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SYSTEMATIC REVIEW

Stress Fractures about the Foot and Ankle in Athletes

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ABSTRACT

Stress fractures of the foot and ankle are a common problem encountered by athletes of all levels and ages. This review summarizes the history and physical exam findings, radiographic imaging, diagnosis and treatment of stress fractures of the foot and ankle in those participating in athletic activities.

These injuries can be divided into low- and high-risk based upon their propensity to heal without complication. A wide variety of nonoperative strategies are employed based on the duration of symptoms, type of fracture, and patient factors such as activity type, desire to return to sport and compliance. Operative management has proven superior in several high risk types of stress fractures. Evidence on pharmacotherapy and physiologic therapy such as bone stimulators is evolving.

A high index of suspicion for stress fractures is appropriate in many high-risk groups of athletes with lower extremity pain. Proper and timely workup and treatment is successful in returning these athletes to sport in many cases. Education of athletes as well as their families, training and coaching staff is important. Attention to training regimens, technique, equipment and proper nutrition is paramount in the prevention of these injuries.

Keywords: Stress fracture, Athlete, Foot, Ankle.

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INTRODUCTION, EPIDEMIOLOGY AND BASIC SCIENCE

Stress fractures are relatively uncommon injuries, accounting for approximately 1 to 7% of all athletic injuries.[1,2] The incidence of these injuries is rising due to earlier and longer participation in sports, the emergence of more extreme sporting activities, and a heightened awareness of the diagnosis.[3]

In contrast to acute fractures which typically occur with a single maximal load, stress fractures occur due to repetitive, submaximal loading of a bone leading to micro-fractures that are unable to heal due to bone resorption and formation imbalances.[1] A bone responds to supra-physiologic stress (whether repetitive or acute in nature) on a continuum from a stress reaction to a fracture.[1,4] The initial stage of bone failure is generally called a stress reaction. This diagnosis is usually made in a symptomatic patient who has bone scan or magnetic resonance imaging (MRI) evidence of bone reactive changes without a true fracture line.[1,4] If the repetitive loading continues, the stress reaction can progress to a true stress fracture.[1,4] This mechanism of injury explains the higher incidence of stress fractures among military recruits, runners, and those involved in jumping sports, though any activity with repetitive loading can lead to stress fractures.[1,2,4-6] Most bones have reported cases of stress fractures in the literature, but the lower extremities have the highest prevalence.[1] In a study of 320 athletes, the tibia (49.1%), tarsals (25.3%), and metatarsals (8.8%) were the most frequently involved bones affected by a stress fracture.[7]

Table 1: Intrinsic and extrinsic factors related to stress fractures of the foot and ankle[34,45,51]

Intrinsic factors	*Extrinsic factors*
Cavus feet	Type of activity
Leg length discrepancies	Excessive/new training regimen
Excessive forefoot varus	Poor equipment/footwear
Tarsal coalitions	Improper technique
Prominent posterior calcaneal process	Type of training surface
Tight heel cords	Sleep deprivation
Osteopenia/osteoporosis	
Poor vascular supply	
Abnormal hormonal levels	

Intrinsic and extrinsic etiologies of a stress fracture have been described (Table 1). Intrinsic factors consist of the patient's anatomy and biology including cavus feet, leg length discrepancies, excessive forefoot varus tarsal coalitions, a prominent posterior calcaneal process, tight heel cords, poor bone density or vascular supply, and abnormal hormonal levels.[2,3,8] Extrinsic factors such as type of activity, excessive or new training regimens, equipment and footwear issues, training surfaces and techniques, and nutrition can also play a role.[2,3,8] Several studies have described the biomechanical effects of muscle fatigue on the development of stress fractures.[9,10] Muscle fatigue can result in changes in the normal loading patterns of the foot and is postulated to be a factor in stress-related injuries.[10]

Females have a higher incidence of stress fractures.[2,8,11-13] A wider pelvis and higher incidence of genu valgum result in a compensatory increased Q-angle and foot pronation.[3,14] On average, females have 25% less muscle mass than males. This can result in increased tension on smaller areas of bone due to less muscle to dynamically distribute the force.[3] The 'female athlete triad' (eating disorder, amenorrhea and osteoporosis) is found among competitive female athletes, particularly those involved in long distance running, figure skating, and gymnastics.[2,14,15] High level endurance athletes from both genders are in danger of osteoporosis based on

Table 2: High and low-risk stress fractures of the foot and ankle[12,13,34]

High risk	*Low risk*
Medial malleolus	Calcaneus
Talus	Cuboid
Navicular	Cuneiforms
Fifth metatarsal base	Lateral malleolus
Sesamoid	

the effects of estrogen and testosterone on bone remodeling; low levels of testosterone and estrogen have been measured following rigorous aerobic training sessions in both males and females.[16] Sex steroids normally inhibit osteoclasts and enhance osteoblasts, thereby slowing the resorption process, and prolonged periods of subphysiologic levels correlate with low bone mineral density.[16]

Two main categories, low and high risk, (Table 2) have been used to determine the relative risk of progression to complete fracture or to nonunion. This distinction aids in guiding workup and treatment.[1,2,4] Low risk stress fractures, such as those of the calcaneus, have a better prognosis and can often be diagnosed clinically and treated with activity modification.[1,2,4] Those at high risk, such as the navicular, talus, medial malleolus, proximal fifth metatarsal, and sesamoids will often need more advanced imaging, periods of non-weightbearing and, possibly, surgical fixation.[1,2,4]

