Magnetic Resonance Imaging Findings in Horses With Recent and Chronic Bilateral Forelimb Lameness Diagnosed as Navicular Syndrome

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High field strength MRI is an effective method for diagnosing injury to structures within the feet of horses with navicular syndrome. The prevalence of different pathologies was determined in 151 horses with normal radiographs but clinical signs of bilateral navicular syndrome. Authors' address: Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman, WA 99164; e-mail: sarahs@vetmed.wsu.edu. © 2008 AAEP.

1. Introduction

Navicular syndrome is one of the most common causes of forelimb lameness in many types of athletic horses.1–4 Before magnetic resonance imaging (MRI), definitive diagnosis of pathologic change within the horse's foot was determined only at post-mortem examination. Radiography continues to be the first diagnostic imaging step used to try to define pathology associated with navicular syndrome, but it has become more apparent with the availability of MRI that radiography is inadequate in this region both for diagnosis of bone and soft-tissue pathologies. This is made clear by the reports of pathology diagnosed with MRI over the last 10 yr.3–17 The ability to use MRI to further define navicular syndrome in live horses has enabled a greater understanding of the disease processes in the foot of the horse that contribute to heel pain. The normal anatomy of the foot seen with MRI has been defined,18–20 and the increasing number of horses imaged continues to improve our understanding of individual variation between horses as well as the variety of pathologic changes that can occur in each horse.4–10,12–17,19,21,22

Navicular syndrome can now be defined as multiple disease processes resulting in damage to the navicular bone (NB), the collateral sesamoidean ligament (CSL), the impar ligament (IL), the navicular bursa, and/or the deep digital flexor tendon (DDFT).4–6,10,13–14,17,23 Navicular syndrome is described as heel pain that blocks to a palmar digital nerve (PDN) block on both forelimbs; radiographs may or may not have visible pathology. Furthermore, it has been reported that horses with navicular syndrome can have a variety of other structures involved that are not directly associated with the navicular region itself. Examples of these are pathologic changes in the distal digital annular ligament, proximal, middle, and distal phalanges, distal interphalangeal joint cartilage and subchondral bone, proximal interphalangeal joint cartilage and subchondral bone, collateral ligaments of the distal
and proximal interphalangeal joints, lamina (including keratoma formation), and digital flexor tendon sheath. Pathologic change can occur in any of these structures as the only manifestation of disease within the foot; however, multiple structures can be affected within the foot of one horse. Pathologic change within the bone or soft tissues, respectively, within the horse’s foot has been missed when only radiography and/or ultrasonography are used as diagnostic aids.

MRI remains a valuable diagnostic tool for orthopedic problems in humans for both bone and soft-tissue injury. Multiple MRI studies have been performed on cadaver specimens to evaluate horses with chronic, end-stage navicular disease. Many of the horses in these post-mortem studies have had flexor cortical erosion or other severe bone abnormalities visible on radiographs. MRI has enabled evaluation of the soft tissues associated with the NB in live horses for the first time, and, surprisingly, it has proven how much MRI has to offer in the diagnosis of orthopedic injury in the horse.

The objective of this study of horses with recent and chronic signs of navicular syndrome was to determine the prevalence of injury to different structures within the horse’s foot when lameness has been present for variable periods of time. Additionally, this study will compare these findings on two groups of horses based on whether or not they had recent or chronic signs of navicular syndrome. MRI observations in these two specific groups of horses restricted to those with bilateral forelimb lameness diagnosed as navicular syndrome without radiographic changes have not been reported.

2. Materials and Methods

The horses in this study had MRI performed on both front feet at Washington State University Veterinary Teaching Hospital between May 1998 and November 2007, because they had developed clinical signs of bilateral navicular syndrome. Horses that had been diagnosed with clinical signs of navicular syndrome were accepted if they did not have abnormalities on radiographs of the front feet. Information recorded from medical records of horses included in the study consisted of history, physical examination findings, results of lameness examinations, previous treatments, MRI findings, treatments based on MRI findings, and the suggested rehabilitation program.

A thorough lameness evaluation was performed in all horses. Injection of 1.5–2 ml local anesthetic over the medial and lateral PDN of the lame leg proximal to the collateral cartilage caused each horse to switch to lameness in the opposite forelimb. Injection of local anesthetic over the medial and lateral PDN of the opposite forelimb then eliminated the horse’s lameness. All horses were examined at a trot in a straight line and in small circles in each direction before and after PDN blocks. Lameness grades were 0 (no lameness observed), 1 (mild lameness without an observed head nod), 2 (subtle head nod observed), 3 (obvious head nod observed), 4 (severe head nod with <50% of normal weight supported on the limb), and 5 (limb not bearing weight). Radiographs, including a 60° dorsopalmar projection, a palmaroproximal to palmarodistal oblique (skysline) projection of the distal sesamoid (navicular) bone, and a lateromedial projection of the foot were evaluated and found to be normal for the age and use of the horse for all horses included in the study.

