

Histopathology in horses with chronic palmar foot pain and age-matched controls. Part 1: Navicular bone and related structures

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Keywords: horse; foot pain; navicular disease; distal sesamoidean impar ligament; collateral sesamoidean impar ligament; navicular bursa; histopathology

Summary

Reasons for performing study: Causes of palmar foot pain and the aetiopathogenesis of navicular disease remain poorly understood, despite the high incidence of foot-related lameness.

Hypotheses: Abnormalities of the collateral sesamoidean ligaments (CSLs), distal sesamoidean impar ligament (DSIL), deep digital flexor tendon (DDFT), navicular bone, navicular bursa, distal interphalangeal (DIP) joint or collateral ligaments (CLs) of the DIP joint may contribute to palmar foot pain.

Methods: Feet were selected from horses with a history of unilateral or bilateral forelimb lameness of at least 2 months' duration that was improved by perineural analgesia of the palmar digital nerves, immediately proximal to the cartilages of the foot (*Group 1*, n = 32); or from age-matched control horses (*Group 2*, n = 19) that were humanely destroyed for other reasons and had no history of forelimb foot pain. Eight units of tissue were collected for histology: the palmar half of the articular surface of the distal phalanx, including the insertions of the DDFT and DSIL; navicular bone and insertion of the CSLs; DDFT from the level of the proximal interphalangeal (PIP) joint to 5 mm proximal to its insertion; synovial membrane from the palmar pouch of the DIP joint and the navicular bursa; CLs of the DIP joint and DSIL. The severity of histological lesions for each site were graded. Results were compared between *Groups 1* and *2*.

Results: There was no relationship between age and grade of histological abnormality. There were significant histological differences between groups for lesions of the flexor aspect, proximal and distal borders, and medulla of the navicular bone; the DSIL and its insertion and the navicular bursa; but not for lesions of the CSLs, the dorsal aspect of the navicular bone, distal phalanx and articular cartilage, synovium or CLs of the DIP joint.

Conclusions: Pathological abnormalities in lame horses often involved not only the navicular bone, but also the DSIL and navicular bursa. Abnormalities of the navicular bone medulla were generally only seen dorsal to lesions of the FFC.

Potential relevance: Adaptive and reactive change may be occurring in the navicular apparatus in all horses to variable degrees and determination of the pathogenesis of lesions that lead to pain and biomechanical dysfunction should assist specific preventative or treatment protocols.

Introduction

Palmar foot pain is a common cause of forelimb lameness in the horse, but there are only a limited number of in-depth histopathological investigations (Wilkinson 1953; Doige and Hoffer 1983; Poulos 1983; Svalastoga *et al.* 1983; Pool *et al.* 1989; Poulos *et al.* 1989; Wright *et al.* 1998; Bowker 2003), the majority of which have focused on the navicular bone in horses with radiographic abnormalities consistent with navicular disease. It is well recognised that, in association with advanced navicular disease, fibrillation of the opposing dorsal aspect of the deep digital flexor tendon (DDFT), with or without adhesion formation between the tendon and the navicular bone, are common features. Pathological abnormalities of the navicular bursa (Svalastoga and Nielsen 1983; Wright *et al.* 1998) and the origin of the distal sesamoidean impar ligament (DSIL) (Poulos *et al.* 1989; Wright *et al.* 1998) have been described in conjunction with navicular disease and in some age-matched control horses (Wright *et al.* 1998). The latter authors also examined the collateral sesamoidean ligaments (CSLs), but found no abnormalities in either age-matched control horses or those with navicular disease. Pool *et al.* (1989) described degenerative changes of the distal interphalangeal (DIP) joint in conjunction with navicular disease.

None of the previously published studies of lame horses described histology of the insertional region of the DSIL or the collateral ligaments (CLs) of the DIP joint, although pain associated with these regions may contribute to palmar foot pain.

In publications referring to the clinical aspects of foot-related lameness, there has been considerable confusion over definition of the term 'navicular syndrome'. Some authors (Colahan 1994; Field *et al.* 1995) have used it synonymously with any cause of pain in the palmar aspect of the foot, whereas others (Pool *et al.* 1989; Turner 2001) have used it to imply pain associated with the navicular bone prior to the development of radiological abnormalities.

It was hypothesised that abnormalities of the CSLs, DSIL, DDFT, navicular bone, navicular bursa, DIP joint or CLs of the DIP joint may contribute to palmar foot pain; that ageing degenerative changes may be seen in horses free from lameness; and that horses with lameness were likely to have a greater severity of abnormalities than age-matched horses with no history of foot pain.