HISTORY, PHYSICAL EXAM AND IMAGING

Patients typically present with a progressive onset of pain with weightbearing activity over a period of days to weeks.[17] A history that includes a rapid increase in mileage, intensity, or duration of activity, changes in playing surface or sport, or inadequate rest periods should raise the suspicion of a stress fracture.[1-5,7-9,13,18-20] A thorough history including diet, nutrition, medications, daily activities, footwear, and menstrual cycles in females should be discussed.[1,2,4,5,8,13,14,21] On physical exam, pain may be elicited with weightbearing, joint motion, or palpation, and in more superficial areas, edema, warmth, ecchymosis or even a palpable callus may be present.[1-5,7-9,13,18-20]

Assessment of limb alignment and length discrepancies, gait, passive range of motion, tendon function, and callosities provides information about repetitive stresses placed on the symptomatic area.[22] Noting alignment and flexibility of any foot or ankle deformities can provide insight into the underlying causes of the pathology.[22] Both stiff, as well as ligamentously lax joints are indicators of abnormal forces across a joint and are postulated to put the patient at higher risk of stress fracture.[22]

Imaging studies including radiographs, computed tomography (CT) scans, MRI and bone scintigraphy can be helpful when the diagnosis is questionable or is suspected in a high risk bone given the possible sequelae of a missed or late diagnosis.[1] Plain films will often be negative for the first 2 weeks following a stress fracture, until resorption, sclerosis, or callus formation occurs.[1,4] Radionuclide bone scan has been shown to be a sensitive imaging modality since the 1970s, as changes can be seen within 48 to 72 hours of injury.[1,23,24] Uptake in all three phases of a technetium-99m diphosphonate scan is characteristic of a stress reaction/fracture. A soft tissue injury will only show increased uptake in the first phase (angiographic and blood pool/soft tissue imaging, respectively).[24] MRI has replaced bone scan as the imaging modality of choice in most cases due to its superior specificity and resolution relative to bone scintography.[25] CT scan can be used to identify incomplete and complete fractures, but cannot aide in identification of stress reactions.[25] However, CT scan is thought by some to be more helpful than MRI in following the healing of stress fractures.[26] Burne et al proposed that as a stress fracture heals, the initial edema and hematoma seen well on MRI for diagnosis is replaced by sclerosis, which is better seen on CT scan.[26] This, in combination with a thick periosteum in some locations, impairs the ability of MRI to detect subtle fracture lines.[26]

HIGH RISK STRESS FRACTURES

Some foot and ankle stress fractures have a relatively low propensity for spontaneous healing due to various factors such as blood supply, shearing forces across their surface, and location.[1,2,8] Strict non-weightbearing, immobilization, and, not uncommonly, surgery are frequently needed to obtain a solid union.[1,2,8]

Medial Malleolus

Stress fractures of the medial malleolus are uncommon and generally found in athletes involved in running, jumping, and kicking sports, although abnormal forces at the ankle due to tibial and talar osteophytes has also been implicated.[26-28] The first series of six patients reported by Shelbourne in 1988 described a diagnosis based on medial malleolar tenderness, ankle effusion and pain during running activities. Radiographs revealed a vertical radiolucent line extending from the plafond in these patients.[26] Although most medial malleolar stress fractures occur in skeletally mature patients, adolescent cases have been reported.[28]

A patient with concerning symptoms but negative radiographs should undergo a bone scan or MRI. Figure 1 shows the typical appearance on MRI imaging. The presence of increased uptake on bone scan with stress reaction but no true fracture line on MRI can be treated with cast

immobilization and non-weightbearing.[26,28,29] Almost all of these injuries will heal with appropriate nonoperative treatments, but earlier return to competitive sports, often within 1 to 3 months, has been shown with internal fixation.[26,28,29] Several series of medial malleolar stress fractures in athletes were reviewed by Shabat et al.[28] Their findings demonstrated that both time to healing and return to sport were longer with nonoperative treatment. They concluded that early surgical intervention yields better results.

Talus

Talar stress fractures were first described in 1965 by McGlone.[30] It is a relatively rare injury, and athletes and military recruits performing repetitive axial loading activities are most prone to this injury.[31,32] Advanced imaging, particularly MRI, is often required to obtain a radiographic diagnosis (Fig. 2).[31-33] Physical exam findings are variable, including point tenderness, ankle effusion or soft tissue swelling.[30-33] Excessive subtalar pronation or plantar flexion is noted clinically in many patients with lateral process stress injuries, thought to be due to impingement of the lateral process of the calcaneus on the posterolateral talus.[33] The superior part of the talar head is most frequently involved, and the posterolateral talar body fracture will usually be seen extending into the subtalar joint.[31,33]

