Clinically relevant osteochondrosis of the seventh lumbar vertebra in a Beagle

Klinisch relevante osteochondrosis van de zevende lumbaalwervel bij een Beagle

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ABSTRACT

This case report describes the occurrence of clinically significant osteochondrosis of the seventh lumbar vertebra in a 10-year-old, female Beagle with progressive monoparesis, lumbosacral pain and urinary incontinence. A presumptive diagnosis was made using radiography and computed tomography. The dog was surgically treated by a dorsal laminectomy and the detached bone fragment was removed. A postoperative computed tomography scan demonstrated the complete removal of the osteochondrotic tissue. Although the dog recovered remarkably after surgery, she developed a clinical relapse after several weeks. The owners declined further diagnostic tests and the dog was euthanized. Histopathological examination of the removed tissue was in agreement with a diagnosis of osteochondrosis of the seventh lumbar vertebra.

SAMENVATTING

In deze casuïstiek wordt het voorkomen van klinisch relevante osteochondrose van de zevende lumbaalwervel beschreven bij een 10 jaar oude, vrouwelijke Beagle met progressieve klachten van monoparese, lumbosacrale pijn en urinaire incontinentie. Een vermoedelijke diagnose werd gesteld aan de hand van radiografie en computer tomografie. De hond werd chirurgisch behandeld door middel van een dorsale laminectomie met verwijdering van het losse fragment. Een postoperatieve computer tomografiescan toonde een nagenoeg volledige verwijdering van het fragment aan. Hoewel de hond onmiddellijk postoperatief duidelijk verbeterde, kende zij een klinisch herval na enkele weken. De eigenaars weigerden verder onderzoek en de hond werd geëuthanaseerd. Het histopatologisch onderzoek van het verwijderde weefsel was in overeenstemming met de diagnose van osteochondrose van de zevende lumbaalwervel.

INTRODUCTION

Osteochondrosis (OCD) is a multifocal disease of endochondral ossification leading to cartilaginous overgrowth, sometimes resulting in the separation of cartilage flaps (Ekman and Carlson, 1998). This disease may cause clinical signs in young, rapidly growing animals of many species (Guthrie *et al*., 1991). In dogs, it is most frequently reported in the appendicular skeleton (Kippenes and Johnston, 1998) with only few descriptions of OCD of the vertebral column (Hime and Drake, 1965; Lang *et al*., 1992; Hanna, 2001). Although lumbosacral OCD is typically considered to be an incidental finding, it can contribute to clinical signs of cauda equina syndrome (Lang *et al*., 1992). The latter describes sensory and/or motor neuronal dysfunction of the terminal part of the spinal cord and respective nerve roots. Common clinical signs are lumbosacral pain, reluctance to move, pelvic limb ataxia or paresis, sometimes in combination with urinary and/or faecal incontinence and other sensory deficits (De Risio *et al*., 2000). The sacral endplate is typically affected, with eventual separation of a mass at its craniodorsal corner (Lang *et al*., 1992; Hanna, 2001). There is a strong breed and gender predisposition with German Shepherds and male dogs being overrepresented (Lang *et al*., 1992). Lumbosacral OCD can be diagnosed by a variety of diagnostic modalities and different treatment options are proposed to assess this disorder. The purpose of this case report is to describe the clinical and diagnostic features of an unusual clinical presentation of lumbosacral osteochondrosis.

CASE REPORT

A 10-year-old, female, neutered Beagle (bodyweight 13.5 kg) was presented at the Department of Medicine and Clinical Biology of Small Animals, Ghent University, for the investigation of progressive

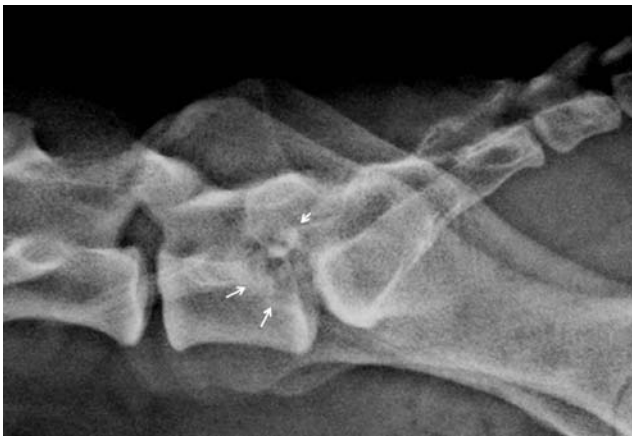

Figure 1. Lateral pelvic radiograph in neutral position. A triangular radiolucent area at the caudodorsal aspect of L7 (white arrows), associated with a radiopaque structure in the vertebral canal (white arrowhead) can be noticed.

