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General Articles

High detail radiography and histology of the centrodistal tarsal joint of Icelandic horses age 6 months to 6 years

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Keywords: horse; Icelandic; bone spavin; OA; distal tarsal joints; high detail radiograpy; histology

Summary

Reasons for performing study: **Osteoarthrosis (OA) in the distal tarsal joints, bone spavin, is a well known condition which is common in Icelandic horses age 6–12 years.**

Objectives: **To determine the nature, location and age of appearance of early radiographic and histological changes in the centrodistal tarsal joint (CD) of young Icelandic horses.**

Methods: **Slab sections from the CD of young Icelandic horses were examined by high detail radiography (age 6 months to 6 years, n = 111) and histology (age 6 months to 4 years, n = 82) to detect and describe the early changes indicative of OA. Horses younger than 5 years were unridden.**

Results: **Chondronecrotic lesions histologically similar to those described in the early pathogenesis of OA were seen in 33% of the joints, located both medially and laterally. Radiographic sclerosis of the subchondral bone was recorded in 60% of the specimens, most often medially. Medial location was not associated with chondronecrosis, but was strongly related to age. Sclerosis was an infrequent finding on the lateral side, and was probably secondary to chondronecrosis in the corresponding part of the joint. Small defects in the subchondral bone were considered to be the most specific radiographic sign of OA as they were strongly associated with chondronecrosis.**

Conclusions: **The high prevalence of chondronecrosis in the young horses indicates an early onset and slow progression of the disease. The early appearance also shows that the initiation of the disease is unrelated to the use of horses for riding. As clinical manifestation of OA in the distal tarsal joints is most often described in mature or old horses, the first stages of the disease are not likely to result in clinical signs. Subchondral bone sclerosis did not appear to be a primary factor in the development of OA in the CD but was considered to reflect an uneven distribution of biomechanical forces within the joint.**

Potential relevance: **The development of OA in the CD of young Icelandic horses seems to be due to poor conformation or joint architecture rather than trauma or overloading. These aetiological factors are likely to be of importance for OA in the distal tarsal joints in other breeds as well. The influence of hindlimb conformation and the architecture of the distal tarsal joints on the biomechanics of joints need to be investigated, preferably by locomotion analysis in young horses.**

Introduction

Osteoarthrosis (OA) is a chronic disorder of synovial joints characterised by progressive deterioration of articular cartilage and reactive changes in the joint margin and joint capsule (Pool and Meagher 1990). It is accompanied by changes in the bone and soft tissue of the joint, including subchondral bone sclerosis and marginal osteophyte formation (McIlwraith and Vachon 1988).

OA in the distal tarsal joints (bone spavin) is a well known condition which has been reported to be common in Icelandic horses and strongly related to age (Eksell *et al*. 1998; Björnsdóttir *et al*. 2000a). In a study of Icelandic horses age 6–12 years, used for riding, the prevalence of radiographic signs of OA in the distal tarsus increased from 18.4% in horses age 6 years up to 54.2% in 12-year-old horses. The centrodistal tarsal joint (CD), also named the distal intertarsal joint (DIT), was affected in 93% of the affected limbs and 65% of the horses had radiographic signs bilaterally. (Björnsdóttir *et al*. 2000a). The same distribution between joints and limbs was found in 99 Icelandic horses radiographed and scintigraphed in Sweden (Eksell *et al*. 2000). Unilateral radiographic signs were distributed evenly between left and right tarsus in these 2 studies.

In Icelandic horses, radiographic signs of OA in the distal tarsal joints were most often detected in the dorsolateral, central and dorsal areas of the joints and a high scintigraphic uptake was found in the central and dorsal areas (Eksell *et al*. 1999; Eksell and Carlsten 2000). In other breeds (Hartung *et al*. 1983; Laverty *et al*. 1991; Barneveld and van Weeren 1999), the dorsomedial part has been regarded as the most specific location of osteoarthrosis.

