



## DISCOSPONDYLITIS

Case Report  
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### Signalment:

“Rudy”  
7-year-old MN Rhodesian Ridgeback  
7/9 BCS

### History:

Rudy was initially presented to VMSG on September 16<sup>th</sup>, 2008 for evaluation of difficulty rising and laying down. He was intermittently crying out in pain and was observed to be shifting his weight when standing. Rudy had a history of loose stool and at presentation was being treated with famotidine, Baytril and metronidazole. He had no history of recent travel, tick exposure or vaccination.

Rudy was presented to his primary veterinarian on September 18<sup>th</sup>, 2008 as he continued to be reluctant to ambulate and he had recently developed diarrhea. Pain was localized to the mid-thoracic vertebral region, and spinal radiographs revealed a collapsed disc space at T8-T9. Medical therapy was initiated with Robaxin, Deramaxx and tramadol. The Deramaxx was discontinued on September 23<sup>rd</sup> when Rudy developed melena. Rudy was presented to VMSG again on September 27<sup>th</sup> for evaluation of melena. At that time he continued to be painful and hesitant to ambulate.

### Clinical Exam:

September 16<sup>th</sup>: Shifting leg lameness and hesitant to lie down. No joint effusion appreciated and no pain on palpation of any peripheral joints. Good range of motion in all joints. No pain on palpation of spine and no cervical pain. Neurologically appropriate.  
September 27<sup>th</sup>: Hyperthermic (105.3°F at presentation), mildly dehydrated and painful during rectal palpation and palpation of both the thoracic and lumbar spine. Rudy was slow to lie down and rise. No neurological abnormalities were appreciated.

### Laboratory Findings:

September 16<sup>th</sup>:  
Right carpal and tarsal joint taps were performed. No fluid was obtained from the tarsal joint and only minimal fluid was obtained from the carpus. Cytological findings were unremarkable.  
September 28<sup>th</sup>:  
Blood cultures were submitted and revealed the presence of a *Pseudomonas* species.  
A urine culture revealed the presence of *Pseudomonas*.  
A CBC, chemistry panel, UA and T4 revealed mildly elevated globulins and were otherwise unremarkable.  
A Spec cPL was not diagnostic for pancreatitis.

### Diagnostic Imaging:

Spinal radiographs (T3 – caudal) revealed thoracolumbar and lumbar articular facet osteoarthritis, lumbar and lumbosacral spondylosis and an indistinct intervertebral disc space at T8-T9.

An abdominal ultrasound was unremarkable; all structures were successfully visualized, and no abdominal pain was appreciated during the ultrasound.

A thoracolumbar spine MRI was performed at AVMI by Dr. Fundak/Dr. Gavin. Their findings included a regional STIR hyperintensity of the T8-T9 intervertebral disc space, contiguous endplates and regional paraspinal soft tissues. The endplates of the T8-T9 intervertebral disc space were mildly irregular with loss of distinct signal void endplate cortical margination; moreover, regional ventral to lateral mild STIR hyperintense modeling of the disc space was present. Following intravenous administration of the gadolinium based contrast media there was mild to moderate contrast enhancement of the T8-T9 intervertebral disc space, ventral hypaxial soft tissues, adjacent endplates and T8-T9 intervertebral modeling with additional moderate diffuse enhancement of the STIR hyperintense region within the left epaxial musculature over the L2 vertebra and the left caudal articular facets.

Interpretation: Findings are consistent with discospondylitis of the T8-T9 intervertebral disc space with concurrent endplate and peridiscal soft tissue infection to inflammation with additional focus of infection within left epaxial musculature over the L2 vertebra with probable extension into the left caudal L2-L3 articular facet joint resulting in infectious arthritis and moderate to marked

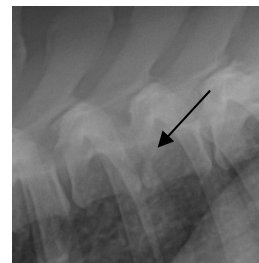


Figure 1 – indistinct disc space at T8-T9.

regional modeling of the L2-L3 articular facet joint. Multifocal mild variable intervertebral disc dehydration/degeneration are also noted.