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[Paper received for publication 24.11.04; Accepted 04.05.05]

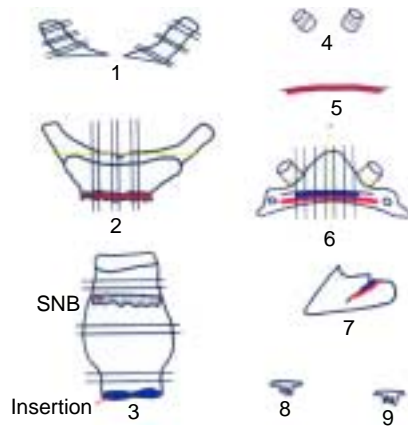


Fig 1: Diagram to show the sites (black lines) for collection of specimens for histopathological examination. 1 = collateral sesamoidean ligaments (CSLs); 2 = navicular bone, distal sesamoidean impar ligament (DSIL) and CSLs (yellow line denotes where CSLs detached); 3 = deep digital flexor tendon (DDFT) (blue) with synovium from navicular bursa (SNB); 4 = collateral ligaments (CLs) of the distal interphalangeal (DIP) joint; 5 = DSIL (red); 6 = distal phalanx, CLs of DIP joint and insertions of DDFT and DSIL; 7 = midline section of distal phalanx with insertions of DDFT and DSIL; 8 = synovium from navicular bursa; 9 = synovium from DIP joint.

The purposes of this study were to describe and compare histopathological abnormalities in horses with no history of and those with chronic palmar foot pain. Comparison in the DDFT is described elsewhere in this issue (Blunden *et al.* 2005).

Materials and methods

Feet were selected from horses with a history of unilateral or bilateral forelimb lameness of at least 2 months' duration, improved by perineural analgesia of the palmar digital nerves immediately proximal to the cartilages of the foot (*Group 1*); or from age-matched control horses (*Group 2*) that were humanely destroyed for other reasons and had no history of forelimb foot pain.

Fifty-one front feet in *Groups 1* (n = 32; 15 horses bilateral and 2 unilateral) and 2 (n = 19) were collected within 6 h of death from horses age 4–15 years (mean 8.3 years) and 5–15 years (mean 7.4 years), respectively. The feet were stored at -20°C and thawed for radiographic and magnetic resonance imaging (MRI) examinations (Murray *et al.* 2006a,b); they were dissected immediately afterwards and macroscopic changes recorded. Eight units of tissue were collected for histology (Fig 1): the palmar half of the articular surface of the distal phalanx, including the insertions of the DDFT and DSIL; navicular bone and insertion of the CSLs; CSLs; synovial membrane from the proximal palmar pouch of the DIP joint; synovial membrane from the proximal recess of the navicular bursa; CLs of the DIP joint and DSIL; and DDFT (Blunden *et al.* 2005). Eight sagittal plane cuts were made through the distal phalanx including the insertions of the DDFT and DSIL, and 3 cuts made through the navicular bone including the axial insertion of the CSL and the origin of the DSIL. Three equally spaced transverse cuts were made through each CSL, and one transverse cut through the DSIL and each CL of the DIP joint. Three transverse cuts were made through the DDFT immediately proximal to the navicular bursa, at the level of the middle third of the bursa and close to the tendon's insertion.

The samples were fixed in 10% neutral buffered formalin. Ligament samples were softened in 4% phenol in 70% alcohol for

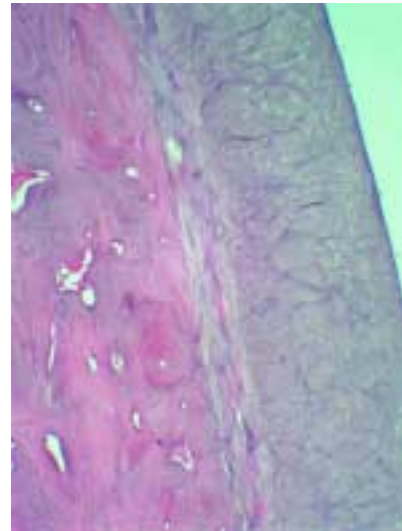


Fig 2: Palmar aspect of a normal navicular bone (palmar to the right). The palmar surface is smooth and there is clear demarcation between the uncalcified and calcified layers of the flexor fibrocartilage and subchondral bone. H&E; magnification x100.

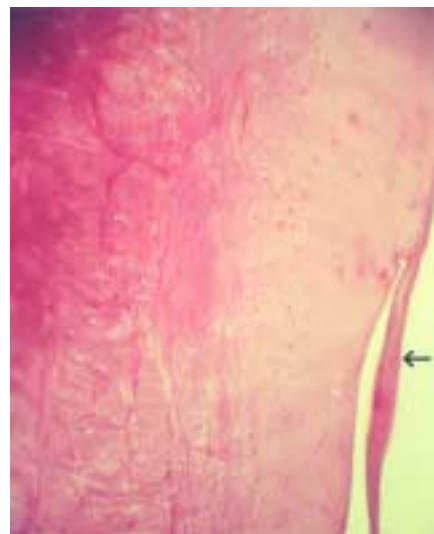


Fig 3: Mild (grade 1) abnormalities of the flexor aspect of a section from the distal half of a navicular bone (palmar to the right). There is surface fibrillation of the fibrocartilage (arrow), loss of cellularity in the superficial aspect of the fibrocartilage, and the matrix is more amorphous in appearance compared with Figure 2. H&E; magnification x100.

