Review Article

Navicular Stress Fractures

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ABSTRACT

Navicular stress fractures are multifactorial injuries due to chronic overload on the navicular, particularly in young athletes. The navicular is subject to unique stresses and has a complex blood supply, making it susceptible to stress fractures and potentially delayed union or nonunion. Expeditious diagnosis is critical to prevent a delay in treatment and a poor outcome. Advanced imaging is essential in making the diagnosis and monitoring healing. Both nonsurgical and surgical treatments have demonstrated good results. Nonsurgical management consists of a period of immobilization and nonweight bearing, and surgical management typically involves open reduction and internal fixation. Patients need to be appropriately counseled regarding expectations for these challenging injuries.

Navicular stress fractures were first described by Towne et al[1] in 1970. Although rare in the general cohort, estimates suggest up to 35% of all foot and ankle stress fractures occur in the navicular.[2,3] Diagnosis has increased in incidence over time likely because of improved symptom recognition and advanced imaging. This injury is most common in running and jumping athletes, especially short distance runners and basketball players.[4-6] In a recent systematic review, Mallee et al[7] found 98.5% of stress fractures were in athletes, and most were men in their mid-20s. The diagnosis can be challenging and is often markedly delayed,[8,9] resulting in pain, disability, or prolonged return to work or sport. Understanding the pathophysiology, risk factors, diagnosis, and management of navicular stress fracture are critical for achieving optimal outcomes in these patients.

Anatomy and Biomechanics

The tarsal navicular bone derives its name from the latin word *navicularis*, referring to its "boat-like" morphology. It is located between the talus and the three cuneiforms on the medial aspect of the foot. Its primary axis is in an oblique orientation in the lateromedial and dorsoplantar directions.[10] Proximally, it is biconcave, completely covered with articular cartilage, and articulates with the talus. The concavity of this surface can vary markedly. On its convex distal aspect are three articular surfaces, the largest articulating with the medial cuneiform. The three articular surfaces converge plantarly to create the transverse tarsal arch.[10] Its dorsal surface is also convex and

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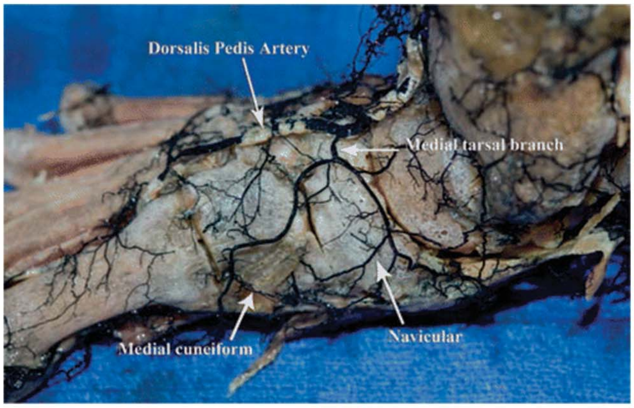
Figure 1



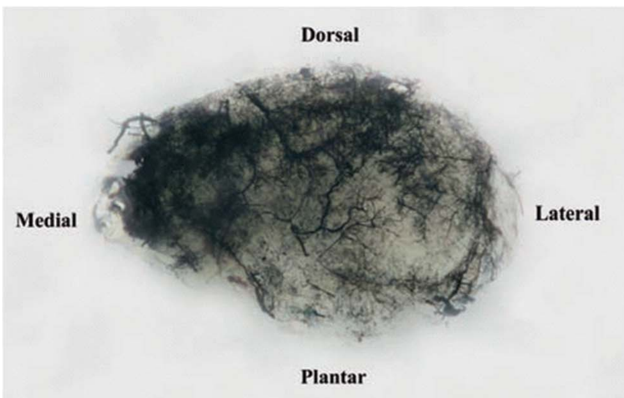
Cadaveric dissection of the navicular demonstrating the blood supply to the navicular. (Reproduced with permission from McKeon KE, McCormick JJ, Johnson JE, Klein SE: Intraosseous and extraosseous arterial anatomy of the adult navicular. *Foot Ankle Int* 2012;33[10]:857-861.)

provides several capsular and ligamentous attachment sites. Medially, the navicular tuberosity is the most prominent structure and serves as the attachment site for the plantar and medial navicular ligaments, as well as the tibialis posterior tendon. In up to 21% of people, an accessory navicular ossicle can occur in this region.[11] The lateral surface is small, convex, and inconsistently articulates with the cuboid. The bifurcate ligament inserts superolaterally. Dorsally, the navicular is convex and has several capsular and ligamentous attachments. The plantar side is concave and contains the attachment site of the plantar calcaneonavicular (spring) ligament—the plantar beak.

A relatively poor blood supply increases the risk of stress fractures and nonunions in the navicular. It receives contributions from the dorsalis pedis and posterior tibial arteries. The medial tarsal branch of the dorsalis pedis supplies the dorsal aspect, and a branch of the posterior tibial artery supplies the medial and plantar aspects (Figure 1). In 1958, Waugh[12] first described an avascular zone in the central one-third of the navicular that was long hypothesized to increase susceptibility to stress fractures. More recently, McKeon et al[13] performed a cadaveric study and determined 58.8% of naviculars had no zones of avascularity (Figure 2). Only 11.8% of naviculars had the classic dorsal and central zone of hypovascularity, suggesting other factors may also contribute to stress fractures (Figure 3).

