

Review: Understanding Radiographic Changes Associated with Navicular Syndrome—Are We Making Progress?

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The etiopathogenesis and radiographic features of navicular syndrome remain controversial. However, as our knowledge of pathophysiology increases, better decisions regarding radiographic interpretation are made. Employment of magnetic resonance imaging and computed tomography will lead to earlier, more accurate diagnosis of navicular syndrome. Authors' address: School of Veterinary Medicine, 1248 Lynn Hall, Purdue University, West Lafayette, IN 47906. © 2002 AAEP.

1. Introduction

Although radiography is often used to validate a diagnosis of navicular syndrome (NS), findings may correlate poorly with clinical evaluation.¹⁻³ The classic radiographic changes associated with NS and their relative importance are well known to equine clinicians and have been debated for years. Unfortunately, standard radiographic evaluation can be misleading because hard tissue changes are primarily a measure of osseous remodeling, and important soft tissue changes cannot be seen. In addition, the projectional nature of radiography can mask subtle, but significant navicular changes. This review details contemporary opinion regarding the significance of radiographic changes of NS and their relation to current concepts of pathogenesis and histopathologic findings. The potential impact of alternate imaging modalities such as magnetic resonance (MR) and computed tomography (CT) is briefly explored.

2. Radiography of Navicular Syndrome

Part of the confusion surrounding radiographic interpretation is anatomic and technical in origin. The equine digit consists of dense and spongy bone, various connective tissues, and the hoof capsule. Unfortunately, standard radiography can only distinguish between cortical and trabecular bone and the soft tissues. Therefore, radiographic assessment is limited to looking for osseous changes in the navicular bone proper, such as osteolysis, osteoproduction, or shape change. Except for dystrophic mineralization, alterations in the ligaments, bursa, or deep digital flexor tendon cannot be seen because these structures tend to have the same radiopacity in health and in disease. Altering radiographic technique will do little to improve visualization of soft tissues; this requires use of other imaging modalities that afford additional soft tissue contrast such as MR.

Ambitious radiographic techniques have been developed to maximize visualization of the navicular bone.³ These include the use of detail film and

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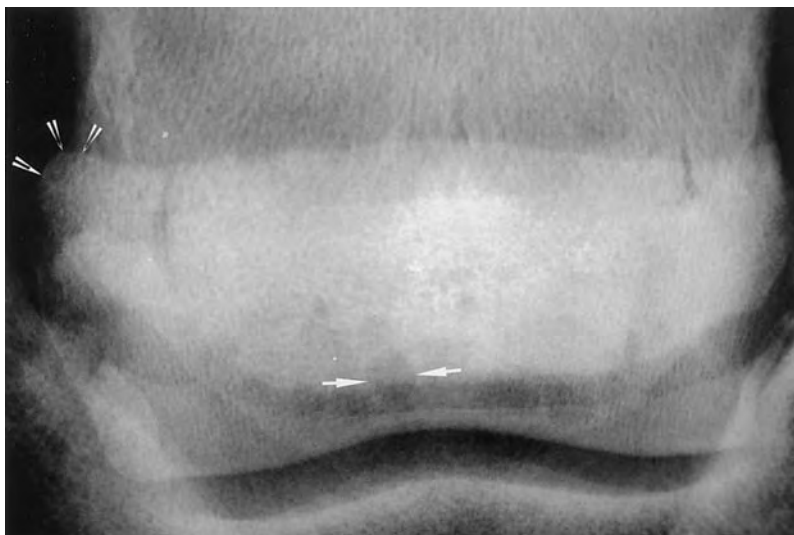


Fig. 1. Dorsopalmar radiograph of the fore digit of a 10-year-old Thoroughbred with clinical signs of navicular syndrome. There is enthesopathy of the extremity (arrowheads) and loss of the normal shuttle shape. Note lollypop-shaped synovial fossae along the distal border (arrows).

screen combinations, grids and strict collimation to reduce scatter radiation, tangential (skyline) projections, and magnification. However, none can overcome the limiting factor of superimposition of surrounding structures or components of the navicular bone itself. For instance, the superimposed middle phalanx can mask subtle changes like fragmentation of the distal border of the navicular bone. Because of its projectional nature, the x-ray beam can provide false information by superimposing an adjacent structure like mineralized collateral cartilage. Obviously technical errors like failing to pack the hoof sulci with radiopaque material, not cleaning the hoof and pastern, motion artifact, and using exhausted screens will compromise radiographic quality and interpretation.

