OSSEOUS LESIONS IN THE METACARPO(TARSO)PHALANGEAL JOINT DIAGNOSED USING LOW-FIELD MAGNETIC RESONANCE IMAGING IN STANDING HORSES

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We report the use of low-field standing magnetic resonance imaging in the standing horse for the diagnosis of osseous lesions in the metacarpophalangeal (MCP) or metatarsophalangeal (MTP) joint that were not apparent using standard radiography. Thirteen horses were studied and all had thickening of the subchondral bone plate and abnormal signal intensity in the adjacent spongiosa in either the condyles of metacarpal/metatarsal III or the proximal phalanx or both. Abnormalities were characterized by diffuse decreased signal intensity on T1-weighting adjacent to the subchondral bone and within the spongiosa in at least two imaging planes; in the absence of increases in signal intensity in fat-suppressed images, this change was interpreted as bone sclerosis. Nine horses also had a diffuse decreased signal intensity on T2*-weighting in the same areas and five had a diffuse increase in signal intensity in fat-suppressed images in conjunction with a decrease in signal intensity on T1- and T2*-weighted images; the increase in signal intensity in fat-suppressed images was interpreted as fluid accumulation. Five horses had a focal area of change in signal intensity within the subchondral bone with apparent loss of definition between the subchondral bone and the articular cartilage. Eleven horses were available for follow up, of which eight were sound and three remained lame. We conclude that lameness originating from the MCP or MTP joint may be associated with osseous damage in horses of any signalment in the absence of radiographic changes. *Veterinary Radiology & Ultrasound, Vol. 50, No. 1, 2009, pp 13–20.*

Key words: Magnetic Resonance Imaging, fetlock, horse, lame.

Introduction

MAGNETIC RESONANCE (MR) IMAGING is used extensively to evaluate joint disease in humans[1] and is now being used for investigation of lameness in horses.[2,3] The equine metacarpophalangeal (MCP) and metatarsophalangeal (MTP) joints are commonly affected by osteoarthritic changes.[4–6] However, as in other joints, this damage may go undetected on radiographic examination, precluding a specific diagnosis. Synovial fluid and serum biomarker analysis has improved the detection of osteoarthritic changes, but it is difficult to accurately establish the degree of articular cartilage damage or subchondral bone disease in any single joint. A combination of biomarkers and other diagnostic techniques may be required to gain maximum information regarding joint integrity.[7]

MR imaging has been used to evaluate the equine MCP joint in cadaveric samples, and to compare MR findings with normal anatomy and results of other imaging techniques.[8–10] Good correlation between the thickness of cartilage and subchondral bone assessed histologically and in MR images has been found.[11] MR imaging with a 1.0 T magnet has also been used to identify subchondral bone damage in the distal limb.[12] These studies have helped to validate the clinical use of MR imaging in the study of equine osteochondral structure and modelling.

Recently, a low-field MR system designed to image the distal limb of standing horses has become available.[13–15] Most studies using this system have concentrated on foot lameness, and a similar range of lesions that has been described using high-field scanners has been recognized.[13,16–19] The objective of this study was to describe the clinical presentation, findings from low-field standing MR imaging, and follow-up of horses with lameness attributable to the MCP or MTP joint that had no or equivocal radiographic abnormalities.

Materials and Methods

Clinical records of horses undergoing standing MR imaging of either the MCP or MTP joint in our clinics between August 2004 and May 2006 were reviewed. Horses with lameness attributable to the MCT or MTP that had evidence of osseous damage on MR imaging that had not been detected radiographically were included. Some of the

horses had radiographic mild periarticular modelling suggestive of mild osteoarthritis.

Thirteen horses were identified, twelve of which were unilaterally lame and one of which was bilaterally lame. All horses were mature with a mean and median age of 9 years (range 4–14 years). There were six Warmbloods, three Thoroughbred or Thoroughbred crosses, two cobs, one Andalucian, and one trotting horse. Four horses were used for general purpose pleasure riding, three were showjumpers, three were used for racing (two flat racing and one trotting), one was used for dressage, and the use of two horses was not recorded.