1–5 days, depending on the hardness of the tissue. Bone specimens were decalcified in rapid decalcifying solution¹ for 24–48 h. Tissues were embedded in paraffin wax and sectioned at ~5 µm for tendon/ligament and ~7 µm for bone, using heavy duty rotary and base sledge microtomes. Sections were stained routinely with Harris's haematoxylin and eosin (H&E).

All sections were examined by a pathologist (A.B.) who was unaware of each foot's group allocation. For each anatomical site examined, the histological findings were scored as: 0 = no changes, 1 = mild, 2 = moderate or 3 = severe abnormalities. Histological grading was compared between groups using a Mann Whitney U test, and a Kendall Rank Correlation was used to test associations between age and grade and for associations in histological grade between structures. In order to check the variations that might pertain to the process of

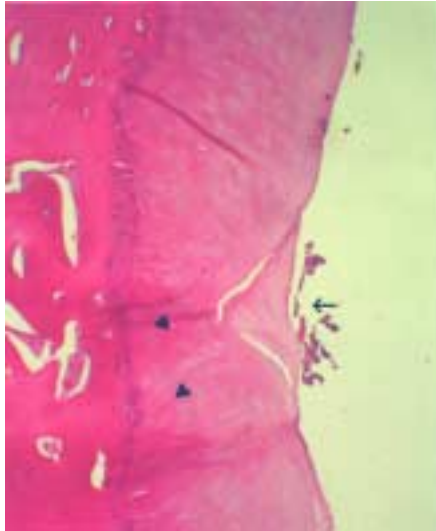


Fig 4: Moderate (grade 2) abnormalities of the flexor aspect of a navicular bone (palmar to the right). There is a central defect (arrow) with a V-shaped area of degenerate fibrocartilage, surrounded by an area of low cell density, with large chondrocytes (arrowhead) and some chondrones. H&E; magnification x20.

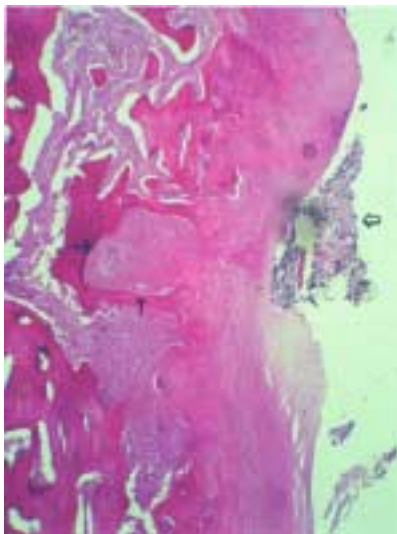


Fig 5: Severe (grade 3) abnormalities of the flexor aspect of a section from the distal half of a navicular bone (palmar is to the right). There is focal complete loss of fibrocartilage, with subchondral bone necrosis dorsally and areas of fibrosis (black arrows). Bone dust (open arrow) is a processing artefact adjacent to the lesion. H&E; magnification x20.

scoring, results were recorded according to a standard protocol; tissue sections from standard sites were examined in a standard manner on recording forms, the observer being unaware of the allocation as to whether the tissues belonged to *Group 1* or *2*. The histological criteria were set out before histopathological examinations were commenced and then histological gradings were made at the end of the process. The process was performed twice and this showed minimal variation, demonstrating the repeatability of the method.

Results

There was no relationship between age and grade of histological abnormality in any of the structures in either *Group 1* or *2*.

TABLE 1: No. feet with each histological grade according to tissue site and compartment

Structure	<i>Group 2</i> (n = 19)				<i>Group 1</i> (n = 32)			
	0	1	2	3	0	1	2	3
Navicular bone								
Fibrocartilage (flexor surface)	1	14	4	0	3	7	10	12
Distal border	15	4	0	0	3	16	7	6
Hyaline cartilage (dorsal border)	0	13	6	0	7	21	4	0
Proximal border	18	1	0	0	21	9	2	0
Medulla	16	3	0	0	19	11	2	0
DSIL								
Ligament [†]	8	6	3	0	2	11	5	2
DSIL insertion	19	0	0	0	24	8	0	0
CSL								
Lateral	12	5	2	0	18	13	1	0
Medial	9	9	1	0	26	6	0	0
Navicular bursa								
Synovium	17	2	0	0	18	12	2	0
DIP joint								
Distal phalanx articular cartilage	4	12	3	0	5	24	2	1
Synovium	16	2	1	0	27	5	0	0
Collateral ligament of DIP joint								
Lateral [‡]	7	8	0	1	8	9	2	1
Medial [§]	6	8	0	1	13	5	0	2

Group 1 = lame horses; *Group 2* = age-matched control horses. 0 = no significant changes; 1 = mild; 2 = moderate; 3 = severe. CSL = collateral sesamoidean ligament; DSIL = distal sesamoidean impar ligament; DIP = distal interphalangeal. [†]*Group 2*, n = 17 and *Group 1*, n = 20; [‡]Lateral: *Group 2*, n = 16; *Group 1*, n = 20; [§]Medial: *Group 2*, n = 15; *Group 1*, n = 20.