3. Radiographic Changes of Navicular Syndrome

Radiographic changes commonly associated with NS are listed below.¹⁻³ Abnormalities of the synovial fossae of the distal border are a time-honored *sine qua non* of the diseased navicular bone. Many investigators and clinicians try to equate presence and/or severity of NS with number, shape, and type of synovial fossae.²⁻⁷ Enlarged fossae are actually synovial invaginations or “inroads” and part of the synovial membrane of the distal interphalangeal joint. In horses with NS, the nutrient vessel is actually very small and is surrounded by hyperplastic synovium; hence, the term “vascular channel” is inappropriate.⁵ Magnetic resonance imaging, which provides superior soft tissue contrast, confirms these histopathologic observations.⁸ The pathophysiology is unclear, but evidence suggests these synovial invaginations are a result of arthrosis of the distal interphalangeal joint. Normal horses usually have 3–5 short cone-shaped synovial fossae along the distal border—these

represent an equilateral triangle. With use and age, these may enlarge, and become wider or even columnar-shaped because of synovial invaginations. Horses with NS frequently have abnormal synovial fossae. When fossae are lollypop or flask-shaped (Fig. 1), many consider them abnormal and a reliable sign of NS.^{5,6} However, lollypop-shaped synovial fossae have been found in 11% of sound horses.⁷ When numerous abnormal synovial fossae are present and/or they are fork-shaped, they may predispose to fracture or fragmentation of the distal border. From a radiologist’s perspective, enlarged abnormally shaped fossae are most significant when they are found along with other radiographic signs of NS.

Fragmentation of the distal navicular border is a “catch all” term for osseous bodies consisting of true navicular chip fractures, mineralization of the impar ligament (enthesophyte production), osseous metaplasia, or possibly separate ossification centers.^{3,9} High-quality dorsopalmar projections are needed to see these changes, and use of a grid is helpful. They can also be seen on a true lateromedial projection. CT and MR imaging have shown that fragmentation of the distal border is frequently underestimated on routine radiographic projections.⁸ Obviously, chip fractures may be significant changes because they reflect underlying pathology of the navicular bone. Confirmation of chip fractures is based on finding donor sites, whereas other causes of fragmentation do not have donor sites. Mineralization and osseous metaplasia of the impar ligament reflect stress and strain and thus may be a part of NS. Fragmentation of the distal border is also found occasionally in sound horses.⁷

Elongation of the medial and lateral extremities is caused by a combination of remodeling (see below) and enthesophyte production or “spur” formation at the



Fig. 2. Lateromedial radiograph of the same horse in Figure 1. Shape change is characterized by elongation of the flexor surface (arrows) and the medullary cavity (M) is sclerotic.

site of attachment of the collateral sesamoidean ligaments. This change is thought to be a result of chronic stress and strain that may be a part of the degenerative process of NS.¹⁻³ Additional new bone formation may be seen along the proximal navicular border and also at the attachment of the collateral sesamoidean ligament. However, enthesophyte production can be seen in normal horses that work hard and or have chronic hoof imbalance but have not developed clinical signs of NS.^{3,10} Young horses with enthesopathy of the extremity may be at risk for development of clinical NS.³ Most radiologists feel that these changes alone do not constitute radiographic evidence of NS.

Shape change refers to loss of the normal shuttle shape of the navicular bone on the dorsopalmar radiograph or elongation of the flexor cortex on the lateromedial projection (loss of trapezoid shape) (Fig. 2). A change in shape is caused by osseous remodeling in response to stress and strain. Shape change should be considered an adaptive response and does not always correlate with clinical NS. However, if osseous remodeling cannot keep up with navicular loading, progressive degeneration occurs.^{1,3} Additional radiographic signs are seen such as medullary sclerosis, flexor cortical thickening, and abnormal synovial fossae. A hereditary predisposition between shape of the proximal articular margin of the navicular bone and development of NS has been shown in Warmbloods.¹¹ Horses with a concave proximal articular border had a higher incidence of radiographic changes of NS. The shape of the offspring was, on the average, the same as the sire, implying a hereditary element. Pathogenesis is unclear, but possibly, shape-dependent distribution of forces is a factor.¹¹ It is unknown whether or not navicular

shape is a predisposing factor in other breeds at risk for NS, such as Thoroughbreds and Quarter Horses.