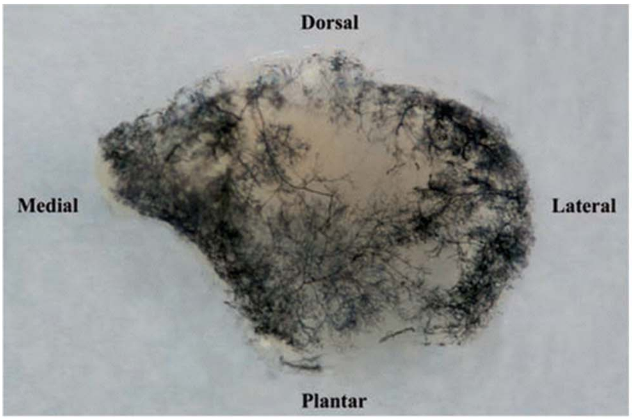
The transverse tarsal joint consists of the talonavicular and calcaneocuboid joints, and work in concert with the

Figure 2



Photograph of an example of a well-vascularized navicular without evidence of hypovascularity. (Reproduced with permission from McKeon KE, McCormick JJ, Johnson JE, Klein SE: Intraosseous and extraosseous arterial anatomy of the adult navicular. *Foot Ankle Int* 2012;33[10]:857-861.)

Figure 3



Photograph of an example of a navicular with central hypovascularity extending to the dorsal cortex. (Reproduced with permission from McKeon KE, McCormick JJ, Johnson JE, Klein SE: Intraosseous and extraosseous arterial anatomy of the adult navicular. *Foot Ankle Int* 2012;33[10]:857-861.)

tibiotalar and subtalar joints throughout the gait cycle. During normal gait, the transverse tarsal joint locks when the subtalar joint is inverted, leading to a rigid midfoot and hindfoot during toe-off. It unlocks when the subtalar joint is everted by bringing the axes of the joints parallel to one another, creating a flexible midfoot and hindfoot for heel strike.[14] Failure of the talonavicular complex can lead to collapse of the medial longitudinal arch causing abduction of the forefoot and valgus deformity of the subtalar joint.[15] The location of the navicular between the talar head and cuneiforms predisposes it to unique stresses that may account for its pathologies. The navicular transmits forces from the first and second metatarsocuneiform joints during foot strike and shares some of these forces with the talar head medially.[16] The lateral aspect of the navicular does not share these forces, leading to shear from the second metatarsal and middle cuneiform forces. The posterior tibial tendon also contributes to tension medially during contraction. These net forces lead to shear stress coursing through the middle third of the bone.

Pathogenesis and Risk Factors

The pathogenesis of navicular stress fractures is multifactorial, and despite significant research is still incompletely understood. These injuries are thought to be a chronic overuse phenomenon due to intense activity without adequate recovery. Repetitive loading of the bone predisposes to microfractures, and ultimately stress fractures. Hence, these fractures are more common in explosive athletes, particularly with sprinting and repetitive jumping exercises. As mentioned previously, the variable blood supply of the navicular likely contributes as well. The relatively hypovascular central third of the bone in combination with higher shear forces in this location weakens the bone. Patients who develop navicular stress fractures likely have some combination of genetic, anatomic, lifestyle, and biomechanical risk factors.

Numerous risk factors have been identified leading to navicular stress fractures. First are factors intrinsic to the patient. These include a history of previous stress fractures, unfavorable foot biomechanics, genetics, and female sex.[17] Navicular stress fractures most commonly occur in young male athletes, but female sex is an independent risk factor for stress fractures. Biomechanical risk factors include metatarsus adductus, equinus contracture, limited subtalar ankle motion, and a short first metatarsal and long second metatarsal.[2,18-20] Reduced ankle dorsiflexion has also been implicated, possibly due to a compensatory increase in navicular excursion leading to impingement.[21] The reduced ankle dorsiflexion can be secondary to achilles contractures, however, can also be due to anterior ankle impingement secondary to an osteophyte. The radiographs must be analyzed critically to look for impingement. A recent study by Becker et al[22] compared foot kinematics between runners with and without a history of navicular stress fractures. They identified several alterations in foot kinematics that correlate with navicular stress fracture, including decreased plantar flexion range of motion, greater hindfoot eversion, and reduced forefoot abduction excursion. Ultimately, an inflexible foot may predispose to these injuries, due to increased stress across the navicular.

Extrinsic factors may also contribute to navicular stress fractures. These include rigorous training regimens, improper shoe wear, and poor nutrition.[6] A randomized controlled trial in military trainees showed that custom orthoses significantly reduced the incidence of lower extremity stress fractures.[23] Barrack et al[24] demonstrated increased stress fracture risk in women "with the female athlete triad." Specifically, there was an increased risk with BMI <21 kg/m^3, oligomenorrhea or amenorrhea, elevated dietary restraint, low bone mineral density, and ≥12 hr/wk of purposeful exercise. In patients with multiple comorbid factors, the relative risk increase of navicular stress fractures can be significant.