The clinical manifestation of the disease is most often described as occurring in mature horses (Barneveld 1983; Pool 1996; Eksell *et al.* 1998; Axelsson *et al.* 1998; Björnsdóttir *et al.* 2000a; Sullins 2002). A frequent occurrence of radiographic changes in the distal tarsal joints was, however, recorded in young

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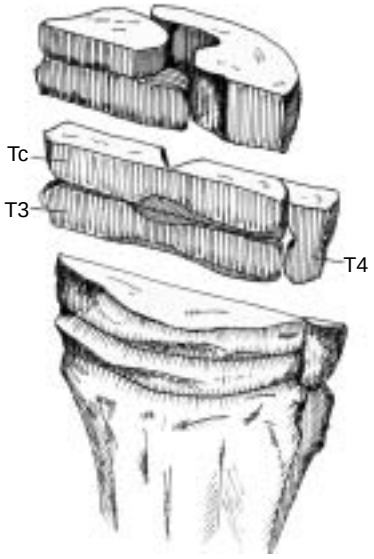

Fig 1: An illustration of the section plane of the distal tarsus and the slab bone section radiographed.

trotters (Hartung *et al.* 1983), and morphological changes of the distal tarsal joints have been observed in young horses by high detail radiography and histology (Laverty *et al.* 1991; Watrous *et al.* 1991; Barneveld and van Weeren 1999). In a macroscopic and microscopic study, articular cartilage fibrillation was described in Dutch Warmblood horses age 6–12 months (Barneveld 1983). In a study of foals of the same breed, predisposed for osteochondrosis and at different exercise levels, degenerative cartilage lesions were found in the distal tarsal joints in 22 out of 43 foals. In two (8%) 5-month-old and ten (53%) 11-month-old foals, major pathological changes of the articular cartilage were identified (Barneveld and van Weeren 1999). Laverty *et al.* (1991) described irregularities in the subchondral bone plate and chondronecrosis of the corresponding articular cartilage in all age groups, including horses younger than age 1 year. Pathological changes, with a focally thickened articular cartilage and cystoid lesions of the subchondral bone, interpreted as a form of osteochondrosis, were found frequently in the distal tarsus of 50 equids age 12–36 months (Watrous *et al.* 1991).

Most cases of OA in man are regarded as a complex interaction of multiple genetic and environmental (including traumatic) factors (Dieppe 1995). Radin (1995) emphasised trauma from malalignment, deformity or injury as the most common cause.

A genetic predisposition to bone spavin has been demonstrated in Icelandic horses (Björnsdóttir *et al.* 2000a; Árnason and Björnsdóttir 2002) and the prevalence of the disease was correlated to the conformation of the tarsus (Axelsson *et al.* 2001). Although the radiographic signs usually first appear when the horses have been broken to saddle, suggesting a connection to riding or workload, a variation in applied training intensity had no effect on the prevalence of radiographic signs of OA in the distal tarsal joints in Icelandic horses (Axelsson *et al.* 2001). Also, different training regimens were not reported to affect the frequency of tarsal OA in Dutch Warmblood foals age 5 and 11 months (Barneveld and van Weeren 1999).

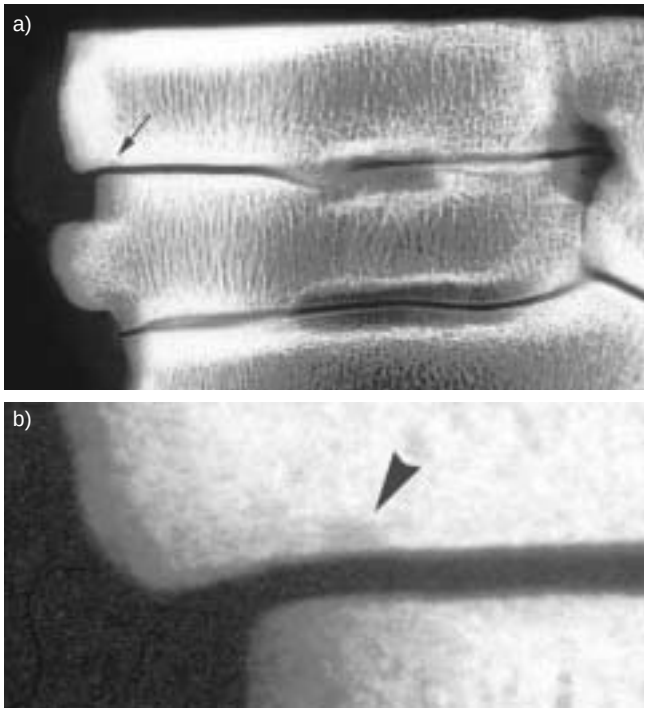

Fig 2: a) Section of the CD joint of a tarsus from a 4-year-old Icelandic horse (same horse as Figure 7). It was graded as 0 for sclerosis, 1 for subchorndral bone defect medially (arrow) and 0 for joint narrowing. b) The image magnified from (a) shows the subchondral defect in detail (arrowhead).

