



Case Report

Osteochondral dysplasia of the coxofemoral joints in a Friesian foal: Clinical findings and methods of diagnosis

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Summary

Diseases of the coxofemoral (hip) joint are infrequently diagnosed in horses. Most cases are presented as an unilateral condition and usually are of traumatic origin. This case report describes a Friesian foal with a clinically obvious thoracolumbar kyphosis, combined with a weight-shifting stance and a shortened stride of both hindlimbs. General clinical and lameness examinations, computed tomographic examination of the pelvis, and macroscopic and histopathological examinations of the coxofemoral joints were performed. This revealed a final, phenotypical diagnosis of a primary osteochondral dysplasia of both coxofemoral joints with secondary osteoarthritis. Similar to the occurrence of this condition in other species and considering the small genetic basis of the Friesian horse breed, a genetic predisposing factor is suspected to play a key role in the developing mechanism of dysplastic coxofemoral joint disease in horses as illustrated with this case. Computed tomography scanning appears to be a useful imaging technique in the detection of coxofemoral joint disease in small horses and foals.

Introduction

The term 'dysplasia' derives from the Greek words dys (abnormal) and plasis (formation) and thus describes an abnormal development. Dysplasia of the coxofemoral joint has been described in numerous animal species, but in dogs it is the most common orthopaedic disease of large breeds (Fries and Remedios 1995; Arnbjerg 1999; Keller *et al.* 1999; Piermattei 2006; Bracken and Ditchfield 2012; Bracken *et al.* 2012; Lopez 2012).

Coxofemoral joint disease in horses is, in contrast to the situation in dogs, not frequently diagnosed, while dysplastic hips are even more uncommon and diagnosed in only a few single case reports using radiographs only (Speirs and Wrigley 1979; Malark *et al.* 1992; Huggons *et al.* 2010). Recently, *juvenile osteochondral conditions* (JOCC) was proposed as a term in horses for developmental disorders related to immature joints or growth plates (Denoix *et al.* 2013). Osteochondral dysplasia is a more general term for a disorder of the development of bone and cartilage (Minor and Farnum 1988; Lachman 1998).

This case report describes a Friesian foal with osteochondral dysplasia of the coxofemoral joints, diagnosed noninvasively using computed tomography (CT). This condition, confirmed by macroscopic and histopathological examinations, has not been described previously in horses.

Case report

History

An 8-month-old Friesian colt (Foal 1) was admitted to the Equine Clinic of Utrecht University with thoracolumbar kyphosis, which had worsened progressively over the preceding few weeks (Fig 1). The foal had been treated with meloxicam (Metacam, 15 mg/ml)¹ for several days, but there had been no improvement of the condition. The owner reported that the foal was reluctant to trot or gallop, and had a weight-shifting stance of both hindlimbs; no traumatic event was reported. The foal had not been weaned yet and was growing rapidly. The referring veterinarian noted swollen femoropatellar joints 3 weeks before referral and suspected the foal of having osteochondrosis. The foal was referred because of progressive worsening of the clinical signs. Two other 8-month-old Friesian foals (Foals 2 and 3) from different owners were admitted to the clinic around the same time for investigation of a disease unrelated to their coxofemoral joints and were used as clinical controls.

Clinical findings

Upon physical examination, the affected foal (Foal 1) was bright and alert and in good body condition. Its vital signs and complete blood count were within normal limits and no abnormalities at ophthalmic examination were found. The foal showed severe kyphosis and a weight-shifting stance of both hindlimbs (Fig 1) with shortened strides during walking and trotting. The femoropatellar joints were somewhat distended, but not painful on palpation. The foal showed bilaterally swollen and firm gluteal muscles. Pain could be induced by deep palpation of the longissimus dorsi muscle at both sides in the thoracolumbar region. No clear pain response was evident on palpation or manipulation of the hindlimbs. The foal appeared bilaterally lame when walking and trotting but hindlimb flexion tests were negative. Local anaesthesia of the tibial and fibular nerves of the left hindlimb was performed, and did not significantly affect locomotion. The control foals (Foals 2 and 3) were subjected to euthanasia for reasons other than an orthopaedic disease.