Clinical Presentation and Imaging

Diagnosing navicular stress fractures can be challenging. The presentation is often nonspecific and insidious, leading to a delay in diagnosis of over 6 months.[8,25,26] Initial symptoms include vague medial midfoot pain and tenderness that occurs only with weight bearing or sport-specific activities. The point of maximal tenderness has been described as the prominence over the proximal and dorsal navicular, called the "N-spot."[27] (Figure 4) The pain progresses over time to all activities and can even occur at night. On physical examination, the patient will have pain with tenderness at the "N-spot" and medial midfoot without any neurologic symptoms. Range of motion is typically normal but can have minimal reduction in dorsiflexion or pain with tarsal abduction/adduction or forefoot inversion/eversion.[9] Positive provocative maneuvers include pain with a single-limb heel rise or single leg hop.[9,28,29]

Imaging should always begin with plain radiographs to evaluate for fractures and rule out other pathology. Three weight-bearing views of both the foot and ankle should be obtained, although these are often normal. In a 1992 study, Khan et al[30] demonstrated that plain radiographs were positive in only 18% (14/77) patients with CT confirmed stress fractures. This is due to the significant osteoclastic resorption that needs to occur before radiographic changes, which can take up to 3 weeks.[31] Radiographic evidence is most often an incomplete fracture that does not penetrate the plantar cortex. If x-rays are negative and clinical suspicion is high, advanced imaging is indicated.

Triple-phase bone scan, CT scan, and MRI are much more sensitive in detecting navicular stress fractures than plain radiographs. Triple-phase bone scans have been shown to have up to 100% sensitivity, but lack specificity.[6,19] In addition, the fracture pattern cannot be accurately assessed on a bone scan, therefore CT and MRI are used more frequently. CT scans provide the best boney detail for visualization of fractures lines and can aid in preoperative planning. They offer higher fracture resolution, visualization of coalitions, sclerosis, cystic changes, and are less expensive than an MRI.[27,28] Furthermore, CT scans are useful to monitor fracture healing. The first sign of healing is

Figure 4

Photograph of the clinical depiction of the "N-Spot."

Table 1. Depicting the Classification as Described by Saxena et al of Navicular Stress Fractures

Saxena CT Classification		
Type 0.5	Normal CT, evidence of stress reaction on MRI	Figure 7
Type 1	Dorsal cortex fracture	Figure 8
Type 2	Fracture extends to midpoint of navicular	Figure 9
Type 3	Both cortexes affected	Figure 10

typically radiographic and can be seen as early as six weeks after injury.[21] Consolidation should be present by 3 to 4 months, but dorsal notching may persist for years later.[8] One classification system was developed by Saxena et al[32,33] and is based on CT interpretations (Table 1, which includes Figures 5–8) as follows:

Type 0.5: stress reaction on MRI
Type 1: dorsal cortex fracture
Type 2: fracture propagating into the navicular body
Type 3: fracture propagating into another cortex or complete fracture

MRI is considered the benchmark in diagnosing navicular stress fractures and is recommended in patients with high suspicion and negative radiographs due to its high sensitivity.[6,34] MRI can detect early bone edema and medullary extension better than CT scans, which can improve detection of early stress reactions and possibly prevent progression to a stress fracture.[27,28] We recommend initial CT scan for diagnosis, as serial CT scans can be useful to assess healing. However, if negative for fracture, MRI is recommended to look for a stress reaction.

Figure 5

Photograph of the operating room setup with intraoperative three-dimensional imaging to aid in appropriate placement of fixation.

Although rare, it is important to consider Mueller-Weiss syndrome in the differential diagnosis of suspected navicular stress fractures. Mueller-Weiss syndrome is a spontaneous adult-onset osteonecrosis of the navicular. Its etiology is multifactorial, and its clinical symptoms can mimic navicular stress fractures. On imaging, plain radiographs may show a comma-shaped deformity and depending on stage, collapse of the navicular. It may occur bilaterally. Treatment for this disease is typically conservative, with surgical management reserved for refractory cases with decompression and bone grafting.