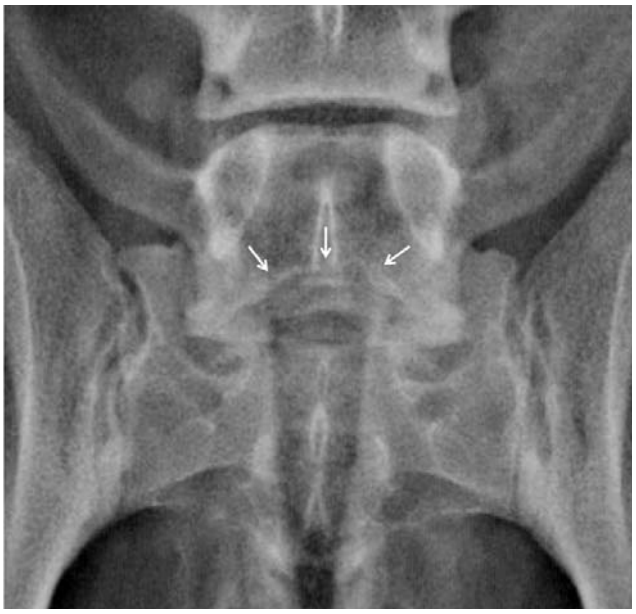

Figure 2. Ventrodorsal pelvic radiograph. A radiolucent and irregular defect of the caudal aspect of L7 can be noticed (white arrows).

clinical signs of monoparesis, trembling, lumbosacral pain and urinary incontinence since 3 months. Oral treatment with carprofen 2 mg/kg twice daily for 8 days gave no improvement. Physical examination, and a pre-anesthetic complete blood count and biochemistry panel were unremarkable. Neurologic examination revealed ataxia, paresis, mild muscle atrophy and proprioceptive deficits of the right pelvic limb. A painful response at the lumbosacral region was elicited by spinal palpation. No further abnormalities could be detected during neurologic examination.

A survey lateral radiograph, submitted by the referring veterinarian, demonstrated a triangular radiolucent defect at the caudodorsal aspect of the seventh lumbar vertebra (L7) together with a mineralized opacity in the vertebral canal at the level of the lumbosacral junction (Figure 1). Except for some mild sclerosis of the sacral endplate, no further abnormalities were noticed. A ventrodorsal radiograph showed a radiolucent area associated with an irregular contour of the caudal aspect of L7 (Figure 2). OCD, epiphysitis, a traumatic fracture or a neoplastic process were considered. To further characterize the lesion, computed tomography (CT) of the lumbosacral region was performed with the dog in dorsal recumbency and the pelvic limbs in extension. The dog was premedicated with a combination of acepromazine 0.02 mg/kg IV and morphine 0.1 mg/kg IV. Anesthesia was induced using propofol 50 mg IV and midazolam 0.2 mg/kg IV and maintained with isoflurane in oxygen. Computed tomography demonstrated a well delineated bone defect at the dorsal aspect of the caudal endplate of L7. The edges of this defect were isodense to cortical bone. A detached fragment was visible within the vertebral canal causing severe reduction of the dorsoventral diameter of the vertebral canal and displacing the cauda equina dorsally. Sporadic bone spurs were noticed bilaterally at the intervertebral foraminae and mild disc bulging was present at the lumbosacral intervertebral disc space (Figure 3). Based on these findings, a presumptive diagnosis of OCD of L7 was made.

The dog was anesthetized for surgery using the aforementioned protocol. Analgesia was achieved by a constant rate infusion of fentanyl (5µg/kg/h). The dog was positioned in sternal recumbency with the caudal lumbar and lumbosacral portions of the spine in a neutral position, the pelvic limbs in frog-leg position, and the tarsal joints level with the ischiatic tuberosities. A standard lumbosacral dorsal laminectomy was performed with preservation of the articular facets. The cauda equina was deviated dorsally over a large fibrous, tissue-covered mass at the caudal edge of L7. No significant disc bulging or other degenerative changes were noticed. The cauda equina was retracted laterally with a nerve hook and the longitudinal dorsal ligament was incised at the cranial border of the fibrous mass. The lesion was firmly attached to the caudal and caudolateral borders of L7 and could not be removed by manual manipulation. The caudal and lateral parts of the fibrous mass were removed by use of a 1 mm pneumatic drill to debulk and liberate the compressing lesion. This elicited the manual removal of the mass. The remaining crater shaped defect was debrided with a 1 mm bone curette. The lumbosacral intervertebral disc was not fenestrated. An autogenous fat graft was placed at the laminectomy site and the wound was closed routinely. The removed fragment was submitted for histopathological examination.

