Navicular Syndrome – Old Problem, New Insights
Annette M McCoy, DVM, MS, PhD, DACVS; University of Illinois College of Veterinary Medicine

Introduction

Navicular syndrome has been described in horses for more than 150 years, although it has gone by many names, including "navicular disease" and "palmar heel pain." The current naming convention reflects the realization that pathology arising from many sources, including the navicular bone (distal sesamoid), navicular bursa, collateral ligaments of the navicular bone, deep digital flexor tendon (DDFT), and distal sesamoidian impar ligament, can result in pain that is responsive to local/regional anesthesia of the palmar foot. Advances in imaging technology, particularly MRI, have revealed the wide heterogeneity of this condition, and offered insight into why clinical manifestations and response to treatment can vary so widely from horse to horse.

The “classic” presentation for navicular syndrome is a middle-aged or older horse in steady work with either a low-grade chronic forelimb lameness that has slowly worsened, or an apparently sudden-onset unilateral forelimb lameness of moderate severity. In either case, when the more lame leg is blocked, lameness in the contralateral forelimb generally (though not always) becomes apparent. Radiographic changes are inconsistent between affected individuals and do not always correlate with clinical signs. Quarter Horses and related breeds are traditionally considered to be predisposed to navicular syndrome, although it is also recognized in Warmbloods, Thoroughbreds, and other breeds. Recent studies have suggested a genetic predisposition for navicular disease specifically in the Hanoverian warmblood, with quantitative trait loci (QTL) identified on equine chromosomes 2 and 10.

The underlying etiology of navicular syndrome is unknown, and the disease cannot currently be reproduced experimentally. Evidence from naturally-occurring disease suggests that there is an accumulation of accelerated degenerative processes in the navicular bone and associated soft tissues in affected horses, although the cause for this acceleration is poorly understood. Hypotheses related to ischemia have largely been disproven, and the theory that is currently most widely accepted is that abnormal biomechanical forces on the navicular bone and surrounding soft tissue structures play an important role in the pathophysiology of disease. Increased force in the DDFT in the early and mid-stance phases has been recorded in horses with navicular syndrome compared to normal horses, which leads to altered loading patterns on the navicular bone. However, whether this is a cause or consequence (or both) of heel pain remains uncertain.

Diagnosis

History, signalment, and clinical signs are often suggestive of navicular syndrome. However, response to local/regional analgesia has historically been considered the strongest indicator for the need to perform diagnostic imaging of the navicular region. Horses with navicular syndrome typically (though not always) respond to perineural analgesia of the medial/lateral palmar digital nerves at the level of the collateral cartilages (1.5-2ml mepivacaine per site), although the lameness may not be completely abolished. It is common for the primary lameness to switch to the contralateral forelimb after blocking; if this limb is also blocked, the lameness may switch back to the first leg. Any remaining lameness is often abolished by performing perineural anesthesia at the level of the base of the proximal sesamoid bones. The navicular bursa can be infused directly with local anesthetic (3-4ml mepivacaine), and this seems to be highly specific
for pain originating from the navicular bone and/or bursa. Intraarticular analgesia of the distal interphalangeal (coffin) joint (6ml mepivicaine) can also improve or abolish lameness in horses with navicular syndrome; due to technical ease, this is the more commonly performed procedure, although, obviously, it is not as specific.

A complete radiographic study of the navicular region should minimally include lateromedial, dorsoproximal-palmarodistal oblique (DPr-PaDiO), and palmaroproximal-palmarodistal oblique (PaPr-PaDiO) views (Figure 1). Importantly, the shoes must be removed to completely evaluate the navicular bone. The DPr-PaDiO view highlights the distal border of the navicular bone, while the PaPr-PaDiO view highlights the flexor surface. Positioning is particularly important for the PaPr-PaDiO, which must be tangential to the palmar aspect of the navicular bone to avoid artifacts. The angle needed to achieve this will vary based on the conformation of the foot.

Radiographic changes seen in navicular syndrome include irregular bony margins (both along the distal border and flexor surface), synovial invaginations along the distal border (“lollipops”), luencies within the medulla, medullary sclerosis, loss of distinction between the cortex and medulla, luencies of the flexor cortex, enthesiophytes, mineralization of collateral ligaments, and mineralized fragments off the distal border (Figure 2). Multiple changes may be seen in a single individual, and the severity of radiographic changes do not always correlate with clinical signs.