MRI was performed on both front feet of every horse after general anesthesia. Each horse had a thorough physical exam, including an electrocardiogram (ECG), complete blood count (CBC), and chemistry profile before anesthesia. Anesthesia was induced with ketamine and diazepam and maintained with 2–5% isoflurane in oxygen. All horses were placed in right lateral recumbency, and their front feet were positioned in a 1.0-Tesla magnet. A human knee quadrature receiver coil was positioned around the foot, and multiple image sequences were obtained using proton density (PD), T2-weighted (T2W), short tau inversion recovery (STIR), and gradient echo (3DGE) sequences. Axial, sagittal, and dorsal sections were obtained using standard MRI protocols for the foot of the horse developed at Washington State University (Table 1). All MRIs were stored on magnetic optic discs for later retrieval and evaluation. All horses were rope recovered from anesthesia in a padded recovery stall.

A standardized format was used to record MRI observations, and images were compared with images previously obtained from normal horses. A primary finding, based on severity and lack of other observed abnormalities, was made in horses where it was clear that one principal abnormality was present. In the other horses, a primary location of inflammation could not be identified from the multiple abnormal findings identified. Based on MRI findings, the limb with the most severe findings was recorded before the lamest limb was known, and these results were later compared with lameness exam findings. If MRI findings were too similar between limbs, this information was recorded.
3. Results

One hundred fifty-one horses were accepted into the study between May 1998 and November 2007. Seventy-two horses had recent onset of signs of navicular syndrome (lameness developed within the last 6 mo); 90 horses had chronic signs of navicular syndrome (lameness had been present for >6 mo).

Of the horses with recent onset, there were 51 geldings, 17 mares, and 2 stallions, and the average age of 8.2 yr (median age = 8 yr, range = 3–17 yr). Forty-two were Quarter Horses, 17 were Warmbloods, 7 were Thoroughbreds, 8 Paint, 2 Arabians, 1 Andalusian, 1 Appaloosa, 1 Lippizaner, 1 Paso Fino, and 1 Morgan. Thirty-two horses were used in Western performance events, 10 horses were used for eventing or jumping, 10 were used as pleasure or show horses, and 9 were used for dressage. The use was unknown in four horses. The mean lameness grade was 3 when the horses were trotted in small circles in both directions. All other horses had a unilateral lameness observed before the PDN block on the lame leg.

MRI Observations

Many different abnormalities were observed on MRIs of the front feet of the horses in this study. The majority in the recent onset group had a primary abnormality present, and the majority in the chronic group had multiple abnormalities present surrounding the navicular region. The prevalence of different pathologic changes and the primary abnormalities are listed in Table 2.

Recent Navicular Syndrome Horses

NB

Abnormal high-signal intensity (compatible with fluid) was observed in the NB on STIR sequences in 61 horses (108 limbs), and this was the most frequently observed abnormality based on the number of horses it affected. Twenty-three of these horses (30 limbs) also had abnormal low-signal intensity (consistent with sclerosis) in the medullary cavity of the NB. One of these horses had a 3-mm NB flexor cortex erosion just medial to the sagittal ridge that was not visible on radiographs. Fluid was seen either as focal high-signal intensity within the proximal, middle, or distal aspect of the NB (Fig. 1A) on the sagittal STIR sequences or as diffuse fluid throughout the medullary cavity (Fig. 1B). Both types were present within the bone in many horses. The amount and strength of increased signal intensity on STIR sequences was graded as mild, moderate, severe, or very severe (Fig. 2). If horses had abnormal high-signal intensity in the NB, it always affected the distal one-third of the bone, either as focal or diffuse high signal. In many horses, it also extended diffusely into the more proximal aspect of the NB. This is seen most clearly on the sagittal STIR sequences. Increased signal intensity in the

Table 1. Magnetic Resonance Imaging Sequences Used for Evaluation of the Foot and Pastern of the Horse Using a 1.0-T Gyroscan Magnet

<table>
<thead>
<tr>
<th>Image Orientation</th>
<th>Sequence</th>
<th>TR (ms)</th>
<th>TE (ms)</th>
<th>FOV/FA</th>
<th>Matrix Size</th>
<th>Slice #/Gap</th>
<th>Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axial DE</td>
<td>TSE T2</td>
<td>2116</td>
<td>100</td>
<td>90</td>
<td>15/15</td>
<td>512 x 512</td>
<td>30/4 mm</td>
</tr>
<tr>
<td></td>
<td>TSE PD</td>
<td>2116</td>
<td>10.5</td>
<td>90</td>
<td>15/10</td>
<td>512 x 512</td>
<td>30/4 mm</td>
</tr>
<tr>
<td>Axial</td>
<td>STIR</td>
<td>1725</td>
<td>35</td>
<td>90</td>
<td>15/15</td>
<td>256 x 256</td>
<td>30/3.5 mm</td>
</tr>
<tr>
<td>Axial</td>
<td>3D GE</td>
<td>47</td>
<td>9</td>
<td>25</td>
<td>10/10</td>
<td>256 x 256</td>
<td>30/3.0 mm</td>
</tr>
<tr>
<td>Navicular</td>
<td>TSE T2</td>
<td>3395</td>
<td>110</td>
<td>90</td>
<td>14/10</td>
<td>256 x 512</td>
<td>22/4 mm</td>
</tr>
<tr>
<td></td>
<td>TSE PD</td>
<td>3395</td>
<td>13.8</td>
<td>90</td>
<td>14/10</td>
<td>256 x 512</td>
<td>22/4 mm</td>
</tr>
<tr>
<td>Sagittal DE</td>
<td>STIR</td>
<td>1500</td>
<td>35</td>
<td>90</td>
<td>14/10</td>
<td>256 x 256</td>
<td>22/3.5 mm</td>
</tr>
<tr>
<td>Coronal DE</td>
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<td>25</td>
<td>10/10</td>
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<td>30/3.0 mm</td>
</tr>
<tr>
<td>DIP CL</td>
<td>TSE PD</td>
<td>3395</td>
<td>13.8</td>
<td>90</td>
<td>13/13</td>
<td>512 x 512</td>
<td>16/3.5 mm</td>
</tr>
<tr>
<td></td>
<td>TSE T2</td>
<td>3395</td>
<td>110</td>
<td>90</td>
<td>13/13</td>
<td>512 x 512</td>
<td>22/3.5 mm</td>
</tr>
</tbody>
</table>

