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# High-Risk Stress Fractures: Diagnosis and Management

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## Abstract

Stress fractures are common overuse injuries in athletes. They occur during periods of increased training without adequate rest, disrupting normal bone reparative mechanisms. There are a host of intrinsic and extrinsic factors, including biochemical and biomechanical, that put athletes at risk. In most stress fractures, the diagnosis is primarily clinical, with imaging indicated at times, and management focused on symptom-free relative rest with advancement of activity as tolerated. Overall, stress fractures in athletes have an excellent prognosis for return to sport, with little risk of complication. There is a subset of injuries that have a greater risk of fracture progression, delayed healing, and nonunion and are generally more challenging to treat with nonoperative care. Specific locations of high-risk stress fracture include the femoral neck (tension side), patella, anterior tibia, medial malleolus, talus, tarsal navicular, proximal fifth metatarsal, and great toe sesamoids. These sites share a characteristic region of high tensile load and low blood flow. High-risk stress fractures require a more aggressive approach to evaluation, with imaging often necessary, to confirm early and accurate diagnosis and initiate immediate treatment. Treatment consists of nonweight-bearing immobilization, often with a prolonged period away from sport, and a more methodic and careful reintroduction to athletic activity. These stress fractures may require surgical intervention. A high index of suspicion is essential to avoid delayed diagnosis and optimize outcomes in this subset of stress fractures.


## Introduction

Stress fractures are common injuries in athletes of all levels. They represent approximately 10% of all overuse injuries in sport, with the incidence in runners as high as 20% [1,2]. Although stress fracture has been described in nearly every bone of the lower extremity, the tibia is reported as the most common site, followed by the tarsals and metatarsals [1,3]. Stress injury occurs when periods of repetitive, submaximal loads are applied to the limb during training and competition without adequate rest, leading to an imbalance between bone resorption and formation. Most stress fractures are uncomplicated and managed conservatively with rest and activity restriction, with a gradual return to sports-specific activity when the patient is symptom free.

There is a subset of high-risk stress fractures (HRSFs) that have a predilection for protracted recovery, progression to complete fracture, delayed union, nonunion, and chronic pain. Specific sites for this type of stress fracture are the femoral neck (tension-side), patella, anterior tibia, medial malleolus, talus, tarsal navicular, proximal fifth metatarsal, and the great toe sesamoids. These locations all have a region of maximal tensile load in a zone of diminished blood flow that is vulnerable to stress injury, with suboptimal healing potential. Although HRSFs are relatively rare, they have important clinical relevance, because they can keep athletes from participating in sports for long periods of time and can cause significant morbidity if not properly treated. Athletes with HRSFs may require surgical intervention to return to play in a timely fashion. A high index of suspicion and early identification are critical to proper treatment and successful outcomes. This article reviews the evaluation and management of HRSFs in athletes.

## Method of Literature Review

A search was conducted of PubMed and Medline databases from their inception to July 2015 with the following search terms: high-risk stress fracture, stress fracture, stress injury, femoral neck stress fracture (FNSF), patellar stress fracture, tibial stress fracture and anterior tibial stress fracture, medial malleolus stress fracture, talus stress fracture, navicular stress fracture (NSF), proximal fifth metatarsal stress fracture


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(P5SF), and sesamoid stress fracture (SSF). The search prioritized articles published from 2000 to 2015. Studies printed in English that focused on pathophysiology, risk factors, evaluation, and management were considered for inclusion. Articles that focused primarily on low-risk stress fracture sites as well as those that compared surgical techniques were excluded.

**Pathogenesis**

During periods of intense exercise without adequate rest, bone formation lags behind bone resorption, resulting in dominance of osteoclast versus osteoblast activity. The result is bone that is vulnerable to microfracture, identified by bony edema on magnetic resonance imaging (MRI), consistent with stress reaction. With continued load, microfractures can propagate into true cortical break, or stress fracture [4,5]. Stress or fatigue fracture occurs with overuse in the setting of normal bone mineral density (BMD). In contrast, insufficiency fracture occurs with relatively normal load in cases of low BMD [5].