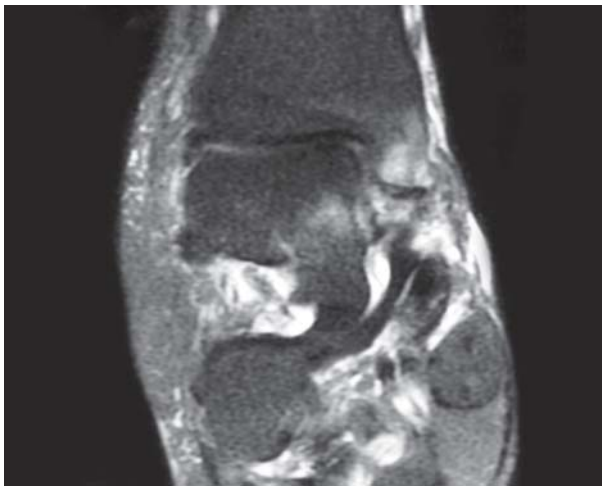

Fig. 1: T2-weighted MRI showing high signal in the medial malleolus

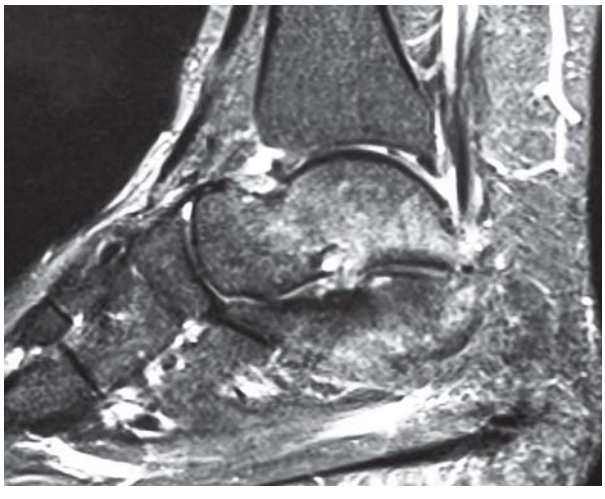

Fig. 2: T2-weighted MRI showing high signal in the talus

A retrospective review of Finnish military recruits found 56 talar stress injuries (including stress reactions and fractures). Five patients had bilateral injuries, and 44 had other associated lower extremity stress injuries. Sixty-seven percent were in the talar head, 25% in the body, and 8% in the posterior portion, similar to previous reports.[31] Talar head injury was associated with navicular stress injury, superior talar body with calcaneal injury, and posterior talus with posterior impingement and presence of a symptomatic os trigonum.[31] Patients who were treated nonoperatively (NSAIDs, relative rest, partial weightbearing as tolerated) until asymptomatic did well with mean duration of treatment of 64 days.[32] In contrast, Bradshaw et al recommended a minimum of 6 weeks of non-weightbearing for this injury due to concern for delayed healing and avascular necrosis based on a small case series.[33] There is no established treatment algorithm for talar stress fractures given the lack of high-level evidence-based data. Patients with excessive pronation may also benefit from orthotics to reduce lateral loading given the coincidence of pronation and lateral talar stress fractures seen in some series.[22,33]

Navicular

Navicular stress fractures are considered high risk due to the rate of nonunion.[1,2] Patients are usually involved in explosive sprinting or jumping activities and complain of pain at the dorsum of the midfoot or along the medial longitudinal arch with activity.[34] Swelling, erythema, and ecchymosis are less reliable indicators, but point tenderness at the dorsal aspect of the navicular known as 'N-spot tenderness' is the most consistent finding.[1,2] Clinical suspicion should prompt radiographic evaluation, and advanced imaging should be obtained if initial X-rays are negative.[34,35] MRI (Fig. 3) and bone scan are sensitive for diagnosis of stress injury to the navicular, and CT scan is often necessary to evaluate the amount of cortical displacement and fracture pattern in this nondiaphyseal small bone, both of which have an impact on treatment.[35,36] The fracture line usually extends from the proximal dorsal border in a plantar and distal direction in the sagittal plane.[35,36]

The central third of the navicular is classically described as a watershed area between blood supplies coming from

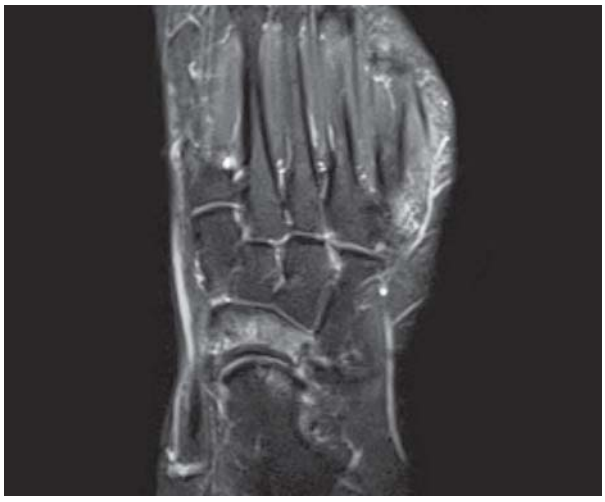

Fig. 3: T2-weighted MRI showing high signal in the navicular

medial and lateral vessels.[34] This is proposed to lead to slower healing of physiologic microfractures in this central area and increase the risk of a stress fracture.[34] During walking and running, this region is also subjected to shear forces from the first and second metatarsals through the cuneiforms along the convex surface distally and the talus at the concave surface proximally.[2,35] Anatomic risk factors include a relatively long second metatarsal, pes cavus, metatarsus adductus, medial narrowing of the talonavicular joint, talar beaking, and limited subtalar or ankle motion.[37]