Immediately after surgery, a second CT was performed. It revealed the extent of the performed laminectomy and demonstrated complete removal of the detached fragment (Figure 4). Postoperative analgesia existed of oral carprofen 2 mg/kg twice daily and morphine 0.2 mg/kg IV each 4 hours.

The dog recovered remarkably during the first days following surgery and she was discharged after 5 days. At that moment, the dog still showed mild ataxia of the right pelvic limb. No pain or neurological abnormalities were apparent. The owners were advised strict

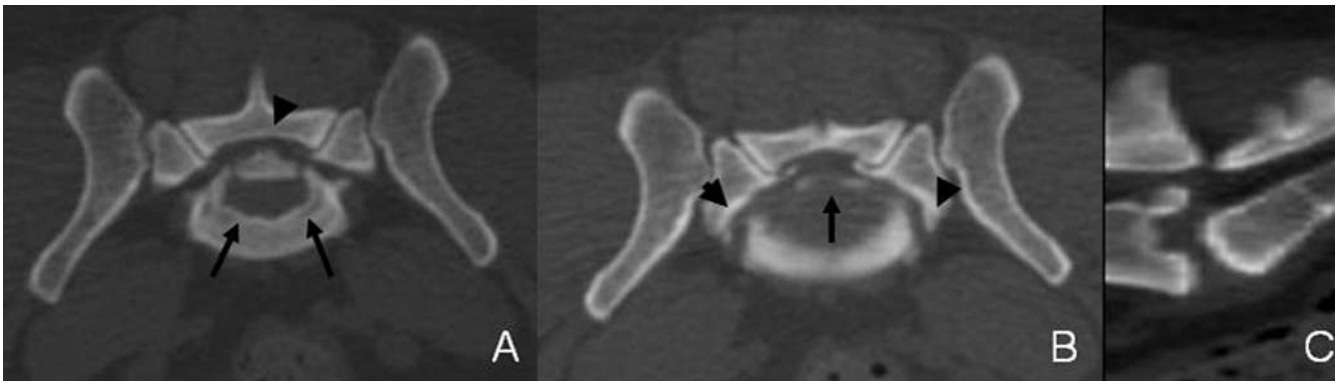

Figure 3. Transverse (A and B) and sagittal (C) pre-operative CT images of the lumbosacral region. There is a well delineated bone defect (A, arrows) at the dorsal aspect in the caudal endplate of the seventh lumbar vertebra. Note the detached fragment (A, arrowhead). New bone formation is visible bilaterally at the intervertebral foraminae (B, black arrowheads) and mild disc bulging is present (B, arrow). Compression and dorsal displacement of the cauda equina are noticed on the sagittal image (C).

