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Osteochondrosis lesions of the lateral trochlear ridge of the distal femur in ponies

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Summary

Lesions of the lateral trochlear ridge of the distal femur (LTR) were investigated in four animals of pony or pony-cross breeding. The animals, all male, were 6-15 months old. Lesions were bilateral in three animals and unilateral in one. Femoropatellar joint effusion and lameness were present in two animals but clinical signs were absent in the others. The proximal LTR was affected in all animals. The radiographic appearance of the lesions was a subchondral defect containing mineralised bodies. Arthroscopic and post-mortem examination findings included an osteochondral flap, a fissured or irregular articular surface and a smooth surface overlying focally thickened cartilage which extended into subchondral bone. Thickened articular cartilage was a histological feature of all the lesions. Amongst the other histological features were
chondronecrosis, chondrocyte clusters, phenotypically abnormal chondrocytes, horizontal fissures at the osteochondral junction and retained blood vessels. The signalment of the four animals, their clinical signs and the pathological features of their lesions were consistent with osteochondrosis of the LTR in the horse, making this the full description of the disease in the pony. The use of multiple criteria was considered to be important to making a specific diagnosis.

Introduction

Although the prevalence of osteochondrosis in the pony has not been investigated specifically, it is generally accepted that the condition does occur in ponies but is rare compared to larger breeds of horses (Jeffcott 1991; Philipsson 1996). From this it has been surmised that ponies may be less susceptible to osteochondrosis than horses (Jeffcott 1991). To date, however, no detailed descriptions of osteochondrosis in the pony have been published and therefore it is not clear whether the clinicopathological features of this condition are the same in ponies and horses.

The objective of this study was to describe the clinical, gross morphological and histological features of localised lesions of the lateral trochlear ridge of the distal femur (LTR) of growing ponies in relation to osteochondrosis lesions at the same site in the horse. This was considered to be a prerequisite to work investigating the basis of the perceived difference in susceptibility to osteochondrosis between the pony and the horse; investigations of this type have the potential to provide insight into the pathogenesis of osteochondrosis.

Materials and Methods

Animals

Four animals with documented pony or pony-cross breeding were used in this study. The signalment of the animals, their medical history and the pertinent findings from clinical examination were obtained by reviewing clinical records or by interview. The signalment of the animals is recorded in Table 1. The animals were all geldings, between 6 to 15 months old. Animals 3 and 4 had no history of orthopaedic disease, whereas animal 1 had been mildly-moderately lame for 3 weeks and animal 2 for 8 weeks. Bilateral femoropatellar joint effusions
were identified in animals 1 and 2. Radiographs were obtained from animals 1 and 2. Animals were humanely killed by an overdose of intravenous barbiturate.

Samples

Lesions were identified by the presence of localised gross abnormalities of the articular cartilage of the proximal half of the LTR, which is recognised as a predilection site for osteochondrosis in the horse (McIlwraith and Martin 1985). The description of the gross appearance of the lesions was obtained from the surgeon who performed the arthroscopic examination or was made at post-mortem examination. Samples of full depth articular cartilage and attached subchondral bone for histological examination were selected from material removed from lesions during arthroscopic debridement or were obtained at post-mortem examination using a saw. The samples were fixed in paraformaldehyde, decalcified in formic acid and embedded in paraffin, prior to cutting and staining sections with haematoxylin and eosin (H&E) for microscopic examination.

Histological examination was standardised by using the presence, or absence, of specific features to evaluate the lesions. The features, which were identified by review of the literature as being characteristic of osteochondrosis lesions of the LTR in the horse, were:

i) Thickened cartilage (Rejno and Stromberg 1978).

ii) Areas of necrosis in the deeper cartilage zones (Rejno and Stromberg 1978). Chondronecrotic areas were recognised by their pale matrix in H&E sections and shrunken chondrocytes containing pyknotic nuclei or lacking nuclei.

iii) The presence of chondrocyte clusters (Henson and others 1997; Savage and others 1993). Chondrocyte clusters are composed of two or more chondrocytes tightly packed within a single lacuna (Henson and others 1997).