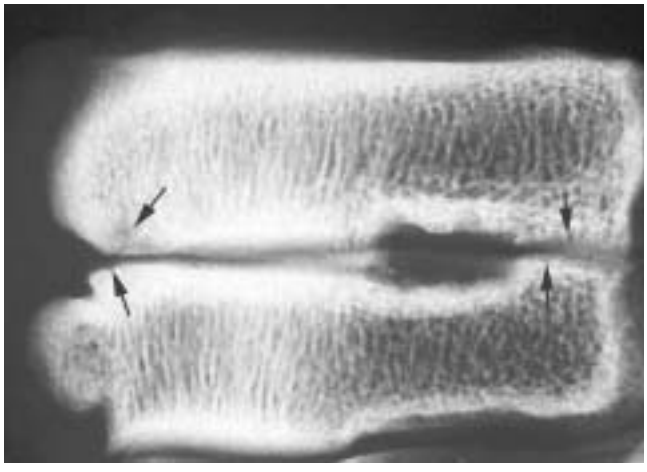

Fig 3: Section of a tarsus from a 1-year-old Icelandic horse (same horse as Figure 8), graded as 1 for sclerosis in the Tc medially, 2 for subchondral bone defect in the Tc medially, 1 in the T3 medially and 1 in both bones laterally and 1 for narrowed joint both medially and laterally.

The purpose of this study was to determine the nature and location of the early radiographic and histological changes in the CD of Icelandic horses, and to correlate radiographic changes with corresponding histological findings.

Materials and methods

Specimens

The CD of the left hind leg, including the central tarsal bone (Tc) and third tarsal bone (T3), were collected *post mortem* from

TABLE 1: Radiographic changes in the subchondral bone of the centrodistal tarsal joints of 111 Icelandic horses and histological defects of the articular cartilage of the centrodistal tarsal joints of 82 Icelandic horses

		Sclerosis		Defects		Narrowing		Chondronecrosis	
Age (years)	n	Medial n (%)	Lateral n (%)	Medial n (%)	Lateral n (%)	Medial n (%)	Lateral n (%)	Medial n (%)	Lateral n (%)
0.5	18	4 (22)	0	3 (17)	0	3 (17)	1 (6)	1 (6)	1 (6)
1.5	17	9 (53)	1 (6)	7 (41)	1 (6)	4 (24)	4 (24)	6 (35)	1 (6)
2.5	22	12 (55)	0	9 (41)	2 (9)	5 (23)	2 (9)	4 (18)	3 (14)
3.5	12	9 (75)	4 (33)	0	1 (8)	1 (8)	0	4 (33)	4 (33)
4.5	13	9 (69)	2 (15)	5 (38)	1 (8)	1 (8)	1 (8)	7 (54)	6 (46)
5.5	14	11 (79)	0	3 (21)	0	0	0	NE	NE
6.5	15	12 (80)	2 (13)	3 (20)	1 (7)	1 (7)	1 (7)	NE	NE
Total	111	66 (59)	9 (8)	30 (27)	6 (5)	15(14)	9 (8)	22(27)	15(18)

n = number of joints affected; Sclerosis = radiographic sclerosis of the subchondral bone; Defects = radiographic defects of the subchondral bone plate; Narrowing = radiographic narrowing of the joint space of the centrodistal tarsal joint; NE = not examined.

Icelandic horses age 6 months–6.5 years at a slaughterhouse in Iceland over 2 consecutive years. The age of the horses was estimated by examination of the eruption, replacement and wear of the incisors (Nickel *et al.* 1979). The age distribution is shown in Table 1. The bones were sectioned with a bandsaw into 8 mm thick slabs in the frontal plane, dorsal to the midline of the central tarsal bone (Fig 1).

In total, joints from 111 horses were examined with high detail radiography. In order to invesitgate age of onset of changes, joints from horses younger than 5 years (n = 82) were then selected for histology.

High detail radiography

The slab bone sections were radiographed (Siemens Polydorus 50)[1] at 48 kV and 4.0 mAs using high detail film (Kodak Min-R mammography)[2] and corresponding high detail intensifying screen.

The CD was evaluated on the high detail radiographs for sclerosis of the subchondral bone, defects of the subchondral bone plate, narrowing of the joint space and periarticular osteophytes. Sclerosis of the subchondral bone was classified on a scale of 0–2. No allowance was made for increased density that might be due to maturation. Scores were allocated as follows: 0 = no changes; 1 = peripheral thickening of the trabecular bone; 2 = all trabecular bone thickened or developed into compact bone. Subchondral defects were graded as: 0 = no defects; 1 = 1 or 2 separate foci of lysis; 2 = more than half of the joint surface with a thin zone or coalescing foci of lysis. Joint narrowing was graded as: 0 = no changes; 1 = narrowing without ankylosis; 2 = narrowing with ankylosis.