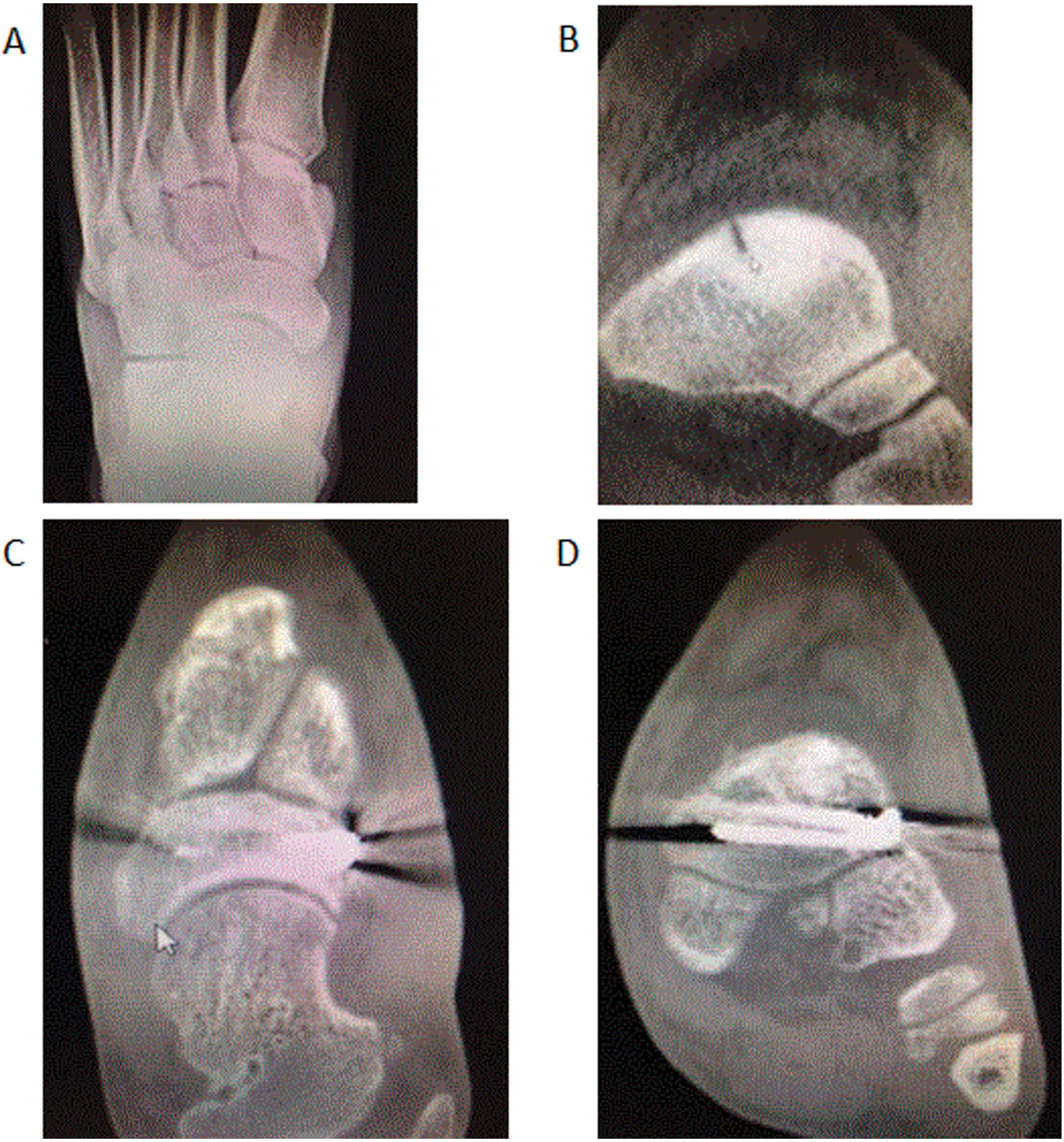
Management

The treatment of navicular stress fractures is challenging and depends on the fracture pattern, degree of displacement, comorbidities, level of activity, and surgeon preference. Historically, stress fractures were treated with nonsurgical management, but recently indications have expanded to help improve union rates and increase return to activity, especially in high-level athletes. Some authors have suggested treatment based on the classification scheme,[9,32] but many studies do not make a differentiation. Reasonable results have been reported with both surgical and nonsurgical modalities. Unfortunately, the ideal management for all navicular stress fractures is unknown due to a lack of high-quality studies in the literature.

Nonsurgical Treatment

Nonsurgical management typically consists of immobilization in a cast or boot with the patient made non-weight bearing. In 1982, Torg et al[8] published their findings in 21 patients with navicular stress fractures. In patients with non-displaced or displaced fractures, they reported excellent success with 6 weeks of cast immobilization and non-weight bearing. Seven patients that were treated with weight-bearing casts or earlier activity developed complications. The average return to sport in this group of predominantly young males was 4.9 months. The same author more recently published an abstract of a meta-analysis of 250 cases treated

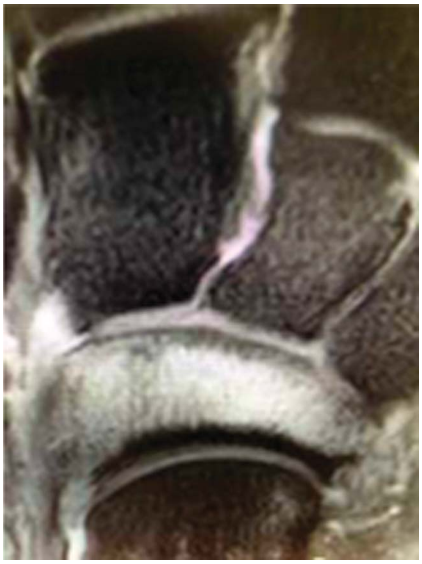
Figure 6



Preoperative radiographs and CT scan (A and B) and postoperative CT scan demonstrating fixation of the navicular stress fracture (C and D).