At least four standard radiographic views of the MCP or MTP joint were obtained in each horse (lateral, dorsopalmar or dorsoplantar, dorsolateral–palmaromedial oblique or dorsolateral–plantaromedial oblique, dorsomedial–palmarolateral oblique or dorsomedial–plantarolateral oblique). Follow up information was obtained through repeat examinations and a telephone questionnaire with owners and referring veterinarians.

Sedation for MR imaging included a combination of romifidine hydrochloride (0.04 mg/kg IV),* butorphanol tartarate (0.02 mg/kg IV)†, detomidine hydrochloride (12 μg/kg IV),‡ and acepromazine maleate (0.02 mg/kg IV).§[13–15] Images were acquired using a 0.27 T open U-shaped magnet (Equine Limb MR Imaging Scanner; Hallmarq Veterinary Imaging Ltd, Guildford, UK). A dedicated transmitter–receiver extremity coil was used. Images obtained included gradient echo (GRE) T1-weighted motion insensitive sequences, GRE T2*-weighted motion insensitive sequences, gradient fat-suppressed sequences (FFS) and fast spin echo short tau inversion recovery (STIR) motion insensitive sequences in sagittal, transverse, and dorsal planes (Table 1). The MR images were compared with the images of horses with no radiographic or ultrasonographic changes and that were free from clinical orthopedic disease (30 cadaver limbs and 10 live horses).

Results

Six horses had sudden onset lameness, four had insidious onset lameness, and the onset of lameness was not recorded in three horses. The horses had been persistently lame for a median of 2 months (range 0.25–12 months). Lameness was graded between 2/10 and 6/10, with grade 0 being sound and grade 10 being nonweight bearing. Effusion was present in four joints and lameness was exacerbated by distal limb flexion in 12 horses. Lameness was further localized to the MCP or MTP joint by significant improvement following intra-articular analgesia, by significant improve-

TABLE 1. MR Sequences Used for Motion Insensitive (MI) Imaging

Sequence weighting	TR (ms)	TE (ms)	Flip Angle (°)	FOV (mm)	Slice Width (mm)	Gap (mm)	Scan Time (s)
T1-weighted GRE MI	49	8	50	170	5	0.5	170
T2*-weighted GRE MI	64	12	28	170	5	0.5	220
FFS GRE MI	65	12	25	170	5	0.5	292
STIR FSE MI	1800	28	90	170	5	0.5	301

MR, magnetic resonance; TR, repetition time; TE, echo time; FOV, field of view; GRE, gradient echo; FFS, fat-suppressed sequences; STIR, short tau inversion recovery; FSE, fast spin echo.

provement using perineural analgesia of the medial and lateral palmar, and medial and lateral palmar metacarpal nerves at the distal aspect of metacarpal bones II and IV after exacerbation of the lameness by distal limb flexion, or by significant improvement using perineural analgesia of the medial and lateral plantar digital nerve at the base of the proximal sesamoid bones.

There were no radiographic abnormalities in eight horses. There was mild periarticular modelling around the dorsal joint margin in four horses. There were equivocal changes in the proximal sesamoid bones in one horse. Ultrasonography of the affected joint and surrounding soft tissue structures was performed in 12 horses, which was normal in seven. Three horses had mild bone irregularity/osteophyte formation around the dorsal joint margin, one of which also had equivocal thickening of the medial attachment of the palmar annular ligament and one of which also had suspensory ligament desmitis. One horse had a questionably thickened lateral collateral ligament of the MTP joint. One horse had a marked effusion of the MTP joint consistent with palpation. The changes observed on radiographs and ultrasounds were considered unlikely to be of clinical significance in all the cases.