Locations of the abnormalities were recorded separately for the Tc and T3 bones and the medial and lateral parts of the bones. Locations of osteophytes were noted.

Histology

For histology, the slabs were fixed in a 4% aqueous solution of buffered formaldehyde. The CD, with the distal part of the Tc bone and the proximal part of the T3, was decalcified in 10% formic acid buffered to pH 4.5 with sodium citrate. The medial and lateral aspects of the CD were trimmed after decalcification, embedded in paraffin, cut into approximately 6 µm thick sections, coded and stained with haematoxylin and eosin (H&E) and Toluidine blue.

The degree of articular cartilage lesions was graded as mild, moderate or severe in the lateral and medial parts of the CD. A focal loss of chondrocytes and more eosinophilic granular extracellular stain (H&E) or loss of stain (Toluidine blue), with formation of chondrocytes in clusters adjacent to the area, was classified as a mild chondronecrosis. When the chondronecrosis was diffuse and contained more cluster formations, the lesion was classified as moderate. Severe lesions were characterised by marked chondronecrosis with loss of cartilage, often together with fibrosis and areas of fusion of the Tc and T3.

Data analysis

To avoid groups with few observations, the results were dichotomised into groups with and without findings. Bivariate logistic regression was used to examine the effect of age on the lesions found by high detail radiography and histology and the association between the findings. The minimum level of significance was chosen as $P<0.05$.

Results

High detail radiography of 111 joints

Sclerosis of the subchondral bone was recorded in specimens from 67 horses (60%), medially or both medially and laterally in all except one. Defects in the subchondral bone plate (Figs 2 and 3) were found in 36 specimens from 30 horses (27%). Locations and severity of the changes in Tc and T3 are shown in Table 2. The joint

TABLE 2: Outcome of high detail radiography of 111 centrodistal tarsal joints split up into the medial and lateral aspects of the Tc and T3 bones

	Medial aspect		Lateral aspect	
	Grading of sclerosis	Grading of defects	Grading of sclerosis	Grading of defects
Tc	0: n = 45 1: n = 59 2: n = 7	0: n = 85 1: n = 16 2: n = 10	0: n = 102 1: n = 9	0: n = 105 1: n = 4 2: n = 2
T3	0: n = 78 1: n = 30 2: n = 3	0: n = 84 1: n = 12 2: n = 15	0: n = 108 1: n = 3	0: n = 107 1: n = 2 2: n = 2

Sclerosis = radiographic sclerosis of the subchondral bone; Defects = radiographic defects of the subchondral bone plate.

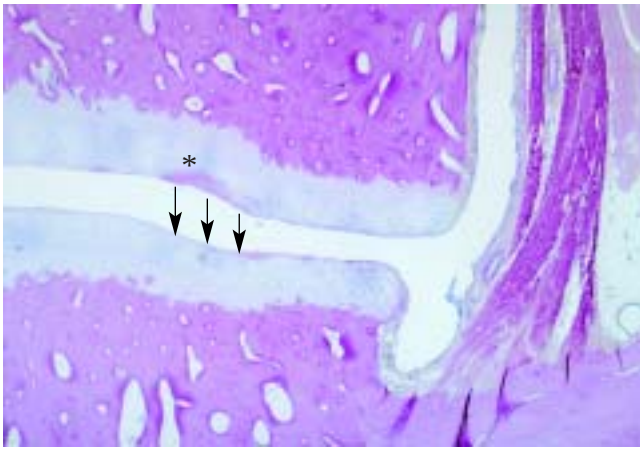

Fig 4a: Microscopic section of the lateral centrodistal tarsal joint of a 2-year-old Icelandic horse with mild chondronecrosis. Chondronecrosis of the distal Tc (*) and proximal T3 (arrows) are present. H&E; x35.

