Stress Fractures in Athletes

Diagnosis and Management

Timothy L. Miller
Christopher C. Kaeding
Editors

Second Edition

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To my family, in particular my wife, Nicole, and our children, Gavin, Ashton, Avery, Sydney, and Giselle, for always believing in me and being supportive when I take on a new project.
To the many athletes who have had their seasons and careers cut short due to stress fractures and other overuse injuries, know that your hard work and efforts have not gone in vain and, in fact, were the inspiration for this book.

To Dr. Christopher C. Kaeding,
Thank you for taking a chance on a farm boy from Barnesville, Ohio. Without your guidance, support, and mentoring I would not be where I am today.

Timothy L. Miller, M.D.

To my wife, Christine, for her bottomless source of patience and support of my academic career in sports medicine with all its time demands, off hours obligations, and unexpected interruptions.
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Christopher C. Kaeding, M.D.

Preface

Stress Fractures in Athletes has been the culmination of many years of experience with overuse injuries both as athletes and as team physicians. Traditional treatment strategies for stress fractures including simply stopping the causative activity or sport are no longer considered practical or acceptable options for many competitive athletes. This textbook compiles the concepts, wisdom, and techniques required to approach and treat the complexities of stress-induced injuries to bone among the athletically active population. The editors would like our readers to understand the value we place on employing a holistic approach to treating stress fractures. A diverse treatment team that seeks to balance training, biomechanics, nutrition, hormonal status, and mental health is required to successfully prevent, treat, and recover from these troublesome injuries. We truly appreciate the contributions of the authors – many of whom are considered pioneers and leaders in the field of Sports Medicine – who have provided their invaluable insights and pearls on the evaluation and treatment of stress fractures and insufficiency fractures. As our athletes continue to break records and push the limits of running, jumping, biking, swimming, skiing, rowing, cross-fit sports, and adventure racing, the branch of Sports Medicine we refer to as *Endurance Medicine* continues to develop to serve their needs. This textbook details the pathology, assessment tools, and treatment options for bony stress injuries throughout the body from the spine and pelvis to the hands and feet. It is our hope that this textbook will be a valuable guide for Sports Medicine physicians, orthopedists, athletic trainers, physical therapists, coaches, parents, and athletes in treating stress fractures and is another step forward for the field of Endurance Medicine.

Columbus, OH, USA — Timothy L. Miller, M.D.

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Part I

General Evaluation Principles for Stress Fractures

1 Risk Factors for Developing Stress Fractures

Donald Kasitinon and Lindsay Ramey Argo

Introduction

Stress fractures are mechanical loading injuries that result from an imbalance between microdamage and bone remodeling and repair [1]. They can range in severity from mild marrow and/or periosteal edema to a visible fracture line [2]. For simplicity, all grades of bone stress injury will be referred to as a stress fracture in this chapter. Unlike acute fractures, stress fractures are typically caused by repetitive, submaximal loading of a bone [3]; like acute fractures, they can lead to significant pain, reduced performance, lost training time, and medical expense [4]. Individual athletes vary in their susceptibility to stress fractures. Risk factors for stress fractures are often categorized as intrinsic or extrinsic and modifiable or non-modifiable to aid clinicians in identifying high-risk individuals and in defining actionable ways to minimize risk [5, 6].

While the quantity of research on this topic is large, the quality of research is more limited, with few high-quality, prospective trials in the athletic population. Most studies analyzing risk factors involve military personnel rather than recreational and competitive athletes [7] and are cross-sectional in design. Further, stress fracture risk factors are often interrelated and difficult to analyze independently [5]. Despite these limitations, multiple risk factors have been established and are reviewed in this chapter (Table 1.1). In addition, proposed prediction algorithms for stress fractures based on these risk factors are outlined.

Intrinsic Risk Factors

Intrinsic risk factors are those that are directly related to the athlete's metabolic or anatomic characteristics [3, 6] and can be subcategorized into non-modifiable and modifiable factors.

Non-modifiable Intrinsic Risk Factors

Non-modifiable risk factors are those that an athlete cannot take measures to change. Non-modifiable intrinsic risk factors include specific demographics such as gender, race, and age; previous history of stress fracture; genetics; and specific anatomic alignment.

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Table 1.1 Risk factors for developing stress fractures

	Intrinsic	Extrinsic
Non-modifiable	Demographics Female gender Caucasian race Age: Undetermined History of stress fracture Genetic predisposition Anatomic characteristics Pes cavus foot morphology Leg length discrepancy Altered knee alignment	Preseason, start of season
Modifiable	Relative energy deficiency syndrome Low energy availability with or without disordered eating Functional hypothalamic amenorrhea Osteoporosis Calcium and/or vitamin D deficiency Low body weight/BMI Poor biomechanics and strength imbalance Medication use Contraceptives Corticosteroids Other Substance abuse Tobacco Alcohol	Training variables High volume Noncompliant or uneven surface Poor pre-training physical condition Equipment variables Old or non-supportive footwear Type of sport Leanness sports: track & field, cross country, gymnastics

Demographics

Gender

The majority of studies have found that females have a higher incidence of stress fractures than males. This is likely multifactorial in nature and due, at least in part, to gender-associated risk factors that include dietary deficiencies, menstrual irregularities, hormonal differences, lower bone mineral densities, and narrower bone width. Slower rates of force development in muscle physiology, especially neuromuscular control, also seem to play a role in the increased incidence of stress fractures among females [5, 8, 9].

The risk of stress fractures in female recruits in the United States military was found to be up to 10 times higher than that in their male counterparts while undergoing the same training program [10]. This increased risk in females has also been noted in multiple studies involving high school and collegiate athletes [11–14]. While some studies have reported no difference in incidence between genders [15–17], these studies did not account for total training volume and should be interpreted with caution [18, 19]. In a recent meta-analysis, female gender was one of two risk factors identified with strong evidence to support an association with stress fractures (OR 2.31) [20].

Race

Military studies have shown the highest incidence of stress fractures among white recruits as compared to other races. Among females, incidence rates are the highest in white followed by Asian and Hispanic females, with the lowest incidence rates in African Americans [21–25]. Similar trends by race have been identified in male military recruits [21–25]. This is thought to be due to differences in bone turnover and peak bone density and not due to race independently [5].

Studies of athletes outside the military have shown mixed results. Two studies involving United States collegiate athletes reported no significant differences in stress fracture incidences between white and African American athletes [17, 26]. However, studies from Japan and Korea display slight differences in stress fracture incidence when compared with white populations. Of note, these studies were not sport specific and differences may be related to variation in activity type between groups rather than race [15, 27, 28]. Evidence within the military suggests that white individuals are at higher risk of stress fracture in comparison to other races, particularly African

Americans; however, evidence outside of the military is limited.

Age

The association between age and incidence of stress fracture is currently unclear. Studies in athletes have not found a consistent correlation between age and risk of stress fracture. Within the military, studies have found similar inconsistent results across age groups [21, 22, 29]. Few studies have reported stress fracture frequency by age group [18]. Within the female athlete population, some studies have found an increased risk of stress fractures with increasing age [22, 23], while others have found decreased risk [21, 24, 29, 30] or no effect [31, 32]. Confounding factors associated with aging, particular in the female population, including changes in hormone levels, bone mineral density (BMD), activity level and activity type, may contribute to the inconsistency presented in the current literature [5]. Studies controlling for these confounding variables are needed to determine whether age is an independent risk factor for stress fractures.

Previous History of Stress Fracture

Previous history of stress fracture has been consistently associated with increased risk of future stress fractures across studies, with odds ratios ranging from 2.90 to 6.36 [16, 19, 33]. These findings were supported by a recent exploratory meta-analysis noting that athletes with a previous history of stress fracture have a five time higher risk of developing a new stress fracture as compared to individuals with no prior history [20].

Genetics

Several observations suggest that genetic factors contribute to stress fracture susceptibility. Case reports have described multiple stress fractures at the same anatomic sites in monozygotic twins after the sixth week of basic training in the army [34] as well as multiple lower limb fractures in the same individual [35]. Findings between twins and their families indicate that differences in traits, such as bone size, shape, and BMD, are largely due to genetic differences and not environmental differences [36]. Additionally, mutations or allelic variants in genes can lead to a variety of bone pathologies such as osteoporosis or osteogenesis imperfecta that can result in increased bone fragility and increased risk of stress fracture. In a Finnish military study, eight genes involved in bone metabolism and pathology were examined in subjects with femoral neck stress fractures versus controls. While details are beyond the scope of this chapter, specific genetic patterns were associated with increased risk of stress fractures, suggesting that genetic factors do play a role in the development of stress fractures in individuals who undergo heavy exercise and mechanical loading [37]. Of note, in the absences of specific mutations that identify a specific bone disorders, it is unclear how this can be used clinically for risk stratification or modification at this time.

Lower Extremity Alignment

Specific anatomic variants have been theorized to increase the risk for stress fractures in the lower extremity, including abnormal foot morphology, leg length discrepancy, and knee alignment. While alignment and biomechanical changes associated with these variants can be minimized with bracing, orthotics, and/or appropriate rehabilitation efforts, here we consider these factors to be non-modifiable as the anatomic variant itself requires surgery for correction.

Foot morphology has long been theorized to play a role in lower extremity stress fractures, as the structure of the foot can affect the absorption and distribution of the ground reaction force during impact exercise. Pes cavus refers to a rigid, high-arched foot and results in more force absorption in the leg (femur, tibia, and fibula) and less force absorption in the foot. Pes planus refers to a flexible, low-arched foot and results in less force absorption in the leg and more force

absorption in the foot [5]. A study among the Israeli military population found that those with the highest arches sustained 3.9 times as many stress fractures as those with the lowest arches [38]. A prospective military study found that those with pes cavus morphology were at increased risk of femoral and tibial stress fractures, while those with pes planus were at increased risk of metatarsal fractures [39]. Studies outside of the military have found similar associations with pes cavus morphology and overuse injuries, particularly among runners [40–42]. However, pes planus morphology has shown inconsistent results outside of the military [43, 44]. In a study of 31 runners with recurrent stress fractures, pes cavus was one of three anatomical factors associated with stress fracture recurrence [45].

Leg length discrepancy has also shown a weak association with stress fractures, particularly in female athletes [43, 44]. In the aforementioned study of 31 runners with recurrent stress fractures, leg length discrepancy was one of three anatomical factors associated with stress fracture recurrence [45]. The degree of leg length discrepancy is thought to correlate with increased risk for stress fracture [46] although a study with male military recruits did not confirm this correlation [5, 47].

Valgus knee alignment and increased quadriceps angle (Q angle >15°) have been proposed as potential risk factors for lower extremity overuse injuries, including stress fractures. However, data to support this is limited. Research in this area is dated and largely focused on males in the military [47, 48]. In a 1996 prospective study of 294 male infantry trainees, those with knee valgus alignment had a significantly higher risk for lower extremity overuse injuries (RR = 1.9) and those with an elevated Q angle had a significantly higher risk for lower extremity stress fractures (RR = 5.4) [47]. However, more recent studies have not reproduced these results in athletes of various sports and mixed gender activities outside of the military [49–51]. While theorized, evidence is lacking to define a clear association between knee alignment and risk for stress fracture outside of the military, and further research is needed to better define this relationship.