iv) Blood vessels (not necessarily associated with cartilage canals) in cartilage from animals older than 6-7 months (Carlson and others 1995; Shingleton and others 1997).
v) Horizontal fissures at the osteochondral junction, possibly accompanied by myelofibrosis (Rejno and Stromberg 1978).

vi) The accumulation of large numbers of randomly arranged small, rounded chondrocytes in the deeper cartilage zones (Henson and others 1997).

vii) An increase in mineralised cartilage and subchondral bone basophilia in H&E stained sections (Henson and others 1997).

No animals with additional lesions within the femoropatellar joint, including generalised pathology suggestive of osteoarthritis, were included in the study.

Results

Radiographic Examination

A concave defect in the subchondral bone of the LTR containing a number of mineralised bodies was identified in the radiographs obtained of the left and right femoropatellar joints of animals 1 and 2 (a lateromedial radiographic view of right stifle is shown in Figure 1(A)). Radiography was not performed in animals 3 and 4.

Arthroscopic Examination

Synovial membrane hyperaemia and thickening were found in the three femoropatellar joints examined arthroscopically (left and right joints in animal 1, left joint in animal 2). Epiphyseal abnormalities were restricted to the proximal half of the LTR. In animal 1, a cartilaginous flap in the left LTR and an area of wrinkled and poorly attached cartilage on the right LTR were identified. Probing a vertical fissure in the surface of the left LTR of animal 2 elevated a cartilaginous flap. A small cartilage nodule was visualised on the surface of the patella opposing the fissure.
Post-mortem Examination

The major abnormalities of the distal femoral epiphysis identified at gross post-mortem examination of the left and right femoropatellar joints of animal 3 and 4 and the right femoropatellar joint of animals 2 and 4 were localised to the proximal LTR. The cartilaginous flap found in the right femoropatellar joint of animal 2 was in a similar position to the fissure visualized arthroscopically in the left joint (Figure 1(B)). A small cartilage nodule was present on the opposing articular surface of the patella. A small dimple, with a cartilage nodule at its base, was identified in the articular surface of the left LTR of animal 3. A focal increase in cartilage thickness of the lateral trochlear ridge of the distal right femur of animals 3 and 4 was identified when the ridge was cut in the sagittal plane - Figure 1(C) shows this feature in animal 3.

The left femoropatellar joint of animal 4 was normal at post-mortem examination.

Histological Examination

The histological features of the lesions are summarised in Table 2 and illustrated in Figure 2. Areas of thickened cartilage were found in all seven lesions. The next most common histological features were chondronecrosis and chondrocyte clusters, both of which were identified in the deep zone cartilage of five lesions. Figure 2(B) shows an area typical of the appearance of chondronecrosis. This figure also illustrates a horizontal fissure (marked by an asterisk); a feature found in four lesions and accompanied by myelofibrosis (star) in three of the four.

Figures 2(C) and (D) depict the appearance of chondrocyte clusters. Figure 2(C) is a low magnification photomicrograph in which a large number of clusters (cartilage adjacent to cross) are visible. A higher magnification photomicrograph (Figure 2(D)) shows more clearly the arrangement of multiple chondrocytes, identified by their nuclei, within a single lacuna.

Retained blood vessels were present in the deep zone cartilage of four lesions. In the example shown, numerous vascular elements are located near to an area of myelofibrosis at the 6 °/clock position and a fissure running from 6 to 8 °/clock (Figure 2(E)).
Areas of deep zone cartilage in which the normal columnar arrangement of chondrocytes was replaced by a randomly distribution of small, rounded chondrocytes were found in three lesions (Figure 2(F)). Figure 2(A) shows the usual arrangement of chondrocytes from an adjacent area of histologically normal cartilage.

An abnormal pattern of staining was not identified in any of the lesions.

Two to six features were present in each lesion (Table 2). Although, there was no clear association between histological features, local myelofibrosis accompanied horizontal fissures at the osteochondral junction in three lesions (representing three animals) of the four in which fissures were identified.

