Case Report

Spinal fracture in a dog with diffuse idiopathic skeletal hyperostosis

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Introduction

Diffuse idiopathic skeletal hyperostosis (DISH) is a systemic disease affecting the axial and appendicular skeleton of humans and dogs (1-4). To distinguish DISH from other diseases such as spondylosis deformans, ankylosing spondylitis, and spinal osteoarthritis, three criteria are widely accepted for diagnosis of DISH in humans and these criteria are also used in veterinary medicine (2, 5, 6). These criteria are as follows: 1) The presence of flowing calcification and ossification along the ventrolateral aspect of at least four contiguous vertebral bodies, with or without associated localized pointed excrescences at the intervening vertebral body–intervertebral disc junctions; 2) Relative preservation of intervertebral disc width in the vertebral segment involved, and the absence of extensive radiographic changes of degenerative disc disease, including vacuum phenomena and vertebral body marginal sclerosis; 3) The absence of articular process joint osseseous ankylosis and sacroiliac joint erosion, sclerosis, or intra-articular osseous fusion. Although based on just a single case-report, Morgan et al. proposed additional criteria to confirm the diagnosis of DISH in dogs that differed mainly by the inclusion of osteophyte formation around the articular process and lumbarosacral joints together with pseudarthrosis formation at the bases of the spinous processes (3). The prevalence of DISH in dogs is postulated to be 3.8% in general and 40.6% in the Boxer breed; it increases with age, and is more frequent in male dogs (6). The thoracolumbar spine is mainly affected (6). Diffuse idiopathic skeletal hyperostosis can coexist with spondylosis deformans in dogs and may be misinterpreted as severe spondylosis (2). A recent study provides support for the supposition of two different aetiologies, but to date the aetiology of DISH is unknown (7). Diabetes mellitus and hyperuricaemia, increasing age, and obesity are thought to be predisposing factors in human patients, and ethnic or genetic factors are suspected (1, 8-10, 11). In veterinary medicine, the Boxer breed is a closed gene pool and therefore may indicate a genetic origin for the disease (6). The clinical relevance of DISH in dogs has so far not been fully determined (2). Restricted mobility and non-painful and progressive reduction of peripheral articular and spinal range-of-motion have been attributed to DISH in two dogs (3, 4). In humans, DISH is considered to be a predisposing factor for spinal fracture and severe spinal cord injury after minor trauma, neither of which has been reported in animals to our knowledge (12, 13). This is the first case report of a dog with DISH-related fracture after minor trauma.

Case report

A six-year-old, spayed female Weimaraner dog, weighing 32.4 kg, was presented with paresis after colliding with a tree while playing with other dogs. Proprioceptive positioning was delayed in both hindlimbs. The patellar reflex was slightly exaggerated bilaterally. Deep pain perception was present in both hindlimbs. The remaining neurological examination was within normal limits. Based on the neurological deficits, the signs were attributed to an upper motor neuron lesion that was localised to the thoracolumbar region (thoracic [T]3-lumbar [L]3 vertebrae). As possible aetiologies,
vertebral fracture or luxation, traumatic intervertebral disc disease, fibrocartilaginous embolic myelopathy, spinal cord infarction, oedema or haematoma were considered.

Laterolateral and dorsoventral radiography of the spine showed signs of ventral and lateral smooth new bone formation on the vertebrae extending from the 11th thoracic vertebra to the sacrum (Figure 1A, B). The ossification was flowing along the ventral and lateral aspects of the vertebra leading to a segmental bony ankylosis. All facet joints showed periarticular osteophytes and increased sclerosis. The width of most of the intervertebral spaces was preserved. Only the L6-7 intervertebral disc space and the lumbosacral transition showed signs of calcification and narrowing of the intervertebral disc space together with mineral opacities in the region of the intervertebral disc and foramen. At the level of the L2-3 intervertebral disc space, a well-defined radiolucent line extending from the intervertebral disc space ventrally was visible. The ventrodorsal radiograph showed signs of mild deviation of the axis of the spine at the level of the second and third lumbar vertebra, caused by a marked widening of the intervertebral disc space at the right side (Figure 1B).