Limited evidence suggests that orthotics to support pes cavus or correct leg length discrepancies decreases risk of stress fracture, but no clear evidence has been shown [52].

Modifiable Intrinsic Risk Factors

Modifiable risk factors are those that an athlete can take measures to change. Identified modifiable intrinsic risk factors include relative energy deficiency syndrome (RED-S) encompassing low energy availability, functional hypothalamic amenorrhea, and osteoporosis; calcium and/or vitamin D deficiency; low body weight or body mass index (BMI); suboptimal biomechanics and strength; and exposure to certain medications or substances.

Relative Energy Deficiency Syndrome

The term “female athlete triad” was initially used to describe a medical condition observed in physically active females involving one or more of the following: low energy availability, menstrual dysfunction, and low BMD [53]. The Task Force on Women’s Issues of the American College of Sports Medicine published the first triad position statement in 1997 describing a syndrome of three distinct but interrelated conditions: disordered eating, amenorrhea, and osteoporosis [54]. Later studies found that athletes were developing negative health consequences of the triad associated with less severe conditions than these clinical endpoints, so the triad was redefined in 2007 as relative energy deficiency syndrome (RED-S) including low energy availability with or without disordered eating, functional hypothalamic amenorrhea, and osteoporosis with a spectra between optimal health and these endpoints (Fig. 1.1) [55]. The goal of presenting the triad symptoms along a spectrum is to emphasize the importance of identifying subclinical abnormalities among athletes to allow for early intervention

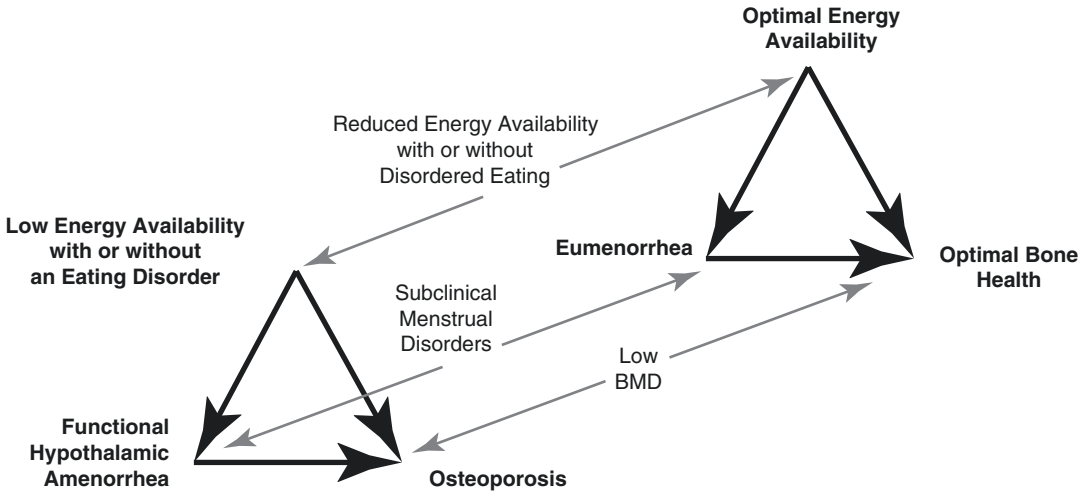

Fig. 1.1 Spectra of the female athlete triad. Spectra along which female athletes are distributed, including energy availability, menstrual function, and BMD. An athlete's condition moves along each spectrum at a different rate based on diet and exercise habits. (Reprinted with permission from Eguiguren, M. L., & Ackerman, K. E. (2016). The female athlete triad. In C. Stein, A. Stracciolini, & K. Ackerman (Eds.), *The young female athlete* (p. 64). Cham, Switzerland: Springer International Publishing. Originally from De Souza et al. [53])

[53]. RED-S is the broader, more comprehensive name as components of the triad are also reported in males [56].

Low Energy Availability with or Without Disordered Eating

Low energy availability is defined as inadequate caloric intake relative to the energy expenditure required for physical activity level [5]. In the absence of disordered eating, it can be difficult to diagnose. Signs of low energy availability include low body weight (a BMI < 17.5 or <85% of expected body weight in adolescents), reduced resting metabolic rate, menstrual dysfunction, low triiodothyronine (T3), excessive fatigue, and impaired immunity/frequent infections [57, 58]. When RED-S is suspected, detailed information regarding food intake and energy expenditure should be evaluated [53].

Low energy availability seems to be the primary mechanism by which female athletes are predisposed to menstrual dysfunction and negative effects on bone health. Anorexia nervosa has been associated with a significantly decreased BMD [59, 60] with nearly 75% of adolescent girls with anorexia having a BMD more than two standard deviation below the normal value [61]. Females with anorexia are at an increased risk of stress fracture development [62, 63]. Similarly, in a group of competitive female track and field athletes, those with stress fractures had significantly higher scores on the EAT-40, an eating attitude test, and tended to weigh themselves more often, indicating a greater preoccupation with weight control among those with stress fractures [64].

Functional Hypothalamic Amenorrhea

Functional hypothalamic amenorrhea (FHA) is a form of hypogonadotropic hypogonadism caused by a disturbance in the normal pulsatile release of gonadotropin-releasing hormone (GnRH) from the hypothalamus [65]. It is a diagnosis of exclusion as the term "functional" indicates that there is no organic disease identified [66, 67]. FHA can result from a combination of low energy availability (weight loss-related, stress-related, and/or exercise-related) and genetics, and it predisposes athletes to osteoporosis [5, 59]. Previous studies have indicated that menstrual dysfunction is

associated with the development of stress fractures [24, 26, 30, 44, 68, 69]. Track and field athletes with a history of oligomenorrhea (defined as four to eight menses per year) were found to be six times more likely to have sustained a prior stress fracture [64]. Female military recruits who reported no menses during the 12 months prior to training were more than five times likely to suffer a stress fracture and more than eight times likely to suffer a pelvic or femoral stress fracture than their eumenorrheic counterparts. Those with secondary amenorrhea, defined as six or more consecutively missed menses during the past year, were more than twice as likely to suffer a pelvic or femoral stress fracture [70]. In a group of female high school competitive runners, multiple menstrual variables were associated with increased risk for stress fracture, as follows: fourfold increase with late menarche at or after 15 years of age, twofold increase with current amenorrhea, and successive decrease in risk with increasing number of periods in the past year—each menstrual period was associated with an 11% decreased risk of suffering a stress fracture [16].

Osteoporosis

Osteoporosis is a condition marked by decreased BMD, resulting in fragile bone and is defined by the World Health Organization as a T score less than −2.5 in post-menopausal females and males aged 50 and over on dual-energy X-ray absorptiometry (DEXA) scans [71]. Those with a T score ranging from −1.0 to −2.5 are considered to be osteopenic. The T score represents the number of standard deviations a person is above or below a reference database of a healthy 30-year-old adult. For pre-menopausal females, males younger than 50, and children, the Z score should be used [72]. The Z score is the number of standard deviations a person is above or below a reference database of similar age, gender, and body size. A Z-score of −2.0 or below is considered outside of the expected range based on the International Society of Clinic Densitometry. This suggests that something other than aging is causing abnormal bone loss, which should prompt an evaluation for secondary causes of osteoporosis [73]. However, the American College of Sports Medicine notes that, as athletes in weight bearing sports typically have higher than normal BMD, a Z-score of −1.0 or below should be considered low BMD in this population and is worthy of further investigation. DEXA alone should not be used to diagnose osteoporosis, but Z-score combined with secondary clinical risk factors for fracture would support a diagnosis of osteoporosis in this younger age group.

When comparing pre-menopausal female athletes and military recruits with and without stress fractures, those with a stress fracture have demonstrated lower BMD by DEXA [74]. The first study to prospectively examine this link found that lower BMD in the lumbar spine and foot were significant predictors of future stress fracture development in female track and field athletes [64]. Studies in male athletes have shown similar, but inconsistent, trends [68, 75, 76]. Cancellous, as opposed to cortical, bone stress fractures have more consistently correlated with lower BMD in both the female [77] and male [78] athletic population.

DEXA testing should be considered in athletes with a stress fracture and any of the following: stress fracture of cancellous (rather than cortical) bone, recurrent stress fracture of any site, low BMI (<18.5 kg/m^2), oligo- or amenorrhea for 6 months or more, history of disordered eating or low energy availability, chronic medical condition associated with bone loss, chronic medication use associated with low BMD and non-weightbearing athletes (cyclists, swimmers).

Calcium and/or Vitamin D Deficiency

Calcium and vitamin D are two nutritional components widely recognized as necessary to maintain optimal bone health and have been shown to improve BMD in the general population [79–81]. This increased BMD has been shown to be somewhat protective against stress fractures as described above in the Osteoporosis section.

However, among athletes of ages 18–35 years, the role of calcium and vitamin D in both bone development and the prevention of stress fractures is not as well-established. Some studies have found an association between calcium/vitamin D and BMD/stress fracture risk in athletes [68, 82], while others have not [23, 31, 44, 83]. Nieves et al. utilized dietary data collected during the course of a randomized trial of the effect of oral contraceptive pills (OCPs) on bone health in female competitive distance runners. Dietary variables were assessed with a food frequency questionnaire and found that higher dietary intake of both calcium and vitamin D were associated with a gain in hip BMD and that higher dietary intake of calcium was associated with a gain in whole-body BMD and lower rates of stress fractures [82]. Lappe et al. performed a double-blind, randomized clinical trial among female navy recruits with one group taking 2000 mg of calcium and 8000 international units (IU) of vitamin D supplementation a day while the other group was given a placebo. They found a 21% lower incidence of stress fractures in the supplemented group [84]. Tenforde et al. performed a prospective study among competitive high school runners and found that female athletes who used a calcium supplement were at three times higher risk of developing a stress fracture, though this was not true in males [16]. However, the results of this study have been brought into question as athletes were asked about regarding calcium supplementation late in the study, meaning that injured athletes may have been prescribed calcium supplementation as part of their treatment plan rather than primary prevention [16, 20]. There is limited data focused on male athletes. More prospective studies are needed to evaluate the relationship between calcium and vitamin D with the risk for stress fracture, particularly studies including males [85].

Based on the current evidence linking calcium and vitamin D to increased BMD and an association with stress fracture incidence in females, dietary intake of both nutrients should be discussed and optimized in all athletes, particularly those at high risk [86, 87]. The National Osteoporosis Foundations recommends at least 1000 mg of calcium daily in women 50 and under and men 70 and under, and 1200 mg daily above these age groups in in the general population. Recommended vitamin D daily intake ranges between 600 and 1000 international units (IU) daily, with an upper limit of 4000 IU daily set by the Institute of Medicine [88]. Dietary intake is preferred to supplementation due to both absorption and potential side effects of long-term supplementation.