However, there were significant histological differences between groups. Results of the histological grading of feet from *Groups 1* and *2* are summarised in Table 1. Results of macroscopic examinations are reported elsewhere (Schramme *et al.* 2005).

Navicular bone

Flexor aspect: Only one horse in *Group 2* and 3 horses in *Group 1* had a normal appearance of the flexor fibrocartilage of the navicular bone (Fig 2). Lesions of the flexor aspect of the navicular bone were focal and located mostly in the central region and/or at the junction between the proximal two-thirds and distal one-third of the bone, with both lesional sites often present together. Changes were graded mild in 74% of feet from *Group 2* and 22% from *Group 1* (Fig 3). *Grade 1* changes were characterised by a slight reduction in depth of the flexor fibrocartilage, mild fibrillation and pitting, and the frequent presence of a superficial narrow amorphous hyaline layer (tending to flake away from the surface of the sections and mainly present either towards the distal or proximal border). There was a mild chondrocyte response characterised by an increased number of and plumper chondrocytes, or formation of chondrones. In both groups there was focal loss of cellularity in the superficial zone of the fibrocartilage, especially one-third proximal from the distal border and/or in the central region of the fibrocartilage; these areas were saucer-shaped with an amorphous or poorly cellular matrix. *Grade 2* abnormalities were present in 21% of feet from *Group 2* and 31% from *Group 1* and were characterised by a focal loss of cellularity or partial loss of the fibrocartilage layer between 30–50% of normal depth, sometimes with pitting and crevicing, typically with a prominent chondrocyte response bordering these focal lesions, with frequent chondrones and plump chondrocytes (Fig 4). *Grade 3* change was present in 38% of feet from *Group 1*, but none from

Group 2. There was focal loss of the uncalcified fibrocartilage from 50–100% of the normal depth, with or without loss of the calcified zone, and a marked chondrocyte response, with many chondrones, deep within the fibrocartilage and often involving the calcified zone. Where the calcified zone was disrupted there was also focal subchondral bone loss and replacement by fibrous tissue (Fig 5). Focal subchondral necrosis was present only dorsal to focal areas of cartilage degeneration and was seen in 11 feet, all from *Group 1*. Some of these lesions extended into cancellous bone, associated with mild widening of the intertrabecular spaces, but without intertrabecular fibrosis. Occasionally, there was thinning only of the subchondral bone dorsal to a fibrocartilage lesion, without osteonecrosis. Sections of navicular bone were prepared after adhesions with the DDFT had been cut during dissection and this relationship was therefore not recorded histologically. However, 8 feet from *Group 1* had gross evidence of adhesions between the DDFT and navicular bone.

The frequent mild changes observed in the fibrocartilage layer in *Group 2* (14 feet) indicate normal wear and tear. The flexor aspect of the navicular bone in *Group 1* had a higher histological grade than in *Group 2* ($P < 0.001$).

Distal border with DSIL origin: In *Group 2*, 79% had no abnormality of the distal border of the navicular bone (Fig 6a) and 21% had only *grade 1* changes, with mild fibrocartilaginous metaplasia at the bone-ligament interface. In *Group 1*, 90% had mild to severe lesions of the distal border, of which 41% were *grade 2* or *3*. *Grade 2* changes were characterised by moderate focal fibrocartilaginous metaplasia in the region of the bone-ligament interface. This was sometimes accompanied by moderate enlargement of the synovial fossa, with synovial invagination into the adjacent cortex, forming a discrete synovial-lined cavity surrounded by a narrow zone of fibrovascular tissue, adjacent to intact subchondral bone. *Grade 3* changes were seen in 6 feet from *Group 1* and characterised by large foci of fibrocartilaginous metaplasia and/or entheses bone formation near to the ligament-bone interface (Fig 6b). This was often also accompanied by marked enlargement of the synovial fossa, with synovial invaginations into the underlying cortical and medullary bone, sometimes with localised osteonecrosis, with a zone of loosely arranged fibrovascular tissue surrounding the synovial invagination.

The distal border of the navicular bone in *Group 1* had a higher histological grade than in *Group 2* ($P < 0.0001$).

Medulla: Lesions that extended into the medulla from the distal or flexor borders of the navicular bone (i.e. subchondral bone necrosis adjacent to lesions of the fibrocartilage or distal border) were not categorised as primary medullary lesions within this grading system, but were included as part of the distal or flexor border grade, as histologically the changes were not part of a distinct central medullary lesion. In one foot there was focal central medullary necrosis, with widened intrabecular spaces, contiguous with a subchondral bone lesion dorsal to a fibrocartilage defect, but generally osteonecrosis was restricted to the subchondral location associated with the flexor lesion. Only one horse from *Group 1* had *grade 3* lesions of the medulla, within the definition of a primary medullary change in this study. The interstitial fat showed loss of integrity of cytoplasmic membranes, indicating early necrosis, with a

prominent arrangement of interstitial capillaries. Many bone trabeculae had a 'moth-eaten' appearance, with necrosis of bone edges (Fig 7). In *Group 1*, 44% of navicular bones were *grade 1* or *2*, while in *Group 2*, 85% were *grade 0* and only 15% *grade 1*. In no feet was there evidence of thrombosis of blood vessels within the medulla.