**Discussion**

The signalment of the four animals described in this study and the clinical, radiological, arthroscopic and pathological findings associated with their LTR lesions are strikingly similar to osteochondrosis of the LTR in the horse. The animals which exhibited clinical signs were young (all <15 months old) when the lesions were identified. For comparison, 90/161 (55.9%) horses with femoropatellar joint OC in one clinical case series (Foland and others 1992) and 8/15 (53.3%) horses with lesions only involving the LTR in another (McIlwraith and Martin 1985) were <24 months old.

Although the lameness and femoropatellar joint effusion exhibited by animals 1 and 2 could have been caused by a number of joint diseases, a bilateral presentation is common for osteochondrosis of the LTR in the horse (McIlwraith and Martin 1985; McIntosh and McIlwraith 1993) and unusual for other conditions. Osteochondrosis of the LTR in the horse is not invariably accompanied by clinical signs (McIntosh and McIlwraith 1993), as was the case in animals 3 and 4 in this study.

While there were no marked differences in signalment between animals 3 and 4, and animals 1 and 2, the gross appearance of their lesions was different. The articular surface of the LTR of animals 3 and 4 was intact, although irregular in the case of the left LTR of animal 3, whereas the
articul surface had been breached in animals 1 and 2. Disruption of the articular surface may have resulted in the release of bone particles into the joint cavity, thereby initiating inflammatory responses by the synovium and articular cartilage (May and others 1992) which were manifest clinically as lameness and joint effusion.

The subchondral defect identified by radiography and the cartilaginous flaps and fissure identified at arthroscopy in animals 1 and 2 are characteristic of osteochondrosis of the LTR in the horse (McIlwraith and Martin 1985), as are the increase in cartilage thickness (McIlwraith and Martin 1985; Rejno and Stromberg 1978) in animals 3 and 4 and the cartilage dimple in animal 3 (Dabareiner and others 1993; McIlwraith and Martin 1985) which were seen at post-mortem examination. In all four animals the lesions involved the proximal half of the LTR, which is a recognised predilection site for osteochondrosis in the horse (McIlwraith and Martin 1985).

Of the histological features associated with osteochondrosis of the LTR in the horse, at least two were identified in each of the seven lesions examined in this study. While an increase in cartilage thickness was the only histological feature consistently present, chondronecrotic areas and chondrocyte clusters were present in 5/7 lesions, fissures at the osteochondral junction and blood vessel retention within cartilage in 4/7 lesions and small, randomly arranged, rounded chondrocytes in 3/7 lesions. A similar frequency of histological abnormalities was found in osteochondrosis lesions of the LTR in a group of horses of mixed breeds (Henson and others 1997), providing further evidence that the LTR lesions in the animals in this study were representative of osteochondrosis.

In the absence of published studies of the development of the LTR in the pony, data from studies performed in the horse were used as a guide for the upper age limit for normal vascularisation of cartilage of the proximal lateral trochlear ridge of the distal femur cartilage in the pony. The upper limit for the horse of 6 months old proposed by Shingleton and others (1997) and 7 months old proposed by Carlson and others (1995) was exceeded by at least 5 months by three animals in this study, suggesting that the blood vessels found within the cartilage of three LTR lesions from these animals were pathological features.
The lack of clear evidence of correlation between histological features in the lesions examined in this study appears to contrast with osteochondrosis lesions of the LTR in the horse, in which chondrocyte clusters are frequently found at the periphery of necrotic areas (Henson and others 1997). However, greater numbers of LTR lesions in ponies would need to be examined to confirm this difference. No association was found between retained blood vessels and areas of chondronecrosis, which is in agreement with the observations of Henson et al. (1997) but not those of Carlson et al (1995) in the horse. Given the likely involvement of disruption of cartilage canal blood supply in the pathogenesis of osteochondrosis (Olstad and others 2008) however, further investigation of this aspect of the histology of LTR lesions in ponies is warranted.

