Diagnosis and treatment of lumbosacral vertebral instability caused by discospondylitis in a dog

De diagnose en behandeling van instabiliteit van de lumbosacrale wervelkolom ten gevolge van discospondylitis bij een hond

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ABSTRACT

A ten-month-old, female canine crossbreed of 40 kg was referred for episodes of severe pain non-responsive to analgesics. Neurological examination revealed pain on palpation of the lumbosacral region and non-weight bearing pelvic limb lameness, which was attributed to pain. Discospondylitis at L7-S1 was diagnosed based on radiographs and magnetic resonance imaging. Blood and urine culture were positive for *Staphylococcus* spp.. Despite ten days of conservative treatment, the dog did not show any improvement. Dynamic radiographs revealed a vertebral subluxation of L7-S1 in flexion. Surgery consisting of a dorsal laminectomy and stabilization of L7-S1 was performed. This resulted in a fast and complete recovery. Follow-up radiographs at six and twelve months after diagnosis showed severe osteolytic changes affecting L7. Despite these findings, the dog remained clinically normal.

SAMENVATTING

Een tien maanden oude, vrouwelijke hond van 40 kg werd doorverwezen omwille van erge pijn die niet verbeterde met pijnmedicatie. Neurologisch onderzoek toonde rugpijn aan in de lumbosacrale regio. De hond kon niet meer steunen op beide achterpoten door uitgesproken pijn. Met behulp van radiografieën en magnetic resonance imaging werd de diagnose van lumbosacrale discospondylitis gesteld. Het bloed- en urine-onderzoek was positief voor *Staphylococcus* spp.. De hond werd gedurende twee weken conservatief behandeld maar dit gaf geen beterschap. Dynamische radiografieën toonden een subluxatie in flexie van S1 ten opzichte van L7 naar ventraal. Er werd een dorsale laminectomie uitgevoerd gevolgd door stabilisatie van L7-S1. Dit resulteerde in een snel en volledig herstel. Radiografieën die genomen werden zes en twaalf maanden postoperatief toonden de aanwezigheid van uitgesproken osteolyse van L7 aan. Ondanks deze bevinding vertoonde de hond geen klinische symptomen.

INTRODUCTION

Discospondylitis is a primary infection of the cartilaginous vertebral endplates with secondary involvement of the intervertebral disc (Thomas, 2000; Burkert et al., 2005). Large, male, older and purebred dogs are predisposed (Thomas, 2000; Burkert et al., 2005). Less commonly, smaller dogs and cats are affected (Burkert et al., 2005; Packer et al., 2005). The most commonly affected site is the L7-S1 segment, followed by the thoracolumbar and cervical vertebral

column (Burkert et al., 2005). In 40% of dogs diagnosed with discospondylitis, multiple sites are affected (Burkert et al., 2005). *Staphylococcus* spp. is the most common recognized infectious agent (Kornegay, 1986; Gilmore, 1987; Burkert et al., 2005). Other common identified agents are *Streptococcus* spp., *Brucella* spp. and *Escherichia coli* (Kornegay, 1986; Gilmore, 1987; Burkert et al., 2005). Most dogs respond well to a long course of antibiotics, anti-inflammatory therapy and rest. In one study, the mean duration of treatment with antibiotics was 53.7



Figure 1. Laterolateral radiograph of the lumbosacral region in a neutral position. There is narrowing of the intervertebral disc space at the lumbosacral joint. The adjacent endplates of L7 and S1 are irregular.

