RELATIONSHIP BETWEEN HISTOPATHOLOGICAL LESIONS AND MAGNETIC RESONANCE IMAGING (MRI) IN DISEASES OF THE FOOT OF THE HORSE

RELATIONS ENTRE LES LÉSIONS ANATOMOPATHOLOGIQUES ET LES IMAGES DE RÉSONANCE MAGNÉTIQUE (IRM) DANS LES MALADIES DU PIED CHEZ LE CHEVAL

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The increasing number of available pathological studies on the various tissues of the foot has helped validate the significance of Magnetic Resonance Imaging (MRI) signal variations documented with various clinical MRI systems used in equine practice. In particular MRI has helped elucidate the different grades of histological abnormalities and possibly also different stages of disease progression in the deep digital flexor tendon and the navicular bone. However, further work is needed to continue to improve our understanding of the different causes of foot lameness and their pathogenesis.

Key words: MRI, deep digital flexor tendon, navicular bone.

SUMMARY

The number croissant d’études histopathologiques portant sur les tissus du pied a permis d’expliquer et de valider les variations des signaux obtenus par l’imagerie de résonance magnétique (IRM) dans la pratique équine clinique. En particulier, l’IRM a permis d’élucider les différents grades des anomalies histologiques et peut-être aussi les différentes étapes de la progression de la dégénérescence du tendon fléchisseur profond et de l’os naviculaire. Cependant, des travaux supplémentaires sont nécessaires pour continuer d’améliorer nos connaissances sur les différentes causes de boiterie du pied et leur pathogénie.

Mot clés: IRM, tendon fléchisseur profond, os naviculaire.

Diseases of the horse’s foot remain an important concern to the clinician, taking into account that they constitute a major cause of lameness in the horse. A detailed clinical examination and current imaging techniques have long been impeded by the presence of the hoof capsule, leading to the use of vague diagnostic terminology like “podotrochlear syndrome” and “navicular syndrome”, to describe the presence of chronic lameness in the palmar aspect of the foot but without specific anatomopathological definition or localization. More recently the advent of magnetic resonance imaging has filled this void by allowing us a detailed anatomical window into all anatomical structures of the foot. Magnetic resonance imaging (MRI) is now widely used in the diagnosis of equine foot lameness (table 1).

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It has been able to provide diagnostic information not available from other imaging techniques. Understanding of the significance of alterations in signal intensity and patterns relies on the study of pathological changes that occur in the various disease processes that affect different tissues in the foot. The purpose of this paper is to review the current histopathological knowledge on various anatomical structures of the foot and the suitability of MR imaging for their identification.

**NAVICULAR BONE**

The most common type of MR signal abnormality seen in the navicular bones of horses with lameness associated with navicular disease is short tau inversion recovery (STIR) signal hyperintensity in the medullary cavity of the navicular bone with or without additional areas of T2 and PD signal hypointensity (figure 1). Medullary STIR hyperintensity may be focal near the distal border of the navicular bone, or extend from the distal border in a vertical band along the palmar cortex to the proximal border of the bone, or spread diffusely throughout the medullary cavity (Sampson et al. 2009). In horses with chronic navicular disease, abnormal signal hyperintensities at the level of the palmar surface of the navicular bone are equally common as those in the medullary cavity. These can be areas of subtle, focal increase, caused by synovial fluid pooling at a site of early fibrocartilage loss (figure 2a) (Schramme et al. 2009), or more extensive signal increase extending deeper within the cortical bone of the flexor cortex (Sherlock et al. 2008).

Degenerative changes in the palmar fibrocartilage of the navicular bone occur in the distal half of the palmar aspect of the navicular bone, especially centered around the sagittal ridge, and may extend into the subchondral bone (figure 2b). Loss of fibrocartilage in this location is the most common lesion significantly associated with navicular disease and most likely represents the earliest pathology of classic navicular disease (Wright et al. 1998). Fibrocartilage loss from this location remains difficult to identify in vivo, even with the use of MRI (Schramme et al. 2009). Progression of fibrocartilage loss may result in cortical bone erosion in the flexor cortex, and even in osteonecrosis and fibroplasia extending into the spongiosa (Busoni et al. 2005; Schramme et al. 2005). Degenerative change of the spongiosa is generally only seen dorsal to extensive fibrocartilage damage. There may also be oedema, congestion and fibrosis of the marrow stroma within the medullary bone. Clinical experience with MRI in horses with foot pain from classic navicular disease provides support for the progression of lesions as outlined above.

However, there is a small number of horses with diffuse abnormalities of the medulla characterized by ‘bone oedema’ signal (increased signal in fat suppressed images) but no detectable abnormalities of the flexor fibrocartilage or cortex. Postmortem examination has revealed evidence of necrosis of medullary fat cells and active remodeling of medullary trabeculae, with both osteoclastic and osteoblastic activity along trabecular surfaces. In other horses with ‘abnormal medullary fluid signal’, intertrabecular edema and perivascular mononuclear cellular infiltration have been identified (Blunden et al. 2006 a). These lesions most likely have a different aetiopathogenesis than that of classic navicular disease and may be acutely traumatic or inflammatory in origin.