The final diagnosis was DISH of the lumbar spine, with fracture of the ventral hyperostotic bone bridge and vertebral subluxation. The fracture was surgically stabilized with standard vertebral body plating. A 5-hole, 3.5 mm compression plate (DCP) was applied with two screws each in the vertebral bodies of L2 and L3. Radiographs taken after surgery showed correct implant position and good axial alignment of the second and third lumbar vertebrae. The dog was discharged from our hospital four days after surgery with instructions to administer metamizole (20 mg/kg p.o. every 8 hours) for five days and for the dog to undergo physical therapy supervised by a professional physical therapist. The dog recovered completely within 10 weeks according to the owner’s information. Radiographs obtained eight months postoperatively revealed unchanged implant position and complete fracture healing. No lameness or neurologic deficits could be detected.

Two years later the patient was presented again with the same history to the local veterinarian. Initial examination revealed monoparesis of the left hindlimb. A combination of phenylbutazone and prednisolone were administered to the dog, and a re-evaluation was planned for the following day. The patient deteriorated neurologically overnight, showed hindlimb paralysis, and was referred to our veterinary medical teaching hospital. On presentation, the patient was non-ambulatory and displayed signs of severe pain. Palpation of the mid-region of the lumbar spine elicited signs of pain. Proprioceptive positioning was absent on both hindlimbs. The withdrawal reflex was depressed bilaterally, and the patellar reflex was absent in the left and depressed in the right hindlimb. Signs of deep pain perception were present in both hindlimbs. The remaining neurological examination was within normal limits. Based on the neurological deficits, the diagnosis was made of a lower motor neuron lesion that was localised to the lumbosacral region (L4-S3). Radiographs (Figure 2A, B) and computed tomographic images were obtained.

Computed tomography was conducted using a multislice helical scanner. The patient was positioned in dorsal recumbency. Helical transverse slices of the mid-lumbar

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A) Lateral and B) dorsoventral radiograph of the lumbar spine showing a fracture of the hyperostotic bone bridge (DISH: diffuse idiopathic skeletal hyperostosis) at the level of lumber vertebrae (L) 2-3 (arrows).

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\(^{a}\) Dynamic Compression Plate: Synthes GmbH, Freiburg, Germany
\(^{b}\) Vetalgin: Intervet Deutschland GmbH, Unterschleissheim, Germany
\(^{c}\) PhenPred: CP-Pharma Handelsgesellschaft GmbH, Burgdorf, Germany
\(^{d}\) Lightspeed QXi: GE Healthcare, Berlin, Germany
spine (L2 to L6) were acquired (120 kVP, 350 mA; bone windowing, slice thickness 1.25 mm; pitch 3.5). Multplanar and three-dimensional reconstructions were used for the assessment of the lumbar spine. The intervertebral disc space L4-5 was widened and showed a discontinuity of the ventral bridging bone formation. The caudal endplate of L4 was mildly irregularly outlined but sharply delineated. Several small linear mineral opacities were visible in the region of the intervertebral disc space. The right facet joint showed a well-defined radiolucent line running in a craniodorsal-caudodorsal direction through the joint space. Mild displacement of the fracture fragments was present. Dorsally reconstructed images showed a deviation of the vertebral column longitudinal axis due to the wedge-shaped widening of the intervertebral disc space. In addition, heterogeneous slightly hyperattenuating material was visible in the right lateral aspect of the vertebral canal, causing mild displacement and deformation of the spinal cord at the intervertebral disc space of L2-3. The heterogeneous material occupied about 30% to 40% of the vertebral canal diameter. Finally, new bone formation between the bases of the spinous processes was detected. The findings were consistent with a fracture of the ventral bridging osteophyte, the caudal endplate L4, and the facet joint L4-5, with subluxation of the facet joint on the left side and subluxation of the vertebrae (Figure 3A, B). Additional findings included bulging L2-3 intervertebral disc material and pseudoarthrosis between the bases of the spinous processes. The state of the previous osteosynthesis at L2-3 was unchanged, while ankylosis of the facet joints and vertebra had increased in comparison with earlier examinations. Spinal instability and clinical deterioration supported the decision to proceed with vertebral stabilisation.