TSE, turbo spin echo; T2, T2-weighted; PD, proton density; STIR, short tau inversion recovery; 3D GE, three dimensional gradient echo; TR, repetition time; TE, echo time; FA, flip angle; rFOV, relative field of view; DIP, distal interphalangeal joint; CL, collateral ligament; DE, dual echo (PD and T2).

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NB was determined to be the primary abnormality in 24 horses.

**NB Fragments**

Thirty horses had the NB sectioned coronally, and nine of these horses (15 limbs) had NB fragments (NBF) off the distal NB margin. Six horses had these bilaterally, and five of those horses had lateral fragments on each foot. The other one horse had medial and lateral fragments on each foot. Three horses had these unilaterally: one horse had a medial fragment, and two horses had lateral fragments. Two of the unilateral fragments were on the most lame limb, and one was on the least lame limb.

**DDFT**

Thirty-two horses (49 limbs) had thickening, irregularity, and/or abnormal high-signal intensity within the DDFT (Fig. 3) that most commonly located in the tendon proximal to the NB. In some horses, abnormalities extended proximally as far as the horse was imaged (midway in the proximal phalanx). Lesions within the DDFT were divided into four locations: distal to the NB (DTNB; 39% of limbs), at the NB (ANB; 35% of limbs), proximal to the NB (PTNB; 92% of limbs), and between the pastern and fetlock joint (31% of limbs). Twenty-four (49%) limbs had lesions in more than one region, and 25 (51%) limbs had lesions in only one region. Of the 25 limbs that had lesions in only one region, 84% had lesions PTNB, 8% had lesions DTNB, and 4% had lesions ANB. No horses were seen that had a DDFT lesion within the pastern region that did not have lesions more distally in the tendon as well. Twenty-seven (84%) horses with DDFT lesions also had enlargement of the CSL.

**CSL**

Thickening, with or without abnormal hyperintensity, of the CSL was observed in 54 horses (86 limbs; Fig. 4). Thirty-nine (45%) limbs had damage to the deep flexor tendon at the level of the CSL. Damage to the CSL was the primary abnormality in 11 horses.

**IL**

Abnormal high-signal intensity and/or thickening of the IL were observed in 26 horses (40 limbs). Ten horses (19 limbs) also had abnormal signal intensity on PD and T2W sequences at the IL insertion onto the NB; this is compatible with sclerosis in this area (Fig. 5). IL damage was determined to be the primary abnormality in seven horses.

**Other Findings**

Six limbs had damage to the distal digital annular ligament. Four limbs had abnormal hyperintensity within the third phalanx. Two horses had osteoarthritis of the distal interphalangeal joint. Three horses had keratomas within the medial or lateral one quarter of the area, and all were found on the most lame limb. These were not visible on dorsopalmar oblique projections of the distal phalanx. One horse had desmitis of one CSL of the distal interphalangeal joint.

**Synovial Structures**

Thirty-six horses (71 limbs) had increased synovial fluid observed in the distal interphalangeal joint (Fig. 6). All of these horses also had other abnor-

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**Table 2. Prevalence of Types of Pathologic Change and Primary Abnormality in the Recent and Chronic Navicular Syndrome Groups**

<table>
<thead>
<tr>
<th>Structure</th>
<th>Recent Group</th>
<th>Chronic Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Horses</td>
<td>Limbs</td>
</tr>
<tr>
<td>Multiple</td>
<td>18%</td>
<td>NA</td>
</tr>
<tr>
<td>NB</td>
<td>85%</td>
<td>75%</td>
</tr>
<tr>
<td>NBF</td>
<td>13%</td>
<td>10%</td>
</tr>
<tr>
<td>FCE</td>
<td>1%</td>
<td>0.7%</td>
</tr>
<tr>
<td>CSL</td>
<td>75%</td>
<td>60%</td>
</tr>
<tr>
<td>DDFT</td>
<td>44%</td>
<td>34%</td>
</tr>
<tr>
<td>PASTERN</td>
<td>44%</td>
<td>31%</td>
</tr>
<tr>
<td>PTNB</td>
<td>92%</td>
<td>NA</td>
</tr>
<tr>
<td>ANB</td>
<td>35%</td>
<td>NA</td>
</tr>
<tr>
<td>DTNB</td>
<td>39%</td>
<td>NA</td>
</tr>
<tr>
<td>IL</td>
<td>36%</td>
<td>28%</td>
</tr>
<tr>
<td>Bursa</td>
<td>44%</td>
<td>40%</td>
</tr>
<tr>
<td>DIP Effusion</td>
<td>50%</td>
<td>49%</td>
</tr>
<tr>
<td>DDAL</td>
<td>8%</td>
<td>4%</td>
</tr>
<tr>
<td>Bone Bruises</td>
<td>6%</td>
<td>3%</td>
</tr>
<tr>
<td>DIP CL</td>
<td>1%</td>
<td>0.7%</td>
</tr>
<tr>
<td>DIP OA</td>
<td>3%</td>
<td>1%</td>
</tr>
<tr>
<td>Laminar defects</td>
<td>4%</td>
<td>2%</td>
</tr>
</tbody>
</table>