Small studies have supported both conservative and operative treatment.[2,17,34,35,38] Nonoperative treatment typically includes non-weightbearing in a short leg cast.[2,17,34,35,38] Operative treatment entails open reduction and internal fixation with or without bone grafting.[2,17,34,35,38] One of the first studies reviewing this injury found good results in patients treated with non-weightbearing in a short leg cast.[34] In a review of 86 navicular stress fractures in athletes, only 26% of patients treated with activity modification returned to sports compared to 86% treated with non-weightbearing in a short leg cast.[17] Six patients initially underwent surgical treatment and five returned to sports at an average of 3.8 months. They concluded that non-weightbearing in short leg cast was the standard of care, though they acknowledged that time to return to sport seemed to be shorter with initial surgical intervention. In an initial evaluation of 22 patients, 11 of 13 navicular stress fractures healed with non-weightbearing in short leg cast, and eight returned to sport at an average of 4.3 months.[38] All nine treated with initial internal fixation, with or without bone grafting, returned to sport at an average of 3.1 months.[38] Later, the same authors compared 19 navicular stress fractures and noted that all six treated with non-weight-bearing in a short leg cast healed and returned to sports at 4 months, and all 13 of those undergoing ORIF returned at 4.1 months.[39] Combining data from these two studies, they found no significant difference between outcome or time to return to sports.[39] A recent systematic review compared outcomes with regard to radiographic healing, clinical healing, and time from onset of symptoms to return to activity between patients who underwent one of three different treatment regimens.[40] Weightbearing permitted, non-weightbearing in short leg cast, and surgical intervention groups were analyzed and no statistically significant difference between non-weightbearing in short leg cast and surgical intervention was found. Weightbearing-permitted treatments were inferior in all outcome measurements. This study concluded that the previous recommendations for an initial 6 to 8 weeks of non-weightbearing in a short leg cast should be the standard of care for navicular stress fractures.[40]

Metatarsal

Metatarsal stress fractures occur most frequently in the second and third metatarsals, and are relatively common.[1,2,4,8] They can be considered high or low risk, depending on location. They are common injuries in runners, military recruits, ballet dancers, and basketball players, and most patients will report a recent increase in training.[1-8,10,13,20,22,41-58] Forefoot pain with weight bearing, inability to toe walk, point tenderness, and swelling are present on exam.[1-8,10,13,20,22,41-58] Radiographic workup with plain films of the foot may show callus formation about the metatarsal at around 2 weeks.[1-4,6-8,20,22,41,43-45,48,49,52,53,56] As with other stress fractures, bone scan, MRI (Fig. 4) can differentiate between stress reaction or fracture and soft tissue abnormalities, and CT scan can show cortical irregularities, if present.[2,8,56]

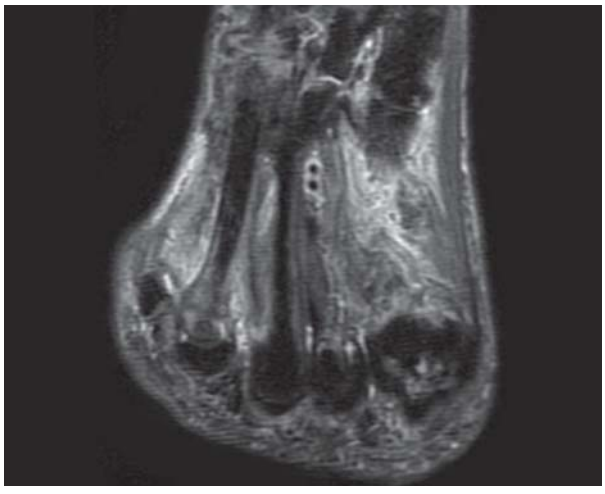

Fig. 4: T2-weighted MRI showing high signal in the distal 4th metatarsal

Distal second metatarsal stress fractures are cited as the most common among metatarsal stress fractures, and possibly among all stress fractures.[22] During walking and running, the second and third metatarsals see the highest bending strain and shear force.[48] The fixed bases and proximal hinged metatarsophalangeal joints cause a bending moment at the proximal diaphysis of the metatarsal, as well as 2.7 times more force at the metatarsal heads during the stance phase of gait.[48,59] A relatively long second metatarsal and an excessively mobile first ray, sometimes termed Morton's foot, have been shown to increase this force even further.[3,48,59] Additionally, females have been found to have a higher middle forefoot loading force than males.[55] These findings describe the potential for these anatomical and biomechanical characteristics to play a role in the development of stress fractures.[48,55] Many providers prescribe orthotics for prevention, and a recent cadaveric biomechanical study reported that both custom and semi-custom orthotics decrease tension and shear strain on the second metatarsal, with custom orthotics being superior.[59] Clinical studies describing benefit from orthotics are largely from military recruit databases.[47,60] Treatment of established distal metatarsal stress fractures initially involves non-operative management. This includes activity modification for 6 to 8 weeks, gradual return to sports when asymptomatic, and usually produces good results.[22] The addition of a stiff-soled shoe, midfoot taping, walker boot, or short leg walking cast can be used for comfort.[2,4,22]