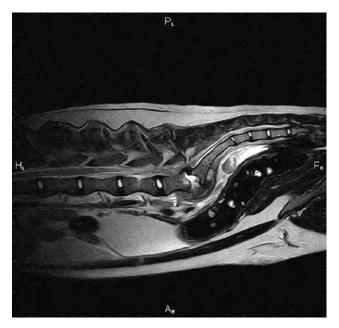


Figure 2. Sagittal T2-weighted magnetic resonance image of the lumbosacral region. Note the widening of the intervertebral disc space at L7-S1, with irregular appearance of the endplates and abnormal hyperintens signal and dorsal herniation of the intervertebral disc.

weeks (Burkert et al., 2005). Surgical treatment is indicated when no improvement is achieved within one or two weeks of medical treatment, when neurological deficits are present or when bone destruction causes vertebral instability (Kornegay, 1993). Possible surgical treatments are decompressive procedures (laminectomy or hemilaminectomy), stabilization of the vertebral column, curettage of the intervertebral disc and bone grafting (Auger et al., 2000; Burkert et al., 2005; Tellegen et al., 2015). In this case report, a dog diagnosed with lumbosacral discospondylitis, not responding to medical management is described. Lumbosacral vertebral instability was diagnosed with the aid of dynamic radiographs and was treated surgically. The clinical and radiographic findings during a one-year follow-up period are discussed.

CASE REPORT

A ten-month-old, female, intact, 40 kg crossbreed dog was referred to the neurology and neurosurgery service with a ten-day history of lethargy, lameness of the right pelvic limb, severe pain and fever. Despite treatment with NSAID's, gabapentin and tramadol during ten days, the dog continued to deteriorate. The dog became non-weight bearing lame on the pelvic limbs and remained extremely painful.

On presentation, physical examination was unremarkable. Rectal temperature was 38.3°C. Neurological examination was limited because of the severity of pain. The dog refused to put any weight on the pelvic limbs but voluntary movement was present in both pelvic limbs. Postural reactions were normal. The withdrawal reflex in the right pelvic limb was reduced. Myotactic and withdrawal reflexes in all other limbs were intact. Spinal palpation revealed severe pain at the lumbosacral level. Neuroanatomical localization was consistent with the L4–S3 spinal cord segments.

Complete blood cell count revealed mild neutrophilia [13.3 × 10⁹/L; reference range (RR): 3-11 × 10⁹/L] and mild monocytosis [1.6× 10⁹/L; RR: 0.0- 1.3×10^{9} /L]. Serum biochemical profile revealed mild elevated creatine kinase activity [250 U/L; RR: 0-190 U/L] and alkaline phosphatase [173 U/L; RR: 0-50 U/L].

Radiographs of the entire vertebral column were taken. Laterolateral radiographs of the lumbosacral region revealed narrowing of the intervertebral disc space at the lumbosacral transition. The adjacent endplates of L7 and S1 were irregular (Figure 1). The remain-



Figure 3A. Laterolateral radiograph of the lumbosacral region in a flexed position. There is a ventral subluxation of the sacrum in respect to the L7 vertebra.



Figure 3B. Laterolateral radiograph of the lumbosacral region in an extended position. There is narrowing of the intervertebral disc space at the lumbosacral joint. The adjacent endplates of L7 and S1 are irregular.

ing part of the vertebral column was unremarkable. Magnetic resonance images (MRI) of the lumbosacral vertebral column were obtained with a 0.4 Tesla MRI unit (Hitachi, Aperto). T2-weighted (T2W) images were obtained in sagittal and transverse planes. T1weighted (T1W) images pre- and post-contrast administration [intravenous administration of gadolinium 27.9 mg/kg body weight (BW)] were obtained in sagittal planes. The intervertebral disc space at L7-S1 was markedly widened with a markedly hyperintense appearance on T2W images of the intervertebral disc (Figure 2). The endplates had an irregular appearance and heterogeneous signal on T1W and T2W images with moderate contrast enhancement. The lumbosacral intervertebral disc was dorsally herniated causing mild focal vertebral canal narrowing. The nerve roots at the lumbosacral intervertebral foramina were bilaterally thickened and showed a heterogeneous hyperintense signal on T2W images and also marked contrast enhancement. Soft tissues ventral, and to a lesser extent, dorsal to the caudal lumbar spine and sacrum showed heterogeneous hyperintense signal on T2W images and contrast enhancement on T1W postcontrast images. Thoracic radiographs, abdominal ultrasound and echocardiography were within normal limits.