NA, not applicable.
malities observed in the NB or supporting soft tissue structures, although they may not have been the primary abnormality noted. Thirty-two horses (58 limbs) had increased synovial fluid observed in the navicular bursa. In most horses, however, a relatively small increase in fluid was observed. In 17 limbs (29%), there was severe bursitis, and 12 of these (71%) had deep flexor tendonitis within the navicular bursa. Although a severe increase in synovial fluid was observed in the bursa in 17 horses, a large amount of fluid consistent with bursitis was not considered the primary abnormality in any of these horses; however, one horse with multiple other chronic lesions and an acute lameness history did have severe fluid in the bursa. Six limbs had effusion of the proximal interphalangeal joint.

**Multiple Abnormalities**

Multiple abnormalities were observed in 13 horses in which a primary abnormality could not be determined. Abnormalities were not observed in the NB or its supporting soft tissues in four horses.

**Correlation of MRI Findings and the Lamest Limb**

Fifty-six of the 72 horses had obvious differences in severity of pathology between limbs. In 93% (52 of 56) of horses, the most severe abnormalities occurred in the limb on which the horse was most lame. In 7% (4 of 56) of horses, the worst findings were in the opposite limb. Sixteen of the 72 horses had similar findings on both limbs.

**Lameness Follow-Up**

Follow-up information was obtained for 76% (55 of 72) of horses by reevaluation of horses or by telephone conversations with trainers or owners. This information is shown in Table 3.

**Chronic Navicular Syndrome Horses**

**CSL**

Thickening, with or without increased abnormal signal intensity, of the CSL was observed in 72 horses (137 limbs; Fig. 4). This was the most frequent abnormality observed based on the number of limbs it affected, although less total horses were affected compared with those with increased signal intensity in the NB. Ligament thickening was slightly more likely to be seen bilaterally than increased signal intensity within the medullary cavity of the NB, which is described below. Thirty-six horses (65 limbs) had adhesions present between the CSL and the DDFT. Of these limbs, 38 had damage to the tendon at the level of the CSL. One horse had damage to the tendon distal to this level (at the level of the NB). Damage to the CSL was determined to be the primary abnormality in 19 horses.

**NB**

Abnormal high-signal intensity was observed in the NB on STIR sequences in 73 horses (133 limbs), and this was the most frequently observed abnormality based on the number of horses it affected; however, slightly fewer limbs were affected than with thickening of the CSL. Eighteen horses (26 limbs) had abnormal decreased signal intensity on PD and T2W sequences that was located within the central medullary cavity of the NB, which is compatible with sclerosis (Fig. 7). In 8 horses, this sclerosis was associated, at least partially, with a flexor cortical erosion (Fig. 8). Pathologic change within the NB was determined to be the primary abnormality in 13 horses.

**NBF**

Forty-two horses had their NBs imaged in a coronal plane. Of these horses, 15 (36%; 19 limbs) had NBF (Fig. 9). Four horses had distal margin fragments on both limbs. Of these four, one horse had medial and lateral fragments on each limb, one

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**Fig. 1.** These are sagittal STIR images showing (A) abnormal high-signal intensity (fluid) in the distal one-third of the NB and (B) spread diffusely throughout the medullary cavity of the NB.
horse had medial and lateral fragments on one limb and a medial fragment on the other limb, and two horses had lateral fragments. Ten horses had distal margin fragments on one limb only: six were medial, and 4 were lateral. One horse had an avulsion fragment on the proximo-lateral aspect of the NB within the CSL. Of the horses with unilateral fragments, seven were found on the lamest leg, and four were found on the least lame leg.

Fig. 2. These are sagittal (left) and axial (right) STIR images showing (A) mild, (B) moderate, (C) severe, and (D) very severe increased signal intensity, which indicates inflammation of the NB.
Eight horses in the study were found to have flexor cortical erosions, and these were not visible on radiographs. All erosions involved the flexor surface in the distal one-third of the bone over or in association with the mid-sagittal ridge. One of these horses had defects in both NBs, and the remaining seven horses had a single defect in only one NB—four in the left front, and three in the right front. The horse with bilateral lesions had a small erosion on either side of the sagittal ridge on one limb. Erosions measured 3–6 mm, depending on the horse, in a medial to lateral dimension. Of the horses with unilateral erosions, five were found on the lamest leg, and two were found on the least lame leg.

**IL**

Abnormal high-signal intensity and/or thickening of the IL were observed in 50 horses (95 limbs).

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**Fig. 3.** These are axial PD images of the pastern and foot arranged (top) proximally to (bottom) distally that show (A) mild, (B) moderate, and (C) severe lesions within the DDFT at different levels of the limb.