The medulla of the navicular bone in *Group 1* had a strong trend to a higher histological grade than in *Group 2* ($P = 0.059$).

Dorsal hyaline cartilage: The dorsal hyaline cartilage was graded similarly to the fibrocartilage. There were *grade 1* changes in 68% of feet from *Group 2* and 66% from *Group 1*. Lesional sites were localised to the proximal and distal border angles of the hyaline cartilage, corresponding to impact points with the opposing cartilage of the navicular facet of the middle and distal phalanges. *Grade 2* abnormalities were only present in 10 feet, 6 from *Group 2* and 4 from *Group 1*. Severe changes were not seen in either group. There was no significant difference between groups.

Proximal border with CSL insertion: Grading parameters for the proximal and distal borders of the navicular bone were similar. *Grade 1* and *2* lesions of the proximal border of the navicular bone were seen in 31% of feet in *Group 1*, but only 1 of 19 feet in *Group 2* had *grade 1* lesions. In one foot, there was a small proximal fossa synovial invagination. *Grade 3* changes were not recorded in either group. The proximal border of the navicular bone in *Group 1* had a significantly higher histological grade than in *Group 2* ($P = 0.018$).

Distal sesamoidean impar ligament

The DSIL was composed of well-delineated fascicles, containing fibres running longitudinally between the navicular bone and distal phalanx. The fascicles were separated by a well vascularised and plentiful interstitium, containing pockets lined by synovial membrane. In some ligaments these pockets were prominent, suggesting synovial hyperplasia and invagination into the ligament, but these may be anatomical differences rather than reactive change. Subsynovial and perivascular haemosiderin deposition was present in 10 feet, all from *Group 1* (31%), indicating previous haemorrhage (Fig 8). However, no significant synovial inflammatory cell infiltration was observed in any foot. Blood vessels were particularly prominent in the central area of the ligament. Changes were graded on the basis of degree of fibrocartilaginous metaplasia. Mild to moderate fibrocartilaginous metaplasia was seen in 25 feet (47% of feet from *Group 2* and 50% from *Group 1*) and severe changes were noted in only 2 feet from *Group 1* (Fig 9). In feet with *grade 2* or *3* lesions, there was also focal vacuolar degeneration of the collagen, with associated fibroplasia and increased vascularisation in the adjacent tissue. The DSIL in *Group 1* had a significantly higher histological grade than in *Group 2* ($P = 0.0215$).

Insertion of the DSIL

Changes were graded on the basis of the degree of fibrocartilaginous metaplasia. Mild changes consisting of early fibrocartilaginous metaplasia were seen in the DSIL in 25% of feet from *Group 1*, but none from *Group 2*. Histological grading for the DSIL insertion was significantly higher in *Group 1* than *2* ($P = 0.019$).

Collateral sesamoidean ligaments

Grade 1 change of the CSLs was seen commonly in both groups, usually represented by a smooth transitional fibrocartilaginous metaplasia towards one edge of the section of ligament. This was regarded as an adaptive change near to the insertion of the ligament, compared with degenerative change, characterised by focal, irregular fibrocartilaginous metaplasia replacing an area of ligament fascicles where this would not usually be anticipated. *Grade 2* lesions, rare in both groups, had moderate focal fibroplasia and/or fibrocartilaginous metaplasia. There was no difference in histological grade between the groups for the CSLs, but histological changes were more frequent in the lateral than the medial CSL.

Navicular bursa synovium

Grading was based on the degree of inflammatory cell infiltration. There was mild lymphoplasmacytic infiltration beneath the synovial lining with associated synovial hyperplasia and sometimes surface deposition of fibrinous material without a neutrophil response in 14 feet, 38% of feet from *Group 1* and only 10% from *Group 2*, with *grade 2* changes in 2 feet, both *Group 1* (6%). Occasionally, there was focal subsynovial fibroplasia. Focal subsynovial haemosiderin deposition was present in 22% of feet from *Group 1*, but none from *Group 2*. The feet in *Group 1* had a significantly higher histological grade than in *Group 2* ($P = 0.0132$).

Distal phalanx

The hyaline cartilage of the distal phalanx was graded similarly to that of the navicular bone. *Grade 1* changes were seen in the majority of horses from both groups; in 63% from *Group 2* and 75% from *Group 1*, with *grade 2* lesions in 16% and 6% of feet, respectively. *Grade 3* lesions were present in only one horse from *Group 1*. Lesions were most frequent at the angle of the navicular facet and, sometimes, within the more palmar edge of the cartilage near to the insertion of the DSIL. Occasionally, defects were present just dorsal to the angle of the navicular facet of the distal phalanx. No specific changes were noted in the subchondral bone. There was no significant difference in grade between *Groups 1* and *2*.