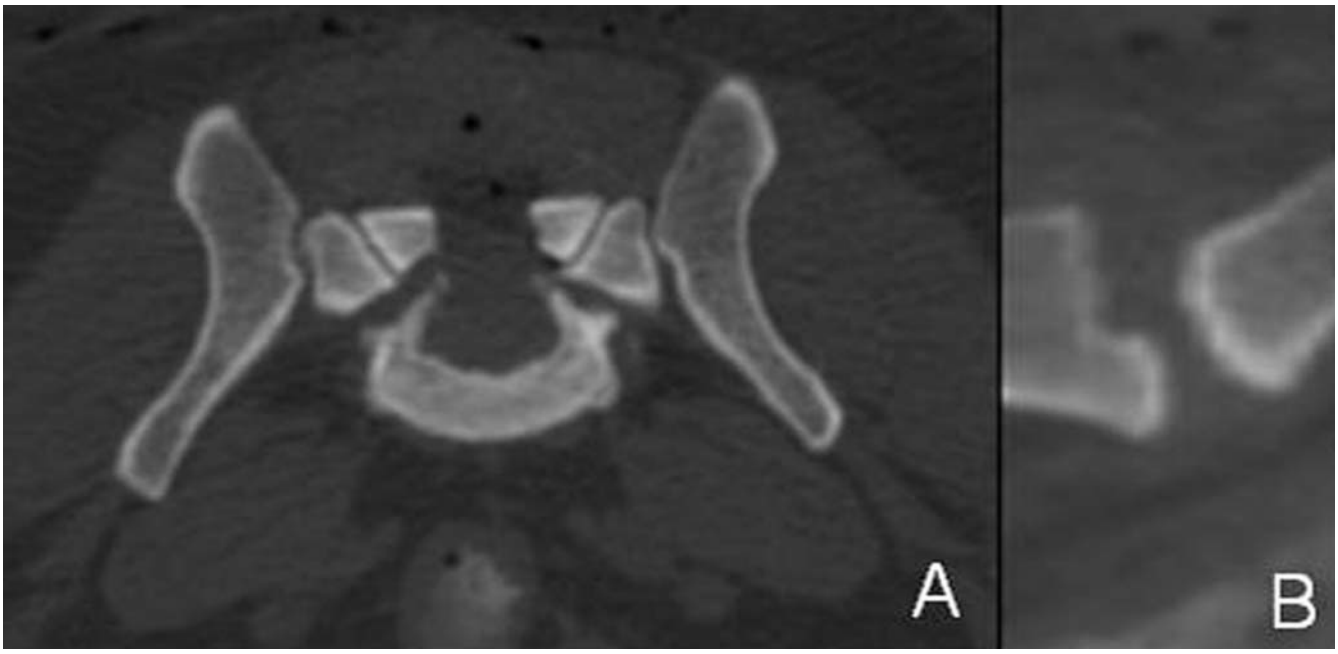

Figure 4. Transverse (A) and sagittal (B) postoperative CT images. Note the removed fragment.

rest for the dog for 6 to 8 weeks and to administer carprofen at 2 mg/kg twice daily orally for the following 14 days.

Telephone contact with the owners 1 week after discharge, confirmed further improvement of the clinical status. Difficulties with the rest confinement were reported. For this reason acepromazine 1 mg/kg orally twice daily was added to the treatment protocol. Five weeks after discharge the referring veterinarian was contacted. Deterioration of the dog's clinical status was reported. This deterioration consisted of lumbosacral pain and bilateral proprioceptive deficits. The owners declined further diagnostic tests or treatment and the dog was euthanized. The owners declined a post-mortem pathological examination. Histopathological examination of the submitted tissue revealed normal trabecular and focal necrotic bone tissue. It was covered by hyaline cartilage tissue with local formation of a cartilaginous flap (Figure 5). No neoplastic or inflammatory processes were observed.

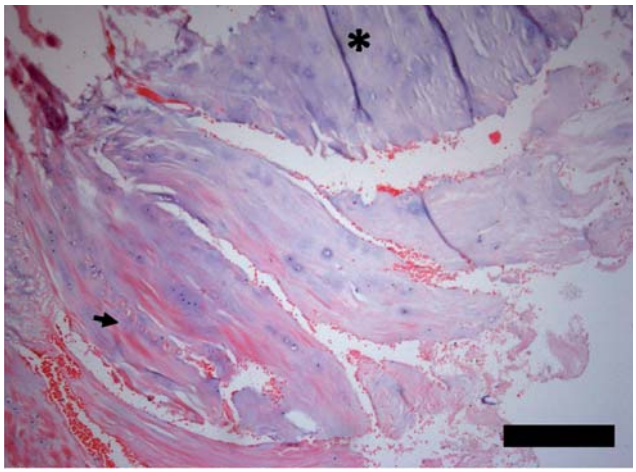

Figure 5. Removed tissue during the surgical procedure stained with hematoxylin and eosin (HE). The histopathological examination revealed normal trabecular and focal necrotic bone tissue (arrowhead). This was covered by hyaline cartilage tissue (*) with local formation of a cartilaginous flap (magnification 100x; bar = 250µm).

DISCUSSION

Lumbosacral OCD has been described in 68 German Shepherds, 4 Boxers, 3 Rottweilers, 2 Great Danes, 2 Springer spaniels, 2 Mastiff dogs, a Newfoundland dog and a Bernese mountain dog. (Lang *et al*., 1992; Snaps *et al*., 1998; Kriegleder, 2000; Hanna, 2001; Glyde *et al*., 2004; Michal *et al*., 2004; Mathis *et al*., 2009). To the authors' knowledge this is the first case reported in a Beagle. Moreover, the occurrence of such a lesion appears to be extremely rare in a medium sized dog.