Second and Third Metatarsal Base

Metatarsal base stress fractures are reported to be most common in female ballet dancers, but have been reported in nondancers as well.[41,43] The insidious onset of vague midfoot pain is often overlooked or misdiagnosed.[41,43,49,51-53,56] The second and third metatarsals are most affected. They are at risk during ballet in the 'en pointe' position due to the locking of the second metatarsal base and cuneiforms in extreme plantar flexion.[41,43,49,51-53,56] Intrinsic risk factors include a pronated foot and poor ankle plantarflexion causing a so-called 'over-pointe' foot with compensatory plantarflexion through the Lisfranc joint.[41] This causes the center of gravity to be anterior to the metatarsal shaft, creating more force at the base. MRI is appropriate if plain films are negative and suspicion is high.[41] The first series was described by Micheli in 1985, who reported four female ballet dancers with midfoot pain.[51] Three were treated successfully with a short period of immobilization and rest, and one required operative debridement of necrotic bone due to delay in diagnosis.[51] Several subsequent studies have also reported good results and returned to dancing with conservative management ranging from wooden-soled shoe to short leg walking cast.[52,53] The incidence of nonunion is traditionally thought to be low, however some recent studies suggest a higher rate of nonunion.[43,52,56] Comorbidities including diabetes, chronic steroid use, the female athlete triad, cancer, and metabolic bone disease seem to be associated with a higher rate of nonunion.[43] Ballet trainees treated with medium energy external shock wave treatment and ultrasound along with a period of 3 to 5 weeks of weight bearing rest had 100% return to dancing at a mean 4.6 weeks, return to full pointe at a mean 18 days later, and no subsequent pain or nonunion.[41] Overall, nonsurgical management seems to yield good results in the majority of studies, and can include weightbearing in a regular tennis shoe, hard-soled shoe, CAM walker boot, or walking cast as well as non-weightbearing in a short leg cast.[41,43,49,51-53,56] Surgical treatment of nonunion following nonoperative treatment is successful and can include drilling procedures and open reduction internal fixation.[56]

Fifth Metatarsal

Fifth metatarsal stress fractures differ from acute fifth metatarsal fractures. Pain has usually been present for several weeks, and the fracture is located at the diaphyseal-metaphyseal junction.[57] This region is between the insertion of the peroneus brevis and tertius tendons, and is distal to the tuberosity, the intermetatarsal ligaments, and the metatarsocuboid ligamentous structures.[57] Repetitive adduction force with the ankle in plantarflexion often causes these stress fractures due to the pull of the plantar fascia. As such, they are frequently seen in basketball players.[1,2,45,54,58] A 2 to 3 weeks prodrome of lateral foot pain with activity, tenderness about the fifth metatarsal base, and pain with passive inversion stretch are clues to the diagnosis.[1] A cavovarus foot or restricted hindfoot eversion is thought to predispose patients to fifth metatarsal base stress fractures by increasing the force on the lateral aspect of the foot.[2] Torg created a classification system for these more distal fifth metatarsal base stress fractures based on history and radiographic appearance that helps to guide treatment.[57] Type I fractures are acute fractures by history and have sharp fracture lines with no radiographic signs of healing. Type II fractures are considered delayed unions. There is no history of previous fracture, but radiographs show periosteal new bone, resorption and sclerosis at the fracture line. Type III fractures are considered nonunions. History reveals history of pain with recurrent symptoms, likely representing repetitive insults, and on plain films the fracture line is widened with medullary canal replaced by sclerosis. Initial treatment recommendations of non-

weightbearing in a short leg cast for type I injuries and curettage and bone grafting for types II and III injuries were based on Torg's experience with 46 fractures.[57] Operative treatment has yielded good results using an intramedullary malleolar screw.[45] Placement of these screws can be technically challenging as the surgeon inserts a straight screw into the curved proximal metatarsal. Intraoperative fracture of the metatarsal shaft, bicortical penetration, and skin irritation proximally are potential complications of intramedullary fixation. A reasonable goal for return to full sport activity following screw fixation is 7 to 10 weeks postoperatively, and this has led many to recommend screw fixation for not only Torg types II and III, but also for athletes wishing to return to sport more quickly.[45,54] Curettage and bone grafting in addition to screw fixation is also an option for types II and III surgical treatment.[1,2]

Currently, most authors agree with the opinion of Torg, and it is recommended that type I injuries undergo a 6 to 8 weeks trial of non-weightbearing in a short leg cast and types II and III injuries undergo surgical fixation.[54,57] An exception is a high level athlete who wishes to return to sport sooner, in which case surgical fixation of an acute stress fracture may be considered.[54] These patients must be warned, that refracture in spite of intramedullary fixation is possible, and returning too soon is a risk factor.[50,58] Some suggest functional bracing or orthotics upon return to sport after a high rate of refracture was seen in their series.[50,58]

Sesamoid

The medial sesamoid is more commonly affected by stress injuries than the fibular sesamoid due to its position directly under the first metatarsal head.[61] Activities involving repetitive, forceful dorsiflexion of the toes are most commonly associated.[22,61] Sometimes, swelling or even bulging soft tissues are appreciated, and pain is reproduced with forced dorsiflexion.[22,61] Radiographic differential includes bipartite sesamoid, which is difficult to distinguish on plain films.[22,61] Often, the gap between proximal and distal fragments of a bipartite sesamoid is wider with smoother margins than a fracture.[22] The overall incidence of bipartite sesamoid in the general population is 5 to 30%, 25% of which are bilateral.[22] In contrast to other stress fractures, sagittal cuts on CT scan may be superior to MRI or bone scan when advanced imaging is needed for diagnosis.[61] Initial treatments include shoe modification, immobilization, cessation of sport, partial or non-weight-bearing, systemic anti-inflammatories, and steroid injections.[22,61-63] Failure of nonoperative treatment is commonly reported.[61-63] Operative treatment including sesamoidectomy,[63,64] partial sesamoidectomy,[61,65] closed reduction and percutaneous screw placement,[66] curettage and bone grafting[67] have been described. Each of these reports has small numbers, but success rates of 90 to 100% union and 100% return to sport have been reported.[61,63-67] Complications of complete and partial sesamoidectomy include hallux valgus and flexor hallucis brevis tendon dysfunction, so careful dissection of the sesamoid out of the flexor tendon sheath is important.[61-64] In a report of two athletes with sesamoid stress fractures and combined hallux valgus deformities the authors determined that the hallux valgus was the inciting deformity.[68] Because of this finding, treatment included osteotomies along with open reduction internal fixation with cannulated screws to reduce the sesamoid. Both of these patients returned to their previous level of activity with excellent subjective outcomes.[68]