The dog was positioned in sternal recumbency for surgery. A standard dorsoventral approach was performed. A 5-hole 3.5 mm compression plate with four screws was used for vertebral body stabilisation. The plate was positioned beneath the spinal nerve root. Two screws were placed in each of the vertebral bodies L4 and L5. Radiographs obtained postoperatively revealed unsatisfactory fracture reduction with unchanged widening of the intervertebral disc space ventrally. Revision surgery was performed the next day, at which time better fracture reduction was achieved. Postoperative radiographs revealed correct axial alignment of the vertebrae and position of the implants. Only mild widening of the intervertebral disc space was present. Following surgery, supportive care included the administration of buprenorphine (20 μg/kg SC every 8 hours) and metamizole (20 mg/kg IV every 8 hours), and wound management.

As part of the postoperative care, physical rehabilitation, pelvic limb passive range-of-motion, and massage were implemented. One day postoperatively the dog was still non-ambulatory. Signs of deep pain perception were present in both pelvic limbs with absent motor function. Voluntary motor function recovered four days after surgery. The patient showed signs of urinary incontinence. On day seven, the patient was discharged from the hospital at the owners’ request. The administration of metamizole (20 mg/kg orally every 8 hours) was continued for seven days. The owners continued simple rehabilitation exercises, including passive range-of-motion and sling-assisted walking four times a day for 15 minutes, and nursing care. In addition, professional physical rehabilitation was performed twice weekly.

At the first re-evaluation ten days after surgery, the dog was still non-ambulatory, but the neurologic deficits had improved slightly. At the second re-evaluation, 15 weeks postoperatively, the dog was able to walk for 10 minutes three times a day. Radiographs obtained under sedation with...
medetomidine\(^{f}\) (20 µg/kg IM) revealed signs of incomplete bone healing and bone callus formation. No signs of implant failure were detected on these images. At the third re-evaluation, seven months postoperatively, the patient was still mildly paretic. Proprioceptive positioning was absent on the left hindlimb and delayed on the right hindlimb. Spinal reflexes were normal. On follow-up radiographs, the position of the implants was unchanged. Signs of ankylosis of the entire lumbar vertebrae were present. The facet joints from L1 to L6 were ill-defined, and all vertebrae showed a homogenously reduced overall opacity. In addition, lateral smooth new bone formations flowed caudally along the lumbar spine up to the level of the lumbar-sacral junction. The current state showed an ongoing fracture healing and ongoing ankylosis (Figure 4A, B). Telephone communication 11 months postoperatively with the owner revealed further slow, but continuous improvement. The patient was able to walk for three hours twice daily.

**Discussion**

Diffuse idiopathic skeletal hyperostosis is a systemic skeletal disease in humans and dogs (1-4). The criteria for diagnosis in humans and dogs differ, and the use of these criteria in veterinary medicine was recently discussed (3, 5, 14). The radiographic findings in this dog fit the criteria proposed by Morgan et al. regarding the initial presence of periarticular osteophytes surrounding facet joints and the assumption that formation of pseudoarthrosis between the bases of spinous processes — noted from computed tomography, not radiographs — already existed on first presentation (3). Ankylosing spondylitis and spinal osteoarthritis, which produce changes within the spine seen in our patient.

