL/kg) 8.4 mL IV, additional T1-weighted images were acquired in the same 3 planes. Henry’s MRI revealed multiple vertebral endplates that were irregularly shaped and marginated with T2-weighted and T1-weighted hypointensity relative to the medullary cavity. The irregularities were centered at the disc space of T10 to T11, T13 to L1, L1 to L2, and the lumbosacral junction. Secondary compression of the lumbosacral spinal segments and associated nerve roots were also noted. On the basis of Henry’s signalment, history, and radiographic findings, his MRI results were most consistent with multifocal discospondylitis and right-sided compression of the cauda equina secondary to extruded disc material, a cyst, or an abscess.

After the MRI, cystocentesis for urine culture was attempted but a sample could not be collected. Blood was drawn for serologic testing for Brucella antibody. Three individual blood samples were also drawn aseptically from the jugular and both the right and left lateral saphenous veins at 30-minute intervals for blood cultures. Because animals may be intermittently or continuously bacteremic, obtaining multiple, separate samples is recommended to increase the likelihood of identifying the pathogen.7 While the blood results were awaited, Henry was continued on the tramadol, deracoxib, and gabapentin for pain relief, and cephalixin 24 mg/kg (500 mg) PO q12h was initiated. The blood samples sent to Cornell University showed a positive result for agglutination and negative result for agar gel immunodiffusion II. These results were inconclusive, and it was recommended that the test be repeated in 4 to 6 weeks. Within several days, all 3 blood cultures grew Brucella species, confirming that Henry was Brucella positive with secondary discospondylitis.

At this point, Henry was now back in the care of his general practitioner. Brucella infection is a reportable zoonotic disease, and the following treatment protocol was made by the State Public Health Veterinarian. The cephalixin was to be discontinued, and Henry was to be started on doxycycline, 12.5 mg/kg (525 mg) PO q12h, and amikacin sulfate, 10 mg/kg (420 mg) SC q24h. The doxycycline was to be continued until 2 negative test results were obtained, while the amikacin was to be given for 1 week, followed by a rest period for 2 weeks, then repeated again on week 4. Because of the renal toxicity effects of aminoglycosides, it was recommended that Henry be monitored closely through biochemical profiles and urinalysis to confirm the use and continuation of amikacin; testing was to be done before week 1 and week 4 of drug therapy to confirm a negative result, based on the provided protocol. If a positive result remained, then the aminoglycoside protocol was to be repeated, along with continuation of doxycycline.

Because of Brucella’s zoonotic potential, hospital employees who were exposed to Henry were notified. The university’s employee health service was notified, and each employee was briefed about the disease, symptoms, and Centers for Disease Control and Prevention protocol. Thankfully, no employees contracted the disease.

Unfortunately, Henry was lost to follow-up in the months after his Brucella diagnosis, and his outcome is unknown. Henry’s case provides an excellent example of why it is important to consider B canis as an infectious agent in all suspected discospondylitis cases. It also emphasizes the importance of always using precautions, such as routine hand washing, minimization of exposure to urine, and wearing gloves while handling the patient, until Brucella has been ruled out as a cause.
for culture. Unfortunately, the originating site of infection cannot always be identified. In these cases, empirical therapy is chosen according to the most commonly isolated infectious organisms.

**TREATMENT**

The treatment of discospondylitis consists of antibiotics, pain medications, and cage rest. Long-term antimicrobial drug therapy must be instituted, ideally for a minimum of 6 to 8 weeks. In some cases, treatment may need to be extended by many months. While culture results are awaited, treatment for the most common pathogen, *Staphylococcus* species, should be initiated. Common antibiotics include cephalixin or amoxicillin (**TABLE 1**). In patients with severe neurologic compromise or signs of sepsis, IV antibiotics, such as cefazolin, should be considered for the first 5 to 7 days. If fungal disease is confirmed, discospondylitis should be treated with antifungal drugs, such as fluconazole or itraconazole. Dogs with fungal discospondylitis may require lifelong treatment with antifungal drugs. In cases of *Brucella*-positive dogs, combination therapy consisting of doxycycline and an aminoglycoside is indicated. Corticosteroids are generally contraindicated because of possible impairment of the immune system.