Urine and blood samples were collected aseptically for aerobic and anaerobic bacterial cultures. Urine was collected by cystocentesis. The blood was added to a commercially available bacterial culture medium (Signal blood culture system, Oxoid, Basingstoke, Hampshire, UK). Both urine and blood samples were positive for Staphylococcus spp. (coagulase positive). The susceptibility test showed sensitivity to amoxycillin clavulanic acid, cephalexin, marbofloxacin, erythromycin, fusidic acid and clindamycin. Intravenous amoxicillin clavulanic acid (Augmentin®, Beecham Group Ltd, Uxbridge, Middlesex, UK), 20 mg/kg BW, q8h and metronidazole (Metronidazole 500 mg/100ml®; Braun, Melsungen, Germany), 10 mg/kg BW, q12h were initiated. Analgesia was provided with a combination of meloxicam (Metacam®, Boehringer Ingelheim, Bracknell, Berkshire, UK), 0.1 mg/kg BW, IV, q24h; methadone (Physoseptone®, Martindale Pharmaceuticals, Romford, Essex, UK), 0.3 mg/kg BW, IV, q4h; gabapentin (gabapentin 300mg®, Double-E Pharma LTD, Dublin, Ireland) 20 mg/kg BW, orally, q8h and paracetamol (Paracetamol 500mg®, M&A Pharmachem LTD, Bolton, England, UK) 10 mg/kg BW, IV, q12h. The dog failed to improve clinically and remained extremely painful ten days after the treatment was initiated. Therefore, it was decided to take dynamic radiographs to look for vertebral instability.

Laterolateral radiographs of the lumbosacral vertebral column were obtained (Figures 3A and 3B). Radiographs were taken in neutral, flexed and extended position. Radiographs taken in flexed position showed a ventral subluxation of the sacrum in respect to the L7 vertebra (Figure 3A). Otherwise, the findings were

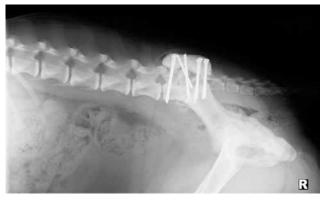


Figure 4A. Laterolateral radiograph of the lumbosacral region taken immediately after surgery. This radiograph demonstrates a good position of the screws and pins, which are dorsally embedded in PMMA.



Figure 4B. Ventrodorsal radiograph of the lumbosacral region taken immediately after surgery. This radiograph demonstrates a good position of the screws and pins.

similar to the radiographs taken ten days earlier. The vertebral instability could explain the persistent pain despite medical management, and surgical management was advised.

A standard dorsal laminectomy at L7-S1 was per-



Figure 5. Laterolateral radiograph of the lumbosacral region six months after surgery. There is marked osteolysis of the vertebral body of L7. One of the two cranial screws is broken. New bone formation ventral to the lumbosacral junction is more marked compared to the initial radiographs.

formed. Inspection of the spinal canal showed the presence of abnormal appearing epidural fat which was removed. A discectomy was performed, followed by stabilization of the lumbosacral vertebra. A 3.5 mm cortical screw was placed on each side in the pedicles of L7 just caudal to the base of the cranial articular process of L7. Two 3.5 mm cortical screws were placed in each side of the vertebral body of the sacrum just caudal to the caudal articular facet of L7 and behind the dorsal foramen of S1. Transarticular 3.2 mm pins were placed across the L7-S1 articular facets. Subsequently, all screws and transarticular pins were embedded in gentamycin impregnated PMMA bone cement. Postoperative radiographs showed a good position of the implants (Figures 4A and 4B). Samples from the epidural fat and a swab of the intervertebral disc were send for culture and the results came back negative.