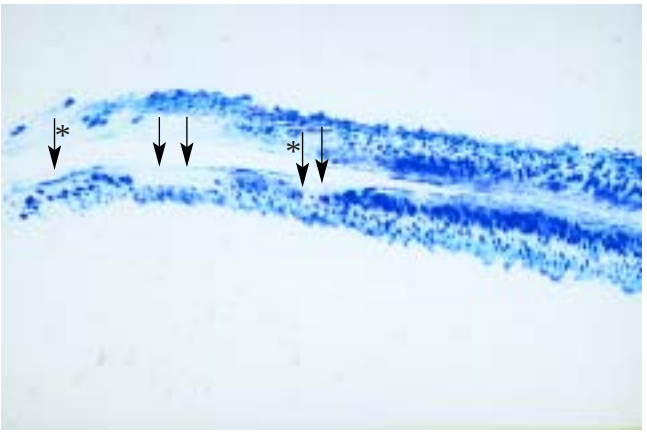

Fig 5a: Microscopic sections of the medial centrodistal tarsal joint from a 3-year-old Icelandic horse with moderate articular cartilage lesions. Chondronecrotic areas (*) of the distal Tc and proximal T3 (arrows) are seen. Toluidine Blue; x35.

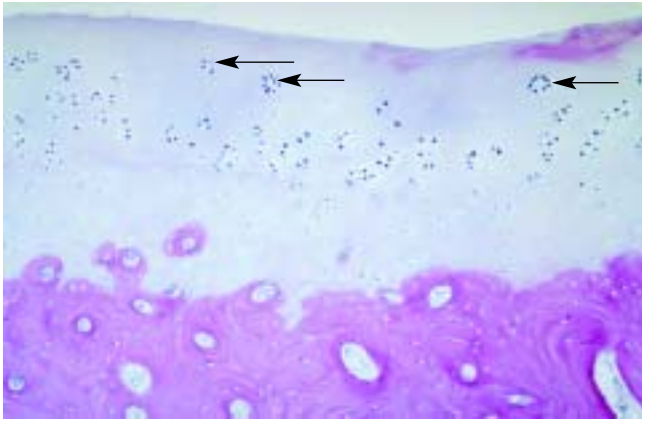

Fig 4b: High detail of the chondronecrosis in proximal T3 (from Fig 4a). Note formations of chondrocytic clusters (arrows). H&E; x140.

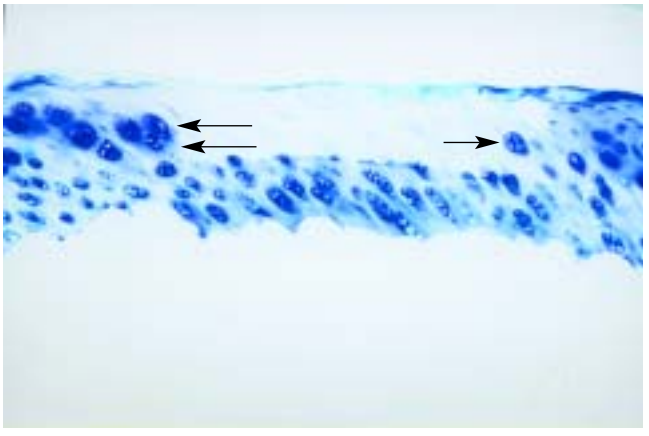

Fig 5b: High detail of the chondronecrosis in proximal T3 with loss of chondrocytes and many cluster formations (arrows) surrounding the necrosis. Toluidine Blue; x140.

space was narrowed in 15 cases (14%), of which all were affected medially and 9 were also affected laterally. Seven had ankylosis and were graded as 2. The age distribution of the radiographic findings is shown in Table 1. The presence of sclerosis on the medial aspect of the bones was related significantly to age ($P<0.001$). Narrowing on the medial aspect was negatively associated with age ($P<0.05$) while the other radiographic changes were unrelated to age (Table 1). Periarticular osteophytes were found in 6 specimens, all of which had defects in the subchondral bone. They were all located on the medial border of the joint, 2 on the Tc, 2 on the T3 and 2 connected to both Tc and T3.

Histology of 82 joints

The morphology of the articular cartilage changed with age. The basal layer was more cellular in younger horses than older. A layer of calcified cartilage with a marked 'tidemark' between the noncalcified and calcified articular cartilage was present in all horses. A thinner, less cellular noncalcified cartilage layer characterised the older horses.

Mild articular cartilage lesions were present in both medial (n = 13) and lateral (n = 7) sections. Focal areas with loss of chondrocytes and matrix stainability with a granular eosinophilic matrix (Figs 4a,b) characterised the lesions. A few clusters of chondrocytes were found in the border of the necrosis. The lesions were most often located in the periphery, close to the synovial cavity. The articular cartilage layer was sometimes mildly fibrillated superficial to the necrosis, but the cartilage bone interface mostly had a normal morphology. However, focal disruptions of the cartilage/bone interface were found in association with necrotic cartilage of the medial side from 3 horses. In one case (age 3 years), pannus-like connective tissue with synoviocytes covered the focal cartilage lesion of the lateral region.