Overall, most stress fractures occur in the cortical region of bone, at the diaphysis of long bones, or at the shell of square bones where remodeling is slower [5]. Stress fractures occur less frequently in cancellous bone, where more active remodeling occurs, at the metaphysis and epiphysis of long and square bones. Stress fractures of cancellous bone more often correlate with low BMD [5].

There are common characteristics of bone regions involved in HRSF. They frequently occur at sites of maximal tensile load and regions of hypovascularity. Stress fractures at the tension-side of bone have a risk of displacement because of the summative distracting force created by gravity and muscular attachments, leading to instability and risk of poor healing. Diminished blood flow to these sites contributes to delayed healing, risk of nonunion, and prolonged time out of sport. HRSFs often do not respond to conservative management and can result in significant morbidity. In contrast, stress fractures at the compression-side of bone heal well with nonoperative care because of the summative compressive force that induces osteogenesis, promotes stability, and makes nonunion unlikely [4].

**Risk Factors: Opportunity for Prevention and Intervention**

Careful investigation and modification of risk factors are essential to direct appropriate treatment and prevent future injury. Risk factors are commonly divided into intrinsic and extrinsic categories. Extrinsic risk factors are modifiable and include training variables such as volume, intensity, and duration of activity. Greater running mileage has been associated with risk for stress fracture, but this threshold is likely different for each athlete [6]. Implementing periods of rest from running is considered protective. Athletic terrain and shoe wear have been implicated as risk factors. It is generally recommended that training shoes be updated every 6 months or every 300-500 miles [7]. Shock-absorbent insoles and orthotics may decrease the risk of lower extremity stress fractures, particularly in athletes with pes planus [7].

Athlete behaviors that can negatively impact bone health include caffeine intake, alcohol consumption, and tobacco use [8]. Nutritional factors, such as calcium and vitamin D, can have a positive influence on bone health. Vitamin D contributes to calcium and phosphorus absorption from the gut and resorption from bone and appears to be important in fracture healing. However, studies are mixed regarding the correlation of vitamin D level and risk for stress fracture [9]. One blinded, randomized controlled trial found that female Navy recruits receiving calcium and vitamin D supplementation were 20% less likely to suffer stress fracture [10]. In a systematic review by Tenforde et al [11], female subjects who consumed $\geq$1500 mg of calcium daily exhibited the largest risk reduction in stress fracture, among the many variables they investigated. Because it is difficult to meet the daily recommendations of vitamin D and calcium with diet alone, supplementation is often necessary.

Systemic medical conditions that affect metabolism and nutritional status also can influence bone health, including thyroid dysfunction and gastrointestinal malabsorptive conditions such as gluten sensitivity and celiac disease. Medical treatment should be optimized.

Two well-known intrinsic risk factors for stress fractures are history of previous stress fracture [12] and female gender [13]. Independent risk factors in female athletes include body mass index $<$19, late menarche ($\geq$15 years), and participation in selected leanness sports [12]. The female athlete triad is a combination of (1) low-energy availability, with or without disordered eating, (2) menstrual dysfunction, and (3) low BMD. This condition ultimately leads to a low estrogen state that disrupts the normal osteoclastic/osteoblastic response to repetitive stress and can result in vulnerability to fracture. Screening for female athlete triad is essential in managing stress fracture and preventing future injury, as well as optimizing overall health in this population.

Identifying intrinsic biomechanical risk factors also can present an opportunity for intervention. Muscle strength, endurance, flexibility, and lower extremity alignment have been proposed as risk factors for different stress fractures [1,14]. Sports-specific form and technique also may play a role. Kinematic and kinetic variables influence the way forces are transmitted throughout the bones of the lower extremity and likely have an impact on stress injury. Injury-specific risk