Low Body Weight/BMI

Low body weight and/or low BMI is often seen in RED-S and has been associated with stress fracture incidence, particularly among female athletes. In one study conducted on a group of competitive high school runners including both males and females, a BMI < 19 was found to be one of the strongest independent predictors for developing a stress fracture with a two- to threefold increase in the rate of stress fracture [16]. Lower adult weight was also noted to increase the likelihood of stress fracture in a study of female army recruits [5, 23]. On the contrary, in a study conducted among a group of competitive collegiate runners including only males, BMI risk scores were not associated with increased risk of stress fracture. This may be due to the need for different criteria required to define low BMI among male athletes [75].

Biomechanics and Strengthening

The amount of force a bone can withstand is proportional to its cross-sectional area and moment of inertia, and military studies have found these parameters to be significantly lower among those who develop stress fractures [5, 89–91]. Faulty biomechanics can contribute to stress fracture risk. These can either be due to abnormal forces or abnormal motions. Increased forces on a normally aligned lower extremity can result in abnormal bone loading, as can normal forces on a malaligned lower limb. Increased forces on a malaligned lower limb are thought to further

increase the risk of stress fracture. Runners with abnormal loading are thought to be at higher risk of stress fracture; individuals with a history of stress fracture have been found to have greater vertical ground reaction force loading rates and peak accelerations [92–94]. Torsional loads have been associated with similar risks [95], and increases specifically in peak hip adduction, absolute free movement, and peak rearfoot eversion have been associated with an increased risk of prior tibial stress fracture [93].

There seems to be a close mechanical relationship between muscle and bone, and it has been hypothesized that muscle is protective rather than causative of stress fractures. Muscles are thought to act as a shock absorber for bones during impact loading, and when they become fatigued, this may lead to increased loads on the bones. For example, fatigue in laboratory studies have shown to lead to a decrease in shock reduction [96, 97], an increase in ground reaction force loading rates and peak acceleration [98, 99], and an increase in bone strain magnitude and rate [100, 101]. Additionally, fatigue can result in altered kinematics, which may alter the direction a bone is loaded into a direction that the bone is less accustomed to bearing force [102, 103]. Studies have shown that stress fracture risk is directly related to muscle size and strength [104, 105]. Increased muscle strength has been shown to be protective from stress fractures in numerous studies including military studies that have shown that previously inactive or less active military recruits have a higher incidence of stress fractures compared to those who are active before beginning basic training [1, 21, 30, 31]. Aerobic fitness and flexibility may also play a role in this, though this has not been as well-defined [5, 19, 29, 44, 106, 107].

Medication Use

Contraceptives

The role of OCPs in the development of stress fractures remains unclear. Some studies have reported a protective effect of OCPs against stress fractures in female athletes [26, 64, 68], while others have found no association [16, 20, 31, 33, 44, 70, 108]. To complicate things further, several small studies of amenorrheic or anorexic females found individuals on OCPs to have higher BMDs in the lumbar spine and hip than those who were not [109–111]; however, other similarly designed studies showed no difference [69, 112] or a slight decrease in BMD with OCP use [113]. Conflicting results and lack of well-controlled, prospective studies make it difficult to assess the independent effects of OCPs on skeletal health in normally menstruating females and females with a menstrual disturbance. While OCPs may help to minimize further bone loss in females with menstrual disturbances, this should be done only after appropriate nutritional counseling focused on achieving and maintaining a healthy weight and well-balanced diet. Caution must be taken to ensure that the resumption of menses following OCP use is not disguising an underlying nutritional disorder, as this will not normalize metabolic factors that impair bone formation and bone health [5]. Depo-medroxyprogesterone shots may also contribute to low BMD and should be avoided in young women [114]. More research is needed on OCPs and their role in the prevention of stress fractures [70].

Other Medications

Other medications such as corticosteroids, thyroid hormone [5], antiepileptics [115], antidepressants [116], aluminum-containing antacids (such as aluminum hydroxide) [117], and proton pump inhibitors [118] are thought to impair bone health and may increase an athlete's risk of osteoporosis. Chronic corticosteroid use has been associated with a rapid loss of BMD, with loss of up to 5–15% of bone density found during the first year of medication use. In addition, individuals on chronic corticosteroids have shown a rapid, increased risk of fractures within 2–3 months of initiation, though this is reversible when medication is discontinued [119]. Oral orticosteroids have been associated with an increased risk of stress fracture within the military, though

time-dependence and reversibility is unknown [23]. While associations with low BMD have been explored, the remaining medications have not yet been investigated as risk factors for stress fractures in athletes [5].

Substance Abuse

Tobacco and Alcohol Consumption

Cigarette smoking and alcohol consumption are known to increase the risk for osteoporosis. Among female army recruits, those with a history of current or past smoking or alcohol consumption of ≥10 drinks per week were at increased risk of a stress fracture [5, 23].

Extrinsic Risk Factors

Extrinsic risk factors are those found outside the body and include poor training habits, improper equipment, type of sport or activity, and time of season. Of these, all are modifiable except time of season.

Non-modifiable Extrinsic Risk Factors

Time of Season

Stress fracture incidence has been reported to be highest during the pre-season and first 6 weeks of training in two recent prospective studies. In a study among National Collegiate Athletic Association (NCAA) athletes of all sports, there was a 42.6% increased risk of stress fracture in the pre-season when compared to the regular season based on rates per athlete exposure [120]. Similarly, among National Basketball Association players, while most injuries occurred during the regular season, nearly half occurred within the first 6 weeks of the 6-month season [121]. These results suggest that athletes are at increased risk of stress fracture during the initiation of training for a new season. It is likely that deconditioning, decreased physical fitness levels and training volume during the off-season, followed by a rapid increase in activity at the start of the season play a role in this trend, as detailed below. Regardless of causation, training and medical staff should be aware of this trend.

Modifiable Intrinsic Risk Factors

Training Variables

Heavy training volume is a major risk factor for the development of stress fractures. Higher weekly mileage is associated with an increased incidence of stress fractures [43] and overuse injuries [122–124] in runners across multiple studies [5]. Running volumes greater than 20 miles per week significantly increase risk for stress fracture [16, 20, 125, 126]. Training more than 5 hours per day is associated with a significantly higher risk of stress fractures among ballet dancers [127]. Conversely, fewer stress fractures were seen in military recruits after reducing intensity or frequency of training [128–130]. In addition to overall volume, sudden changes in duration, frequency, and/or intensity of training also alter an athlete's risk for stress fracture [5]. Altered loading associated with large changes in training may contribute to microdamage accumulation and development of stress fracture. Scaling a running program up too rapidly or too frequently is thought to disrupt the balance between bone microdamage formation and removal [105]. In addition, those with poor physical fitness and low activity prior to starting a new training program are at increased risk of stress fracture [131]. Overall, a gradual and individualized progression in training volume and intensity, with adequate recovery periods, is recommended to minimize risk for stress fracture. No specific cutoffs have been established as "too much" or "too fast," as this varies between individuals and is related to the multiple intrinsic risk factors detailed above.

Training surface has been postulated to play a role in stress fracture risk in athletes. Conceptually, uneven training surfaces can predispose to stress fractures by increasing

muscle fatigue and redistributing bone loading. Engaging in high-impact exercise on less compliant surfaces, such as cement, has been proposed as a potential risk factor. However, studies have shown inconsistent results; some have noted correlations [122, 132] while others have displayed no effect [43, 123, 124]. Interestingly, treadmill runners have been found to be at lower risk for developing tibial stress fractures when compared to overground runners, but they demonstrated less significant improvement in tibial bone strengthening [132]. The difficulty in controlling and quantifying training surfaces is a barrier in further studying this potential association [5]. Whether the increased loading rate associated with less compliant surfaces actually leads to an increased risk for stress fracture remains unclear. What may be the key with regard to stress fracture risk is whether there has been a recent change in training surface to which the runner has not adjusted [105].

Equipment Variables (Footwear and Inserts)

Athletic footwear and inserts (orthoses and insoles) are thought to decrease ground impact forces and provide stability by controlling foot and ankle motion [133, 134]. Through these two mechanisms, shoes and inserts may affect bone loading and thus the risk for stress fracture [105]. A study conducted among United States Marines found that military recruits who trained in shoes older than 6 months were at greater risk of developing a stress fracture. Shoe cost, on the other hand, has not shown any association with stress fracture risk, implying that shoe age may be a better indicator of shock-absorbing quality than shoe cost [21]. Proper orthoses have been shown to reduce stress fracture risk in military recruits [135–137]. However, there is no clear evidence that the same benefits are observed in runners [5, 105]. To complicate matters further, potential protective factors involving adopted gait mechanics (forefoot and midfoot strike gaits) by runners who run barefoot or in minimal shoes have put the role of shoes as an injury prevention tool in question [138].

Type of Sport

Type of sport engagement plays a role in risk for stress fractures. Overall, sports that place a competitive or esthetic value on leanness, often referred to as "leanness" sports, have demonstrated higher reported rates of stress fractures. In one previous study among collegiate athletes at a major university in Australia, the percentage of athletes per season who had stress fractures were found to be as follows: softball 6.3%, track 3.7%, basketball 2.9%, tennis 2.8%, gymnastics 2.8%, lacrosse 2.7%, baseball 2.6%, volleyball 2.4%, crew 2.2%, and field hockey 2.2% [11]. A more recent study looking at NCAA athletes found similar trends with cross-country, women's gymnastics, and track athletes being at highest risk based on stress fracture per athlete exposure and ice hockey, swimming and diving, and baseball/softball athletes being at lowest risk [120]. Among track and field athletes, sprinters, hurdlers, and jumpers have been found to have more foot fractures, while middle- and long-distance runners have more long bone and pelvic fractures [19]. Increased rates of rib stress fractures have been seen in rowers and golfers [139, 140].

A prior history of playing ball sports has been proposed as a protective factor in the development of stress fractures. The jumping, cutting, and sprinting involved in ball sports are thought to provide high-impact, multidirectional loading to the skeleton that may promote peak bone mass accrual and improve geometric strength [141]. Athletes who have participated in these high-impact, multidirectional loading sports, such as basketball and soccer, consistently display greater BMD and enhanced bone geometric properties when compared to those who have participated in lower-impact sports such as running [142, 143]. In the military population, infantry recruits who had participated regularly in ball sports (primarily basketball) for 2 or more years before basic training were found to have a 46–84% reduction in stress fracture risk [144].

In an elite track and field population, those who had participated in ball sports during youth were half as likely to sustain a stress fracture later in life [145]. In a group of competitive high school runners, the boys who had participated in basketball were noted to have an 82% reduction in stress fracture risk [16]. It is important to keep in mind, however, that this potential protective benefit of ball sports for long-term bone health must be weighed against the immediate injury risks that also accompany ball sports [141].