Two days after surgery, the dog improved markedly and was able to get up and walk without showing any signs of pain. The dog developed a seroma postoperatively, which improved spontaneously. Analgesia was gradually ceased and the dog was discharged ten days after surgery. Antibiotic treatment was continued in oral form (amoxicillin clavulanic acid (Synulox 500 mg®, Pfizer, Louvain-La-Neuve, Belgium) 20 mg/kg BW, q8h). The metronidazole was stopped as soon as the results of the blood and urine cultures were received.

On follow-up examination six months after the diagnosis, the dog was reported to be clinically normal by the owner and was still being treated with antibiotics. Physical and neurological examination was within normal limits. Radiographs of the lumbosacral region revealed marked osteolysis of the vertebral body of L7 (Figure 5). The most cranial screw on the right hand side was broken, the cranial screw on the left hand side showed a radiolucent halo around its head and body. New bone formation ventral to the lumbosacral junction was more marked compared to

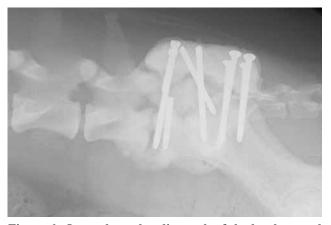


Figure 6. Laterolateral radiograph of the lumbosacral region twelve months after surgery. L7 has a more radiopaque appearance compared to the previous radiographs (Figure 5).

the initial radiographs. Blood and urine cultures were repeated and the results came back negative. Based on the radiographic findings, it was decided to continue the antibiotic administration for at least three more months.

The owner reported the dog to be still clinically normal at the follow-up examination twelve months after diagnosis. The dog was still being treated with the antibiotics. Physical and neurological examination was still within normal limits. Radiographs of the lumbosacral vertebral column were repeated, and L7 had a more radiopaque appearance compared to the previous radiographs (Figure 6). The radiolucent halos around the screws became smaller. There was some new bone formation at L6-7, which was thought to represent reactive changes or a sign of ongoing osteitis/osteomyelitis. Blood and urine cultures were repeated and the results came back negative. At that point, it was decided to stop the antibiotics. During the last telephone update, 18 months after diagnosis (i.e. six months after the antibiotics had been stopped), the owner reported that the dog had remained clinically normal.

DISCUSSION

The exact pathogenesis of discospondylitis remains unknown. Infection might be established in the highly vascular, slow flowing metaphyseal and epiphyseal capillary beds with extension into the disc (Kerwin, 2015). It is less likely that the intervertebral disc becomes infected directly as a healthy disc has few (if any) blood vessels (Sharp and Wheeler, 2005).

The source of the infection may be autogenous or iatrogenic. The majority of cases are thought to result from hematogeneous spread of an infection from a distant site, such as the genitourinary tract, skin, heart or teeth (Burkert et al., 2005). In humans, discospondylitis is most frequently diagnosed as a postoperative complication (Rhode et al., 1998; Lehovsky, 1999). Iatrogenic discospondylitis occurs in 2-7% of humans undergoing vertebral column surgery (Rhode et al., 1998; Lehovsky, 1999). In a retrospective study by Canal et al. (2016), discospondylitis was diagnosed in 8 out of 372 dogs (2.2%) as a postoperative complication after spinal decompression surgery for intervertebral disc herniation. In dogs, urinary tract infection is the most commonly diagnosed concurrent disease condition (Burkert et al., 2005). Also in the case reported here, a urinary tract infection was the most likely underlying cause. Staphylococcus spp. was identified in the blood and urine cultures of the dog. *Staphylococcus* spp. and *Escherichia coli* are the most common identified agents in dogs with urinary tract and prostatic infections (Krawiec and Heflin, 1992; Johnston et al., 2000).