The dog in this case report was 10 years old. This is in agreement with the previous reported literature (Hanna, 2001). In contrast, osteochondrosis of the appendicular skeleton usually induces clinical signs early in life, during the developmental stages (Ekman and Carlson, 1998). This difference is probably due to the fact that the lumbosacral joint is not a synovial joint and the separation of an osteochondral fragment does not result in pain after the synovial fluid penetrates to the subchondral bone (Glyde *et al*., 2004). Generally, the sacral endplate is affected (Lang *et al*., 1992). Osteochondrosis of the caudal endplate of L7, as in this dog, has only been described in 3 other cases (Hanna, 2001).

There is uncertainty about the clinical relevance of lumbosacral OCD. It has been reported as an incidental finding in clinically normal dogs and it is common for affected dogs with typical clinical signs of cauda equina compression to have concurrent degenerative lumbosacral stenosis (DLSS) (Lang *et al*., 1992). It has been suggested that clinical signs of dogs with lumbosacral OCD are more likely related to secondary degenerative changes of the intervertebral disc and vertebral canal than to the detached bone fragment itself (Lang *et al*., 1992; Hanna, 2001). It has been reported that dogs with clinical signs of DLSS with lumbosacral OCD are on average two years younger than dogs with DLSS without lumbosacral OCD (Lang *et al*., 1992). Since imaging and surgical findings in the present case did not reveal any additional significant degenerative abnormalities, it was presumed that the cauda equina compression was primarily caused by the detached bone fragment itself. It is also possible that a recent traumatic insult or repeated microtrauma contributed to the separation of the osteochondrotic lesion with subsequent onset of clinical signs. Mild disc bulging observed on the initial CT scan, was not noticed during surgery. This could be related to differences in patient positioning at the moment of CT and surgery (Suwankong *et al.*, 2006).

Different medical imaging modalities have been described to establish a diagnosis of lumbosacral OCD and associated cauda equina compression (Lang *et al*., 1992; Snaps *et al*., 1998; Michal *et al*., 2004). Although survey radiographs are very helpful in reaching a diagnosis, the superposition of the iliac wings makes it difficult to detect smaller detached fragments (Lang *et al*., 1992; Kriegleder, 2000). Although CT has been used for dogs with lumbosacral osteochondrosis (Michal *et al*., 2004; Mathis *et al*., 2009), there is little known about the CT features of this disease. The CT findings in the dog of the present case were very helpful to further characterize the abnormality and to visualize the severe reduction of the dorsoventral vertebral canal diameter and cauda equina compression. Computed tomography also proved to be useful in assessing the degree of surgical removal of the detached bone fragment.

In agreement with the study of Hanna (2001), conservative treatment was unsuccessful in this dog. Several surgical procedures have been described to treat this disease. Dorsal laminectomy with or without excision of the detached fragment and dorsal laminectomy with additional fusion are generally recommended (Hanna, 2001). Although the dog improved remarkably after surgery, she experienced a recurrence of clinical signs after several weeks. Although this complication has also been described in the study of Hanna (2001), no specific cause of such a clinical relapse has yet been identified. Since the owners declined further investigations, the authors can only hypothesize about the possible reasons of this recurrence. It is possible that further disk herniation occurred several weeks after the surgical procedure. Although no significant disk herniation was noticed intraoperatively, the defect in the caudal endplate of L7 could have caused altered biomechanics and could have induced further disk degeneration and protrusion. It is also possible that the dorsal laminectomy combined with extensive iatrogenic damage to the dorsal longitudinal ligament caused a significant decrease in stability of the lumbosacral junction. Although a recent veterinary cadaveric study failed to demonstrate a decrease in stability after a standard dorsal laminectomy (Meij *et al.,* 2007), several cadaveric human studies have demonstrated an important role of the posterior longitudinal ligament in resisting flexion forces (Gillespie and Dickey, 2004; Heuer *et al*., 2007). Further studies are necessary to determine whether additional stabilization is favorable in comparable surgical procedures. Other reported causes of recurrence of clinical signs after the completion of a dorsal laminectomy are scar formation, infection, residual compression, herniation of the dural sac through the laminectomy defect, and facet fracture after excessive thinning of the base of L7 facet at laminectomy (Sharp and Wheeler, 2005). Although only a part of the abnormal tissue could be submitted for histopathological examination, the results were in agreement with the original description by Lang *et al.* (1992), supporting the diagnosis of lumbosacral OCD. Since it was not possible to perform a post-mortem pathological examination of the seventh lumbar vertebra, this diagnosis could not be definitively confirmed.

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