**Fig. 4.** These are (left) sagittal and (right) axial PD images showing (A) mild, (B) moderate, and (C) severe enlargement of the CSL.

**Fig. 5.** These are sagittal PD images showing diffuse thickening of the IL (A) with and (B) without abnormal low-signal intensity at the insertion of the ligament on the distal navicular bone (arrow).
Twenty-five horses (36 limbs) also had increased low-signal intensity on PD and T2W sequences at the IL insertion onto the NB, which is compatible with sclerosis in this area (Fig. 5). IL damage was determined to be the primary abnormality in six horses.

**DDFT**

Thirty-nine horses (58 limbs) had thickening, irregularity, and/or abnormal high-signal intensity within the DDFT, and it was graded as mild, moderate, or severe at four separate levels of the tendon within the horse’s distal limb (Fig. 3). Twelve horses (13 limbs) had damage DTNB, 19 horses (24 limbs) had damage ANB, 38 horses (56 limbs) had damage PTNB, and 21 horses (27 limbs) had damage within the pastern region. Tendon damage was most commonly located in the tendon just proximal to the NB, and it was least likely to be located distal to the NB. Tendon damage DTNB was not seen bilaterally in any horse, and it was found in the lamest limb in 8 of 12 horses (67%). Tendon damage in the pastern region was never seen without tendon damage also occurring in a more distal portion of the tendon in the same limb. In five horses,

![Image](image1.png)

**Fig. 6.** These are (A) sagittal and (B) axial STIR images that show (arrows) increased fluid in the distal interphalangeal joint. There is also effusion of the DDFT sheath visible on the sagittal image.

![Image](image2.png)

**Fig. 7.** This is a sagittal proton density image from a horse with abnormal low signal intensity centrally located within the navicular bone, indicating remodeling of the trabeculae resulting in sclerosis.

**Table 3.** Comparison of Follow-Up Lameness Information Between the Recent and Chronic Navicular Syndrome Groups

<table>
<thead>
<tr>
<th>Performance Level</th>
<th>Recent Group</th>
<th>Chronic Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 yr</td>
<td>3 yr</td>
</tr>
<tr>
<td>Same level of work</td>
<td>51%</td>
<td>33%</td>
</tr>
<tr>
<td>Lower level of work</td>
<td>13%</td>
<td>11%</td>
</tr>
<tr>
<td>Total</td>
<td>64%</td>
<td>44%</td>
</tr>
</tbody>
</table>

![Image](image3.png)

**Fig. 8.** These are axial (A) 3DGE and (B) PD images of a horse with abnormal high-signal intensity in the (arrows) flexor cortex of the NB at the mid-sagittal ridge. This horse also has an area of abnormal low-signal intensity within the medullary cavity associated with the flexor cortex lesion visible on the (arrowhead) PD image in B.
tendonitis of the DDFT was the primary finding observed.

Navicular Bursa
Fifty-six horses (96 limbs) had increased synovial fluid observed in the navicular bursa; this was graded as mild, moderate, or severe (Fig. 10). Twenty-eight horses (49 limbs) also had abnormal tissue present in the proximal aspect of the navicular bursa that appeared to be fibrous tissue between the DDFT and the proximal suspensory ligament of the NB (Fig. 11). In these limbs, the bursitis was often less pronounced because of obliteration of bursal space by scar tissue. A large amount of fluid consistent with bursitis was considered the primary abnormality in one horse.

Distal Interphalangeal Joint
Thirty-six horses (62 limbs) had increased synovial fluid observed in the distal interphalangeal joint. All horses also had other abnormalities observed in the NB or its supporting soft-tissue structures. One horse (1 limb) had damage to one CL of the distal interphalangeal joint. This horse had damage at the insertion of the medial CL on the left front leg. There was associated abnormal high-signal intensity on STIR images, which indicates inflammation, and abnormal low-signal intensity on PD and T2 images, which indicates increased bone density (sclerosis; Fig. 12). Three horses (4 limbs) had osteoarthritis of the distal interphalangeal joint. This horse had cartilage and subchondral bone damage that was not visible on radiographs as well as some changes in the navicular region (Fig. 13).
Distal Digital Annular Ligament

Eight horses (11 limbs) had thickening, with or without increased signal intensity, in the distal digital annular ligament (DDAL; Fig. 14). Of those horses, 5 had unilateral injury, and four had bilateral injury. Of the unilateral injuries, 3 were on the lamest leg, one of which had adhesions to the digital flexor tendon sheath and DDFT. One was on the least lame leg. Of the bilateral injuries, one horse had similar injuries on both limbs, one had a worse injury on the lamest leg, and one had a worse injury on the least lame leg. The DDAL was not determined to be the primary abnormality in any horse.

Bone Bruises of Phalanges

Five horses (6 limbs) had increased signal intensity in one or more of the phalanges seen on STIR sequences.

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Fig. 12. The top two images are (A) axial PD and (B) T2W images showing (arrows) abnormal increased signal intensity within the medial collateral ligament of the distal interphalangeal joint at its insertion onto the distal phalanx. The (C) STIR image shows increased signal intensity within the distal phalanx at the CSL insertion, which is indicative of inflammation within the bone. The (D) coronal T1-weighted image shows the (arrow) abnormal decreased signal intensity within the distal phalanx at the insertion of the CSL, which indicates sclerosis in this area.