In this case, initial antimicrobial therapy consisted of amoxicillin-clavulanic acid combined with metronidazole. In general, first-generation cephalosporin and amoxicillin-clavulanic acid are recommended for initial treatment as they have an activity against Staphylococcus spp., Streptococci spp. and Escherichia coli, which are common identified bacteria in dogs with discospondylitis (Thomas, 2000; BSAVA and SAMS, 2011; Sykes and Kapatkin, 2014). Metronidazole was added in this case to broaden the anaerobic spectrum. As results of culture and susceptibility testing should always define further treatment (Thomas, 2000; Burkert et al., 2005; BSAVA and SAMS, 2011; Sykes and Kapatkin, 2014), metronidazole was ceased once the results were known. Combined results of blood and urine microbial cultures yield a reported 30-78% success rate for the detection of an infectious agent (Kerwin et al., 1992; Fischer et al., 1997; Burkert et al., 2005).

The lumbosacral disc is the most commonly affected site in dogs (Burkert et al., 2005). This can be attributed to the high mobility of this intervertebral disc space. A possible explanation is the intermittent venous occlusion or stasis of blood flow at the lumbosacral junction during locomotion, which may lead to focal endplate necrosis. An episode of bacteremia could then lead to focal colonization (Eisenstein and Roberts, 2003).

The diagnosis of discospondylitis is most commonly based on radiographic changes of the vertebrae. Although the radiographic findings in this case report were diagnostic for discospondylitis, MRI was performed to rule out epidural empyema (Plessas et al., 2013). The limitation of radiographs is the time gap between the onset of clinical signs and the first appearance of the radiographic findings, as well as the disassociation between the clinical and radiographic signs during recovery (Thomas, 2000; Shamir et al., 2001). In a previous study, it was demonstrated that dogs that were admitted less than 20 days from the onset of clinical signs, either had no radiographic abnormalities, or had initial signs of collapsed disc space with or without bony lesions (Shamir et al., 2001). In the same study, worsening of the radiographic changes continued despite improvement of the clinical signs after the antibiotic treatment was started (Shamir et al., 2001).

In young dogs (less than one year old), radiographic evidence of improvement (bridging and sclerosis) has been evident at a three-weeks follow-up examination. In older dogs, radiographic evidence of improvement has been documented at six- to twelveweeks follow-up examination (Shamir et al., 2001). In the dog of this case report, severe osteolysis of L7 was present six months after diagnosis. Osteolysis together with the radiolucent halos seen around the screws, raised the suspicion of ongoing infection. Possible ongoing infection may result from an undetected foreign body, infection by fungal organisms, colonization by antimicrobial-resistant bacteria, or the inability of antimicrobial drugs to penetrate into the target tissue (Burkert et al., 2005). Another possible explanation is bone loss because of disuse of L7 immobilized by screws and cement. The only sign of improvement was the increased amount of new bone formation at L7-S1 compared to the initial radiographs. Despite the severe changes seen on the radiographs, the dog did not show any signs of ongoing infection. A follow-up MRI examination could have provided more valuable information about a possible ongoing infection but the presence of metallic implants would have caused significant susceptibility artefacts likely precluding an adequate evaluation of the area of interest.

Dynamic radiographs were important in this case to detect the vertebral instability. Only the radiographs in flexion revealed lumbosacral instability. It is questionable if the dynamic radiographs should have been taken on the first day. The instability might have been detected earlier but conservative management would still have remained the first choice of treatment as surgery is a very invasive procedure with high risk of complications due to the already present infection. Instability is most likely caused by the extensive bone destruction due to infection, and is a feature of latestage discospondylitis. The combination of vertebral instability together with the compression of the cauda equina by the pyogranulomatous material coming from the intervertebral disc probably played a major role in the persistent pain experienced by the dog in this case report. The goal of the surgery was to decompress the cauda equina and to stabilize the lumbosacral vertebrae. Retrospectively, the significant improvement of the dog shortly after surgery demonstrated the need for surgical treatment of this dog.

In conclusion, this is a case report about a dog with severe discospondylitis and secondary vertebral instability treated surgically. In dogs with discospondylitis not responding to conservative treatment, persistent pain might be caused by vertebral instability. Surgical stabilization of the affected instable vertebrae may result in fast and drastic clinical improvement.

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