<table>
<thead>
<tr>
<th>Sequence</th>
<th>T2</th>
<th>T1</th>
<th>Proton Density</th>
<th>Inversion Recovery</th>
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</tr>
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<tr>
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<td>grey</td>
<td>grey</td>
</tr>
<tr>
<td>Tendon</td>
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<td>black</td>
<td>black</td>
<td>black</td>
</tr>
<tr>
<td>Ligament</td>
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<td>grey to black</td>
<td>black</td>
<td>black</td>
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<td>white</td>
<td>black</td>
</tr>
<tr>
<td>Fluid</td>
<td>white</td>
<td>dark grey</td>
<td>light grey</td>
<td>white</td>
</tr>
</tbody>
</table>

Table 1: The signal intensity of different tissues in different magnetic resonance contrast weightings.

**Figure 1:** Sagittal short tau inversion recovery (STIR) image of the foot of a horse with lameness that is abolished by anesthesia of the palmar digital nerves. There is marked STIR hyperintensity of cancellous bone in the medullary cavity of the navicular bone (arrow) indicating the presence of abnormal medullary fluid, medullary fibrosis or medullary fat necrosis.
In some other horses, fluid-filled osseous cyst-like lesions have been seen in the distal aspect of the bone, apparently separate from synovial invaginations, and not associated with any detectable abnormality of the flexor aspect of the bone. Such lesions have not yet been characterised histologically and their etiology remains speculative, but recent evidence suggest that their presence is associated with degenerative changes in the impar ligament (Dyson et al. 2010). In recent postmortem studies, osseous fragments associated with a defect in the distal margin of the navicular bone were more common in horses with navicular disease than in age-matched controls (figures 3a and 3b) (Busoni et al. 2005; Schramme et al. 2005; Blunden et al. 2006; Wright et al. 1998). Histologically, distal border fragments have variously been described as avulsion fractures, separate centers of ossification, osseous metaplasia of the impar ligament or synovial osteoma but pathological evidence elucidating their pathogenesis remains elusive. More recently it was shown that the presence of these fragments was associated with varying degrees of histopathological damage of collagen fibers and fibroblasts in the axial third of the impar ligament (Dyson et al. 2010).

**DEEP DIGITAL FLEXOR TENDON (DDFT)**

Tendinopathy of the DDFT in the foot is almost exclusively an MRI diagnosis. Tendon damage is seen as focal signal increase within the normal contour of the hypointense tendon lobes, on both T1- and T2-weighted sequences, variably accompanied by enlargement of the affected lobe. There is a good correlation between the MRI appearance of DDFT lesions and their pathological classification into core lesions, sagittal plane splits, dorsal plane tears, insertional lesions and dorsal abrasions (Busoni et al. 2005; Schramme et al. 2005; Murray et al. 2006; Blunden et al. 2006 b; Blunden et al. 2009).

Core lesions result in focal, circular areas of signal increase in the center or near the dorsal border of the affected lobe, but are completely surrounded by normal ‘black’ tendon signal (figure 4). Histologically they consist of various amounts of collagen necrosis, fibroplasia and fibrocartilagenous metaplasia resulting in loss of normal fascicular architecture. Core necrosis was seen more frequently in horses lame for less than 6 months. In horses with lameness of more than six months’ duration, core lesions consisted predominantly of fibroplasia and/or fibrocartilaginous metaplasia (Blunden et al. 2006 b; Blunden et al. 2009).
Sagittal plane or oblique splits form linear hyperintensities of variable depth arising from the dorsal surface of the tendon and progressing palmarly (figure 5). Histologically, disruption of the deep dorsal layer of the tendon by deep splits extending from the surface can be observed (Murray et al. 2009). Splits propagate mostly along septal lines, with chondrones clustered around fibrillating tissue and around crevices but no evidence of inflammatory cells (Blunden et al. 2006 b).

Insertional lesions are limited to the distal 20 mm of the DDFT, distal to the distal border of the navicular bone, near the tendon’s insertion on the distal phalanx (figure 6). They consist of small core lesions, sagittal plane splits or osseous changes of the insertion site (Blunden et al. 2009).

Severe dorsal border abrasions of the DDFT usually cause signal increase extending from the dorsal surface towards the center of the affected lobe (figure 7). Histopathologically, dorsal DDFT fibrillations, erosions and abrasions consist of longitudinal strips of superficial fiber damage extending the proximodistal length of the navicular bursa. Bundles of fibers torn away from the surface of the tendon have a tendency to curl up proximally in the navicular bursa. Crevices, splits and fibrillations of the dorsal border of the DDFT may be accompanied by either small, discrete or large, circumscribed foci of necrosis (Wright et al. 1998). Chondrocyte clusters can be seen to produce chondroid matrix in areas of degenerative change (Blunden et al. 2009).