Nuclear scintigraphy was performed in three horses all of which had diffusely increased radiopharmaceutical uptake associated with the affected joint. One horse had mildly increased radiopharmaceutical uptake in the medial proximal sesamoid bone in the absence of change on MR imaging. Two horses had increased radiopharmaceutical uptake in the presence of decreased T1- and T2*- and increased signal intensities in FFS in the distal condyles of metacarpal/metatarsal III.

All 13 horses had thickening of the subchondral bone plate and abnormalities in the signal intensity of the adjacent spongiosa in either the distal condyles of metacarpal/metatarsal III or the proximal phalanx or both. The abnormalities were characterized by a diffuse decreased T1-weighted signal intensity within the spongiosa adjacent to the subchondral bone in at least two imaging planes (Fig. 1A and B). Nine horses also had a diffuse decreased T2*-weighted signal intensity (Fig. 2) and five horses had a diffuse increase in STIR and FFS signal intensity in the
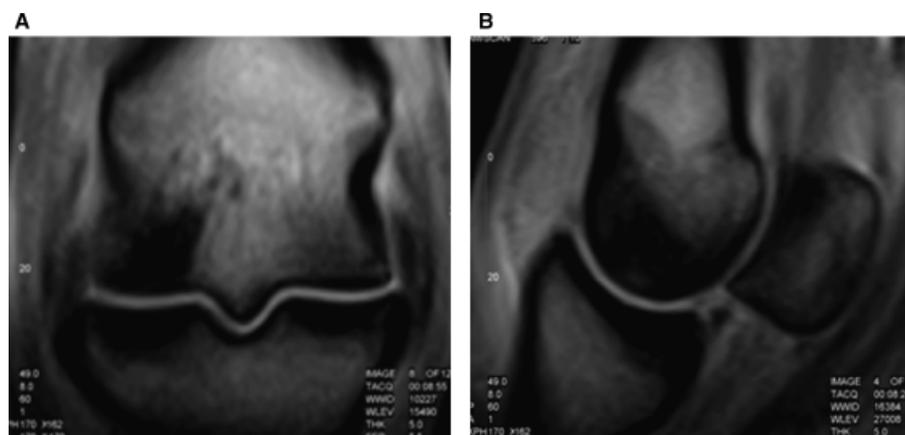
Fig. 1. T1-weighted gradient echo images of the right metatarsophalangeal (MTP) joint. (A) Dorsal image. Note the decreased signal intensity in the lateral condyle of metatarsal III and of the proximal glenoid of the proximal phalanx. (B) Sagittal image through the lateral aspect of the MTP joint. Note the decreased signal intensity in the lateral condyle of metatarsal III and the lateral glenoid of the proximal phalanx.

same area as the decrease in T1-weighted signal intensity (Fig. 3A and B). Six horses had a well-defined focal area of increased signal intensity on both T1- and T2*-weighted and FFS within the subchondral bone with loss of definition of the adjacent articular margin (Fig. 4A–C). Two horses had evidence of abnormalities within the sagittal ridge characterized by decreased signal intensity on T1-weighting, and increased signal intensity in STIR and FFS sequences in one horse, and modeling of the proximal dorsal border with thickened subchondral bone in another horse. Nine horses had ill-defined areas of abnormal signal intensity in the region of the articular cartilage, characterized by mild signal change of the presumed cartilage layer and loss of the presumed normal definition of the in-

terface between the cartilage and the subchondral bone. Eleven horses had periarticular osteophytes of the dorsal joint margin.

One horse with decreased signal intensity on T1- and T2*-weighting in the spongiosa of metacarpal III and the proximal phalanx also had a low signal intensity (T1- and T2*-weighting) well-defined linear pattern within the spongiosa of the proximal phalanx radiating perpendicularly from the endosteal surface of the cortical bone (Fig. 5). Another horse with decreased signal intensity on T1- and T2*-weighting in the spongiosa of metatarsal III and the proximal phalanx also had a low signal intensity (T1- and T2*-weighting) well-defined linear pattern running in a dorsoplantar direction through the spongiosa of metatarsal III (Fig. 6A and B).