Fig. 13. These are (A) axial STIR, (B) PD, and (C) 3DGE images showing a cartilage and subchondral bone defect in the medial aspect of the distal phalanx (arrows).
Two horses (2 limbs) had abnormalities in the second phalanx, and 3 horses (4 limbs) had abnormalities in the third phalanx. All these horses had obvious pathologic change in the navicular region of both front feet, and it was suspected that the inflammatory change (bone bruise) within bones other than the NB was a result of a traumatic incident subsequent to the development of navicular syndrome; however, this cannot be verified. Two horses had unilateral inflammation in the third phalanx at the insertion of the IL, which is most likely a result of IL injury at the bone/ligament interface on the distal phalanx. Both of these horses had IL thickening and abnormal increased signal intensity on the same foot. The other horse with inflammatory changes in the distal phalanx had changes along the dorsal surface of the bone, but this was not associated with laminar thickening or inflammation.

Lamina

Three horses (three limbs) had irregularity of the lamina on one foot near the toe. None of these horses had rotation of the distal phalanx within the hoof capsule, but they did have focal thickening of lamina in areas near the toe that may represent unhealthy lamina as a result of an old abscess or seedy toe. One of these horses had the laminar irregularity in the least lame leg, and the other two horses had the laminar irregularity in the lamest leg. These changes were not determined to be a primary abnormality in any horse, and these horses were not sensitive to hoof testers over the toe in any case.

Correlation of MRI Findings and Lamest Limb

Of the 79 horses evaluated with MRI, 13 horses (16%) had very similar findings on both limbs, and 66 horses (84%) had obvious differences in severity of pathologic change between limbs. In the horses with obvious differences between limbs, 90% (59 of 66) had the most severe and highest number of findings on the most lame leg, and 11% (7 of 66) of the horses had the most severe and highest number of findings on the least lame leg.

Lameness Follow-Up

Fifty-six horses (71%) were available for follow-up evaluation by phone calls with the owner or trainer. Twenty-five (45%) of these had remained lame and out of work after the diagnosis and MRI. Twenty-nine (52%) had remained in the same or lower level of work for the next 6–12 mo after diagnosis. Two horses (4%) had died of an unrelated cause shortly after diagnosis. Within 1 yr of diagnosis, eight horses died of unrelated causes or were euthanized for their lameness: four were euthanized for ongoing lameness, two sustained a broken leg in pasture, one was attacked by a cougar, and one was euthanized because of a tumor. Follow-up lameness information is available in Table 3.

4. Discussion

This is the first report of MRI observations in a group of horses with clinical signs of navicular syndrome without radiographic changes that correlates MRI findings with lameness and follow-up information. It is clear that clinical signs of navicular syndrome can result from a variety of abnormalities, which has been shown previously.4–17 Additionally, abnormalities like NB edema/sclerosis, CSL desmitis, IL desmitis, navicular bursitis, and deep digital flexor tendonitis may be related and may occur together in the same horse.

It is clear in this study that horses with a chronic history of navicular syndrome have a higher number of structures undergoing pathologic change than those with recent signs of navicular syndrome. This is evident by the large number of chronic horses (35 of 79) in which a primary abnormality could not be determined based on which structure was affected more severely than the others. These horses grouped in the multiple abnormality category had moderate to severe changes in at least two structures, which included at least two of the following structures: CSL, IL, NB, DDFT, navicular bursa, distal interphalangeal joint, distal interphalangeal joint collateral ligament, DDAL, second phalanx, or third phalanx. The majority of horses in this category had more than two structures moderately to severely involved, and it was not possible to determine which of the pathologic changes initiated the subsequent disease process. Aging these lesions and determining the chronological order of different pathologic changes remains impossible at this time. It is known that some horses may develop inflammation of the NB as an early onset of disease, whereas other horses may develop desmitis of the CSL as the initial onset of the disease.4–17 We have
also seen that horses with changes to the NB and its supporting structures can go on to develop DDFT injuries that seem to be a progression of a disease process centered around the NB. In other horses, primary deep flexor tendonitis occurs without any pathologic change to the NB or its ligamentous supporting structures.

In this study, the majority of horses diagnosed with navicular syndrome were Quarter Horses, and there were also many Warmbloods and Thoroughbreds. Because these breeds of horses perform in a wide variety of events, it suggests that the activity itself may not be an important factor in the disease process. The horses in this study often had one limb that was much more lame than the other limb, which explains the reason that some of these horses present to veterinarians as a unilateral lameness. It is only after the lame limb is blocked successfully that it becomes apparent that there is an obvious lameness in the contralateral limb, which was seen in 50–76% of horses in this study. Because of the bilateral nature of navicular syndrome, the majority of horses are seen to be moderately lame on one limb before nerve blocks. This does not always indicate the actual degree of lameness in these horses, because they are protecting both feet to some extent, even when circling in small circles. To truly determine the lameness grade of the lamest limb, lameness would have to be eliminated from the least lame limb and vice versa. This is impractical in a clinical setting and was not done with the horses in this study. Distal limb flexion does exacerbate

Fig. 15. These are (left) sagittal and (right) axial STIR images showing different areas of abnormal increased signal intensity seen in horses in this study. (A) Focal increased signal intensity at the (arrows) insertion of the IL on the distal phalanx. (B) Diffuse increased signal intensity in the NB and medial one-half of the middle phalanx. (C) Focal and slightly diffuse abnormal increased signal intensity within the dorsodistal middle phalanx.
lameness in some of these horses, and this would suggest that compression-type pressure over the NB and its supporting structures was painful. It has also been shown that extreme extension of the distal interphalangeal joint can exacerbate lameness in navicular syndrome horses. More than 50% of the horses in this study had sensitivity to hoof testers over the middle of the frog on at least one limb, and this correlates with the location of the pathologic change in many of these horses. This area is directly under the NB and its supporting structures. The area of the DDFT within the foot was also most commonly affected with pathologic change.