Distal interphalangeal joint synovium

In most feet there was no significant inflammation of the synovium. In 7 feet (16% of feet from *Group 1* and 11% from *Group 2*) there was a mild subsynovial lymphocytic inflammatory infiltration and occasional focal haemosiderin deposition. One foot from *Group 1* had moderate synovitis. No feet had severe synovitis. There were no significant differences between groups.

Collateral ligaments of the DIP joint

Changes of the CLs of the DIP joint were graded on the basis of fibrocartilaginous metaplasia within the ligament, which was the only significant change observed. The most marked change consisted of multiple fissuring within fibrocartilaginous areas, together with reactive chondrone formation. Two feet out of 16 sampled had *grade 3* lesions in the lateral CL, one in each

group; and 3 out of 15 feet sampled (2 in *Group 1* and one in *Group 2*) had severe changes in the medial CL. One foot had an epidermal inclusion cyst in the medial CL. This unusual finding may represent a congenital anomaly. *Grade 1* changes, consisting of a transitional fibrocartilaginous metaplasia towards one edge of the section, were frequently seen and considered to be adaptive, probably associated with insertion of the ligament. There was no difference in histological grade between *Groups 1* and *2*.

Correlation between structures

There was a positive correlation between the grading for the distal ($P = 0.04$) and proximal ($P = 0.04$) aspects of the navicular bone. There was a trend towards an association between the grades for the proximal aspect of the navicular bone and the CSL ($P = 0.06$). No significant associations in histological grade were detected between the DSIL and distal aspect of the navicular bone, or for the DSIL and DSIL insertion on the distal phalanx. There was also no association between the grades for the navicular bursa and the flexor aspect of the navicular bone. Associations between the DDFT and other structures are described by Blunden *et al.* (2005).

Discussion

The results of this study support the hypothesis that horses with foot-related lameness were likely to have a greater severity of abnormalities in a variety of anatomical structures than age-matched horses with no history of foot pain. However, contrary to our hypothesis, there were no differences in histological grade in the DIP joint or the CSLs. Age-related degenerative changes were not identified. However, the study was biased by case selection, with a significant proportion of horses having relatively advanced radiological abnormalities of the navicular bone and not representative of all horses with palmar foot pain, in which a greater variety of pathological abnormalities of the soft tissue structures of the foot have been identified using MRI (Dyson *et al.* 2004). This study also gives fairly limited information about the early stages of navicular disease. The age range of horses in *Group 2*, lacking immature horses, may have been too small to demonstrate age-related degenerative changes.

Navicular bone

There is currently no consensus on the pathogenesis of pathological lesions of the navicular bone, although current theories tend to support a biomechanical aetiology, rather than vascular abnormalities (Pool *et al.* 1989; Wright and Douglas 1993). This is supported by the current study, in which there was no evidence of generalised ischaemic bone disease leading to erosions of the fibrocartilage as described previously (Olsson 1954; Colles 1979). In this study, subchondral bone necrosis occurred only in association with deep and focal ulceration of the fibrocartilage. It is possible that the lesions of the fibrocartilage are occurring concurrently with degeneration of the opposing surface of the DDFT or that the DDFT lesions are primary in the aetiopathogenesis.

Radiological diagnosis of navicular disease has been based on identification of an increased number of radiolucent zones along the distal borders of the navicular bone, of abnormal shape and size, and solitary lucent zones within the medulla, with or without penetration through the flexor cortex or modelling of the bone



Fig 6a: Junction of a normal navicular bone (top) and distal sesamoidean impar ligament. There is a well demarcated bone-ligament interface. Note the regular orientation of the ligament fascicles. Compare with Figure 6b. H&E; magnification x20.

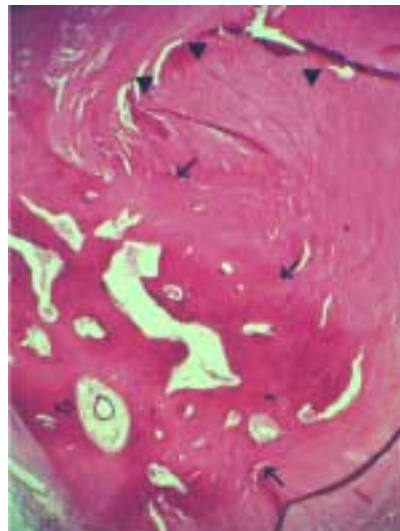


Fig 6b: Junction of the navicular bone (top) and distal sesamoidean impar ligament (DSIL). There is a large enthesophyte (outlined by black arrows), within which are marrow spaces enclosing vessels (open arrow). There is disorganisation of the fibre orientation within the DSIL (arrowheads). H&E; magnification x20.

margins (Butler *et al.* 2000). In a recent histopathological study of horses with navicular disease, rounded areas of medullary lysis were noted in 4 out of 10 feet (Wright *et al.* 1998) and intertrabecular myxoid stromal change was observed in 6 feet. Distal palmar medullary lesions were identified histologically as pseudocysts and feet without pseudocysts had areas of replacement of intertrabecular marrow by myxoid fibrous tissue. In the current study, no feet had distinct discrete encapsulated cysts; areas of focal subchondral bone loss were replaced by fibrous tissue. In one foot, there was focal central trabecular necrosis with widened intertrabecular spaces contiguous with a subchondral bone lesion dorsal to a fibrocartilage defect. There was no evidence of direct continuity between subchondral bone necrosis dorsal to the fibrocartilage and localised osteonecrosis near the distal synovial invaginations, although the 2 lesions were close in some feet where the subchondral bone lesion occurred one-third proximal from the distal border. Contrary to the findings of Wright *et al.* (1998), there was no evidence of generalised fibromyxoid stromal change within the medullary interstitium.