Pain management is also an important part of the treatment of discospondylitis. Analgesics, such as nonsteroidal anti-inflammatory drugs, fentanyl patches, tramadol, gabapentin, or amantadine, may be indicated. Veterinary technicians play an important role in the assessment and management of pain. While in the hospital, patients should receive regular pain assessments. When the patient is being treated at home, the veterinary technician can keep in close contact with the owner to ensure patient comfort.

Physical rehabilitation can also be used to help relieve pain and maintain full range of joint motion, limit loss of muscle mass, and prevent contractures and deterioration of joints during recovery from neurologic injury. This is achieved through controlled therapy techniques, such as passive range of motion, massage, thermotherapy, and neuromuscular electrical stimulation.

Strict confinement is essential to avoid worsening discomfort in severely affected patients. Restrictions should include cage rest, restriction of exercise, and leash walking only for urination and defecation. Activity restriction may also prevent vertebral subluxations and herniation of

<table>
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<tr>
<th>INFECTIOUS AGENT*</th>
<th>ANTIBIOTIC</th>
<th>DOSING</th>
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<tbody>
<tr>
<td><em>Staphylococcus intermedius</em>&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Cephalexin</td>
<td>20-30 mg/kg PO q8h</td>
</tr>
<tr>
<td></td>
<td>Cefazolin</td>
<td>20 mg/kg IV q6h</td>
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<tr>
<td><em>Streptococcus species</em>&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Amoxicillin</td>
<td>20 mg/kg PO q12h</td>
</tr>
<tr>
<td><em>Escherichia coli</em>&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Enrofloxacin</td>
<td>5-11 mg/kg PO q24h</td>
</tr>
<tr>
<td><em>Brucella canis</em>&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Doxycycline</td>
<td>12-15 mg/kg PO q24h</td>
</tr>
<tr>
<td></td>
<td>Gentamicin sulfate</td>
<td>9-14 mg/kg IV, IM, SC q24h</td>
</tr>
<tr>
<td><em>Aspergillus species</em>&lt;sup&gt;4&lt;/sup&gt;</td>
<td>Fluconazole</td>
<td>2.5-5 mg/kg PO q24h</td>
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<tr>
<td></td>
<td>Itraconazole</td>
<td>5 mg/kg PO q24h</td>
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*Superscript numbers are reference citations.
disc material. Although surgical decompression is rarely needed, destruction of the vertebrae can cause vertebral instability with secondary compression; in this case, decompressive surgery and stabilization may be indicated.2

Clinical improvement of signs associated with systemic illness and pain is expected in the first week of antibiotic therapy.3 It is important that patients return for follow-up neurologic exams and radiography every 1 to 2 months to monitor for disease progression and to help direct therapy. Progress is characterized by resolution of sclerosis of the bone, lysis of the bone, and vertebral fusion. Laboratory tests may also be repeated depending on the original source of the infection. Antibiotics should be continued until bone lysis resolves completely.5

**PROGNOSIS**

Prognosis for patients with bacterial discospondylitis, especially those with minor neurologic deficits, is generally favorable. The prognosis is more guarded in patients with *B canis* infections, resistant bacterial infections, and severe neurologic deficits. Fungal discospondylitis is associated with poor prognosis; chronic recurrence and progression are likely.1

**CONCLUSION**

Veterinary technicians play a key role in the diagnosis, treatment, and management of discospondylitis. With the initiation of proper therapy, most patients carry a favorable outcome and the case can be a successful and rewarding one. Until zoonotic diseases, such as *Brucella* infection, have been ruled out as a cause of discospondylitis, the entire veterinary staff should use protective measures, such as routine hand washing, wearing of exam gloves, and minimizing exposure to urine and other body fluids. **TVN**

**References**