Five horses had evidence of desmitis of one of the collateral ligaments of the joint as shown by swelling and increased T2*- and fat-suppressed signal intensity of the affected ligament, with or without increased T2*- and fat-suppressed signal intensity in the periligamentous tissues. Three horses with more severe osteochondral abnormalities at the medial aspect of the joint, had desmitis of the lateral collateral ligament. One horse with signal abnormalities in both condyles and glenoids had desmitis of the lateral collateral ligament. One horse with osteochondral abnormalities at the lateral aspect of the joint also had desmitis of the lateral collateral ligament of the MCP joint.

The proximal sesamoid bones had evidence of change in four horses. One horse had modeling of the base of the medial sesamoid bone; one horse had marked diffuse decrease in T1- and T2*- signal intensities and a STIR signal within the lateral sesamoid bone; one horse had a diffuse STIR signal in the medulla of the medial sesamoid concurrently with signal abnormalities in the distal condyles of metacarpal III and the proximal phalanx; and one horse had a loss of homogenous T2*- signal within the medulla of the medial proximal sesamoid bone.
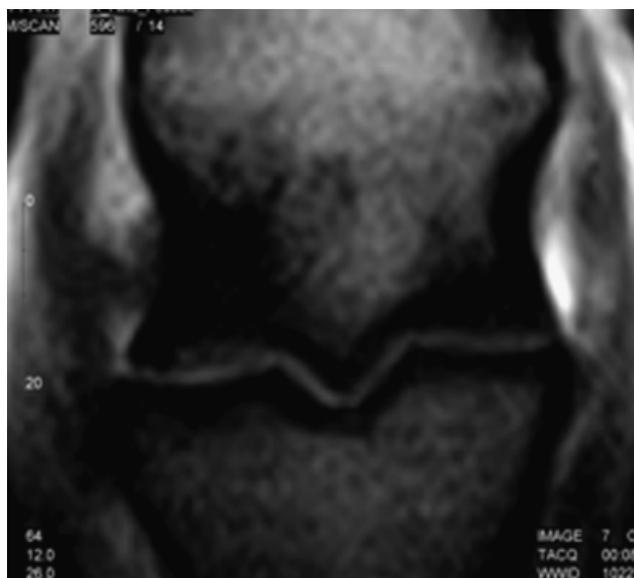

Fig. 2. Dorsal T2*-weighted gradient echo image of the right metatarsophalangeal (MTP) joint. Note the decreased signal intensity in the lateral condyle of metatarsal III suggestive of sclerosis in the absence of increases in fat-suppressed signal intensity.
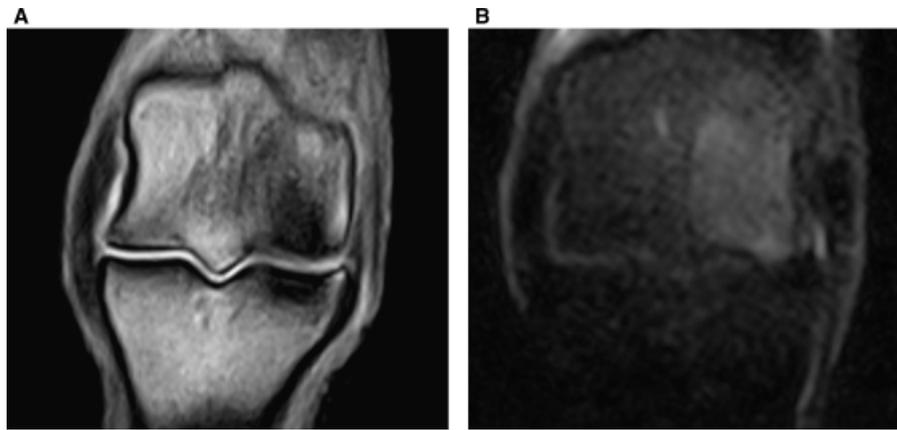
FIG. 3. (A) Dorsal T1-weighted gradient echo image of the right metatarsophalangeal (MTP) joint. Note the decreased T1 signal intensity in the medial condyle of metatarsal III and of the proximal glenoid of the proximal phalanx. (B) Dorsal short tau inversion recovery image of the right MTP joint. Note the increased signal intensity in the lateral condyle of metatarsal III and of the proximal glenoid of the proximal phalanx, suggestive of fluid accumulation.