nonsurgically and noted 96% success.[35] Further studies have demonstrated similar findings advocating for strict non-weight bearing protocols. Khan et al[30] reviewed 86 patients with navicular stress fractures and demonstrated 86% healing with return to full activity at 5.6 months in those that were made non-weight bearing for a minimum of 6 weeks. In contrast, they reported a mere 20% success in the group prescribed weight bearing casts or less than 6 weeks non-weight bearing. Torg et al[26] did not find a statistically significant difference between successful non-surgical and surgical management, but there was a trend favoring immobilization and non-weight bearing (96%

Figure 7

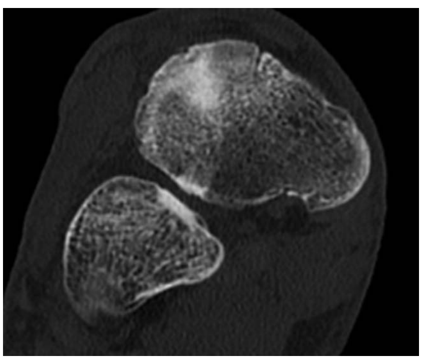


MRI example of a type 0.5 navicular stress fracture (coronal cut).

success vs 82% success). However, it is important to consider the indications for surgical intervention could indicate a more challenging fracture. Again, they showed weight bearing had substantially worse clinical outcomes compared with non-weight bearing.

It is important to consider the risks and benefits of a hard cast versus a removable boot. A cast is the typical recommendation; however, we rarely employ these in high level athletes due to "cast disease." This can lead to significant stiffness and atrophy of the calf. Also, it can be challenging to use some bone stimulators or shockwave therapy with a hard cast employed. A boot allows the patient to maintain ankle range of motion and lessen calf atrophy. The decision of a cast versus boot must be patient specific, as compliance of non-weight bearing is critical to healing.

Figure 8



CT example of a type 1 navicular stress fracture (coronal cut).

Other nonsurgical modalities have been proposed including the use of low intensity pulsed ultrasonography, extracorporeal shockwave therapy, vitamin D, bone marrow aspirate,[36] and teriparatide.[6]

Currently, there is no literature on the treatment of navicular stress fracture using teriparatide. However, previous literature has demonstrated that the use of intermittent recombinant parathyroid hormone improved quality and strength of fracture callus in a randomized, placebo-controlled trial, and that those benefits remained even after the medication was stopped.[37] In another small randomized control trial, intermittent recombinant parathyroid hormone led to increased bone formation markers and better healing on MRI for lower extremity stress fractures in premenopausal women.[38] While the use has not been studied specifically for navicular stress fracture, there is enough evidence on its positive effects on bone formation, that treatment should be considered. It is important to note that the use of teriparatide for this indication is considered off-label.

Currently, no literature exists on the use of shock wave therapy in treating stress fractures; however, it has been studied in the treatment of non-unions with positive results.[39-41] The use of this is non-invasive, and has the potential to accelerate the healing process. Barring issues with cost (treatment is often not covered by insurance), we advocate for its use in the treatment of navicular stress fractures (both surgically and nonsurgically), along with external bone stimulation.

Operative Treatment

Operative treatment typically involves a medial approach spanning the talonavicular joint. A lateral-based incision can be considered in more lateral fractures or to assist in hardware positioning. Determination of incision is based on the positioning of the fracture. If only a small dorsal cortex fracture is present, bone grafting and closure can often be sufficient to heal the fracture. However, we advocate for placement of fixation across the navicular to prevent propagation of the fracture. Open reduction and internal fixation (ORIF) is most commonly performed with 1 to 2 partially-threaded headed 4.0 or 4.5 mm cannulated screws across the fracture site to achieve compression. Postoperative protocols commonly being with 2 weeks of splint immobilization, followed by conversion to a cast or boot with complete non-weight bearing for the first 6 weeks. Gradual, progressive weight bearing in a walking boot or cast begins after 6 weeks, and full weight bearing begins typically between 8 and 12 weeks and is guided by symptoms as well as imaging. In high level athletes, return to sport is possible as early as 4 to