Increased basophilia of mineralised cartilage or of subchondral bone was not observed in any of the sections stained with H&E. This may indicate that pony LTR lesions all belonged to only one of the two histologically distinct types of lateral trochlear ridge of the distal femur osteochondrosis lesions proposed for the horse (Henson and others 1997). However, type VI collagen immunocytochemistry would be necessary to confirm this classification (Henson and others 1997). Therefore, whether the lateral trochlear ridge of the distal femur lesions are generally of one histological type and, perhaps more importantly, whether this has any implications for the pathogenesis of osteochondrosis lesions of the LTR in ponies when compared to horses, remains to be seen.

Two of the animals described in this study (animals 3 and 4) were a sample from 43 ponies which were subjected to post-mortem examination at the Department of Clinical Veterinary Medicine, University of Cambridge over the 6 year period from October 1992 to October 1998. Although the nature of this population makes comparison with other studies difficult, the prevalence of osteochondrosis of the LTR in these ponies (4.7%) was lower than might be anticipated from the results of radiographic surveys of Thoroughbred yearlings presented for sale (Kane and others 2003) and of Warmbloods (Dik and others 1999). The absence of published reports of osteochondrosis lesions of the LTR in the pony compared to the numerous reports in the horse also suggests that the prevalence of osteochondrosis of the LTR in the pony is lower than in the horse. This implies that the pony is either less susceptible than the horse to osteochondrosis of the LTR or that differences in husbandry reduce exposure of the pony to, or
protect the pony from, aetiological factors for the disease. Further knowledge of these two possibilities could provide new insights into the aetiopathogenesis of osteochondrosis in equids.

The role of genetic factors in susceptibility to osteochondrosis is evident from the large breed-to-breed variations in the prevalence of osteochondrosis related to selection for rapid growth that occur in the pig (Reiland and others 1978b) and broiler chicken (Reiland and others 1978a), and in horses from the findings of studies performed in Standardbreds (Grondahl and Dolvik 1993; Philipsson and others 1993; Schougaard and others 1990) and Warmbloods (van Grevenhof and others 2009). It seems likely that genetic factors also influence the prevalence of osteochondrosis in the pony; the differences in growth rate between the pony and horse (Green 1969, 1976; Hintz and others 1979; Jordan 1977) may reflect a role for the genetic determinants of growth rate in the susceptibility of the horse and pony to osteochondrosis.

This work is the first full description of osteochondrosis of the LTR in the pony: the signalment, physical signs, radiological signs and gross and histological features of the LTR lesions in the ponies were consistent with osteochondrosis lesions at the same site in the horse. The use of clinical, gross pathological and histological criteria to diagnose osteochondrosis was important in overcoming the lack of specificity of individual criteria, such as histological features (Pool 1993), and thus in defining the lesions as osteochondrosis (Douglas 2003).

While the limitations of this study made it impossible to address the question of the relative susceptibility of ponies and horses to LTR osteochondrosis, it is evident that the pony cannot be regarded as wholly insusceptible. Investigation of the basis for any differences in susceptibility of ponies and horses to osteochondrosis of the LTR, for instance, through comparing aspects of metabolism of the articular-epiphyseal growth cartilage complex, might provide insight into the pathogenesis of osteochondrosis.

Acknowledgements

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Table and figure legends

Table 1. Summary of the signalment and clinical signs of four ponies with lesions of the lateral trochlear ridge of the distal femur and gross appearance of the lesions

Table 2. Analysis of the histological features of lesions of the lateral trochlear ridge of the distal femur in four ponies

Figure 1. Radiographic and gross appearance of lesions of the lateral trochlear ridge (LTR) of the distal femur in ponies
(A) Lateromedial radiographic view of right stifle of animal 2. Arrows indicate margin of concave defect in contour of subchondral bone of LTR. A number of indistinct mineralised bodies are visible within the defect.
(B) Gross appearance of right LTR of animal 2 at post-mortem examination. Proximal is to the top. Arrows outline raised cartilaginous flap centred on the apex of the proximal LTR.
(C) Gross sagittal section of lesion of right LTR of animal 3 showing focal increase in cartilage thickness (arrows). Subchondral bone is labelled with an asterisk.