A horse with bilateral hind limb lameness had similar abnormalities in both hind limbs; diffuse decreased T1- and T2*-weighted signal intensities within the distal condyles in conjunction with a well-defined focal area of increased signal intensity on T1- and T2*-weighting and fat-suppressed images adjacent to, and apparently involving the articular surface. These lesions had the appearance of cystic lesions surrounded by sclerosis.

FIG. 4. (A) Sagittal T1-weighted gradient echo image of the left metatarsophalangeal (MTP) joint. Note the focal area of high T1 signal intensity present in the subchondral bone of the lateral condyle of metatarsal III and the loss of definition between the subchondral bone and overlying articular cartilage. There is also decreased T1 signal intensity in the condyle surrounding the high signal lesion (B) Sagittal T2*-weighted gradient echo image of the left MTP joint. Note that the area of high T1 signal intensity in the subchondral bone of the lateral condyle of metatarsal III in part (A) also has high T2* signal intensity. The area of low T1 signal intensity in the condyle surrounding the high signal lesion also shows low T2* signal intensity. (C) Sagittal fat-suppressed image of the left MTP joint. Note that the area of high T1 and T2* signal intensity in the subchondral bone and overlying articular cartilage in parts (A) and (B) also has high signal intensity on fat-suppressed images. This is suggestive of hemorrhage, immature fibrosis or proteinaceous fluid.
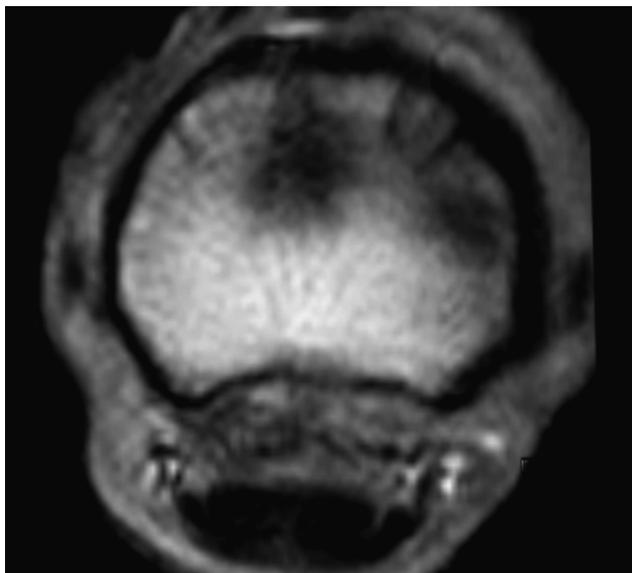
FIG. 5. Transverse T1-weighted gradient echo image through the proximal part of the proximal phalanx of the right fore. Note the low signal linear pattern within the spongiosa radiating perpendicularly from the endosteal surface of the dorsal cortical bone suggestive of sclerosis of the trabeculae.

Two horses had follow-up MRI scans after approximately 3 months. One had resolution in the STIR signal in the medial condyle and resolution in the T2*- and STIR signal surrounding the lateral collateral ligament. The other had a reduction in the cross-sectional area of the injured lateral collateral ligament and resolution of the periligamentous T2*- and fat-suppressed signal.