Nuclear scintigraphy is highly sensitive for bone turnover, and increased radiopharmaceutical uptake in the palmar foot may be seen in both the pooled (soft tissue) and bone phase. However, this finding is not specific to this condition and false positives can occur. Computed tomography (CT) and contrast-enhanced CT findings for horses with foot lameness have been described; however, magnetic resonance imaging (MRI) tends to be the preferred
advanced imaging modality because of improved visualization of soft tissue structures within the foot. Ultrasound has been investigated as an alternative to diagnose soft tissue lesions in cases where MRI is not possible. Diagnostic images of the navicular bone and associated soft tissue structures can be obtained through the frog when it is trimmed and soaked, although there are anatomical limitations. In one report, lesions were successfully detected ultrasonographically in 39 horses with lameness localized to the foot, but no radiographic abnormalities. The majority of these lesions were located within the DDFT.

MRI has been the subject of most research focused on the diagnosis of navicular syndrome in the past 5-10 years. Low-field MRI is most commonly available (<0.4T), although increasingly, high-field units (1.5-3T) are being installed at veterinary teaching hospitals and large referral centers. Standing MRI has gained popularity because it eliminates the risk inherent to general anesthesia, but the field strength is limited (0.27T) as is the scanning window (i.e. you can scan the foot, but not the foot, pastern, and fetlock all at once). This modality is more sensitive than radiographs for bony lesions, and perhaps more importantly, allows the diagnosis of lesions localized to the soft tissues and cartilage that are not visible radiographically (Figure 3). It should be noted that lesions visible radiographically are typically confirmed on MRI, and radiographic measurements have been shown to be predictive for MRI-diagnosed pathology. As this modality has become more widely used, techniques to detect subtle pathology have become more refined. For example, saline arthrography (of the coffin joint and/or navicular bursa) has been demonstrated to improve evaluation of the structures surrounding the navicular bone, particularly in cases in which there is little fluid in the navicular bursa or in which adhesions are present.

Unquestionably, MRI has contributed to our understanding of the complexity of navicular syndrome; the challenge that still remains is determining the clinical relevance of MRI findings. Changes in signal intensity on MRI have been shown in multiple studies to correspond to histologic lesions within the navicular bone and associated soft tissues, so the issue is not false-positive findings (aside from some well-known technical artifacts). Rather, the difficulty lies in sorting through multiple abnormalities to determine which is/are the primary cause of lameness and should be the focus of treatment. For example, in one study of 72 horses with recent onset of clinical signs due to navicular syndrome, nearly all had multiple abnormalities detected on MRI, and in 13 of these horses (18%) a primary abnormality could not be identified. In another study of 403 horses with more chronic disease, 92 (23%) had both navicular bone lesions and injuries of the DDFT or other soft tissues.

Navicular bursa endoscopy provides the opportunity for direct observation of the flexor surface of the navicular bone, the navicular bursa, the overlying DDFT, and the collateral sesamoidian ligaments. Both direct and transthecal approaches to the bursa have been

![Figure 3: 3D gradient echo T1-weighted low-field MRI (0.25T) images of the same foot as Figure 2B. Pathology in the navicular bone (arrow) and DDFT (chevron) are present.](image-url)
described. The direct approach offers more maneuverability because the scope passes through less soft tissue, but a recent study found that inadvertent penetration of the coffin joint and/or flexor tendon sheath occurred in 45% of limbs. These authors described an alternative direct approach that significantly decreases the risk of inadvertent penetration of these neighboring synovial structures; however, this comes at the cost of decreased maneuverability and visualization of structures. Debridement of DDFT and fibrocartilage lesions can be performed endoscopically, with a reasonable expectation for a good outcome. A recent report of 92 horses treated in this manner found that 61% of horses were sound enough for work, and 42% returned to their previous level of work. Needle arthroscopy (camera the diameter of an 18ga needle) of the navicular bursa has also been described, but is restricted to use as a diagnostic procedure only.

Treatment and Prognosis

There is no cure for navicular syndrome; rather, treatment strategies are aimed at managing the condition so that affected horses can remain comfortable and in work for as long as possible. Use of rest and non-steroidal anti-inflammatory drugs (NSAIDs), along with trimming/shoeing changes are typically the cornerstones of treatment early in the course of disease. Common recommendations for trimming/shoeing include shortening the toe to ease breakover and elevating the heel with a wedge shoe or pad to relieve pressure from the navicular bone. Some practitioners prefer the use of egg bar or heart bar shoes, although a variety of shoes have been reportedly used with success. Routine radiographs are recommended to evaluate medial-lateral balance and the hoof-pastern axis. For horses with a severely broken-back hoof-pastern axis, correction with trimming and shoeing will need to occur in stages. Owners should be warned that a temporary increase in lameness may occur after trimming/shoeing.