In one foot, there were unique abnormalities consisting of a generalised nibbling necrosis of the bone trabeculae accompanied by necrosis of the interstitial fat. In this foot there were no fibrocartilage or subchondral bone lesions or synovial invaginations, and the pathogenesis of the lesions may be different to other navicular bone lesions, although there were concurrent sagittal splits in the DDFT. In this study, it was decided not to designate subchondral bone necrosis as a separate medullary lesion if the changes were clearly associated with a focal flexor or distal border lesion. In previous descriptions, it is not clear which anatomical areas are involved when medullary lesions are described. Large foci of fibrocartilaginous metaplasia and/or entheseous bone formation were identified in 6 feet from *Group 1* (distal border fragments). However, histology did not reveal the presence of distal border fragments in 7 other feet, in which these had been previously identified at *post mortem* examination or radiographically. These were located at the junction of the medial or lateral extremity, areas that were not represented in the

parasagittal slices examined axial to these sites. Therefore, a specific study of enthesophytes would require correlation of imaging with precise anatomical-histological site selection.

CSL and DSIL: The significance of entheseous changes within the CSL and DSIL attachments to the navicular bone is controversial. Pool *et al.* (1989) suggested that these were not primary features of navicular syndrome, but may have been a source of pain during their formation. It was concluded that mature enthesophytes were probably asymptomatic and did not contribute to the signs of navicular syndrome. However, Verschooten *et al.* (1989) indicated that modelling of the proximal and distal borders of the bone was part of the navicular disease complex. In the current study, there was a significantly higher histological grading for distal border changes in *Group 1* than in *Group 2*. The positive correlation between distal and proximal navicular bone changes suggests that these changes occur concurrently, but are more marked in the distal border region.

Within the central segment of the DSIL, feet from *Group 1* had significantly higher grade changes than *Group 2*, including fibrocartilaginous metaplasia, but without formation of metaplastic bone. No mature enthesophytes were identified in the insertional area of the DSIL onto the distal phalanx. The degree of change, including fibrocartilaginous metaplasia, was greater within the insertion of the DSIL than of the DDFT. Selection of samples from predetermined sites may have resulted in underestimation of the degree of entheseous change, which could more accurately be assessed by examination of the intact navicular bone and distal phalanx after boiling and cleaning.

Although changes in the DSIL were generally not severe, the importance of any degenerative change in this ligament should not be underestimated because the DSIL contains an extensive network of sensory nerve fibres (Bowker *et al.* 1995; Van Wulfen *et al.* 2002). Therefore, it is possible that relatively low-grade degenerative changes within the ligament might cause significant pain. The development and continuation of pain within the pathogenesis of navicular disease is poorly understood.

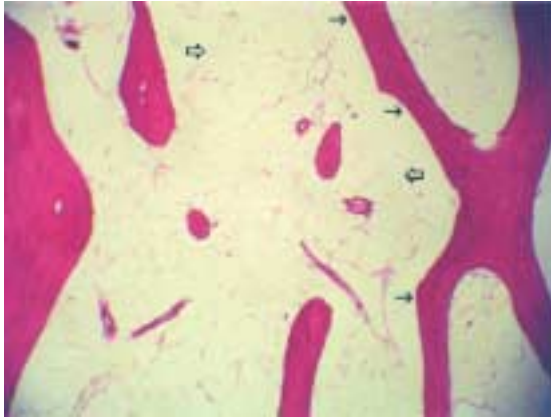


Fig 7a: Normal appearance of the medulla of a navicular bone. The trabeculae are sharply demarcated with smooth edges (black arrows). There is a uniform architecture of the adipose tissue (open arrows). The white spaces between the adipose tissue and trabeculae are processing artefacts. H&E; magnification x200.



Fig 7b: Generalised necrosis of the medulla of a navicular bone, characterised by a 'moth-eaten' appearance of the trabeculae (black arrows) and the loss of uniform architecture of the adipose tissue (open arrows). H&E; magnification x200.