Diagnostic imaging

Standard radiographs of both stifle joints and of the thoracolumbar spine were taken of the affected foal (Foal 1) in the standing position; no bony abnormalities were seen. Merely for financial reasons, it was decided at this stage to





Fig 2: All foals were positioned in right lateral recumbency with the femora at an angle of around 90° to the pelvis and the stifle joints flexed.

continue the diagnostic process with a CT examination of pelvis and coxofemoral joints, so additional radiographic or ultrasound examinations of these areas were not performed. For the selected CT examination a single slice helical CT scanner² was used. The affected foal (Foal 1) was premedicated with detomidine hydrochloride (Domosedan, 0.01 mg/kg bwt, i.v.)³ and butorphanol (Dolorex, 0.02 mg/kg, i.v.)⁴. General anaesthesia was induced with ketamine (Narketan 10, 2 mg/kg bwt)⁵ and midazolam (Midazolam Actavis, 0.06 mg/kg bwt)⁶ and maintained with 'triple drip' intravenous anaesthesia using guiaphenesin (Gujatal, 50 g)⁷, ketamine $(1 \text{ g})^5$ and detomidine $(10 \text{ mg})^3$ at 1 ml/kg bwt/h. The CT examinations of the control foals (Foals 2 and 3) were performed in a similar manner, but post mortem. All foals were positioned in right lateral recumbency with the femora at an angle of around 90° to the pelvis and the stifle joints flexed (Fig 2). All scans were performed using a 1 s tube rotation time, serial slices, a pitch of 1 and the bony algorithm.

It appeared that the affected foal (Foal 1) had incomplete and abnormally developed acetabula bilaterally. The dorsal

acetabular rims were most affected. The intra-articular bony lesions in both coxofemoral joints consisted of many patchy, mineralised areas and fragments of irregular shape and structure. The acetabula were very shallow, and, as a result, the femoral heads were subluxated. In fact, they were positioned against the remaining portions of the dorsal acetabular margins. On the caudal surfaces of the femoral heads, several irregular and ill-defined, erosions and cyst-like contour defects were evident in the subchondral bone. Some bony lipping was also visible bilaterally at the margins of the proximal femoral physis and was consistent with coxofemoral joint arthrosis (Fig 3). Conversely, the normal foals (Foals 2 and 3) had complete and normally developed bony acetabular rims, which were sharply defined and regular in structure and delineation. The femoral heads were positioned deeply inside the acetabula and no subluxation was seen (Fig 3).

Pathological findings

In view of the progressive nature of the clinical signs and the severity of bilateral disease, the owner elected euthanasia for the affected foal (*Foal 1*) upon our advice and gave consent for pathological examination. During gross pathological examination, the foal (*Foal 1*) exhibited irregular erosions 2–3 cm in diameter of the articular cartilage of both acetabula, exposing the underlying subchondral bone. The articular cartilage of the opposing femoral heads was roughened and irregular with small foci of ulcerative erosions. The epiphyseal plate of the femoral head had a loose cartilage was irregular with multifocal, small-to-large-sized areas of erosion, while the adjacent cartilage contained multiple clusters of irregularly proliferating, degenerative chondrocytes.

On further histological examination, the numerous focal surface cartilage lesions with underlying necrotic bone loss were filled with granulation tissue with high numbers of fibroblasts and many small blood vessels. The adjacent subchondral bone showed marked thickening of the trabecular bone. The growth plate showed irregular thickening on the epiphyseal side, necrosis and a moderate number of degenerative chondrocytes. These bilateral abnormalities of



Fig 3: a) Computed tomography images (plain 2D) of both coxofemoral joints in the transverse plane of (A) a normal foal (Foal 2) and (B) the affected foal (Foal 1). b) 3D reconstruction of computed tomography images of the coxofemoral joints of (A) a normal foal (Foal 2) and (B) the affected foal (Foal 1). Note the absence of a smooth acetabular rim and a normal femoral head curvature in both coxofemoral joints of the affected (Foal 1) in contrast to that of a normal foal (Foal 2).

the cartilage of the coxofemoral joints and femoral growth plates in *Foal 1* were consistent with osteochondral dysplasia (**Fig 5**). In contrast, the normal foals (*Foals 2* and 3) showed no histopathological abnormalities of the coxofemoral joints. Finally, the anatomical preparations of the affected and of the normal foals were compared (**Fig 6**) and the diagnosis osteochondral dysplasia was confirmed.