Prediction Algorithms Based on Risk Factors

The ability to utilize these risk factors to identify athletes most susceptible to stress fractures is vital in order to take early action to prevent injury. The 2014 Female Athlete Triad Coalition used six criteria to create a Female Athlete Triad Cumulative Risk Assessment Tool, scored as follows:

1. Low energy availability with or without DE/ED:
   (a) No dietary restrictions = 0 points
   (b) Some dietary restriction by self-report *or* low energy intake on diet logs *or* current/past history of disordered eating = 1 point
   (c) Meets diagnostic criteria for ED/DE = 2 points
2. BMI (absolute BMI cut-offs should not be used for adolescents):
   (a) BMI ≥ 18.5 *or* ≥90% estimated weight *or* weight stable = 0 points
   (b) BMI 17.5 to <18.5 *or* <90% estimated weight *or* 5% to <10% weight loss per month = 1 point
   (c) BMI ≤ 17.5 *or* <85% estimated weight *or* ≥10% weight loss per month = 2 points
3. BMD by z-score on DXA:
   (a) Z-score ≥ −1.0 = 0 points
   (b) Z score −1.0 to −2.0 for weight-bearing athletes = 1 point
   (c) Z score ≤ −2.0 = 2 points
4. Prior stress reaction/fracture:
   (a) No prior stress reaction/fracture = 0 points
   (b) One prior low-risk stress reaction/fracture = 1 point,
   (c) Two or more prior low-risk stress reactions/fractures OR one prior high-risk stress reaction/fracture = 2 points
5. Delayed menarche:
   (a) Onset of menarche at <15 years = 0 points
   (b) Onset of menarche at age 15 to <16 years = 1 point
   (c) Onset of menarche at ≥16 years = 2 points
6. Oligomenorrhea/amenorrhea over 12 month period (current or past):
   (a) 9 or more periods = 0 points
   (b) 6–9 periods = 1 point
   (c) <6 periods = 2 points

The summative score of these six domains is used to define an athlete as low risk (0–1 points), moderate risk (2–5 points), or high risk (≥6 points) [53]. In 2016, Tenforde and colleagues were the first to report the prevalence of stress fractures within each risk category. Athletes assigned to the higher risk categories were found to be more likely to prospectively develop a stress fracture. Nine of 169 (5.3%) low-risk athletes, 11 of 61 (18.0%) moderate-risk athletes, and 5 of 9 (55.6%) high-risk athletes sustained a stress fracture. Adjusted for cross-country participation and age, relative risk was 2.6 for moderate- versus low-risk athletes and 3.8 for high- versus low-risk athletes [146].

In 2019, Kraus et al. looked into a modified Female Athlete Triad Cumulative Risk Assessment tool to predict stress fracture risk in male athletes in a similar manner. The menstrual-related risk factors that cannot be applied to males were taken out of the assessment tool, resulting in four rather than six domains and a total possible risk score of 8 rather than 12, as follows [75]:

1. Low energy availability with or without DE/ED:
   (a) No dietary restrictions = 0 points
   (b) Some dietary restriction by self-report *or* low energy intake on diet logs *or* current/past history of disordered eating = 1 point
   (c) Meets diagnostic criteria for ED/DE = 2 points

2. BMI (absolute BMI cut-offs should not be used for adolescents):
   (a) BMI ≥ 18.5 = 0 points
   (b) BMI 17.5 to <18.5 = 1 point
   (c) BMI ≤ 17.5 = 2 points
3. BMD by z-score on DXA:
   (a) Z-score ≥ −1.0 = 0 points
   (b) Z score − 1.0 to −2.0 for weight-bearing athletes = 1 point
   (c) Z score ≤ −2.0 = 2 points
4. Prior stress reaction/fracture:
   (a) No prior stress reaction/fracture = 0 points
   (b) One prior low-risk stress reaction/fracture = 1 point,
   (c) Two or more prior low-risk stress reactions/fractures OR 1 prior high-risk stress reaction/fracture = 2 points.

Athletes were not categorized into low-, moderate-, and high-risk, but risk was instead looked at based on point increases in cumulative risk scores. Two regression analyses were performed, and both models showed an increased risk for prospective stress fracture with each point increase in cumulative risk score. Depending on the model used, each risk assessment point was associated with a 27–37% increased risk for stress fracture [75].

Such risk assessment models for female and male athletes, respectively, show potential in helping to identify athletes at higher risk of stress fractures. Ideally, athletes who fall in the moderate and high-risk groups, or those with high cumulative risk scores, should be identified early and modifiable risk factors should be assessed and optimized prior to the development of a bone stress fracture.

Conclusion

Identifying risk factors for developing stress fractures is the first step to optimum care of athletes. There are intrinsic and extrinsic risk factors, some of which can be modified to decrease risk. Recognizing these risk factors can help identify athletes who are at high risk of developing a stress fracture and can help guide the management of athletes to ensure their peak health and performance. Prediction algorithms utilizing these risk factors have been tested and show viability.

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2 Sideline and Training Room Evaluation and Treatment for Suspected Stress Fractures

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Philosophy Behind the Decision-Making on the Sideline and in the Training Room

Evaluating a player on the sideline is different from evaluating them in the office, as it focuses on determining whether a player is safe to return to the game or not. This requires a different set of critical thinking and precise decision-making skills to ensure effective management.

Having a high index of suspicion to diagnose a stress fracture in an athlete is important since it has a vague natural history and is a diagnosis of exclusion. The differential diagnosis often includes contusion, muscle strain, tendinopathy, ligament sprain, bursitis, periostitis, avulsion injury, exertional compartment syndrome, nerve entrapment, infection, and neoplasm. An algorithmic approach should be followed when making the decision of whether an athlete with a suspected stress fracture can return to play or not. Boden et al. classified stress fractures as high or low-risk fractures based on the biomechanical environment and the natural history of the fracture, and they proposed that high-risk fractures had a higher rate of nonunion, progression to complete fracture, operative intervention, protracted recovery, and recurrence rates [1–3] (Tables 2.1 and 2.2). Simply, the high-risk stress fracture locations are loaded in tension, have a zone of diminished blood flow, and have a subop-

Table 2.1 Anatomic location of high-risk fractures

Anatomic location of high-risk stress fractures
Femoral neck (tension side)
Patella (tension side)
Anterior tibial cortex
Medial malleolus
Talar neck
Dorsal tarsal navicular cortex
Fifth metatarsal proximal metaphysis
Sesamoids of the great toe

Table 2.2 High- and low-risk fractures

	High-risk fractures	Low-risk fractures
Loading	Tensile	Compression
Natural history	High delayed union or nonunion rate	Low delayed union or nonunion rate
Management	Aggressive Complete: Surgery Incomplete: Strict NWB or surgery	Symptomatic: Activity modification Asymptomatic: No Rx needed observation

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timal healing potential [3, 4]. Low-risk fractures are loaded in compression, have better prognosis, and are unlikely to progress to complete fracture [4]. The sideline and training room management of stress fractures should depend on the risk classification of the suspected fracture.

The team physician must have a sound knowledge of the classification to be able to make the right decision. Overtreatment of a low-risk fracture keeps an athlete out of the game who could be playing. On the other hand, undertreatment of a high-risk injury puts the athlete at risk of significant long-term complications [5]. Low-risk stress fractures may be treated with relative rest and activity modification. When compared with low-risk fractures, high-risk fractures are not likely to heal without complete rest and possibly surgical stabilization. The fundamental decision point is this: if an athlete is suspected of having a low-risk stress fracture, return to play is allowed. However, if the athlete is suspected to have a high-risk stress fracture, the athlete must be removed from the game.

This chapter will be organized by discussing the algorithms for diagnosing stress fractures, both high risk and low risk, and how to determine from a given clinical picture whether or not an athlete can return to play. Refer to Chap. 7 of this textbook for a more detailed description of a holistic approach to treatment of the injuries.

Evaluation

History

Athletes in the earlier stages of developing a stress fracture typically report gradual onset of a vague, non-traumatic pain that is classically not present at the start of the physical activity but does occur after or toward the end of physical activity. In later stages of stress fracture development, pain begins earlier in the activity and even with normal daily activities [6–8]. It is important for a team physician to be familiar with the athlete's current health status, diet and training program, since history of stress fractures generally reveals an abrupt increase in the duration, intensity, or frequency of physical activity without adequate periods of rest during the preceding 2–6 weeks [9, 10]. Therefore, when there is a suspicion for a stress fracture, the athlete must be asked about a change in his/her training regimen, training on a harder surface or track, or any new footwear [11]. In addition, menstrual status, history of smoking, and nutritional irregularities must be noted in female athletes [12].

Physical Examination

Physical examination usually reveals point tenderness with palpation, swelling, and periosteal thickening over the fracture site for superficial bones [2]. However, this may not be elicited in deeper located bones. A gentle range of motion or provocative tests, such as fulcrum test, single leg stance test, and hop test, may elicit pain in these fractures [5].

Imaging

In normal fracture care, radiographs are typically the imaging modality relied on to evaluate for injury and healing progress. Most, if not all, training rooms at the Division I college and professional level have a portable radiograph machine readily available to facilitate evaluation of the players without having to transport them to an emergency room or other healthcare facility. While this is helpful in many clinical scenarios, the radiographs in setting of a potential stress fracture can sometimes be deceiving [11, 13]. Only some types of stress fractures show abnormalities on radiographs, while others go undetected on this imaging modality [11, 13]. For example, an early "stress fracture" of the hip cannot be seen on plain radiographs, but is easily visible on an MRI.

When the team physician is concerned for stress fracture, it is of the utmost importance to use the history and physical exam to determine the exact location where the stress fracture is most likely, as the location will determine the

ability to use radiographs as a diagnostic tool [11]. If it is any location where radiographs do not show stress fractures well, such as in the hip or proximal tibia, then the physician should know that negative x-rays do not mean the athlete lacks a stress fracture, but it means that an MRI or bone scan, which are much more sensitive, is required to further evaluate for a stress fracture [11, 13].

Stress Fractures of the Hip

History

Stress fractures of the femoral neck account for approximately 11% of stress fractures in athletes and can occur on the tension (superior) or compression (inferior) side of the femoral neck [11, 14]. In any athlete, especially a distance runner who presents with hip, thigh, or groin pain, hip stress fractures should be considered [8]. Athletes with hip stress fractures typically present with insidious onset of gradually worsening groin pain that is worse with activity as early as 2 weeks after increasing exercise intensity [9].

Stress fractures of the femoral neck are classified as the distraction or tension type and the compression type. The tension type is classically located on the superior cortex and is a high-risk stress fracture. The compression type stress fractures are located at the cortex of the mid or lower medial margin of the femoral neck and are intermediate stress fractures.

Physical Examination

A comprehensive physical examination, including the affected and contralateral hip, knee, pelvis, and lumbosacral spine, is required. Athletes may have an antalgic gait, the muscle tone and bulk is normal, and swelling is absent. Symptoms are worse with weight bearing. Athletes may have pain with "logrolling" of the hip joint, passive extremes of motion, and straight leg raise [10]. Hop testing is also useful but should be performed with caution if the athlete has severe pain with single leg stance.

Imaging

Radiographs of hip stress fractures are generally negative in the first 2 weeks. In those athletes with a suspected hip stress fracture, MRI or bone scan are most helpful for making the early diagnosis (Fig. 2.1).

Shin et al. suggested that MRI might be more specific than a bone scan in detecting stress fractures of the hip and in differentiating other causes of hip pain including synovial pit, iliopsoas muscle tear or tendinitis, obturator externus tendinitis, arthropathy, or focal bone abnormalities such as avascular necrosis or cysts [8].