Low Risk Stress Fractures

Low risk stress fractures of the foot include those of the calcaneus, cuboid, cuneiform bones, and the lateral malleolus.[2,4,7,8,22] They have a high propensity to heal with conservative management. Cuboid, cuneiform, and lateral malleolus stress fractures are rare and reported in the literature predominantly as case reports.[18,19,42,69,70] Much like high-risk stress fractures of the foot, low-risk stress fractures of the foot are common in patients undergoing new occupations or physical training regimens involving repetitive motions.[4,7,18-20]

Calcaneus

Calcaneal stress fractures are considered low risk by most authors due to their high likelihood of uncomplicated healing.[2,4] The incidence is highest in military recruits and long distance runners.[2,71,72] The history often reveals two to 4 weeks of vague total heel pain following a sudden increase in training intensity.[2,71,73,74] On exam, a positive calcaneal compression test with some amount of swelling is usually present.[2,71,73,74] The diagnosis is often missed initially due to similarity in presentation to plantar fasciitis, Baxter's nerve entrapment, insertional Achilles tendonitis, atrophic heel pad, retrocalcaneal bursitis, and calcaneal apophysitis (Sever's disease) in adolescents.[2] Plain films will often show sclerotic or radiolucent line after 2 to 3 weeks of symptoms.[2,72] A bone scan or MRI (Fig. 5) can also be helpful to determine the cause of pain since the differential diagnosis is broad.[72] Three of the largest reports are early studies from the US military and suggested that calcaneal stress fractures could be adequately treated with activity modification without casting or surgical intervention.[71,73,74] In a more recent Finnish military study, Sormaala reviewed 34 stress injuries (stress reactions and

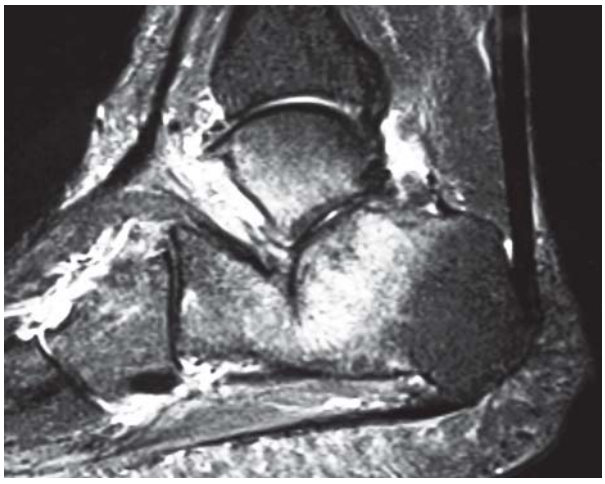

Fig. 5: T2-weighted MRI showing high signal in the calcaneus

fractures) diagnosed over a 96-month period by radiographic diagnosis.[72] Most common were posterior part stress injuries, followed by anterior, and least commonly middle. Sixty-five percent were associated with talar, cuboid or navicular stress injuries, especially when the calcaneal injury was in the upper part of the bone. Conservative management was used, without any casting or surgical intervention, and the recruits were noted to be asymptomatic at an average of 77 days. They concluded that these injuries can be treated conservatively with suspension from activity only.[72]

Cuneiforms, Cuboid, Lateral Malleolus

Cuneiform stress fractures were first reported in by Maseritz in 1936 as a March Fracture of the first cuneiform in conjunction with a second and fifth metatarsal fracture also associated with an os calcis (calcaneal enthesophyte).[18-20] In 1943, Childress described a stress fracture of the second cuneiform.[42] In 1980, Meurman provided a case report of a stress fracture of the third cuneiform in a military recruit.[19]

In a report of 827 military recruits, only three fractures in the cuneiform bones (two medial and one lateral) and one fracture in the lateral malleolus were found.[19] All patients experienced swelling and had point tenderness on exam.[19] These clinical findings are consistent with other reports of stress fractures of these bones.[7,18,20]

Unlike bones with a diaphysis, the cuneiform and cuboid may not display the usual periosteal callus.[18-20] Instead, stress fractures can be appreciated as transverse sclerotic zone across the zone of injury.[18-20] MRI is the imaging modality of choice of some authors if a suspected low-risk stress fracture is not visible on plain films.[18] These injuries will demonstrate bone marrow edema on both T2 and T1 weighted images. A fracture line can be appreciated as low signal intensity in conjunction with bone marrow edema on all sequences.[2,4,18,20]