Eleven of the thirteen horses were available for follow-up. Six were sound and had returned to full work, two were sound but at a reduced performance, and three remained lame. Four of the horses with diffuse increased signal intensity in FFS were available for follow-up. Three were sound and one was lame. One of the sound horses underwent repeat MR imaging and there was resolution of the

increased signal intensity on fat-suppressed images. All six horses with focal increased signal intensities on T1-, T2*-, and fat-suppressed images within the subchondral bone were available for follow-up, five of which were sound. Treatment included stall/box rest, field rest, nonsteroidal anti-inflammatory (phenylbutazone) therapy, extra corporeal shock wave therapy, intra-articular medications with corticosteroids, hyaluronic acid, interleukin 1 receptor antagonist protein (IRAP) and remedial farriery.

Discussion

MR imaging in clinically lame horses has revolutionized the understanding of, and approach to, lameness originating from the foot.[2,13,16–19,20–26] Other areas of the distal limb are now being imaged.[12,27–29] Enhancement of hardware and software, and development of motion correction sequences for use in low-field systems, have led to improved images of the equine limb proximal to the foot without an increase acquisition time.[30] This has allowed clinical use of low-field systems to the level of the carpus and tarsus in adult horses. Unfortunately, there are few studies where low-field MR images have been compared with gross or histologic findings, and it was not possible to perform any pathologic studies on these clinical patients. However, the appearance of the subchondral and adjacent spongiosa bone abnormalities in this study are similar to those previously reported using a high-field system.[27]

Although subchondral bone injury is well recognized in Thoroughbred racehorses,[31] it has only recently been reported as a significant problem in mature sports horses.[27] The largest category of horses in this study group was general purpose pleasure horses, thus subchondral and adjacent spongiosa bone injury can occur in the MCP or MTP of horses of any signalment. Males were over-represented in our study group (77%) in contrast to previous findings.[27]
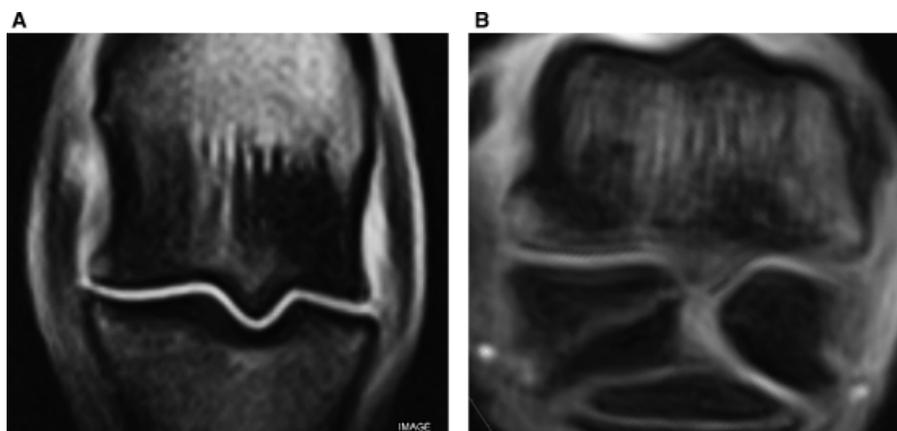

FIG. 6. (A) Dorsal T1-weighted gradient echo image of the left metatarsophalangeal (MTP) joint. Note the low T1 signal linear pattern through the spongiosa of metatarsal III. (B) Transverse T1-weighted gradient echo image of the left metatarsal III. Note the low T1 signal linear pattern running in a dorsoplantar direction through distal metatarsal III.

In this study, all horses had MCP or MTP osseous damage identified on images acquired using a low-field, standing MR system. The different tissues forming these diarthrodial joints have very close physical and biochemical relationships.[32] The articular cartilage is responsible for a small proportion of the energy transmitted across the joint (1–2%), the subchondral bone and subsequently the deeper spongiosa is responsible for dissipation of the vast majority of energy (30–50%), and the remaining energy is dissipated through the surrounding soft tissue structures.[33] In this study, 5 of the 13 horses had signal abnormalities or asymmetry within the soft tissues concurrently with subchondral bone damage, which highlights the importance of evaluating all joint components when assessing joint integrity and joint injury.