Moderate changes were also found in both medial (5 sections) and lateral (4 sections) locations. They were characterised by a more diffuse chondronecrosis with thinner articular cartilage and fraying of focal areas of the superficial articular cartilage covering the chondronecrotic parts (Figs 5a,b). In 2 cases, the cartilage/bone interface was disrupted in focal areas in association with the necrotic cartilage of the medial side. In 2 cases (age 6 months and 1.5 years), a pannus-like connective tissue covered and replaced part of the articular cartilage laterally (Fig 6).

Severe lesions were found in 5 of the medial and 3 of the lateral sections. A diffuse chondronecrosis with an acellular, pale cartilage matrix (Toluidine blue) that stained more eosinophilic

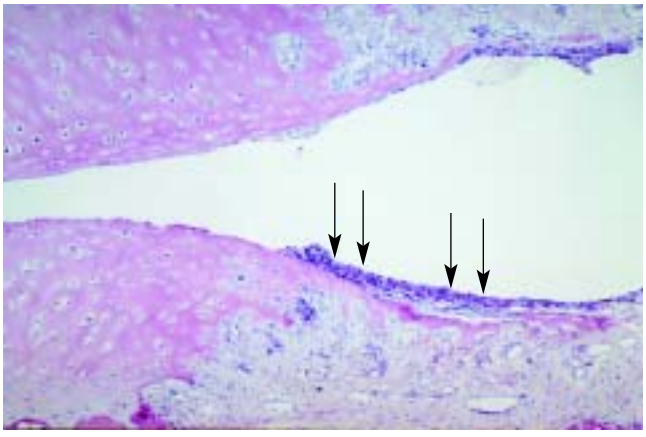

Fig 6: Microscopic section of the lateral centrodistal joint from a 6-month-old Icelandic horse with a moderate articular cartilage lesion. Note the pannus-like connective tissue (arrows) replacing part of the hyaline cartilage in the periphery of the joint. H&E; x140.

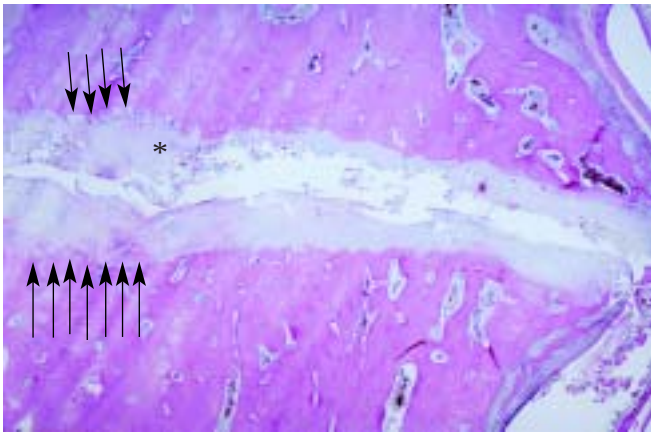

*Fig 7a: Microscopic section of the lateral centrodistal tarsal joint from a 4-year-old Icelandic horse with severe articular cartilage lesions. Thin articular cartilage with diffuse chondronecrosis of the proximal T3 and distal Tc can be seen. Disruption of cartilage/bone interface is also present (arrows). *High detail section of area (see Fig 7b). H&E; x35.*

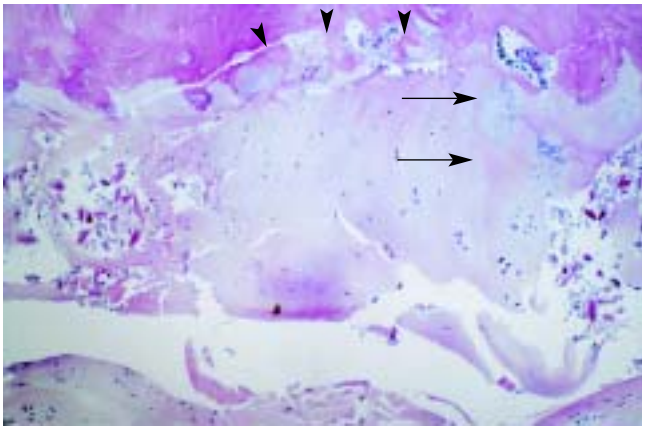

Fig 7b: Cluster formations of chondrocytes (arrows) are present and a disruption of the cartilage/bone interface can be seen (arrowheads). H&E; x140.