Many investigators consider radiographic changes in the flexor cortex to be reliable indicators of NS.^{3,6} However, one should realize when abnormalities are found in the flexor cortex or medullary cavity most *other* radiographic changes are already present as well! Cortical changes play a role in the development of NS.^{1,12} Degeneration of the fibrocartilage of the flexor surface is thought to develop as a result of loading that exceeds physiologic limits. Later on, remodeling of the underlying cortical and trabecular bone must occur to keep pace with demand of increased biomechanical forces. Cortical defects represent lysis of subchondral bone of the flexor cortex and are linked to disease of the overlying fibrocartilage and deep digital flexor.¹³ Fibrocartilaginous degeneration occurs in a fashion not unlike the deterioration of articular cartilage seen in osteoarthritis. In fact, some believe that the etiopathogenesis of primary osteoarthritis and NS are similar.^{1,13} Cortical defects or erosions are seen on the palmaroproximal-palmarodistal radiographic projection (Fig. 3). Improper projection or omission of this projection are serious errors that can lead to a false negative diagnosis. Cortical defects may be focal or diffuse and are rarely seen in sound horses. An artifactual, crescent-shaped lucency within the sagittal ridge (flexor eminence) may be found on the palmaroproximal-palmarodistal projection and should not be confused with a cyst or erosion.^{14,15}

Thickening of the medullary cortex may be a radiographic feature of NS; however, one should keep in mind that cortical thickening is an adaptive response to biomechanical forces. In addition, the thickness varies with breed and technical factors such as object film distance and beam angulation.⁷ Studies¹ have

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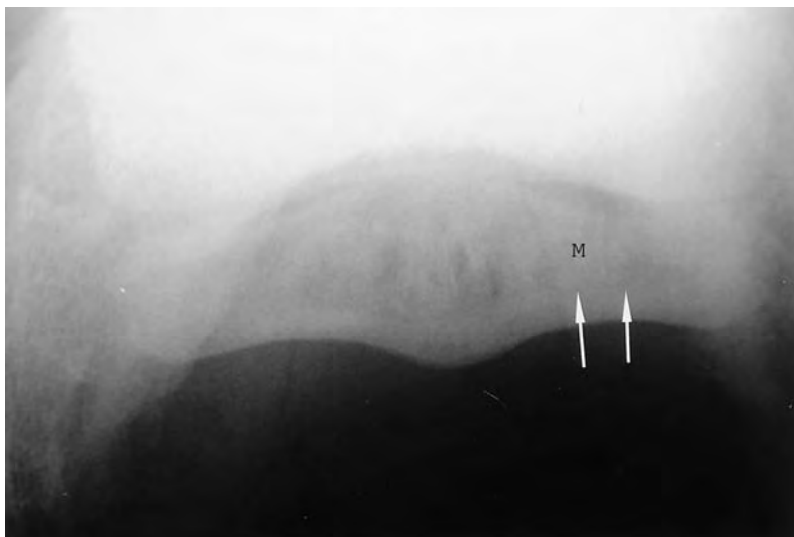


Fig. 3. Palmaroproximal-palmarodistal radiograph of the same horse as in Figures 1 and 2. Note medullary sclerosis (M) and loss of demarcation between the cortex and medullary cavity. There is slight irregularity of the cortex on either side of the midsagittal ridge (flexor eminence).

also shown that the flexor cortex of horses in training *normally* thickens—another example of a radiographic change being an adaptive response. With work, loading of the flexor surface of the navicular bone undergoes osseous remodeling. The normal result is thickening of the cortex and the underlying trabecular bone. One could argue that if over-training does not occur, osseous remodeling remains in synchrony with demand and does not reach a disease state. Apparently, if bone remodeling rates are gradual enough, further changes leading to clinical NS may not occur.¹

Sclerosis of the medullary spongiosa and loss of demarcation of the flexor cortex and medulla are considered reliable signs of NS.^{1,6,16} Although these changes can be seen in sound horses, they are clearly more prevalent in horses with NS. Sclerosis of medullary bone is part of the remodeling process of NS¹ and may cause venous congestion within the navicular bone.¹⁶ Venous congestion increases intramedullary pressure and is a cause of navicular pain. Therefore, the finding of medullary sclerosis has significant diagnostic and therapeutic implications. Obtaining a correct palmaroproximal-palmarodistal projection is mandatory for correct interpretation of the medullary cavity. The correct projection angle is approximately 45° relative to the horizontal. If the beam angle is too steep or too flat, there will be a false impression of medullary sclerosis.