Discussion

The manifestations of a more generalised form of osteochondral dysplasia differ between domestic animals and humans and, based upon radiological and clinical criteria, nearly 500 skeletal dysplasias are known in man (Minor and Farnum 1988; Smit *et al.* 2011). However, in several dog breeds,

the generalised form of osteochondral dysplasia is clearly evident in animals small for their age, particularly in regions of rapid growth (limbs, vertebrae), resulting in disproportionate dwarfism. Particularly in Labrador Retrievers, it can manifest in malformation of both coxofemoral joints, but, in selected cases, also with concurrent shortened vertebrae, ocular defects, and a severely affected locomotion (Smit *et al.* 2011). Disproportionate dwarfism has also been reported in Friesian foals resulting from osteochondral dysplastic physeal growth retardation in limbs (e.g. metaphysis of the distal metacarpus and metatarsus) and ribs (e.g. costochondral junction; Back *et al.* 2008). In addition, Huggons *et al.* (2010) presented a 2-month-old severely lame dwarf Friesian filly with a unilateral primary coxofemoral subluxation from a (traumatic) rupture of



Fig 4: A macroscopic view of a coxofemoral joint in the affected foal (Foal 1) showing (a) a dysplastic acetabular rim and (b) cartilaginous ulceration of the opposing femoral head.



Fig 5: Histological confirmation of the coxofemoral arthritis as clinically evident in the affected foal (Foal 1) showing disorganisation of chondrocytes in the distal portion of the epiphyseal cartilage of the femoral head and similar to chondrodysplasia found in the growth plates of Friesian dwarf foals. Haematoxylin and eosin staining, 4× magnification.

the round ligament. At age 5 months, a ventral-dorsal radiograph of the pelvic region revealed subluxation of the left coxofemoral joint, secondary dysplastic femoral head flattening, excessive anteversion of the femoral head, and severe degenerative osteoarthritic changes to the acetabulum and femoral head.

In the affected foal of our study (Foal 1), both coxofemoral joints were abnormally developed, with severe osteochondral dysplasia of the acetabular margins, subsequent abnormal development of the coxofemoral joints and subluxation of the femoral heads caused by acetabula that were bilaterally very shallow. As a result, the radiological and pathological findings in that foal were thought to resemble the generalised, manifestation of osteochondral dysplasia, similar to those found in man such as multiple epiphyseal dysplasia, spondyloepiphyseal dysplasia congenita and Stickler syndrome, and in dogs such as chondrodysplasia and disproportionate dwarfism in Labradors (Lachman 1998; Lachman *et al.* 2005; Couchouron and Masson 2011; Smit *et al.* 2011; Veeravagu *et al.* 2012; Frischknecht *et al.* 2013). However, our affected foal (Foal 1) showed no signs of dwarfism or ocular defects. Given that, in this case, the only abnormalities evident were located in the coxofemoral joints, osteochondral dysplasia of the coxofemoral joints may, for the time being at least, be the most plausible, phenotypical diagnosis.

In dogs, radiography is the most definitive technique for diagnosing coxofemoral joint disease (Ohlerth *et al.* 2003; Ginja *et al.* 2010; Verhoeven *et al.* 2012). Diagnosing coxofemoral joint disease in horses often requires radiographs. Principally, high-quality radiographs can only be obtained under general anaesthesia with the horse in dorsal recumbency (Kangstrom 1972; Butler *et al.* 2008). Nonetheless, under more practical, field conditions, techniques have been described to be used in the standing horse, such as standing lateral oblique pelvic radiography. The disadvantages of this procedure would include limited visibility of the pelvis, which is further reduced in larger horses with inability to assess right/left symmetry, and due to the longer exposure time increased exposure to radiation for personnel (Barrett *et al.* 2006; Geburek *et al.* 2009).