The two forces thought to be responsible for cuneiform stress fractures are that of bending and compression.[18,19] Bending forces are applied across the cuneiforms due to their location in the midfoot. The medial cuneiform also lies in the same axis as the first metatarsal and is susceptible to compression-type stress fractures.[18,19] A large proportion of body weight passes through this axis, and muscular insertions on each side of the first cuneiform exert strong opposing forces across a small area.[18,19] The plantar fascia plays a critical role in stress fractures of the third cuneiform.[18,19] The third cuneiform is the key stone of the arch and has six articulations with six different bones leading to forces in several vectors across this bone.[18,19] Plantar fasciitis and plantar fasciotomy have been theorized as possible etiologic factors in the development of cuneiform and cuboid stress fractures. This is due to increased lateral column stress because of the plantar fascia's relationship to stability of the arch and its effect on gait.[18,20,42,70,75]

The standard of care for low-risk stress fractures is with nonoperative modalities.[2,4,7,8,19,20] Initially, partial weight bearing on the affected extremity from 2 to 6 weeks with or without immobilization is recommended, followed by progression to low impact activities.[2,4,7,8,19] Return to full activities and athletics may resume once the patient can perform low impact activities without pain or discomfort.[2,4,7,8]

OTHER TREATMENTS

Bisphosphonates

Bisphosphonates have the potential to decrease the incidence of stress fractures by decreasing the amount of bone turnover by inhibiting osteoclast function.[46,76,77] One prospective, randomized trial of 324 military recruits determined no difference in the incidence of stress fractures of the lower extremities between those receiving prophylactic risedronate and placebo.[77] Similar observations have been made in animal studies.[76] Rat ulnae were subjected to repetitive loading (simulating a stress fracture mechanism) and then treated with short term dosing of alendronate. There was no protective effect and actually a trend toward a harmful effect of bisphosphonate treatment, possibly due to inhibition of remodeling of microfractures from woven to lamellar bone.[76] The 25 years experience of the Israeli Army on prevention of stress fractures was published in 2008.[46] Sleep minimums and training modifications, but not bisphosphonate treatment, were shown to decrease the incidence of stress fractures.[46]

Bone Stimulators

External stimulators in the treatment of stress fracture are an attractive treatment idea due to their simplicity for the

patient, low risk of side effects, and the potential positive impact on the athlete's psyche that active, rather than passive, treatment is being undertaken. There are two types of stimulators, electromagnetic stimulators and ultrasound stimulators.

Electromagnetic stimulators generate electromagnetic fields due to the application of coils on either side of the fracture.[78] The idea was introduced following the discovery that mechanical stresses causing fluid flow around and through bones stimulates electrical currents through the interstitial spaces around cells. These currents open calcium channels in cell membranes increasing calmodulin, which begins the cascade that controls DNA proliferation and thus cell proliferation.[78] A wide variety of internal and external electromagnetic stimulators are available with different electrical settings, treatment durations and needs for simultaneous immobilization. Very few controlled studies are available that evaluate the efficacy of these stimulators in stress fractures. One such study found no significant difference in time to healing between the placebo group and those using a capacitive coupled field stimulator.[79] Interestingly, when only higher grade stress fractures were compared there was significantly shorter time to healing noted, though power was not sufficient to draw conclusions. Similarly, when compliance was taken into account, the use of electromagnetic stimulators correlated to shorter healing time.[79] Cohort studies have yielded promising results without major complications, but are difficult to evaluate due to the lack of control groups.[68]

Pulsed ultrasound bone stimulators have been studied extensively in the past 20 years but still remain a controversial modality for the treatment of acute fractures and stress fractures.[80] At the cellular level in response to pulsed ultrasounds, there is an increase in VEGF and FGF which promote angiogenesis, as well as an increase in levels of alkaline phosphatase, bone sialoprotein, and intracellular calcium, all of which are markers of increased metabolism within bone.[80] Most studies available evaluate their utility in the treatment of acute fractures. A systematic review of the results of pulsed ultrasound found low to moderate grade evidence for a positive effect, however there was an overall 33.6% decrease in radiographic healing time.[81] Stress fractures may respond differently to pulsed ultrasound because they heal through intramembranous remodeling instead of endochondral remodeling as acute fractures do. Literature specifically on stress fractures treated with pulsed ultrasound is sparse.[82,83] In a military study of 43 tibial shaft stress fractures there was no significant difference in time to healing using low intensity—pulsed ultrasound.[83] In a rat ulnar stress fracture model, low intensity pulsed ultrasound alone produced better results than ultrasound and NSAIDs combined, and both of these were better than controls.[82] Overall, there is no conclusive evidence that electromagnetic or pulsed ultrasound bone stimulators are efficacious in treating stress fractures.

Table 3: Recommended calcium intake per day

Age	*Recommended calcium intake*
9-18	1,300 mg
19-50	1,000 mg
> 50	1,200 mg

Adapted from AAOS guidelines[2], NIH office of dietary supplement guidelines[64,65]

Table 4: Recommended vitamin D intake per day

Age	*Recommended vitamin D intake*
0-50	200-1,300 IU
51-70	400-1,000 IU
>70	600-1,300 IU

Adapted from AAOS guidelines,[2] NIH office of dietary supplement guidelines,[64,65] American Academy of Pediatric guidelines[36]