Cyst-like radiolucent structures are also reliable evidence of NS; however, their pathogenesis is unclear.¹ These areas have been termed pseudocysts and may or may not communicate with large synovial fossae.¹³ In one study,⁸ these so called “lytic” areas contained amorphous material consistent with old hemorrhage and new bone

formation. These were actually wide medullary spaces that may have developed because of extensive osteolysis, which proceeds new bone formation in the process of bone turnover. This suggests repeated trauma with the absorptive and formative stages of bone turnover out of balance. Medullary cyst-like changes can be best seen using the palmaroproximal-palmarodistal projection, but large radiolucencies are also visualized on the dorsopalmar projection. However, large lollypop-shaped synovial invaginations can mimic medullary cyst-like changes with the dorsopalmar projection.

Mineralization of the deep digital flexor tendon is one of the few soft tissue changes that can be seen on standard radiographs. Unfortunately, this is generally a late finding and is a poor prognostic sign because it indicates widespread degeneration of the tendon and adjacent bursa. The deep digital flexor tendon and bursa are frequently involved when severe radiographic changes are seen in the flexor cortex.

4. Discussion

Equine practitioners are well aware that one should not commit to the diagnosis of NS based solely on radiographic changes. Radiology of NS remains a vague commodity for several reasons. First, the pathogenesis is incompletely understood so the absolute correlation of radiographic changes with clinical signs is not possible. There is disagreement as to the exact origin and developmental sequence of radiographic changes of NS. Does it start as bursitis and extend to the deep digital flexor or does it begin with failure of the flexor fibrocartilage with extension to the subchondral bone and navicular bursa and tendon? Do vascular changes lead to excessive osseous remodeling with extension to adja-

cent soft tissue structures? These questions are best answered with serial imaging beginning early in the course of NS, using a modality like CT or MR that allows visualization of soft tissues.

Second, many radiographic abnormalities occur in sound horses, confounding our ability to determine their significance. Because most radiographic changes of NS reflect remodeling they denote adaptive changes, not necessarily disease. Later, these same horses may develop NS because their navicular apparatus cannot withstand the workload. At this stage, a clinical diagnosis of NS may be evident. An important, but unanswered question is—when does remodeling become pathologic? A controlled exercise study¹⁷ has shown that treadmill exercise causes increased subchondral bone remodeling and mild radiographic changes in the navicular bone of normal horses. Further longitudinal studies with this model may provide the answer to the above question.

Third, histopathologists have determined that the soft tissues of the navicular apparatus are intimately involved,^{1,6,13} but we cannot evaluate them on conventional radiographs! The literature regarding the importance of pathologic changes of soft tissues in NS is convincing. There is some evidence⁸ that by the time good radiographic criteria of NS are present, many underlying soft tissue changes may be advanced beyond our ability to effectively treat them. The need to explore and develop imaging modalities such as MR imaging and CT, which better reveal soft tissue and bone structure, is critical. Use of these modalities shows promise for earlier diagnosis and therapeutic intervention. Although an invasive procedure, navicular bursography may be helpful in identifying changes in the bursa of horses with NS.¹⁸

Finally, NS is not a disease, but a complex, multifactorial condition associated with a constellation of morphologic, pathophysiologic, and radiographic changes. It is unknown whether or not these abnormalities may represent a continuum or a group of unrelated events.³ Again, soft tissue imaging would be helpful in further understanding the sequence of changes and instituting effective therapy. It is unreasonable to expect a diagnostic test like radiography to be specific for NS and the same can be said for its treatment. However, radiology should not be completely condemned because it does help to substantiate a diagnosis of NS and it excludes other diseases that may cause heel pain.¹⁹

5. Conclusion

Based on numerous studies of radiographic changes of NS and current knowledge of bone physiology, radiography still has a place in evaluation of horses with NS. The best approach is to look at the entire spectrum of navicular changes when making the determination as to whether or not radiography supports a diagnosis of NS. The more changes, the more likely the horse has NS. In the authors' view, the most reliable signs are those seen in the flexor cortex (e.g.,

erosions, mineralization of the deep digital flexor) and those in the medullary cavity (medullary sclerosis and cyst-like areas). It is noteworthy that when these areas are diseased, most other changes like abnormal synovial fossae, enthesopathy, etc., are also evident. When there are few isolated changes of NS, caution is advised when assigning significance to radiographic changes. Last but not least—radiographic changes of NS should always be interpreted in light of a thorough lameness examination and the work history of the horse.

Obviously, CT and MR imaging are superior to standard radiography for evaluating NS. Presently, they are not cost effective and require general anesthesia. Standing MR units are being developed for imaging the human knee and may eventually become available for equine use, eliminating the need for general anesthesia. A technique for diagnostic ultrasonography has recently been developed for imaging the normal equine navicular apparatus and may be useful for evaluating NS.²⁰

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