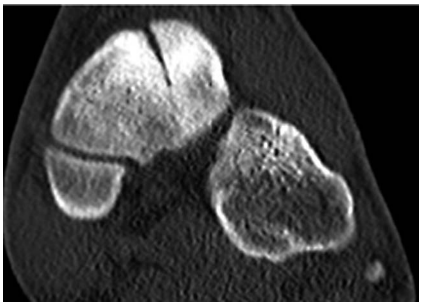
6 months. Surgical management is recommended in complete or displaced fractures, elite athletes, and patients with high levels of activity.[6,9,28] Although initial displacement may play a significant role in selecting an appropriate treatment option, many studies do not differentiate between nondisplaced and displaced fractures. This makes assessing the optimal management more challenging. Saxena and Gross propose surgical management for type 2 and type 3 fractures due to their higher risk of failure, and reported excellent outcomes in surgically treated patients.[9,32] McCormick et al[25] noted 80% union in surgically treated navicular stress fractures, but 50% nonunion in those that were displaced. Therefore, surgical management is likely beneficial in displaced fractures or those with significant fracture extension. One impetus for surgical intervention is the possibility of earlier return to sport. In the aforementioned studies by Saxena, surgical management resulted in a similar or quicker return to sport than nonsurgical treatment. Mallee et al[7] completed a systematic review of 200 navicular stress fractures and found the mean return to sport in surgically treated patients was 16.4 weeks compared with 21.7 weeks treated conservatively, but no statistical analysis was performed. Overall, the literature is mixed on the optimal treatment of navicular stress fractures. There may be a benefit to surgical management in type 2 and type 3 fractures, especially in high-level athletes, but further high-quality studies are needed to determine this. Healing of the injury is typically followed by CT scan, with complete healing required before full release.

Authors' Preferred Surgical Technique

The authors' preferred technique involves the use of three-dimensional imaging, bone marrow aspirate, bone autograft, and one or two cannulated screws. Three-dimensional imaging may improve the accuracy of hardware positioning, but is not required. We have found it useful in reducing implant malposition, and it is readily available at our facilities. The patient is positioned supine on the operating table, and a popliteus block is performed. Bone marrow aspirate is obtained from the iliac crest under sterile technique, and concentrated with a centrifuge down to 5 mL. Six bone graft sleeves are also obtained through the same entry site.

Small stab incisions are made over the medial aspect of the navicular using intraoperative fluoroscopy. Smooth Kirschner wires are advanced under image-guidance from medial to lateral in the navicular, trying to get a perpendicular as possible to the fracture line. Three-dimensional imaging (Figure 9) is then obtained with a CT scan to confirm the position of the Kirschner wire. A 2.7 mm drill bit is used to drill over the wire, and then a 3.5 mm bit is used to overdrill the near cortex to create compression by technique. A few milliliters of the iliac crest bone marrow aspirate is placed into the fracture site. Additional bone marrow aspirate can be placed in the dorsum of the navicular over the fracture site through a small stab incision once the bone has been scraped and prepared. Six sleeves of iliac crest bone autograft are lightly packed into the drilled hole. A 3.5 mm partially-threaded cannulated screw is placed in compression. A second Kirschner wire and screw can be placed if there is adequate space. A final three-dimensional CT scan is performed intraoperatively to confirm position (Figure 10). The wounds are closed in multiple layers, and the patient is initially immobilized in a splint or boot and made non-weight bearing.

Figure 9



CT example of a type 2 navicular stress fracture (coronal cut).

Current Recommendations

Currently, our recommendations are for surgical treatment for all primary and recurrent navicular stress fractures in athletes, with the exception of a type 0.5 (stress reaction), which can be treated with a period of non-weight bearing in a boot. In the athletic cohort (competitive, elite,

Figure 10

CT example of a type 3 navicular stress fracture (coronal cut).

or high-level recreational), we perform ORIF with one versus two screws, as well as bone marrow aspirate concentrate and autograft bone, as above. CT scan is performed at 6 and 10 weeks. Once the CT scan demonstrates healing, the athlete can return to play. We recommend adjuvant therapy with vitamin D, Calcium, daily bone stimulator, and biweekly shockwave therapy. Due to its noninvasive nature, we recommend the addition of shockwave therapy to all patients, if feasible. If there is evidence of delayed union (>3 months), or nonunion, we recommend the addition of teriparatide, however the use of it for this indication is considered off label.

In the non-athletic cohort, a trial of non-weight bearing can be attempted (boot versus cast) and the adjuvant therapy above should be implemented. Repeat CT scan at 6 and 10 weeks are to be performed and return back to activity occurs after documented healing (both radiographically and clinically).

In cases of recurrent navicular stress fractures, we recommend surgical treatment with ORIF with one to two screws, as well as bone marrow aspirate concentrate and autograft bone. The postoperative course on these patients is similar to above, however these patients must be followed with CT scans until complete healing.

Complications and Outcomes

Complications of navicular stress fractures include pain, stiffness, delayed union, nonunion, arthritis, osteonecrosis, and navicular collapse resulting in a deformity. Sequelae of these complications can lead to significant morbidity for patients, particularly when causing an inability to return to previous levels of activity. Nonunion is of particular concern in stress fractures. Nonunion rates have been reported at 20%, and higher in completely displaced fractures.[25] These are difficult problems to treat, and many patients require revision ORIF based on their symptoms. Conservative management can be considered with custom midfoot orthoses. Successful surgical treatment has been reported but is limited to case reports. Vascularized bone grafts have been proposed as a viable treatment modality, but further research is needed in this area.[42] Vascularized bone grafts are typically reserved for nonunions, or evidence of osteonecrosis of the navicular.