All horses in this study had thickening of the subchondral bone of at least one articular surface characterized by decreased signal intensity on T1- and T2*-weighting that extended beyond the normal depth of the subchondral bone into the spongiosa. Eleven horses also had thickening of the subchondral bone of the opposing articular surface. Overall there were six horses whose medial condyle was more severely affected and five horses whose lateral condyle was more severely affected, and two horses whose lateral and medial condyle appeared equally affected. In the forelimbs, the distribution of the most severe abnormalities was similar between the lateral (2) or medial (2) or both (2) condyles. In the hind limbs, the medial condyle was most severely affected in four horses and the lateral condyle was most severely affected in three horses. This is in contrast to previous studies in racehorses that have undergone training programs, where more intense and expansive increases in bone density of the lateral condyle relative to the medial condyle are reported. It is proposed that this is due to increased loading of the lateral condyle[34] as it has a smaller surface area than the medial condyle, and the articular margin slopes distally towards the lateral articular margin in contrast to the medial condyle which is perpendicular to the long axis of the bone. The condylar distribution of subchondral change in a population examined previously[34] may vary from the population in this study as there were a variety of horses and not just intensely trained young horses. Of the two Thoroughbred racehorses in this study, one had abnormalities predominantly within the lateral condyle of the hind limb and one had abnormalities predominantly within the medial condyle of the forelimb.

When examining the subchondral bone for changes in thickness and bone density, it is essential to consider asymptomatic anatomic variation. The subchondral bone of the condyles of metacarpal/metatarsal III can vary in thickness in both dorso-palmar/plantar, and abaxial–axial planes. The subchondral bone of the palmar/plantar distal aspect of the condyles of metacarpal/metatarsal III is denser than that of the dorsal aspect of the condyles.[35] This variation is dependent on training and is generally more marked in animals that have undergone intense training.[5,36] It is proposed that fast speeds increase hyperextension of the joint which the suspensory apparatus resists. Therefore, a focal increase in pressure is formed by the proximal sesamoid bones either side of the sagittal ridge in the palmar/plantar aspect of the joint. When the leg is in hyperextension, there is more than twice the force on the palmar/plantar condyle than the dorsal condyle and the force is not distributed evenly across the entire distal palmar/plantar aspect of metacarpal/metatarsal III as the palmar/plantar ligament does not produce as much pressure as the proximal sesamoid bones.[4]

There was one horse that had a sagittal linear pattern of low signal intensity within the spongiosa of metatarsal III on T1- and T2*-weighted images. This pattern is most probably a consequence of remodelling of the microtrabeculae in response to loading. The trabeculae of the deep subchondral bone of the distal condyles of the cannon bone are aligned predominantly in a sagittal plane and are connected in a mediolateral plane by "studs" only, which is not appreciable radiographically.[37] This is presumably a physiologic response to the normal plane of movement of this joint. Another horse had a low signal intensity (T1- and T2*-weighting) well-defined linear pattern within the spongiosa of the proximal phalanx of the MCP joint, apparently radiating perpendicularly from the endosteal surface of the cortical bone into the spongiosa. In normal horses undergoing nuclear scintigraphy, the most marked area of radiopharmaceutical uptake associated with the MCP joint was the proximal aspect of the proximal phalanx.[38] This implies that this is a site of increased bone turnover due to mechanical loading which might account for the abnormalities in trabecular pattern. It is possible that other horses in the study had this linear pattern but it was not detected due to volume averaging artifact.