Oral Contraceptive Pills

Low levels of estrogen and testosterone are associated with low bone mineral density.[16,84] Abnormally low levels of sex hormones are seen for 24 to 48 hours in endurance athletes following rigorous training sessions, and secondary amenorrhea occurring in female athletes causes a longer term hormone deficient state.[16,84] Hormone replacement therapy via oral contraceptive pills (OCPs) in the athlete to prevent stress fractures is controversial. There is some data to suggest that hormone replacement in amenorrheic women and endurance athletes improves bone mineral density (BMD).[84-86] A recent study randomized 150 young female runners to low dose OCP or no treatment.[21] BMD as well as incidence of stress fractures was followed for 2 years.[21] Oligo- and amenorrheic runners who used OCPs as prescribed gained 1% per year in BMD, which was statistically significant, and stress fracture incidence trended lower in the OCP-treated group but was not statistically significant. A military study of female recruits found an over five-fold increase in the incidence of lower extremity stress fractures in women who had been amennorheic over the year prior to training, though OCP use did not have a significant protective effect.[21]

If a physician chooses to administer OCPs for this purpose, factors such as nutrition or other hypothalamic perturbations must be excluded.[85] To highlight the effect of nutrition and bone density, a recent article reviewed exercising women and grouped them into four categories: Energy and estrogen deficient, energy or estrogen deficient

alone, or energy and estrogen replete. Those who were energy replete, regardless of estrogen status, had no abnormalities in lab values associated with bone turnover. Those who were energy and estrogen deficient had the most abnormalities.[15] A case report of a female runner who had very low BMI (15.8) was followed with bone density scans over a course of 8 years. During training alterations and hormone replacement therapy she did not have significant change in BMD, but following purposeful weight gain to BMI of 21.3 BMD returned to normal range at 94 to 96% of normal.[87] In the above-mentioned study of 150 female runners, dietary calcium intake and weight gain were also independent predictors of increases in BMD over their 2 years study.[21]

Calcium and Vitamin D

Adequate dietary intake and supplementation of calcium and vitamin D has been shown to improve BMD, but mixed conclusions on preventing stress fractures are reported.[5,12,21,44,88-90] The role of calcium and vitamin D supplementation in preventing stress fractures of the foot and ankle specifically has not been studied. In previous studies, no significant difference was found with increased calcium and vitamin D intake and incidence of all types of stress fractures in track and field athletes and military recruits.[5] In a large study population that included female military recruits, the cohort prescribed 2,000 mg of calcium and 800 IU of vitamin D daily had a 20% lower incidence of stress fractures during basic training than those receiving placebo.[88] Each cup of skim milk consumed daily by female distance runners in a different study lowered the rate of stress fracture by 62%.[12] These reports support several previous studies suggesting that low dietary calcium and vitamin D is associated with increased risk of stress fracture, and adequate intake or supplementation can reduce the risk of stress fractures.[89,90] Currently, the recommended daily dose of calcium depends on age, and is uniform between societies.[91-93] The recommended daily vitamin D intake is somewhat more controversial and the subject of increasing research (Tables 3 and 4).[91,94] The specific amount of calcium and vitamin D needed to prevent stress fractures has not been determined. Supplementation of 500 to 800 mg of calcium and 400 to 800 IU vitamin D is shown in some studies to improve BMD and decrease fractures (not specifically stress fractures) risk significantly.[95,96] No reports of treating stress fractures with calcium and vitamin D alone are reported, and thus no conclusions can be made regarding this method alone. However, adequate calcium and vitamin D are needed for bone healing, remodeling, and maintenance of bone mineral density. They may be helpful adjuvants during the treatment of stress fractures.[89,90,95]

Calcitonin

Calcitonin inhibits osteoclasts, the offending agent in the imbalanced remodeling process of stress fractures.[97-99] To date there have been no studies examining the effectiveness supplemental calcitonin specifically on stress fracture healing in the foot. Increased bone mineral density and biomechanical properties has been shown with calcitonin use,[97,98] but its role in fracture healing is controversial.[98,99] At this time, there can be no conclusions drawn on the use of calcitonin for enhancing healing of stress fractures due to lack of scientific studies.

Orthotics

Several biomechanical studies have shown predictable, repetitive stress patterns in the foot and ankle with weight bearing.[55,59] Much of the data regarding orthotics for prevention or treatment of stress fractures comes from military studies.[46,47,60] There is inconclusive data to support or criticize the use of orthotics for prevention of stress fractures of the foot and ankle.[46,47,60,100] A recent systematic review of five articles on orthotics and stress fractures concluded that orthotic use reduced the overall rate of stress fractures of the proximal femur and tibia in military personnel.[100] Although a slight overall benefit was seen, no definite conclusion could be made regarding prevention in stress fractures of the foot and ankle or in athletes specifically.[100]

CONCLUSION

Stress fractures of the foot and ankle in athletes are relatively uncommon.[1] However, a heightened awareness of this condition along with more rigorous training has contributed to the increasing incidence of stress fractures.[3] A change in training conditions such as increased time or distance, new impact activities, change in training surface, improper technique, and poor nutrition are implicated as contributing factors in the development of stress fractures.[2] Additionally, in the female athletic population, coaches, athletes, and families should be educated and alerted to the adverse effects of eating disorders and hormonal abnormalities.[14,16,21,84] Knowledge of which types of these injuries are high and low risk, as well as the demands of the patient, should guide management decsions.[1,2,4] At this time there are mixed results with the use of adjuvant therapy such as bone stimulators,[54,68,79-82] bisphosphonates,[46,76,77] hormone replacement,[13,16,21,84-86,] and dietary supplementation of

calcium and vitamin D[5,12,50,89,90] for prevention or treatment of stress fractures of the foot and ankle. There is no data to support or refute the use of calcitonin.

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