Return to Play Current Game

The athlete with a femoral neck stress fracture should not be allowed to return to game. Both high and intermediate severity hip stress fractures carry the risk of progression to complete fracture.

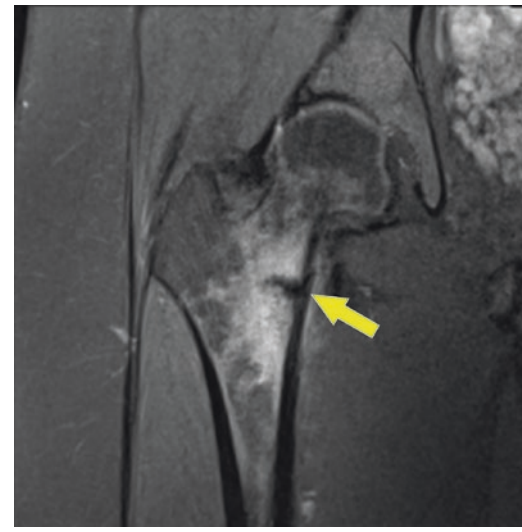

Fig. 2.1 Coronal MRI image of a stress fracture on the tension side of the right hip

Game Day Treatment

After the decision to keep the patient out of the game is given, weight bearing should not be allowed until further imaging is obtained. The use of crutches is essential. Radiographs are obtained first but are usually negative. MRI should be obtained urgently for the definitive diagnosis.

Training Room Treatment

Weight bearing should be restricted to non-weight bearing (NWB) using crutches for both high-risk fractures and intermediate fractures. X-rays should be obtained first for diagnosis. However, X-ray is usually normal initially, unless there is a chronic or complete fracture. MRI is the choice of diagnostic imaging and needs to be obtained as early as possible.

Decision Points

Stress fractures of the hip have the highest risk for eventual displacement, nonunion, or avascular necrosis [10].

High-risk fractures warrant immediate restriction from weight bearing, followed by percutaneous screw fixation using 6.5 or 7.3-mm cannulated screws [11]. Intermediate hip stress fractures including compression (inferior) sided injuries are usually managed nonoperatively, which most often yields excellent results. Mostly, they either do not have an identifiable fatigue fracture line on MRI or have a fracture line that involves less than 50% of the femoral neck [10]. However, if the fracture line involves more than 50% of the width of the femoral neck, urgent surgical intervention is indicated [10, 11].

Stress Fractures of the Femur

History

Stress fractures of the femoral shaft represent approximately 3–20% of stress fractures in athletes [12]. Athletes with a femoral shaft stress fracture (FSSF) usually present with insidious onset of pain in the groin, thigh, or knee [2, 12]. Long-distance runners, jumpers, dancers, female athletes, and older athletes are at the highest risk for developing femoral stress fractures [15].

In the initial stages, the pain is often attributed to a quadriceps muscle strain or tear. If training continues after the onset of symptoms, the pain progresses to the point where it is experienced not only with running but also with ambulation and even during rest at night [8].

Physical Examination

Because of the anatomic location of the fracture, it may be difficult to localize the site of injury. Physical examination only rarely reveals swelling, limited range of motion, or pain with forced rotation [16]. The fulcrum test may aid in making the diagnosis [2, 12, 17]. For this test, the athlete is seated on the examining table with the lower legs dangling. The examiner places his/her arm under the athlete's thigh to use as a fulcrum and moves it from distal to proximal thigh while applying gentle pressure to the dorsum of the knee with the opposite hand. At the point of the fulcrum under the stress fracture, gentle pressure on the knee produces a sharp and localized pain [18].

Imaging

Radiographs are usually negative initially, as in other stress fractures. MRI shows periosteal as well as bone marrow edema involving the medial aspect of the femur approximately at the junction of the proximal and middle thirds of the femoral diaphysis, sometimes with a fracture line present as well.

Return to Play Current Game

The decision to allow returning to current game is controversial. The decision should be based on the symptom severity and physical exam findings.

Game Day Treatment

Symptoms are controlled first. Athlete should be considered for relative rest and should use crutches with no weight bearing allowed initially. Radiographs are obtained for diagnosis and are usually normal. MRI is then obtained for definitive diagnosis if radiographs are inconclusive.

Training Room Treatment

The athlete should be considered for the relative rest program. X-rays of the femur are obtained first for diagnosis. However, X-ray is usually normal initially. MRI or bone scan are most often confirmatory of the diagnosis [12].

Decision Points

Femoral shaft stress fractures (FSSFs) are considered to be low-risk stress fractures. The initial approach should include relative rest and activity modification to a pain free level [19, 20]. Depending on the athlete's symptoms and clinical findings, this may range from discontinuation of the aggravating activity, discontinuing all training activities, or limiting the athlete to only non-weight bearing activities for 4–8 weeks. Once the athlete is pain-free and non-tender on physical examination, weight bearing should be gradually increased. The return to sports is usually allowed by 12 weeks [19, 21].

Stress Fractures of the Tibia

History

Stress fractures of the tibia present with pain localized to the fracture site with axial and/or impact loading of the tibia, and they are one of the most common types of stress fractures [11]. Running athletes are particularly at risk for this type of injury [11]. As with all stress fractures, the localization of pain by the athlete during the history is a key component of making the correct diagnosis. The two sites where the fractures most often occur in the tibia, and thus this pain will most often occur, are the proximal tibia or the midshaft tibia. It is important to note that, if no diagnosis or treatment is made, the pain will eventually progress to the point that it stops the athlete from training or competing.

Physical Examination

On physical examination, a proximal tibia stress fracture will show tenderness to palpation most commonly in the proximal and medial tibia. If the fracture is in the midshaft area, the tenderness to palpation will be anterior in the lower leg and toward the middle of the tibia.

Imaging

Radiographic evaluation of the tibia is inconsistent for demonstrating a stress fracture. If there is a proximal stress fracture, the radiographs are initially negative. However, in the setting of an anterior tibia midshaft stress fracture, the radiographs may be positive if the injury is chronic. Either an MRI or a bone scan is required to confirm the diagnosis to be a stress fracture [13] (Fig. 2.2).

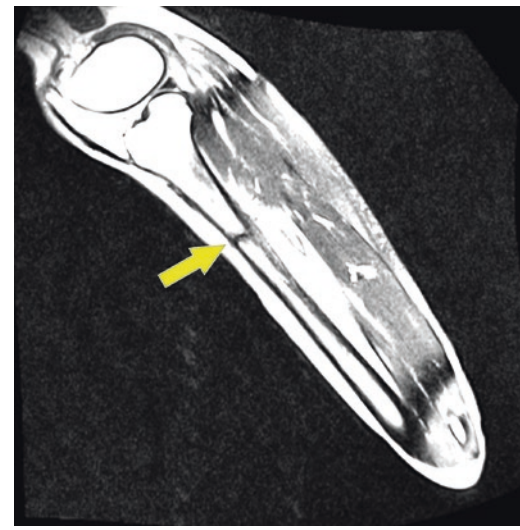

Fig. 2.2 Sagittal MRI image of a stress fracture of the shaft of the tibia

Imaging is especially important in the midshaft tibia because of the variety of stress fractures that can present there [11]. The most common is a fracture in the posteromedial cortex, which is on the aspect of the tibia that undergoes compression during walking and running [11]. Another stress fracture type that can occur is the longitudinal stress fracture [11]. These two stress fracture types fall in the low-risk category. However, stress fractures can also occur less commonly in the anterior (tension) aspect of the midshaft tibia, and these are known as the "dreaded black line" fractures, because they are high risk, difficult to treat, and, when they appear on radiographs, are visualized on a lateral radiograph as a radiolucent line on the anterior aspect of the tibia [1].

Return to Play Current Game

If the team physician suspects a stress fracture of the tibia, the location of that stress fracture informs the appropriate return to play management. If the athlete has a proximal tibia stress fracture, he or she is able to return to the current game. However, if a midshaft tibia fracture is suspected, that athlete must be held out of the game because an anterior aspect fracture cannot be ruled out without advanced imaging.

Game Day Treatment

For a proximal tibia stress fracture, the only treatment required is to control symptoms. In the setting of the more significant midshaft stress fracture, the athlete should be made non-weight-bearing until further imaging is obtained. The use of a pneumatic walking boot can also be considered. In the case of the dreaded black line, surgical stabilization with either a compression plate or intramedullary rod is often required to allow for healing and return to sports activities.

Training Room Treatment of Stress Fractures

To treat these stress fractures in the training room (long-term), a variety of approaches are required. The proximal stress fracture requires relative rest and symptomatic management [11]. If the midshaft stress fracture is confirmed to be one of the two low-risk types (posteromedial cortex or longitudinal), relative rest is the best approach [11]. However, if the stress fracture is of the "dreaded black line" (anterior side) variety, the optimal treatment approach is not as well-defined. The fracture healing for anterior midshaft fractures is often resistant to the relative rest approach [11]. Multiple additional treatment modalities have been explored including pneumatic walking boots [11, 22, 23] and intramedullary nailing [13, 24–27]. Data on intramedullary nailing to treat these difficult fractures has been promising, although the level of evidence is low and there are significant concerns for bias [24, 28].

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3 Pathophysiology and Epidemiology of Stress Fractures

Oisín Breathnach, Kelvin Ng, Kurt P. Spindler, and David N. Wasserstein

Abbreviations

BMD	Bone Mineral Density
BMI	Body Mass Index
MRI	Magnetic Resonance Imaging
PTH	Parathyroid Hormone
SF	Stress Fracture
WNBA	Women's National Basketball Association

Stress Fracture Pathophysiology

To understand the pathophysiology of stress fractures in bone, a review of basic bone biology including normal bone metabolism and turnover is necessary. From this understanding, the pathophysiology of stress fracture development will be outlined. Finally, this section will identify individual clinical parameters that have been linked to the development of stress fractures and summarize their implication and relevance.

Bone Biology

Bone has two forms at the microscopic level: woven and lamellar bone. Woven bone is immature with random orientation and collagen that is not stress oriented. Lamellar bone, in contrast, is mature and organized with stress-oriented collagen [1]. The mechanical properties of lamellar bone can change depending on the direction of the applied force. The macroscopic subtypes of lamellar bone include cortical and cancellous (trabecular) bone. The former is denser and has a low turnover rate. It is composed of packed osteons also called Haversian systems, which are connected by Haversian canals (Fig. 3.1). These canals contain the neurovascular supply of bone. Cancellous bone, however, has a higher turnover and is between 30% and 90% porous depending on the location. Cancellous bone is found more commonly in the metaphysis of long bones, compared to cortical bone, which is found in the diaphysis.