CSLs and CLs of the DIP joint

Transitional fibrocartilaginous metaplasia in the CSL and CLs of the DIP joint was not considered to be of pathological significance, being seen with similar frequency of occurrence in both groups, but was regarded as a local adaptation of the ligament to normal activity. In a few feet there was a more localised fibrocartilaginous metaplasia associated with tissue degeneration, but its clinical significance is unknown. Histological changes were more frequent in the lateral than the medial CSL. Previous radiological studies have demonstrated a higher frequency of occurrence of lateral enthesopathy, which is a common finding in both horses with foot pain and those free from lameness (Kaser-Hotz and Ueltschi 1992). Changes were similar in the medial and lateral CLs of the DIP joint, although all clinical studies to date have demonstrated a greater frequency of desmitis of the medial compared with the lateral CL (Denoix 2000; Turner and Sage 2002; Dyson *et al.* 2004; Dyson and Murray 2004). In our study, the incidence of CL injury was small compared with our clinical experience (Dyson *et al.* 2005), probably reflecting the bias in the current study towards horses with advanced radiological abnormalities of the navicular bone.

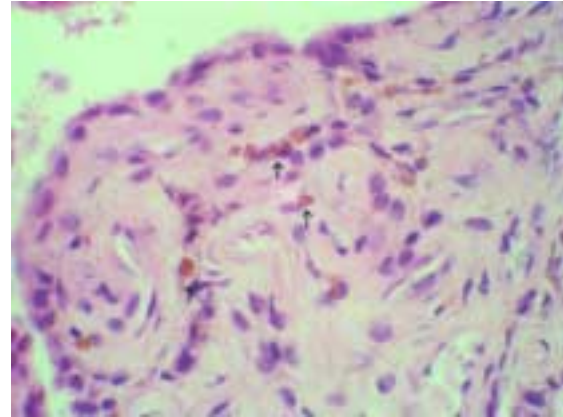


Fig 8: Transverse section of a synovial lined space within the distal sesamoidean impar ligament. There is haemosiderin (arrows) within the synovial tissue. H&E; magnification x400.

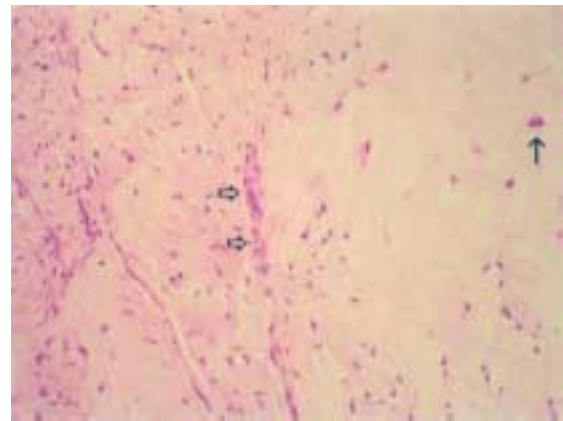


Fig 9: Transverse section of a distal sesamoidean impar ligament midway between the origin and insertion, showing a region of fibroplasia and fibrocartilaginous metaplasia. There is a progression from left to right from moderately cellular fibrous tissue to an area of replacement of ligament fascicles by fibrous tissue (open arrows) and, to the right, an area with loss of cellularity and chondrocytes (black arrow). H&E; magnification x200.

DIP joint and navicular bursa synovium

Examination of synovium from the DIP joint and from the navicular bursa did not reveal evidence of an acute synovitis in any of the feet. The significantly higher histological grade for the navicular bursa synovium in *Group 1* than *Group 2* probably reflects a positive correlation with changes in the superficial dorsal and deep dorsal layers of the DDFT and in the flexor aspect of the navicular bone. Haemosiderosis, indicating previous haemorrhage, in the navicular bursa synovium was detected only in feet from *Group 1*, and may have been associated with previous diagnostic or therapeutic injections of the bursa, or relating to navicular bone and DDFT pathology. Pool *et al.* (1989) found cartilage pathology in the DIP joint in association with navicular disease. In the current study, there were no differences between *Groups 1* and *2*; however, only the palmar aspect of the joint was examined.

Clinical relevance

This study of chronic palmar foot pain has included more extensive histological examinations from larger numbers of

affected feet and has included a wider range of structures than represented in previous studies. Pathological abnormalities in lame horses often involved not only the flexor aspect of the navicular bone, but also the DDFT (Blunden *et al.* 2005), DSIL, CSL and navicular bursa. Investigation of individuals with less advanced navicular disease should assist knowledge of which structures are primarily involved, or whether such changes occur concurrently. However, such specimens are less readily available. On the basis of this study and recent experience with MRI (Dyson *et al.* 2004, 2005; Dyson and Murray 2004), we suggest that the term 'navicular syndrome' should be avoided, because it is now possible to give a more precise diagnosis for the spectrum of injuries that contribute to pain in the palmar aspect of the foot. Further research into the sensory nerve supply to the navicular apparatus and the DDFT may help in understanding what causes pain and, therefore, lameness. We believe that there must be further focus on the interrelationship between the navicular bone and its supporting ligaments and the opposing DDFT. It is postulated that degrees of adaptive and reactive change may be occurring in the navicular apparatus in all horses, but pain results when significant lesions develop as outlined in this report.

Acknowledgements

This study was funded by The Home of Rest for Horses. We thank the many referring veterinary surgeons who provided feet for this study and Ray Wright for invaluable assistance in preparing the histological specimens.

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