(H&E) and had a more fibrillar appearance with deep fraying and cleft formations was found (Fig 7b). The cartilage was thinner, often fused with the opposite articular cartilage, causing obliteration of the synovial cavity. Areas of disruption of the

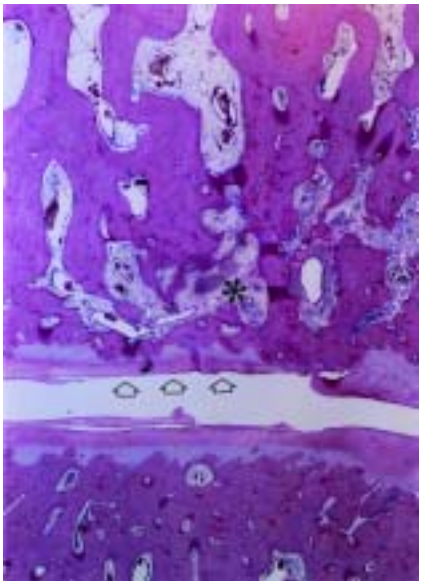

Fig 8a: Microscopic section of the medial centrodistal joint from a 1-year-old Icelandic horse with severe pathological lesions in the articular cartilage and subchondral bone of the distal Tc and proximal T3. Loss of the articular cartilage (arrows) and diffuse chondronecrosis is present, together with a cystic lesion in the subchondral bone of distal Tc (*). H&E; x35.

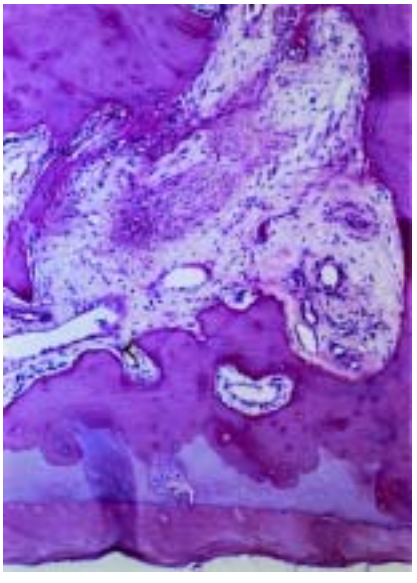

Fig 8b: High detail area of the cystic lesion (*) from Figure 8a. Intertrabecular granulation tissue can be seen. H&E; x140.

cartilage/bone interface and sometimes granulation tissue that crossed the synovial cavity could be seen. In a horse age 1.5 years, a subchondral cyst-like lesion with granulation tissue was found at the medial side (Figs 8a,b).

Chondronecrosis of different grades was seen in 27 joints (33%). There was no significant difference in frequency between the medial and lateral locations and they were both affected in 11 joints. The presence of chondronecrosis distributed in relation to age is demonstrated in Table 1. A significant association between the presence of chondronecrosis and age was found in both the medial ($P<0.05$) and lateral ($P<0.01$) parts of the joint.

No macroscopic or microscopic changes could be detected in the periarticular ligaments or synovial capsule of the 82 joints. The

microscopic examination of the soft tissues was, however, restricted to the synovial capsule adjacent to the bones in the medial and lateral areas and the thin periostal part of the attached ligaments.

Association between findings on high detail radiography and histology

The presence of radiographic bone sclerosis medially was not related to chondronecrosis, but bone sclerosis laterally was significantly associated with chondronecrosis of the adjacent part of the articular cartilage ($P<0.05$). The strongest association was seen between the presence of radiographic defects in the subchondral bone and chondronecrosis, both medially ($P<0.01$) and laterally ($P<0.01$). Defects of the subchondral bone on the medial aspect were also associated with chondronecrosis laterally ($P<0.05$). The presence of radiographic joint narrowing was not associated with chondronecrosis. All specimens with periarticular osteophytes had findings on histology.

Discussion

Icelandic horses are usually broken to saddle at age 4 or 5 years. Until then they are free-roaming, usually on large areas, and fed outside during the winter. As a result of the breeding management, an excess number of horses are bred for the riding horse market. The least promising horses, according to conformation, motion (gaits) and breeding index, are therefore selected for culling at a young age and used for consumption. The present material consisted of CD joints from these horses, collected at slaughter houses, and was not regarded as representative of the whole population of young horses. Clinical information was not available for the material.

Although the selected section plane included known sites of degenerative lesions, the thin slab sections of the joint examined by high detail radiography (8 mm) and histology (6 µm) represented only a small part of the joint tissues. Hence, the number of lesions recorded probably underestimates the pathological changes in this material; and explains the sometimes greater frequency of radiographic subchondral bone defects than histological lesions in Table 1. Conversely, histology is a more sensitive method if a small lesion is present, which may not be seen on the thick slice radiographed.