The matrix of bone is approximately 40% organic and 60% inorganic. The organic portion of bone is primarily type-1 collagen – the component which provides tensile strength. The remaining organic portion (~10%) consists of proteoglycans, which provide compressive strength, and matrix

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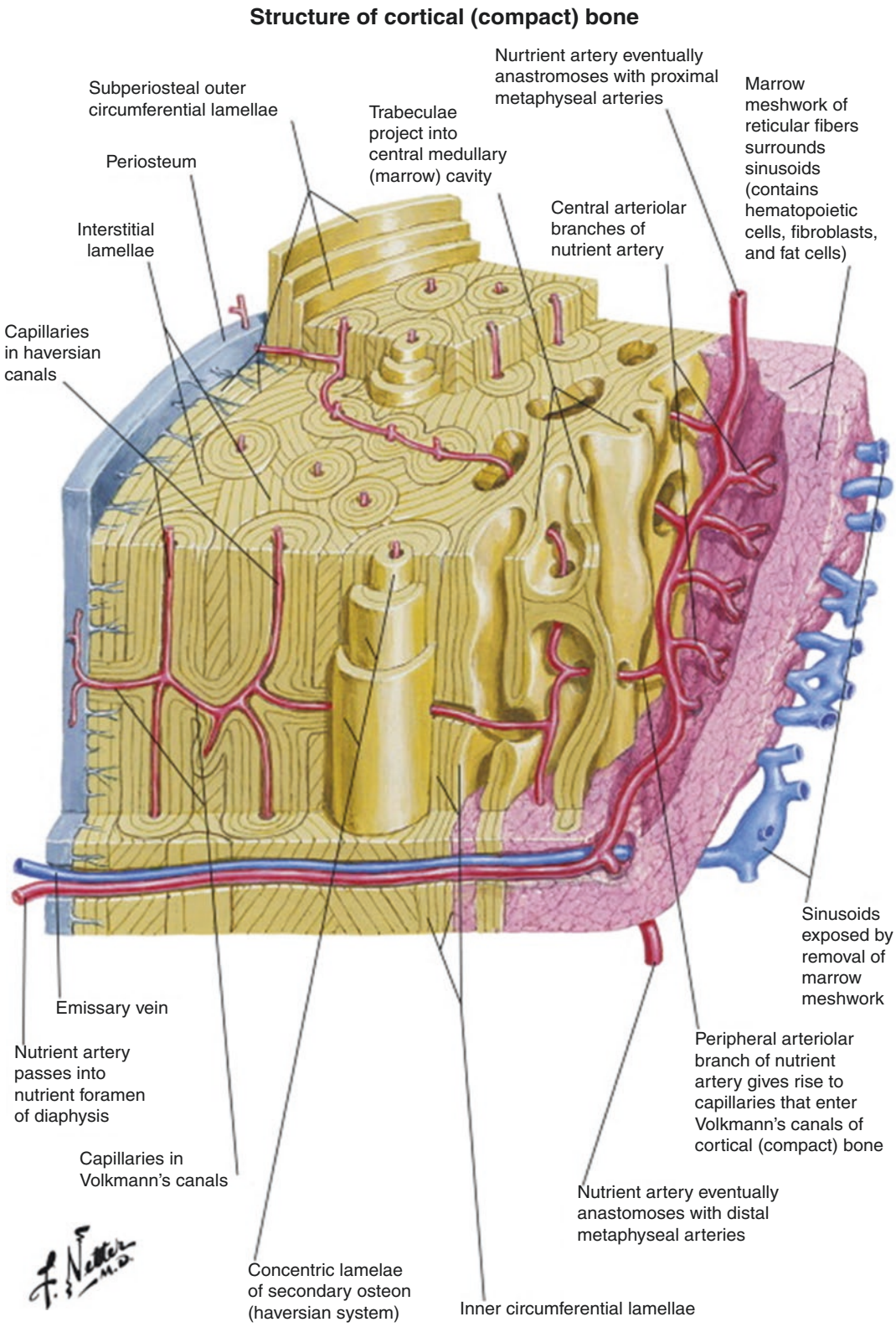

Fig. 3.1 Illustration of the Haversian system and vascular supply in cortical bone. (Reprinted with permission from Elsevier Books License 2012)

proteins. The function of these matrix proteins (e.g., osteocalcin) is to promote mineralization and bone formation. The inorganic component includes calcium hydroxyapatite, which is responsible for compressive strength, and osteocalcium phosphate. The inorganic component is also the mineral portion, which plays a role in calcium metabolic pathways.

Normal bone metabolism is a balanced sequence of bone turnover that includes bone breakdown, known as osteoclastogenesis and bone formation, known as osteoblastogenesis. Osteoclasts are the cells primarily responsible for osteoclastogenesis, and osteoblasts for osteoblastogenesis. Many endogenous hormones regulate metabolism, including parathyroid hormone (PTH), calcitonin, growth hormone, thyroid hormone, estrogen, and testosterone. Endogenous and exogenous steroids, including vitamin D and glucocorticoids, also regulate both calcium and bone metabolism. Factors that promote bone formation do so by either promoting osteoblastogenesis (e.g., PTH, vitamin D) or suppressing osteoclastogenesis (e.g., calcitonin, estrogen). Factors that promote bone breakdown typically suppress osteoblastogenesis (e.g., glucocorticoids).

When stress is applied to bone, Wolff's Law dictates that bone will remodel in response to mechanical stress. The exact method by which bone remodels is not truly understood, but two theories predominate. In the Piezoelectric charge theory, tensile sided strain is said to create electropositive forces that stimulate osteoclastogenesis, while the compression side is subject to electronegative forces that stimulate osteoblastogenesis. The result is the formation or remodeling of bone to increase bone mass on the compressive side in response to mechanical stress. A second theory, the Hueter–Volkmann law, states that bone remodels in small packets of cells in a process called osteoclastic tunneling. Here, there is bone resorption followed by capillaries to introduce blood supply and osteoid producing cells to lay down new osteoid.

Bone Pathophysiology in Stress Fracture

"Stress fracture" constitutes a spectrum of injury which includes bone strain, stress reaction, and stress fracture. The etiology is repetitive loading in the setting of inadequate bone remodeling. The spectrum of injury reflects to some degree the quantity of strain, although exact thresholds are not known and likely mediated by numerous individual host factors in addition to the inciting activity. In general, repetitive injury is more likely to occur in the lower extremity, which sees greater loads than the upper extremity in ambulatory athletes, and with activities that are high volume and offer repetitive loading. Running, for example, produces ground reaction forces approximately five times greater than walking. The result of excess strain is an accumulation of micro damage leading to fatigue reaction or fatigue failure. When the area of fatigue failure is inadequately repaired, it can result in crack initiation in the bone [2] (Fig. 3.2).

Stress injury may also occur with normal strain, but this is typically in the setting of depressed bone remodeling. These injuries are known as insufficiency reactions or fractures. They are more common in the setting of metabolic diseases, hormonal imbalances, and osteoporosis. In the setting of older persons with osteoporosis, both reduced remodeling and structural changes in the trabecular and cortical bone

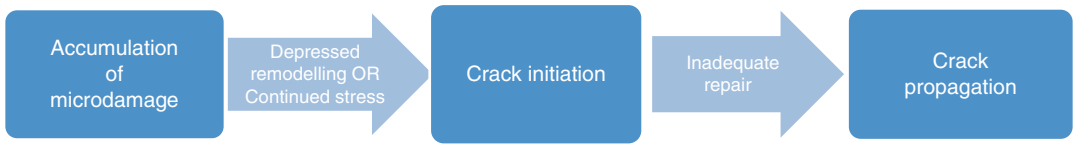

Fig. 3.2 Crack initiation in bone

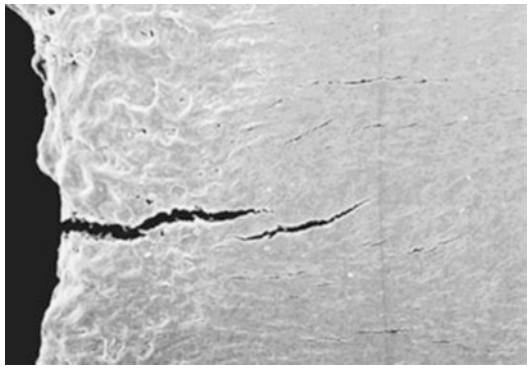

Fig. 3.3 A simple model for the propagation of stress injury in bone (Reprinted with permission from Nalla et al. [2])

leading to reduced biomechanical strength and contribute to the susceptibility to insufficiency fracture at physiologic loads [3]. The dichotomy of fatigue failure and insufficiency is typically more a continuum with respect to athletes who experience greater than physiologic strain through activity, but often also exhibit risk factors for insufficiency failure, placing these subpopulations of athletes at greatest risk (Fig. 3.3).

Another special consideration in the pathophysiology of stress fractures in athletes is the influence of skeletal muscle. Muscles may protect the tibia during running by producing shear forces that counteract the joint reaction forces and resulting in reduced net shear stresses in the tibia. It has been hypothesized that reduced lower leg muscle strength increases the risk of stress fracture through this mechanism [4, 5]. Significantly lower knee extension power was observed in a case-control study of female runners with and without stress fractures [6, 7]. The potential protective effect of muscle may be diminished due to fatigue associated with excessive training. This may be seen in "new exercisers" and military recruits [8]. Prolonged overload of the neuromuscular system due to muscular fatigue (*neuromuscular hypothesis*) [9] can lead to sustained impairment of nerve conduction and muscle contraction [10].

Finally, there is an oxidation deprivation theory of stress fracture development, which deserves some attention. In this theory, the repeated load of an activity such as running is thought to cause decreased oxygen delivery [11] and brief ischemia [12, 13] in weight-bearing bones. This ischemic environment is thought to stimulate the bone remodeling process, specifically by increasing osteoclastogenesis [14]. The result is weakened bone that is less able to withstand subsequent loads, thereby increasing susceptibility to further stress-related injury. This theory may explain some observations that those new to activity are more at risk [15, 16].

Host Risk Factors for the Development of Bone Stress Injury

Bone Mineral Density and Bone Thickness

Although lower bone mineral density (BMD) is likely a stronger etiological factor in insufficiency fracture development, there is evidence that BMD also plays a role in athletes experiencing fatigue failure-related stress fractures [17, 18]. Female athletes aged 13–22, matched in case-control study following diagnosis of an initial stress fracture, revealed that "cases" of stress fracture had lower spine BMD for their age, despite no differences in menstrual irregularity or physical activity participation. Similarly, the odds of a stress fracture were three times that for persons with a family member diagnosed with osteoporosis.

Another case-control study [6] of female athletes aged 18–45 years with and without stress fractures noted that after adjusting for body weight, those with stress fractures had thinner tibial cross-sectional area, lower trabecular BMD and less cortical area of the posterior tibia.

These associations have been confirmed by prospective studies. The first [19] was a 12-month study of both female and male track and field athletes aged 17–26 years. At baseline, females with lower BMD in the spine were at significantly greater risk of developing a stress fracture. A study of military cadets [20] demonstrated that smaller tibial cortical area, lower tibial bone mineral content, and smaller femoral neck diameter increased risk of developing a stress fracture in

males, and smaller femoral neck diameter was a risk factor in females.

Genetics

There appears to be some genetic susceptibility to stress fracture. Early investigation concluded that ethnicity was a risk factor for the development of stress fracture, with lower rates seen in African American compared to Caucasian and Asian biological females. Much of this difference, however, may be related to inherited differences in bone metabolism [21] through bone calcium mineralization. The association between a family history of osteoporosis in first-degree relatives and increased risk of developing a stress fracture among athletes [6] is also suggestive of a genetic role in bone turnover as a risk factor.