The disease is supposed to be mostly asymptomatic in dogs, although the clinical relevance is not yet determined (2). The present case demonstrates that DISH is not always asymptomatic and may predispose to spinal fracture. A relationship between the first fracture and a second in an adjacent segment is possible biomechanically, especially after surgical stabilization. No changes in intervertebral biomechanics were noted at adjacent segments in a recent study after ventral slot procedure and surgical stabilization of the cervical spine with locked bone plates (22). It was postulated that similarities may exist in different spinal locations, although this is not consistent with earlier studies and was related to different techniques (22). Therefore, an explanation by information currently available in the literature cannot be given. However, ankylosis of the spine may be reasonably more likely.

Vertebral fractures are mainly caused by vehicular trauma (23, 24). This indicates the force required to fracture the vertebral column (24). Diffuse idiopathic skeletal hyperostosis often leads to ankylosis of the vertebral spine, and ankylosis predisposes to spinal fractures (25). Humans with ankylosing spondylitis, congenital fusions, and DISH are at higher risk of developing a cervical fracture and fractures owing to trivial trauma in patients with DISH (12, 13, 26, 27). Reduced bone density was noted in our patient on follow-up examination. Measurement of bone mineral density was thought to explain the higher risk of fracture in people with DISH, but a recent study found lumbar spinal bone mineral density measurement to be of little value for predicting fracture risk (27). Reduced flexibility and elasticity of the spine is supposed to be the reason for fracture susceptibility (27, 28). Decreased overall opacity of the vertebrae in our patient, therefore, was most likely due to disuse atrophy. Decreased flexibility of the spine may also lead to alteration of the facets and furthermore predispose them to fracture. The combination of fracture of the ossified ligament, fracture and luxation of the facet joint, and vertebral body involvement has also been found in humans (12). In summary, DISH alone may be a risk factor for

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\(^{f}\) Dormitor: Pfizer Deutschland GmbH, Berlin, Germany
spinal fracture (27). To the author’s knowledge, a DISH-related spinal fracture owing to minor trauma in a dog has not previously been reported.

The dog recovered completely within 10 weeks of the first surgical stabilization. The prolonged time of recovery after the second surgery may be attributed to several factors. Firstly, more severe clinical and radiographic abnormalities were present. In addition, fracture displacement was greater at the second presentation, suggesting higher stress on the spinal cord. Several small linear mineral opacities were visible on lateral radiographs after the second trauma. It is unclear whether these opacities are related to an endplate or to mineralized disc material. An affection of the endplate is known in humans suffering from DISH (12, 13). Fracture of lumbosacral spondylosis in two German Shepherd dogs has been reported. In these cases the discs became necrotic after ankylosis. After fracture of spondylosis, the degenerated disc became bruised owing to regained mobility and prolapsed to the vertebral canal. Detritus altered the spinal cord. The endplates were also affected and showed microfractures (29).

Hemilaminectomy and removal of any detritus within the spinal canal could have influenced the outcome in our case, but increased instability would have put higher loads on the fracture stabilization. Therefore, a mini-hemilaminectomy might have been a more appropriate approach but was not considered in our patient. In addition, L3-4 foraminotomy could have been alleviated compression of any swelling of the nerve roots. A magnetic resonance imaging examination eventually might have influenced our treatment decisions but was not available for this dog. Furthermore, the delay of almost 24 hours with clinical deterioration prior to surgery, together with the manipulation during two surgical interventions may have contributed to the damage of the spinal cord. Deterioration owing to instability without surgical fracture stabilization has also been reported in humans (3, 12). According to the established criteria regarding instability of vertebral fractures, surgical stabilization with bone plates was performed (30). This decision is supported by an in vitro study in which plate stabilization of canine lumbar vertebra with bicortical screws was found to be more rigid than other techniques, such as the polymethylmethacrylate-pin technique and vertebral body crosspins (31).

In conclusion, DISH is often an incidental diagnosis made from radiographs taken for non-related reasons and it is thought to be mostly asymptomatic in dogs (2). However, DISH may predispose dogs to unstable spinal fracture and acute spinal cord injury, and owners should be informed about the possible risk from relatively minor trauma.

Conflict of interest
None declared

References