Phenylbutazone is the most commonly used NSAID in horses with navicular syndrome, although firocoxib has gained popularity due to the reduced risk of gastrointestinal side effects. Horses with less severe disease may only require intermittent treatment, while others must be maintained long-term on a NSAID. Isoxsuprine, a β-agonist, has also been reported to be successful in reducing lameness in horses with navicular syndrome, although the exact mechanism of action is unknown. These two therapies can be combined.

Intrasynovial injections, typically with triamcinolone and/or hyaluronic acid, are also a common treatment. There is debate as to whether direct injection of the navicular bursa is required, or whether treatment of the coffin joint is adequate. Bursal injection is a more complicated technique that is ideally performed under radiographic guidance; additionally, there has been some concern about iatrogenic damage to the DDFT and/or navicular fibrocartilage, although the clinical significance of a 20ga needle track has been debated. There is evidence that triamcinolone diffuses from the coffin joint into the navicular bursa both in normal horses and horses with palmar heel pain. However, there is less diffusion in horses with more severe radiographic changes. Clinically, improvement after coffin joint injection has been reported to be only short-term, and there is an impression that response to direct bursal injection is more profound. However, coffin joint injections are often tried first in horses with few radiographic changes. Intrabursal injections with botulinum toxin have also been reported to result in temporary improvement in lameness, but this treatment has not gained widespread acceptance. Response to treatment with intraarticular medication varies with the severity and location of lesions. Horses primarily affected with navicular bursitis can be expected to have an excellent
response to treatment, while those with more severe, or multiple, structural lesions are likely to have limited improvement. In one study, only 39% of horses were able to return to their previous level of work for at least 3 months without additional intervention. In another report, although the success rate was higher (74%), horses only remained sound for an average of 7 months.

There have recently been many anecdotal reports of using tiludronate and related bisphosphonate drugs, which work by inhibiting osteoclasts, systemically and as regional limb perfusions for the treatment of navicular syndrome. However, the scientific evidence for their efficacy is equivocal. An initial double-blind placebo-control study showed a non-statistically significant improvement in lameness score between horses treated with 1mg/kg tiludronate IV for 10 days compared to placebo-treated horses. A more recent study found improved vertical ground reaction forces, but no improvement in subjective lameness score, in horses treated with a single 1mg/kg IV dose. In this same study, regional limb perfusion did not improve ground reaction forces, and marginally improved lameness scores at a single time point.

Extracorporeal shockwave therapy (ESWT) has been advocated as a treatment for navicular syndrome, but although anecdotal reports are numerous, there is little research supporting its use. One study looking at radial ESWT found no change in scintigraphic or radiographic findings after treatment, and the authors concluded that beneficial effects on lameness score were due to the well-documented short-term analgesic effects of this therapy. However, to date there have been no studies looking at the effects of focused ESWT on objective disease parameters.

The most common surgical treatment for horses with navicular syndrome is a palmar digital neurectomy. This procedure should only be considered in horses in which lameness is completely eliminated with perineural anesthesia of the palmar digital nerves, and then only as a treatment of last resort. Neurectomy can alleviate the pain from navicular syndrome, but does not address the underlying pathology, which will continue to progress. Complications that have been reported secondary to this procedure include neuroma formation, incomplete resolution of lameness (due to collateral innervation and/or re-innervation), DDFT rupture, navicular bone fracture, coffin joint luxation or subluxation. Numerous methods have been reported to try to reduce these complications, but the two currently favored techniques are the guillotine method, which involves sharp transection and removal of 2-4cm of the nerve through a single incision, and the pull-through method, which involves sharp transection of the nerve through two small incisions 8-10cm apart and then removal of the entire transected nerve segment through one of the incisions. Success rate 12-18 months post-operatively has been reported to range from 65-92% in a handful of studies; however, the time it takes for lameness to reoccur in a given individual can vary widely. Repeat surgery is not generally successful. The importance of meticulous foot care for neurectomized horses cannot be overstated to clients, as there is an increased risk of missing foot abscesses or penetrating wounds of the palmar foot because of the lack of a painful response to these lesions.

Selected references and recommendations for further reading