Figure 2. Histological features of lesions of the lateral trochlear ridge of ponies.
Photomicrographs are orientated so that the articular surface is to the top.
(A) Unaffected area deep zone cartilage of the right LTR of animal 1 showing the usual arrangement of chondrocytes in this zone: lacunae containing single chondrocytes are in short columns (arrows) that are orientated perpendicular to the articular surface.
(B) Lesion of the left LTR of animal 3 showing a focal area of chondronecrosis (arrows) within the deep zone of cartilage. The necrotic cartilage appears as an area of pale staining matrix and chondrocyte loss. An area of myelofibrosis (star) is situated deep to a horizontal fissure (asterisk).
(C) Lesion of the right LTR of animal 2. Numerous chondrocyte clusters (cross), composed of variable numbers of cells, are present in the deep zone of cartilage adjacent to a horizontal fissure (asterisk).
(D) Higher magnification field from (B). Two chondrocyte clusters composed of a large number of cells are arrowed.
(E) Lesion of the left LTR of animal 3 showing a large number of vascular elements (arrows) within the cartilage deep zone.
(F) Lesion of the right LTR of animal 1 with small, rounded, randomly arranged chondrocytes in the deep zone of cartilage.
Key: LTR = lateral trochlear ridge of the distal femur
<table>
<thead>
<tr>
<th>Animal</th>
<th>Breed</th>
<th>Age (months)</th>
<th>Sex</th>
<th>Activity</th>
<th>Lameness</th>
<th>FP joint effusion</th>
<th>Gross appearance of lesion</th>
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<td>1</td>
<td>Pony</td>
<td>14</td>
<td>G</td>
<td>Showing</td>
<td>Bilateral</td>
<td>Bilateral</td>
<td>L: cartilaginous flap proximal LTR(^1) R: wrinkled cartilage proximal LTR(^1)</td>
</tr>
<tr>
<td>2</td>
<td>(\frac{3}{4}) pony x (\frac{1}{4}) TB</td>
<td>6</td>
<td>G</td>
<td>Showing</td>
<td>Bilateral</td>
<td>Bilateral</td>
<td>L: fissure proximal LTR(^1) R: cartilaginous flap proximal LTR(^2)</td>
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<td>3</td>
<td>Pony</td>
<td>12</td>
<td>G</td>
<td>Experimental</td>
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<td>None</td>
<td>L: dimple containing cartilage nodule at base(^2) R: surface normal; focal increase in cartilage thickness seen in sagittal section(^2)</td>
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<tr>
<td>4</td>
<td>Pony</td>
<td>15</td>
<td>G</td>
<td>Experimental</td>
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<td>None</td>
<td>L: no abnormalities detected(^2) R: surface normal; focal increase in cartilage thickness seen in sagittal section(^2)</td>
</tr>
</tbody>
</table>

**Key:** TB = Thoroughbred; G = gelding; FP = femoropatellar; LTR = lateral trochlear ridge of the distal femur; L = left; R = right; \(^1\)= appearance at arthroscopy; \(^2\)=appearance at *post-mortem* examination
Table 2

<table>
<thead>
<tr>
<th>Histological feature</th>
<th>Animal 1</th>
<th>Animal 2</th>
<th>Animal 3</th>
<th>Animal 4</th>
<th>Summary</th>
</tr>
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<td>R LTR</td>
<td>L LTR</td>
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<td>Thickened cartilage</td>
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<td>+</td>
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<td>+</td>
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<td>+</td>
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<td>+</td>
<td>+</td>
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<td>+</td>
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<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Small, rounded, randomly distributed chondrocytes</td>
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<td>-</td>
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</tr>
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<td>Abnormal staining</td>
<td>-</td>
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</table>

**Key:** L = left; R = right; LTR = lateral trochlear ridge of the distal femur; +/- = histological feature present/absent.