Late stage OA of a low-motion joint is characterised by full thickness necrosis of the articular cartilage, focal destruction of the bony support, an absence of synovitis and focal to complete bony ankylosis of the joint (Pool 1996). The characteristics of the histological findings in the present study strongly suggest that they are implicated in the early pathogenesis of OA, as they are in agreement with the description in other investigations (Laverty *et al.* 1991; Pool 1996; Barneveld and van Weeren 1999). There were no similarities with osteochondrosis as described by Watrous *et al*. (1991). Features of osteochondrosis are focally impaired endochondral ossification with a retention of cartilage into the subchondral bone (Olsson and Reiland 1978). Those were not seen in the mild articular cartilage lesions. The disruption of the cartilage/bone interface was degenerative in nature and mostly seen in more advanced lesions as a result of the progressive deterioration of the articular cartilage. The osteochondrosis described by Watrous *et al.* (1991) is a different condition, which probably also leads to OA.

Subchondral bone changes are regarded as being integral with the arthrotic process (Radin 1995). Repetitive impulse loading, a dynamic strain, stimulates bone remodelling which ultimately can result in articular cartilage thinning and loss. In the case of static strain, however, early pathological changes are seen in the articular cartilage without previous subchondral bone changes (Kawcak *et al*. 2001). In the present study, sclerosis of the subchondral bone was estimated by high detail radiography rather than histology, as the bone sections radiographed were thicker and more representative than the histological sections. Subchondral bone sclerosis was commonly found medially on Tc and T3 and was strongly associated with age, but not with the presence of chondronecrosis. It was therefore considered to be an adaptation to dynamic strain. It seems unlikely in this material that it initiateds cartilage damage. On the other hand, all the specimens with subchondral bone sclerosis laterally, although an infrequent finding, had chondronecrosis on the corresponding part of the cartilage. However, as there were always more joints affected by chondronecrosis than bone sclerosis in the lateral location (Table 1), the bone sclerosis could be secondary to the chondronecrosis. These results indicate different biomechanical loading patterns in the 2 parts of the joint, probably with dynamic strain medially and a more static strain laterally. In conclusion, sclerosis of the subchondral bone appears not to be a primary factor in the developing of OA in the CD of Icelandic horses. Defects of the subchondral bone plate were considered to be the most specific radiographic sign of the disease, as the association with the chondronecrosis was strong in this material.

Lesions on both high detail radiography and histology were more common at the medial than the lateral side. This appears to be contradictory to the frequency of lesions on clinical radiography, which suggested that the dorsolateral part was the predilection site of bone spavin in Icelandic horses (Eksell *et al*. 1999). The explanation might be that the two studies were describing different stages of the disease; the present study was directed at the initiation of the disease, while the radiographic study described the repair processes. The static strain laterally might in some cases result in ankylosis in that part of the joint before other radiographic signs develop. According to Dieppe (1995), the initiation and progression leading to the clinical expression of OA are different processes which may have different genetic and environmental risk factors.

The results of the present study, with chondronecrosis in one foal and an increasing prevalence up to age 4 years, suggests that initiation of the disease is unrelated to use of the horses for riding. In many cases, the lesions starts to develop long before the horses are broken in, although they may first be detected on radiography at an age when they are used for riding. The high frequency of histological findings in the young horses, compared with the radiographic findings in horses age 6–12 years (Björnsdóttir 2000a), indicates a very slow progression of the disease in many cases. In fact, some cartilage lesions may not progress to OA, as has been observed in the human foot (Youngman *et al*. 1987). Nevertheless, taking into account the thin sections examined on histology, the pathological lesions appear to be extensive within the joint at an early stage of the disease.

The present study did not support the theory that the disease starts with periarticular trauma (Pool 1996). This should, however, be investigated further by studies which include all supportive ligaments and tendons of the distal tarsal joint.

The low-motion joints, such as the CD, are described as being

susceptible to nonphysiological loading and metabolic disturbance, as the area of maximal weightbearing is nearly stationary during locomotion (Pool 1996). The relationship of subchondral bone sclerosis to chondronecrosis revealed in this study indicates an uneven distribution of biomechanical forces within the CD joint. Hence, it seems that poor conformation or joint architecture, rather than trauma or overloading, is the main aetiological factor of the disease in this particular joint.

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Manufacturers' addresses

[1]Siemens, Munich, Germany.
[2]Kodak, Rochester, New York, USA.

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