Degenerative vascular changes consisting of thrombosis and occlusion of septal arteries and veins are also seen in the distal portion of the DDFT. They were initially described with equal frequency in horses with clinical navicular disease and age-matched control horses (Wright et al. 1998). Although it was suggested that these vascular changes could be age-related (Wright et al. 1998), larger and later pathological studies have found that vascular changes in the DDFT were significantly more common in the DDFT of horses with foot pain (Busoni et al. 2005; Murray et al. 2009; Blunden et al. 2006 b; Blunden et al. 2009). It was therefore proposed that vascular thrombosis and occlusion could result in matrix changes that predispose horses to injury of the distal portion of the DDFT (Blunden et al. 2006 b). As these changes are predominantly seen in the intratendinous septa, there is a strong possibility that they predispose to the development of sagittal splits in the dorsal surface of the tendon along these septal planes. The lack of any histological evidence of hemorrhage or inflammatory cell infiltration in core lesions, splits and abrasions of the distal portion of the DDFT adds further support to the notion that these lesions may be primarily degenerative in nature (Busoni et al. 2005; Blunden et al. 2009).

MR signal intensity varies between different echo sequences not only with severity but also with duration of a tendon lesion. Acute lesions generally have higher T2 signal intensity due to the presence of fluid and increased cellularity, whereas fibrous scar tissue in chronic lesions produces a more intermediate to low T2 signal intensity. It may therefore be possible to use T1-to-T2 signal differences to estimate the age or stage of healing of a tendon lesion (Schramme et al. 2010). In the chronic stages of healing by fibroplasia, signal intensity in core lesions generally decreases in T2.
and STIR images but can remain high in T1-weighted sequences. It was suggested that T2-weighted imaging may be more sensitive for detecting the transition of blood-rich immature granulation tissue to fibrous scar tissue (Fujikawa et al. 2007).

**NAVICULAR BURSA**

Villous hypertrophy, hyperplasia of synovial cells and venous congestion have been described in the navicular bursa of horses with navicular disease (Murray et al. 2009; Blunden et al. 2006a; Svalastoga & Nielsen, 1983). There was a positive association between histological abnormalities of the bursa and lesions of either the dorsal aspect of the DDFT or the flexor aspect of the navicular bone.

**DISTAL SESAMOIDIAN IMPAR LIGAMENT (DSIL)**

Ageing changes have been described in the impar ligament, characterised by change in fibroblast shape and increased proteoglycan content (Bowker et al. 2001). Evidence of inflammation was also seen at the intersection of the DSIL and DDFT in horses with navicular syndrome (Bowker 2003). The functional significance of these findings remains unknown. Using MRI, injury to the impar ligament is generally seen as part of an injury complex that includes other components of the navicular apparatus (Dyson & Murray, 2007). One histopathological study limited the histological grading of impar ligament abnormality to the presence and extent of fibrocartilaginous metaplasia, that was more extensive in diseased than control limbs (Blunden et al. 2006a). In a more recent report, the histological changes in the mid body of the impar ligament included degeneration of collagen, loss of fibroblasts, fibrocartilage metaplasia and reduction in vascularity. These changes were well correlated with MR abnormalities at the origin and the insertion of the impar ligament, specifically the presence of a cystic structure in the distal third of the navicular bone, one or more distal border fragments, the presence of entheseous new bone, or increased signal intensity in fat suppressed images at the insertion of the impar ligament on the distal phalanx (Blunden et al. 2009). Abnormal MR signal in the body of the impar ligament on the other hand, was not associated with the presence of histological abnormalities.

**COLLATERAL LIGAMENTS OF THE DISTAL INTERPHALANGEAL JOINT**

MR descriptions of injuries to the collateral ligaments of the distal interphalangeal joint and associated osseous abnormalities are well documented (Dyson et al. 2004; Dakin et al. 2009). A good agreement has been reported between MR and histopathological findings in collateral ligaments of 25 horses with palmar foot pain (Dyson et al. 2008). Lesions appeared to be degenerative, characterised by extensive fibrocartilaginous metaplasia and development of multiple, intercommunicating fissures within the degenerate collagen in severe lesions.
The presence of entheseous new bone on the proximal border of the navicular bone, reflecting recent insertional desmopathy of the CSL is well documented radiographically and histopathologically, in both clinically normal horses and horses with navicular disease (Pool et al. 1989; Verschooten et al. 1989). Fibrocartilaginous metaplasia has been described in the body of the CSL but there was no difference between horses with navicular disease and control horses (Blunden et al. 2006a).

In conclusion, the increasing number of available pathological studies on the various tissues of the foot has helped validate the significance of MR signal variations documented with various clinical MR systems used in clinical equine practice. It has brought to light a number of injuries involving structures in the foot other than the navicular bone, that had previously not been recognized as important causes of lameness, including tendinopathy of the DDFT, desmopathy of the CSL, the impar ligament, the collateral ligaments of the distal interphalangeal joint, and bone bruising of the distal and middle phalanx. In addition MRI has helped the clinician understand the different pathological processes occurring in the navicular bone. This new understanding of various new causes of lameness in the horse’s foot has resulted in changing treatment strategies in the management of foot lameness. However, further work is needed to continue to improve our understanding of the different causes of foot lameness and their pathogenesis.

**BIBLIOGRAPHIE**