**Diagnosis:**

Discospondylitis of the T8-T9 intervertebral disc space and infectious arthritis and modeling of the L2-L3 articular facet joint.

**Treatment/Management:**

On September 16<sup>th</sup>, Rudy was discharged for the owner to monitor at home. Medical therapy was initiated with tramadol in an attempt to control his pain. When Rudy presented for the second time on September 27<sup>th</sup> with melena and continued pain, he was hospitalized so that his dehydration could be corrected with intravenous fluids. His pain was treated with tramadol, a fentanyl patch, and hydromorphone as needed. Rudy's hyperthermia was monitored closely and resolved without therapy.

After receiving the blood and urine culture results, as well as the MRI findings from AVMI, therapy was initiated with amoxicillin (1000mg PO q8h) and Baytril (544mg PO q24h). Ciprofloxacin was later substituted for the Baytril to reduce treatment costs. Treatment was continued with both tramadol (100mg PO q8-12h) and a fentanyl patch to control the pain associated with discospondylitis.

**Discussion:**

Both cats and dogs have been found to suffer from discospondylitis, but it is seen most commonly in large and giant breed dogs. A 2005 retrospective study performed at the University of Louisiana found male dogs to be twice as likely as females to be affected with discospondylitis. Older, purebred dogs, most notably Great Danes, were also more likely to be affected.

Discospondylitis can affect any area within the vertebral column and is frequently caused by the hematogenous spread of both bacteria and fungi. In Rudy's case, the source of his infection was presumed to be a urinary tract infection as a *Pseudomonas* species was cultured in both his urine and blood. *Staphylococcus* spp, *Brucella* spp, *Streptococcus* spp, and *Escherichia coli* are the most commonly implicated infectious agents in cases of discospondylitis. *Brucella* is most commonly spread hematogenously from a genital infection. Other causes include dental disease and bacterial endocarditis. In 2004 Cherubini, et al. reported in the Journal of Small Animal Practice the finding of discospondylitis caused by *Bordetella* species. A definitive diagnosis was made with a combination of both MRI findings and bacterial culture results.

As we discovered in Rudy's case, clinical signs associated with discospondylitis are often non-specific and can include fever, anorexia, pain, reluctance to ambulate, and weight loss. These non-specific clinical signs can often make a definitive diagnosis difficult, but discospondylitis should always be considered in a patient with a fever of unknown origin. The WBC count in a patient with presumed discospondylitis may be normal or elevated. Spinal radiographs may demonstrate 1) bony endplate destruction, 2) new bone production and/or 3) collapsed disc space at the site of infection (Nelson, 1998). As stated previously, discospondylitis can occur at any site along the vertebral column, and it can also occur at more than one site which makes it imperative to radiograph and examine the entire spine. Radiographic changes associated with discospondylitis may lag behind clinical signs, so advanced imaging, like CT or MRI, are frequently useful in revealing early lesions.

Treatment for discospondylitis involves the long-term use of antibiotics and analgesics. The antibiotic choice should be based on the results of the blood and/or urine cultures, but if no organism is isolated, treatment can be initiated against the most commonly implicated infective organism, *Staphylococcus* spp. Both Clavamox and first-generation cephalosporins (cephalexin and cefazolin) have been found to be effective in treating *Staphylococcus* spp.-induced discospondylitis.

In 2005, Kinzel et al. described the fluoroscopy-guided percutaneous discectomy to treat discospondylitis in dogs. The infected vertebrae were fenestrated, and biopsy samples were collected. The causative agent was identified in 9 out of the 10 biopsy samples. With treatment they found that the clinical signs improved after 2-9 days, and they reported complete resolution in 5-14 days.

During chronic antibiotic treatment, radiographs of the spine should be repeated every 2-3 weeks initially to monitor therapeutic response. The course of treatment is frequently prolonged, and radiographic evidence of lesion resolution could take 6 months or longer.

**References:**

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