In the first 6-12 months of life of human children, ultrasonography is used to confirm coxofemoral joint disease (Alanay and Lachman 2011; Bracken and Ditchfield 2012). Once ossification has progressed and the complete acetabulum cannot be assessed accurately, pelvic radiography becomes the primary imaging modality. Ultrasonography can also be a useful technique to diagnose coxofemoral joint disease in horses under practical, field conditions, but it is limited to the detection of irregularities of bony surfaces and soft tissue changes on the outer parts of the coxofemoral joint only (Brenner and Whitcomb 2009; Geburek et al. 2009). Recently, a study of ultrasonographic examination of the coxofemoral joints of young foals yielded reliable images of the joint (Rottensteiner et al. 2012). However, in older foals and mature horses it is impossible to visualise the complete acetabulum (including the acetabular labrum);



Fig 6: Photographical illustration of the anatomical preparations of the femoral head curvature (a+b) of (a) a normal foal (Foal 2) and (b) the affected foal (Foal 1) and the anatomical preparations of the acetabular rim (c+d) of (c) a normal foal and (d) the affected foal (Foal 1).

only parts of the acetabular rim can be observed. In our affected foal (*Foal 1*), ultrasonography may have provided a presumptive diagnosis but the extent of the degenerative changes could not have been visualised.

A CT examination of the coxofemoral joints of young horses has not been described yet in the literature. The major advantage of CT examination is the lack of superposition of the adjacent tissues, which facilitates the detailed observation of bony structures. In addition, it is possible to create useful multiplanar reconstructions or 3D images of the coxofemoral joint and pelvic bones of young horses in which pelvic or femoral disorders are suspected (Fig 3). Unfortunately, the area of large mature horses cannot pelvic be accommodated by CT equipment, due to the limitations of the size of the gantry. In our cases the choice was made to place the foals in lateral recumbency, as dorsal recumbency would have needed more elaborative, permanent hoisting and thus would have increased the anaesthetic risk (van Loon et al. 2010).

The only possible treatment option for the foal in this case would have been total hip arthroplasty, as described by Huggons *et al.* (2010). However, this was not an option in our foal, due to the presence of bilateral disease and the fact that the foal had not reached its maximal limb growth; this technique may only be successful in full grown, small equine cases (Huggons *et al.* 2010).

Concerning the genetic background of this disease, in man and dogs osteochondral dysplasia has been reported to be a hereditary disease (Lachman 1998; Lachman et al. 2005; Goldstein et al. 2010; Couchouron and Masson 2011; Smit et al. 2011; Shi et al. 2012; Veeravagu et al. 2012). These skeletal forms of osteochondral dysplasias seem to arise following mutations in genes that encode for collagen, which in dogs cause such conditions as short-limbed dwarfism and ocular defects (Goldstein et al. 2010; Frischknecht et al. 2013). In Friesian horses, disproportional dwarfism is described as a congenital, genetic defect (Orr et al. 2010) and osteochondral dysplasia may be the generalised underlying cause (Back et al. 2008). Recently, a local, osteochondral dysplastic malformation of the petrosal bone and, as a result, narrowing of the jugular foramen, has been demonstrated to be the cause of hydrocephalus appearing in the same breed (Sipma et al. 2013). Given the fact that Friesian horses are the product of a relatively small population, it is likely that this or a similar manifestation of osteochondral dysplasia (in a local or a generalised form) might become diagnosed more often in this breed, and thus it may be shown that this disease also has strong genetic predisposing factors in horses.

In conclusion, when encountering a weight-shifting stance in the hindlimbs together with a kyphosis of the back and a

shortened hindlimb gait, bilateral coxofemoral joint disease should be on the differential diagnosis list of possible causes including an initial, primary congenital osteochondral dysplasia and a secondary, deforming osteoarthritis. The pathological findings in the affected foal in this study might resemble a form of osteochondral dysplasia, as earlier described in Friesian horses and further illustrating a possible genetic background in this breed. Nonetheless, CT scanning appears to be a useful imaging technique in the detection of coxofemoral joint disease in small horses and foals and improves diagnostic imaging to recognise osteochondral dysplasia of the coxofemoral joints and secondary osteoarthritis.

Authors' declaration of interests

No conflicts of interest have been declared.

Ethical considerations

We certify that the animals in the study were treated in accordance with all legal and ethical requirements. No procedures were performed other than those needed to treat the patients in this study.

Manufacturers' addresses

¹Boehringer Ingelheim Vetmedica GmbH, Germany.

- ²Philips Secura, Eindhoven, The Netherlands
- ³Orion Corporation, Espoo, Finland.
- ⁴Intervet, Boxmeer, The Netherlands.
- ⁵Vétoquinol S.A, Lure Cedex, France.
- ⁶Actavis, München, Germany.
- ⁷Eurovet Animal Health BV, Bladel, The Netherlands.

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