Nutritional Factors

Dietary and nutritional factors may play a role in the pathophysiology of stress fracture. Calcium and vitamin D are important components of normal bone metabolism and contribute to BMD, with the former being a mineral building block and the latter playing a role in both calcium homeostasis and bone turnover. Military randomized trials of recruits demonstrated a 20% reduction in fracture injuries in females with supplementation of 2000 mg elemental calcium and 800 IU vitamin D compared to no supplementation [22] and also noted reduced levels of bone resorption in males and females when supplemented with calcium and vitamin D during initial training [21]. Other research has been inconclusive as to whether dietary intake of calcium is important in the development of stress fractures [23, 24].

Other macronutrients may play a role in susceptibility to stress fractures, although the potential pathophysiologic mechanisms are unclear. Female military recruits, for example, with both anemia and iron deficiency, were more likely to develop a stress fracture [25].

Menstrual Irregularity

Late onset menarche appears to be a risk factor for stress fracture development [20, 24]. It is unclear whether this is due to low peak bone mass attainment, or whether it is a marker of another influence such as excessive training, or low body weight/body fat. The association is further confounded by the fact that under normal circumstances female athletes appear to reach menarche later than their non-athlete counterparts [26].

Disordered menstruation has also been linked to stress fracture risk. Estrogen functions to increase bone mass by inhibiting osteoclastogenesis. It may also function by reducing the adaptation to stress [27]. As such, numerous studies have demonstrated that female athletes who are amenorrheic [23, 28, 29] or oligomenorrheic [23, 24, 30] are at increased risk of stress fracture. Authors have hypothesized about the combined role of menstrual irregularities and low BMD in some female athletes with the so-called “female athlete triad” (disordered eating, amenorrhea, and decreased BMD). The presence of all three components is seen in 1–14% of female athletes, but up to 78% of female athletes have at least one aspect of the triad at a given time [31].

Summary

Bone stress injury occurs via an imbalance of repetitive stress and normal bone remodeling/recovery in response to that stress. Although the paradigms of fatigue failure (high stress overwhelming normal turnover) and insufficiency failure (normal stress overwhelming disordered turnover) are a simple means of conceptualizing this disorder, components of both will contribute to stress injury in any one individual. This is further complicated when one considers that many of the host factors that influence the pathophysiology of bone stress injury are also inter-related.

From a practical standpoint, the clinician who will diagnose and treat patients with bone stress injuries must understand the basics of bone biology, including stress remodeling. Once a diagnosis has been made, further probing into the potential role of etiologic factors is recommended. This may include diet and nutritional deficiencies, menstrual irregularity, family his-

tory, and training volume. Some of these factors may be modifiable and useful in both the treatment of the current stress injury, as well as the prevention of future injury.

Stress Fracture Epidemiology

The epidemiology of stress fractures is described as the occurrence of stress fractures in athletic populations and is typically expressed based on exposure (e.g., number of stress fractures per athlete-years or per athlete-exposures). One of the challenges in defining the incidence of stress fractures lies in accurately determining the exposure component. Stress fracture cases are comparatively easy to identify, typically through chart records or physician visits. The challenge of a retrospectively designed study is that while it may identify most or all stress fractures over a given time period, accurate information regarding athletic exposure is comparatively lacking. Consistent and accurate injury reporting data is important to identify risk factors, at-risk subpopulations, and monitor the effectiveness of interventions.

A second complicating factor in deciphering the literature defining the occurrence of stress fractures in athletes is the method of diagnosis. Older studies used modalities such as X-ray, which have poor sensitivity in identifying changes [32]. Many newer studies utilized bone scan or MRI techniques, which offer greater sensitivity and will identify stress fractures at an earlier stage. The MRI is so sensitive that it can detect stress reaction, a precursor to stress fractures, and thus, studies utilizing this method of detection will report a greater incidence/occurrence but for a broader spectrum of the clinical disorder, including potentially asymptomatic cases. Many of these topics are explored in further detail in the remaining chapters of this text.

This heterogeneity in diagnosis, study design, and accuracy of exposure preclude the pooling of data to formulate incidence rates by sport or activity, at the current time. Therefore, this chapter will focus on a narrative review of the literature, the most robust of which originates from military populations. Studies from various sports will be reviewed to illicit information on the incidence of stress fractures but also to discuss risk factors common to sports and activities.

Stress Fracture Epidemiology – Military

Military populations are a unique group amenable to epidemiological research on stress fractures, but with less generalizability to non-military populations. Patient follow-up and activity exposure can be well controlled and documented, which allows for more homogeneous comparisons and higher level of evidence designs such as prospective cohorts. Additionally, large numbers of patients can be recruited for study, which is helpful when investigating a condition that typically occurs infrequently, or when performing multivariate analyses to identify risk factors. Most importantly, however, is that military personnel appear to have a higher incidence of stress fractures than the general population, due to the sudden increased and extensive exercise associated with training. Accordingly, multiple military studies on stress fractures have been performed all over the world, including the United States [20, 21, 28, 32–36] Finland [37, 38], Canada [39], India [40] United Kingdom [41, 42], and Israel [43–46]. Patients in these military studies primarily included army and navy recruits.

A common theme in this population is a higher reported occurrence or incidence of stress fractures among females compared to males; 19.1% of females and 5.7% of males reported at least 1 stress fracture [20]. In US Army recruits [33], the incidence of stress fractures was 79.9/1000 female and 19.3/1000 male recruits. A similar pattern was seen among a prospective cohort of 152,095 Finnish conscripts [37], where the ratio of female to male bone stress injury on MRI was 9.2. The overall incidence rate of stress fractures in this population was 311/100,000 person-years (95% confidence interval: 277–345).

There also appears to be a difference in the distribution of stress fracture location between male and female military personnel. Compared to

males, females have higher reported rates of stress fracture for the pelvis, sacrum, and tibia [43].

These sex differences have prompted many researchers to specifically study female recruits. Shaffer et al. [28] identified a stress fracture rate of 5.1% in a cohort (N = 2962) of female US marine recruits. All stress fractures occurred in the lower extremity, most commonly in the tibia, followed by the metatarsal bones, pelvis, and femur. In regression analysis the odds of developing a stress fracture were more than five times higher among recruits who were amenorrheic during the prior year (odds ratio 5.64, 95% confidence interval 2.8–25.8). Lower aerobic performance on a timed run also increased the odds of developing stress fractures in the pelvis and femur.

In a separate study of female US Marine Corps recruits [29], the same authors reported on all overuse injuries of the lower extremity [28]. They determined an incidence rate of lower extremity stress fractures of 1.0/1000 days of training exposure. Having multiple overuse injuries was common, and in multivariate regression analysis, again lower aerobic fitness and amenorrhea predicted increased odds of stress fracture.

Regular exercise prior to entering the military can act as a protective measure against a stress fracture occurring whilst in basic training [38]. Restricting physical exercise to the authorized recruit training program has been identified as a factor to reduce the incidence of stress fractures [46]. Gradual supervised return to army training is recommended following the diagnosis of a stress fracture [34]. Deficient levels of vitamin D in military recruits are a poor prognostic indicator in relation to stress fractures [41, 42] with randomized controlled trials suggesting that supplementation of daily diet with calcium and vitamin D reduced stress fracture rate with female soldiers and reduced bone resorption in both sexes [21, 22]. Further military stress fracture related articles are listed in Table 3.1.

Table 3.1 Military activity and stress fractures

Reference	Country	Study design	Rank of soldier	Notes
Pihlajamäki et al. [38]	Finland	Prospective cohort	Conscripts	Regular physical activity prior to entering the military acts as protective factor for stress fracture IRR = 0.41
Gaffney-Stomberg et al. [21]	United States	Randomized controlled review	Marine conscripts	Recruits supplemented with daily calcium and vitamin D demonstrated improved bone strength and reduced stress fracture rates
Dao et al. [39]	Canada	Systematic review		Review suggests some association between low serum 25(OH)D levels and lower extremity stress fracture in military personnel
Richards et al. [41]	United Kingdom	Retrospective review	Army recruits	Low levels of 25-OHD increased mean time to recovery from stress fracture
Davey et al. [42]	United Kingdom	Prospective cohort	Marine recruits	Low serum 25-hydroxyvitamin D is associated with increased risk of stress fracture
Yanovich et al. [44]	Israel	Case–control	Army recruits	Observed link in female recruits with stress fracture and anemia/iron deficiency
Rice et al. [47]	British	Cohort study	Army recruits	Military foot drill increases tibial forces over a 12-week period
Kunte et al. [40]	India	Prospective cohort study	Cadet	Female army, navy, and airforce cadets present with higher incidence of stress fractures
Milgrom et al. [46]	Israel	Prospective cohort	Infantry recruits	Restricting training to the authorized training protocol was the only training change to decrease incidence of stress fracture
Chalupa et al. [36]	USA	Retrospective review	Army recruits	Observed diagnoses of lower extremity stress fractures reduced following the introduction of the Army Physical Readiness Training program

Stress Fracture Epidemiology – Athletics

Runners are at higher risk of developing stress fractures. A change in activity or increased intensity can precede the development of stress fractures. Muscle fatigue due to prolonged sustained activities, such as long-distance running, can predispose to stress fractures.

A survey study of 1505 runners performed in 1990 [48], identified female long-distance runners at highest risk for stress fracture. Several prospective cohort studies have attempted to better define the epidemiology of stress fractures in runners. One study of 748 competitive high school cross-country and track and field runners identified a 5.4% and 4.0% rate of stress fractures in girls and boys, respectively [49]. The tibia and metatarsal bones were among the most commonly affected. Multivariate models identified late menarche, low BMI, and a prior history of stress fracture as significant contributors to increased risk of new onset stress fracture. A smaller cohort study [50] of 230 competitive high school runners followed for 3 years, stress fractures were identified in 21/230 (9.1%) athletes, representing an incidence of 0.06 stress fractures per athlete exposure. Another 12-month prospective study in a cohort of 53 female and 58 male track and field athletes, aged 17–26 years, highlighted an incidence of stress fractures of 21.1% with most injuries located in the tibia [24].

Stress Fracture Epidemiology – Tennis

The nature of tennis lends the potential for stress fracture development in both the racket hand and lower extremity from running and sudden stops. Abrams et al. [51] reviewed the literature for case reports on uncommon stress fracture locations in tennis players, and identified them in the ischium, first rib, humerus, sacrum, patella, hook of hamate, ulna, and distal radius. Another study [52] examined a case series of high-level junior tennis players, noting seven cases of second metacarpal stress fractures postulated to be related to racket grip.

The largest tennis study followed 139 elite tennis players of a median age 20 years, 65% male and 57% professional status over the course of 2 years [53]. In total, 15 players had 18 stress fractures for a rate of 12.9%.