In horses with a chronic history of navicular syndrome, a higher percentage (91%) of horses were found to have pathologic change within the CSL than in horses with recent onset of navicular syndrome (75%). In chronic navicular syndrome horses, there is a high likelihood of adhesion (46%) between the CSL and the DDFT, even if there is not pathologic change within the DDFT itself. Only 56% of horses with adhesions between the CSL and the DDFT had lesions within the DDFT. Adhesions in this region are associated in many horses with scar-tissue formation within the navicular bursa in the proximal aspect. This adds to the complexity of determining a primary abnormality in these horses and makes it difficult to determine the inciting cause. It would make sense that the CSL becomes inflamed and thickened in horses in which there is not damage to the DDFT, but subsequently forms adhesions to the DDFT because of proximity. The inflammation in this area could then result in bursal inflammation and secondary scar-tissue formation in the navicular bursa. These horses can also have inflammation of the NB, but there are horses that only have inflammation of the NB without surrounding pathologic change to the associated ligaments. Therefore, a cause and effect is not necessarily occurring here, whereas adhesions between the CSL and DDFT were only seen to occur in horses with pathologic change in the CSL.

In chronic navicular syndrome horses, 44% of horses had multiple abnormalities that precluded the determination of a primary abnormality; this is in contrast to the horses with recent onset of navicular syndrome in which only 18% of horses were grouped into the multiple abnormality category. The most common primary abnormality in the chronic horses was pathologic change within the CSL (24% of horses). Horses with recent onset of navicular syndrome had pathologic change within the NB (33% of horses) as the most common primary abnormality compared with the CSL (15% of horses).

NB damage was the second most common finding (16% of horses) in the chronic group of horses and was present in almost as many limbs as pathologic change in the CSL (133 versus 137 limbs in 73 and 72 horses, respectively). In this study, if horses had a difference in the amount of increased abnormal signal intensity in the NB between limbs, the more severe changes were most commonly seen in the lamest leg (78% of horses). In contrast, of 13 horses with abnormal decreased signal intensity in the NB (sclerosis) separate from the IL insertion that was worse in one limb than the other, only 54% (7 of 13) of horses had the most severe findings in the lame leg. Therefore, in determining the limb that may be more painful, it is possible that increased signal intensity on STIR sequences is more important than decreased signal intensity on PD sequences. This is in contrast to horses with small flexor cortical erosions, in which 71% (5 of 7) were found on the most lame leg if they were present unilaterally. Obviously, the size of these lesions, being only 3–7 mm in width, may result in them causing less pain than larger lesions visible on radiographs, and larger lesions may correlate even better with lameness.

The prevalence of distal margin NBFs in horses with chronic navicular syndrome (36%) was comparable with findings in horses with recent onset of navicular syndrome (37.5%). Of the 11 chronic horses with fragments in 1 limb only, 64% were found on the most lame limb. It remains unknown how important these fragments are in the pain caused by pathologic change in the navicular region and how they affect progression of the lesions, if at all. All horses with distal margin NBFs had IL desmitis and/or NB inflammation in the distal one-third of the bone centered over the IL insertion. This suggests that there is a pathologic process resulting in fragmentation of the NB distal margin in these horses.

Deep digital flexor tendonitis was the primary abnormality in only five horses (6%) in the chronic group, which is different than the prevalence in the recent group where 13 horses (18%) had deep flexor tendinitis as the primary abnormality. The overall prevalence of horses with DDF tendinitis in the chronic group compared with the recent group is actually not that different (46% versus 44%, respectively), but the horses in the chronic group have more separate structures involved in the navicular region. This precludes determination of deep digital flexor tendinitis as a primary abnormality in many of the chronic horses because the severity of changes in the NB and supporting ligaments are equal or worse than the DDFT lesions. Of the limbs that had lesions in the DDFT at the level of the CSL in the chronic group, 62% (36 of 58 limbs) had adhesions to this ligament. This indicates that horses with tendinitis in this region may be pre-disposed to forming adhesions to the CSL, which in turn may lead to inflammation within this ligament. Of the 56 chronic limbs with DDFT lesions just proximal to the NB, 43 (73%) of these limbs had increased fluid within the navicular bursa, and 26 (44%) of
these had obvious scar tissue within the proximal aspect of the bursa. Comparison of the locations of deep digital flexor tendonitis in the chronic group and the recent group shows that 50% more of the chronic horses had extension of the deep flexor tendonitis into the pastern region. This would make sense with the chronicity of the lameness in that the tendon lesions would be more likely to worsen and enlarge over time. Also of interest is the fact that none of the horses in the recent or chronic group had a DDFT lesion in the pastern without having a tendon lesion at some point more distally in the same limb. It may be possible to extrapolate from this that, most commonly, the tendon lesion begins at or near the NB and over time extends proximally in some horses. It may also be useful information when using ultrasound on the pastern region in horses, because it is likely that a horse with a DDFT lesion in the pastern visible with ultrasound will have equal or more severe lesions within the foot on the same leg. Furthermore, extension of the tendonitis proximally into the area covered by the digital flexor tendon sheath makes injection of the sheath a possible diagnostic and therapeutic technique for horses with this diagnosis. It could also be considered that horses with tendonitis in the pastern may be candidates for navicular bursa injection based on the high likelihood that there are DDFT lesions within the foot, which is most commonly seen just above the NB in an area covered by the bursal lining. The observation that deep digital flexor tendonitis occurs as a primary problem in some horses with clinical signs of navicular syndrome is an important observation. The observation that injury extends proximally in the tendon above the level of the proximal interphalangeal joint affects treatment for some horses. Rest, rehabilitation, and physical therapy to stretch the DDFT have not been routinely used to treat horses with navicular disease, but they may offer another option for treatment of some of these horses in the future.