Overall, patient reported outcome measures in successfully treated patients are high. As previously discussed, success rates have been reported over 90% with both surgical and nonsurgical treatments. There is a paucity of long-term outcome data on navicular stress fractures in the literature. In 2006, Potter et al[43] reviewed 32 navicular stress fractures and found no significant differences between surgical and conservative management at a mean of 10 years post-injury, however, surgically treated patients had more tenderness over the dorsal navicular even after return to sport years later. Patient reported outcome measures were obtained via a modified version of the Midfoot Scale (developed by the American Orthopaedic Foot and Ankle Society). Seventy-three percent of patients reported a score of at least 50 out of a maximum 60 points. There was no difference between the groups in pain, function, or CT findings between surgically and nonsurgically treated patients.

Summary

Navicular stress fractures are relatively uncommon injuries that predominantly occur in running or jumping athletes. Their diagnosis is challenging and is often delayed, and the ideal treatment is controversial. Success has been demonstrated with both surgical and nonsurgical treatments. Immobilization and strict nonweight bearing for 6 weeks with gradual progression is the treatment of choice for early and nondisplaced fractures (type 0.5, type 1). Surgical treatment may benefit high level athletes and type 2 or 3 injuries with possible improved union rates and early return to sport. In displaced fractures, nonunions are a major problem and can lead to further surgery and worse outcomes. Ultimately, high-quality prospective, randomized trials are needed to elucidate the optimal treatment method. Patients should be counseled on the significance of these injuries and appropriate expectations should be set to improve patient outcomes.

References

References printed in **bold type** are those published within the past 5 years.

1. Towne L, Blazina M, Cozen L: Fatigue fracture of the tarsal navicular. *J Bone Joint Surg* 1970;52:376-378.

2. Bennell KL, Malcolm SA, Thomas SA, et al: Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *Am J Sports Med* 1996;24:810-818.

3. Brukner P, Bradshaw C, Khan KM, White S, Crossley K: Stress fractures: A review of 180 cases. *Clin J Sport Med* 1996;6:85-89.

4. Mann JA, Pedowitz DI: Evaluation and treatment of navicular stress fractures, including nonunions, revision surgery, and persistent pain after treatment. *Foot Ankle Clin* 2009;14:187-204.

5. Snyder RA, Koester MC, Dunn WR: Epidemiology of stress fractures. *Clin Sports Med* 2006;25:37–52, viii.

6. Shakked RJ, Walters EE, O'Malley MJ: Tarsal navicular stress fractures. *Curr Rev Musculoskelet Med* 2017;10:122-130.

7. Mallee WH, Weel H, van Dijk CN, et al: Surgical versus conservative treatment for high-risk stress fractures of the lower leg (anterior tibial cortex, navicular and fifth metatarsal base): A systematic review. *Br J Sports Med* 2015;49:370-376.

8. Torg JS, Pavlov H, Cooley LH, et al: Stress fractures of the tarsal navicular. A retrospective review of twenty-one cases. *J Bone Joint Surg Am* 1982;64:700-712.

9. Gross CE, Nunley JA: Navicular stress fractures. *Foot Ankle Int* 2015;36: 1117-1122.

10. Golano P, Fariñas O, Sáenz I: The anatomy of the navicular and periarticular structures. *Foot Ankle Clin* 2004;9:1-23.

11. Tsuruta T, Shiokawa Y, Kato A, et al: Radiological study of the accessory skeletal elements in the foot and ankle (author's transl) [Japanese]. *Nippon Seikeigeka Gakkai Zasshi* 1981;55:357-370.

12. Waugh W: The ossification and vascularisation of the tarsal navicular and their relation to Köhler's disease. *J Bone Joint Surg Br* 1958;40-B: 765-777.

13. McKeon KE, McCormick JJ, Johnson JE, Klein SE: Intraosseous and extraosseous arterial anatomy of the adult navicular. *Foot Ankle Int* 2012; 33:857-861.

14. Elftman H: The transverse tarsal joint and its control. *Clin Orthop Relat Res* 1960;16:41-46.

15. Sammarco VJ: The talonavicular and calcaneocuboid joints: Anatomy, biomechanics, and clinical management of the transverse tarsal joint. *Foot Ankle Clin* 2004;9:127-145.

16. van Langelaan EJ: A kinematical analysis of the tarsal joints. An X-ray photogrammetric study. *Acta Orthop Scand Suppl* 1983;204:1-269.

17. Wright AA, Taylor JB, Ford KR, Siska L, Smoliga JM: Risk factors associated with lower extremity stress fractures in runners: A systematic review with meta-analysis. *Br J Sports Med* 2015;49:1517-1523.

18. Kitaoka HB, Lundberg A, Luo ZP, An KN: Kinematics of the normal arch of the foot and ankle under physiologic loading. *Foot Ankle Int* 1995; 16:492-499.

19. Pavlov H, Torg JS, Freiberger RH: Tarsal navicular stress fractures: Radiographic evaluation. *Radiology* 1983;148:641-645.

20. Ingalls J, Wissman R: The os supranaviculare and navicular stress fractures. *Skeletal Radiol* 2011;40:937-941.

21. Hossain M, Clutton J, Ridgewell M, Lyons K, Perera A: Stress fractures of the foot. *Clin Sports Med* 2015;34:769-790.