A suspicion of abnormal articular cartilage, as demonstrated by lack of clarity of the interface between the cartilage and the subchondral bone, and apparent thinning and signal change of the cartilage layer, was seen in nine horses. However, it is unclear what level of confidence can be ascribed to this finding, and this apparent abnormality has not been validated by arthroscopic or pathologic evaluation. The low-field systems require the horse to stand with one leg in the magnet and one outside the magnet that may alter normal weight distribution through the limb in a medio-lateral plane. The combination of this artificial stance and the inherent difficulties with differentiation of the articular cartilage layer makes accurate assessment of the depth of the cartilage layer difficult. Although more clinically practical, limitations of arthroscopic evaluation include the inability to visualize the entire articular surface

of the MCP or MTP joint,[27] the inability to validate physiologic changes within grossly normal tissues that are abnormal on MR imaging[39] and the inability to identify subchondral defects in the absence of articular cartilage defects.[11] Until specific studies are undertaken to verify the pathologic nature of suspicious articular cartilage, the possibility that the signal changes are artefacts cannot be discounted. Artefactual cartilage signal abnormalities can occur due to motion or due to volume averaging, magic angle or susceptibility artefacts. The curvature of the condyles and the thin articular cartilage can cause volume averaging artefacts[3,27] exacerbated with the poorer resolution and larger slice thickness used with low-field systems in comparison with high-field systems. Additionally, motion artefact and magnetic field inhomogenicity are increased in low-field systems compared with high-field systems.

Six of the horses had severe abnormalities in subchondral bone extending to the articular cartilage margin that were characterized by a focal area of increased signal intensity in T1- and T2*-weighted, and FFS that was surrounded by a larger area of low signal intensity in the trabecular bone. Focal areas of increased signal intensities on T1-, T2*-, and FFS are suggestive of hemorrhage or proteinaceous fluid[3] or granulation tissue or necrotic bone.[27] These high signal areas may be due to concurrent trauma to the subchondral bone and articular cartilage or damage to one component that predisposes to damage of the other component. These areas have similarities to osseous cyst-like lesions, which are considered to occur either due to a primary defect in the subchondral bone predisposing to collapse of the overlying articular cartilage, or due to articular cartilage damage and subsequent damage to the subchondral bone through the hydraulic pressure of the synovial fluid. One horse with a focal well circumscribed area of increased signal intensity on T1-, T2*-, and FFS in the distal condyle of the left hind also had a similar change in the right hind. This may be due to an underlying bilateral lesion or alternatively due to trauma. Horses with traumatic lesions are most likely to be acutely lame; however, horses with pre-existing conditions sometimes present with acute lameness if they suddenly exceed their pain threshold.[12] Unfortunately, the absence of histopathologic assessment precludes a definitive understanding of lesion pathogenesis in our population.

There was poor correlation between ultrasonography and MR findings of the soft tissues, especially the collateral ligaments of the MCP and MTP joints. This was surprising, as ultrasonography is thought to be a sensitive technique for identification of soft tissue damage around the MCP or MTP joint.[40] However, sonography is very user-dependant and it is possible that more detailed examination of these horses may have led to evidence of collateral ligament damage. Alternatively, the difference between techniques may be attributed to poor contrast between the collateral ligaments and the periligamentous tissues. This may have been caused by movement artefact, disease in the periligamentous tissues reducing definition of the collateral ligament or due to the inferior soft tissue contrast inherent in GRE images.

In repeat MR examinations performed in two horses in this study, there was a reduction in the fluid signal intensity in the damaged soft tissue of both horses, and resolution of the fluid (STIR and FFS) signal within the trabecular bone in one horse which subsequently returned to work. There was no alteration in the T1- and T2*-weighted signal intensities in the trabecular bone between the first and second MR examinations in either horse. Low T1- and T2*-weighted signal intensities are considered to be due to trabecular thickening, sclerosis, or mineralization of the trabecular bone in response to repetitive overload.[3,27]

In summary, we found that lameness originating from the MCP or MTP joint may be associated with osseous damage in horses of any signalment. These osseous abnormalities can be absent on routine radiographs; however, low-field MR imaging can be a useful tool in their diagnosis. Repeat low-field MR studies are practical for monitoring lesion resolution and directing return to work. Further studies are required to investigate the prognosis in the individual lesion types, that is, those with diffuse increase STIR signal and those with subchondral damage. Overall, osseous lesions in the MCP or MTP joints have a good prognosis for return to work with 73% of horses back in work and 55% of horses reaching previous levels of performance.

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