Multiple other abnormalities were also found in the horses in this study, including DDAL desmitis, distal interphalangeal joint osteoarthritis, bone bruises of the first, second, and third phalanges, distal interphalangeal collateral ligament desmitis, laminar abnormalities, and adhesions of the DDFT to the DDFT sheath. It is difficult to relate these possible findings to the changes in the heel region of many of these horses that have multiple abnormalities. The presence of hyperintensity on STIR images, indicative of a bone bruise, within the phalanges does suggest that a rest and rehabilitation program could be helpful in these specific horses, whereas without an MRI, these horses would most likely be kept in work with anti-inflammatory medication and not given a chance for bone healing to occur. The identification of osteoarthritis with cartilage damage visible with MRI provides an argument for arthroscopy to debride lesions that are present. This could potentially give the horse a better chance to return to work in the future compared with the horse that is continually treated as a chronic navicular syndrome horse until lameness forces the horse to stop work. Identification of CL injury also provides an argument for a significant rest and rehabilitation program to be instituted as a means to allow the ligament to heal and the horse to return to its previous work. It is unlikely that many of these bone, cartilage, and ligament lesions would heal if the horse continued in treatment for navicular syndrome, which supports the use of MRI to provide a definitive diagnosis.

In the recent and chronic groups, the severity of MRI findings had a high correlation with the lameness of the horse (93% and 90%, respectively). The multitude of structures affected and the severity of pathology in many of the chronic horses may account for the slight decrease in accuracy in picking the limb with the most severe findings. These horses, in general, had more variation in the number and types of pathologic change in each limb which added greater complexity in determining the changes that accounted for the greatest pain. With more cases, we may eventually be able to determine the lesions that result in lameness more often than other lesions or the specific groups of lesions that cause lameness more frequently. At this point in time, all lesions are given weight in determining the severity of the disease process, although the clinical examination is also taken into account when deciding the relevance of some lesions in a clinical setting.

When comparing prognosis for return to work in the recent and chronic groups, it is clear that horses are much less likely to be able to remain in work as they become more chronically affected (Table 3). Four horses with chronic navicular syndrome were euthanized for ongoing lameness within 1 yr of their diagnosis, and this may provide insight into another reason that some people chose to have their horses diagnosed using MRI. This modality can provide a very specific diagnosis, and it can give owners the information they need to make difficult decisions regarding further treatment of horses or humane euthanasia. It can be very difficult for some owners to stop treatment on a horse when they do not have a specific diagnosis to confirm a poor prognosis.

As was shown in this study, the causes of navicular syndrome and more generally, of pain that is eliminated with a PDN block are numerous. We no longer believe that there is one pathologic process that leads to the myriad of pathologic changes present within the foot of the horse that causes heel pain. This study of navicular syndrome provides information that leads to the conclusion that heel pain can be caused by pathologic change within many separate structures in the foot, and therefore, inflammation of a single structure is enough to cause lameness in the horse. When comparing horses with recent onset of navicular
syndrome to chronic cases, it is clear that the inflammation spreads in many horses within the relatively small space within the heel and affects more structures over time, because the pathologic process is specific to each individual horse. We also believe that trauma to one or both front feet that may go unnoticed by the owner may lead to some horses that have significantly worse findings in one limb, although abnormalities are found in both feet. Horses that have pathologic changes in the navicular region may be more affected by a specific traumatic incident to one foot than horses with healthy, normal feet, or they may be more likely to sustain permanent damage compared with normal, sound horses. This could be a result of a loss of elasticity to inflamed ligaments or tendons within the foot that are already present or a NB that has remodeled to a point where it is not as resilient as a normal bone.

Inflammation of several different tissues in the heel of the horse’s foot can cause similar clinical signs that are currently recognized as one syndrome. For this reason, MRI is a valuable technique for evaluating horses with navicular syndrome, because a specific diagnosis can be made that effects new or further treatment. We are still in the learning stages with MRI, and more horses need to be evaluated before onset of disease and earlier in the disease process to learn more about navicular syndrome. This may enable us to correlate findings with the onset of the specific disease process occurring in different horses, which in turn will enable the evaluation of new diagnostic and treatment methods.

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References and Footnotes

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