22. Becker J, James S, Osternig L, Chou LS: Foot kinematics differ between runners with and without a history of navicular stress fractures. *Orthop J Sports Med* 2018;6:2325967118767363.

23. Franklyn-Miller A, Wilson C, Bilzon J, McCrory P: Foot orthoses in the prevention of injury in initial military training: A randomized controlled trial. *Am J Sports Med* 2011;39:30-37.

24. Barrack MT, Gibbs JC, De Souza MJ, et al: Higher incidence of bone stress injuries with increasing female athlete triad–related risk factors: A prospective nultisite study of exercising girls and women. *Am J Sports Med* 2014;42:949-958.

25. Mccormick JJ, Bray CC, Davis WH, Cohen BE, Jones CP, Anderson RB: Clinical and computed tomography evaluation of surgical outcomes in tarsal navicular stress fractures. *Am J Sports Med* 2011;39:1741-1748.

26. Torg JS, Moyer J, Gaughan JP, Boden BP: Management of tarsal navicular stress fractures: Conservative versus surgical treatment: A meta-analysis. *Am J Sports Med* 2010;38:1048-1053.

27. Burne SG, Mahoney CM, Forster BB, Koehle MS, Taunton JE, Khan KM: Tarsal navicular stress injury: Long-term outcome and clinicoradiological correlation using both computed tomography and magnetic resonance imaging. *Am J Sports Med* 2005;33:1875-1881.

28. Ramadorai MU, Beuchel MW, Sangeorzan BJ: Fractures and dislocations of the tarsal navicular. *J Am Acad Orthop Surg* 2016;24: 379-389.

29. Kahanov L, Eberman LE, Games KE, Wasik M: Diagnosis, treatment, and rehabilitation of stress fractures in the lower extremity in runners. *Open Access J Sports Med* 2015;6:87-95.

30. Khan KM, Fuller PJ, Brukner PD, Kearney C, Burry HC: Outcome of conservative and surgical management of navicular stress fracture in athletes: Eighty-six cases proven with computerized tomography. *Am J Sports Med* 1992;20:657-666.

31. de Clercq PFG, Bevernage BD, Leemrijse T: Stress fracture of the navicular bone. *Acta Orthop Belg* 2008;74:725-734.

32. Saxena A, Fullem B, Hannaford D: Results of treatment of 22 navicular stress fractures and a new proposed radiographic classification system. *J Foot Ankle Surg* 2000;39:96-103.

33. Saxena A, Fullem B, Torg JS: Letter to the editor. *Am J Sports Med* 2010;38:NP3-NP5.

34. Mandell JC, Khurana B, Smith SE: Stress fractures of the foot and ankle, part 2: Site-specific etiology, imaging, and treatment, and differential diagnosis. *Skeletal Radiol* 2017;46: 1165-1186.

35. Torg JS, Pavlov H, Roberts MM, Eremus J, Drakos MC, Arnoczky SP: The tarsal navicular stress fracture revisited: The unequivocal case for conservative, non-surgical management. *Foot Ankle Orthop* 2016;1: 2473011416S00018.

36. Gianakos A, Ni A, Zambrana L, Kennedy JG, Lane JM: Bone marrow aspirate concentrate in animal long bone healing: An analysis of basic science evidence. *J Orthop Trauma* 2016;30:1-9.

37. Alkhiary YM, Gerstenfeld LC, Krall E, et al: Enhancement of experimental fracture-healing by systemic administration of recombinant human parathyroid hormone (PTH 1-34). *J Bone Joint Surg Am* 2005;87:731-741.

38. Almirol EA, Chi LY, Khurana B, et al: Short-term effects of teriparatide versus placebo on bone biomarkers, structure, and fracture healing in women with lower-extremity stress fractures: A pilot study. *J Clin Transl Endocrinol* 2016;5:7-14.

39. Birnbaum K, Wirtz D, Siebert C, Heller K: Use of extracorporeal shock-wave therapy (ESWT) in the treatment of non-unions. *Arch Orthop Trauma Surg* 2002;122:324-330.

40. Haffner N, Antonic V, Smolen D, et al: Extracorporeal shockwave therapy (ESWT) ameliorates healing of tibial fracture non-union unresponsive to conventional therapy. *Injury* 2016;47:1506-1513.

41. Kuo SJ, Su IC, Wang CJ, Ko JY: Extracorporeal shockwave therapy (ESWT) in the treatment of atrophic non-unions of femoral shaft fractures. *Int J Surg* 2015;24:131-134.

42. Toren AJ, Hahn DB, Brown WC, Stone PA, Ng A: Vascularized scapular free bone graft after nonunion of a tarsal navicular stress fracture: A case report. *J Foot Ankle Surg* 2013;52:221-226.

43. Potter NJ, Brukner PD, Makdissi M, Crossley K, Kiss ZS, Bradshaw C: Navicular stress fractures: Outcomes of surgical and conservative management. *Br J Sports Med* 2006;40:692-695.