Stress Fracture Epidemiology – Pediatric/Adolescent Athletes

Particular attention has been directed towards pediatric/adolescents with respect to describing stress fractures. This is an important subpopulation due to potentially open physes and associated metabolic changes that accompany menarche. A national survey study of adolescent girls [54] has followed 6831 girls aged 9–15 years for 7 years. Among them, 267 (3.9%) developed a stress fracture. Multivariate modeling demonstrated that running, basketball, cheerleading, and gymnastics were all significant predictors of developing a stress fracture. An increase in the annual incidence of paediatric stress fractures has been demonstrated in New York, USA. Analysis of 11,475,386 outpatient visits, between 2000 and 2015, showed that the annual incidence of paediatric stress fractures increased from 1.37 cases per 100,000 outpatient visits in 2006 to 5.32 per 100,000 visits in 2015 ($\rho = 0.876$, $P < 0.01$) [55].

Stress Fracture Epidemiology – Other Sports

Individual case reports and series have been published, documenting the occurrence and incidence of stress fractures in various sports. These are reviewed in Table 3.2. Cricket, for example, provides specific incidence rates between innings bowled and hours played. Baseball reports a 5.4% incidence percentage in professional baseballers. Para athletes (260) training for the 2016 and 2018 Paralympic Games reported an almost 10% rate of previous bone stress injury. Some para athletes reported a history of more than one

Table 3.2 Stress fracture epidemiology by miscellaneous sports

Reference	Sport	Study design	*N*	Incidence	Notes
Furushima et al. [56]	Baseball	Case series	200	5.4%	Incidence of olecranon stress fracture
Duckham et al. [57]	800 m running	Prospective	70	3.3%	12-month monitoring. Total body BMD assessed by x-ray absorptiometry
Wright et al. [17]	Athletes	Systematic review	–	OR 4.99/2.31	Risk of stress fracture increased with previous history of stress fracture and female sex
Abbott et al. [58]	Female sports	Systematic review	–	13%	Cross country, gymnastics and lacrosse are high-risk sports
Rizzone et al. [59]	NCAA sports	Retrospective review	671/11778145	5.7/1000000	Women experience more stress fractures, often in pre-season and predominately in the lower limb
Miller et al. [9]	Track/ Field	Case series	57	–	Mean return to play 12–13 weeks
Alway et al. [60]	Cricket	Retrospective review	368	2.46/100	Incidence of stress fractures per 100 bowlers
Frost et al. [61]	Cricket	Prospective cohort	248	51.6/10,000 player-hours	Professional; SF to low back had longest return to play

bone stress injury with most injuries common to the metacarpal/hand region [62].

Stress Fracture Epidemiology – Incidence and Return to Play

Several challenges are outlined in relation to the diagnosis of stress fractures. A delay in diagnosis may lead to a delay in treatment initiation. A delay in return to play is a recognized consequence of stress fractures. A mean return of 12–13 weeks has been reported in athletics. Other sports report delayed returned to sport with navicular and lower spinal stress fractures [61, 63, 64].

Summary – Epidemiology

The reported incidence and occurrence of stress fractures in the literature is variable. The most robust data from the military suggests that new activity (i.e. recruits) and females have the highest incidence of stress injury. Among athletes, the pattern of injury and incidence/occurrence varies by sport and level of competition. Return to sport may be slow. Modification of activity is a key factor in prevention of stress fractures.

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4 Diagnostic Imaging Evaluation of Stress Fractures

Scott S. Lenobel, Jason E. Payne, and Joseph S. Yu

Athletes are particularly prone to injuries that are related to overuse. In the general athletic population, the incidence of stress fractures is about 1% but varies according to activity and can occur in up to 20% of runners [1, 2]. The location where a stress fracture develops also is specific to a particular sporting activity [3, 4]. It is reported that 60% of athletes presenting with a stress fracture have experienced a prior stress fracture [5]. Overuse injuries produce stress induced changes that may alter the architecture of the bone. Stress is defined as any force or absolute load that is applied to a bone. These forces arise from having to bear unusual weight or repetitive load or are created when there is an imbalance of muscular support [6–8]. Wolff's Law dictates that a change in the mechanical environment of a bone from new or intermittent stress elicits the remodeling of the architecture of that bone to adjust to its new environment [9]. Increases in muscular strength often precede strengthening of the bone, which can create an imbalance between the relative strength of these tissues. Furthermore, when muscles fatigue during exercise, the protective effect of muscle tension diminishes, which reduces the ability of bone to resist stress.

A stress injury represents unsuccessful adaptation by a bone under duress [10, 11]. Stress fractures are generally divided into two categories. *Fatigue* stress fractures occur when normal bone is subjected to repetitive stresses, each below the threshold of bone failure. Mechanical failure develops as a consequence of an imbalance between microfractures and the ability of the bone to remodel or repair itself [7, 12]. An example of this occurs when an athlete abruptly changes a training regimen, not allowing sufficient time for bone to remodel in response to the added stress. *Insufficiency* fractures occur when normal stresses are applied to an abnormal or pathologic bone that is incapable of adaptation. Osteoporosis is the most common cause of an insufficiency fracture, along with osteomalacia, prior bone irradiation, and metabolic disorders [12].

Fatigue stress fractures related to overuse are relatively common in certain groups, particularly athletes and military personnel [13, 14]. The incidence of stress fractures among females in the military tends to be higher than in men, but this difference has not been consistently observed in athletes [15–18]. The most common pathologic bone abnormality in older athletes that increases the risk for stress fractures is osteoporosis, with the highest reported prevalence occurring in postmenopausal women [19, 20]. A variety of other conditions associated with abnormal underlying bone also predispose an athlete to insufficiency

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fractures including rheumatoid arthritis, corticosteroid use, and diabetes mellitus [21, 22].

Forces resulting in osseous injury can be classified as compression, tension, and/or shear. It is useful to consider these forces when assessing the morphologic properties of a stress fracture. For instance, distance runners tend to develop stress fractures in the posteromedial aspect of the tibia owing to repetitive compressive forces, whereas dancers and jumping athletes tend to develop stress fractures in the anterior tibial shaft due to tensile forces.

Evolution of Imaging

The imaging appearances of stress-induced injuries change over time and the rate of change is affected by factors such as the bone involved, location of injury, inciting activity, and age [23]. The sensitivity of radiography for early diagnosis of stress fractures is low because forces tend to distribute longitudinally along the cortex, producing subtle changes at the surface of the bone and the periosteum [14, 19]. This early phase is referred to collectively as a stress response or stress reaction. If the cyclic loading continues, progressive deformation of the bony architecture localizes to a focal weakened area of the bone, resulting in a uni-cortical break in the cortex or a true stress fracture. Athletes who develop fatigue fractures often exhibit the following triad: a new or different activity has been introduced in their training, the activity is strenuous, and the activity is repeated cyclically. In a stress reaction, there is still active healing of the microfractures but in a stress fracture, the progressive forces ultimately exceed the elastic range of the bone leading to structural failure.

Stress fractures account for at least 10% of patients encountered in a typical sports medicine practice [1]. Imaging has traditionally provided diagnostic support for evaluation of these patients with modalities depicting variable sensitivity and specificity according to the stage along the continuum of a stress injury [24]. Radiography continues to be a low-cost frontline technique but is limited by a lack of sensitivity, especially early in the process. Other than radiographs, the first modality to have an impact on the diagnosis of osseous stress injuries was whole body bone scintigraphy utilizing technetium-99m-methylene diphosphonate (Tc-99m-MDP). Stress fractures are visible on bone scans days to weeks earlier than radiographs. For many years, it served as the gold standard for early confirmation of stress-induced changes related to increased bone metabolism and osteoclastic activity. The limitation of bone scintigraphy was that it lacked specificity in areas that ordinarily resulted in an increase in radiopharmaceutical uptake; however, the advent of triple-phase scanning with additional angiographic and blood pool phases contributed to improved specificity [25].

Although computed tomography (CT) has shown superior spatial resolution in comparison to other imaging modalities, its role in evaluating patients with stress fractures continues to be limited. Recently, however, utilization of multidetector CT has increased due to the ability to depict the stress fracture line in coronal and sagittal high resolution multiplanar reconstructed CT images as well as 3D volume rendered images [26]. This has increased the utilization of CT for differentiation of stress fractures from other entities such as osteoid osteoma, which may have similar radiographic appearances. Ultrasound also has a limited role in the diagnosis of stress-related injuries although it has the ability to assess the superficial cortical surface in bones close to the skin as well as fracture lines, periosteal reactive changes including callus formation, edema in peri-osseous tissues, and increased perfusion [27, 28].

Most recently, magnetic resonance imaging (MRI) has been shown to be extremely sensitive to the pathophysiologic changes that are associated with stress-induced conditions and provides greater specificity than radionuclide imaging owing to its superior spatial resolution [29, 30]. MRI has been efficacious in characterizing early changes of stress injuries with high sensitivity and specificity including local hyperemia and edema, periostitis, bone marrow changes, and cortical failure. MRI is considered the current gold standard for imaging of stress injuries and

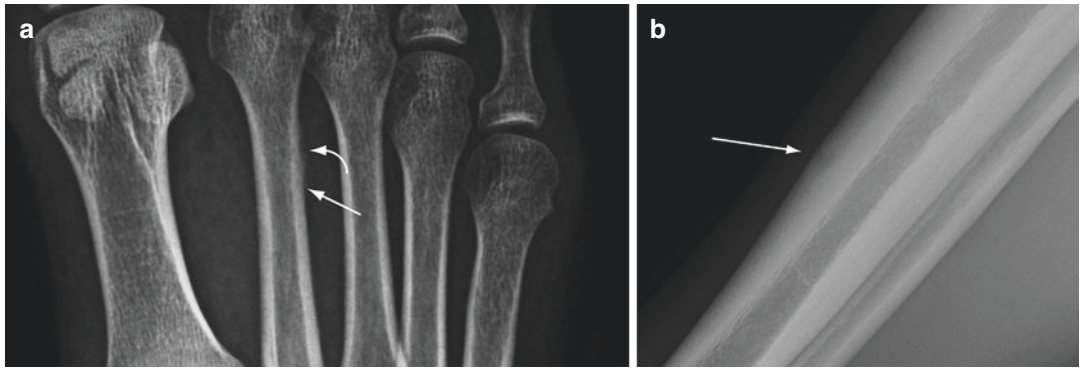


Fig. 4.1 Early stress response on radiography. (**a**) Frontal radiograph of the forefoot shows focal osteopenia of the lateral cortex of the distal second metatarsal shaft (white arrow) and periostitis (curved arrow). (**b**) Lateral radiograph of the tibia shows focal cortical osteopenia (arrow)

also has been useful in determining clinical severity, guiding therapy, and estimating the duration of disability [31, 32].

Imaging Techniques

Radiography

The initial workup of an athlete with pain should begin with a radiographic evaluation of the area involved. Radiography is the least expensive and most widely available imaging modality and radiographs should be obtained in at least two planes [33]. Accuracy is increased when radiographs are optimized and a reliable search strategy is employed [34]. Even with optimal positioning and a detailed history, radiographic findings can be subtle or nonexistent, leading to sensitivities of only 15–35% in initial radiographic imaging [33, 35]. A common approach is to critically evaluate the integrity of the cortex for changes in density (Fig. 4.1), as well as for periosteal reactive and endosteal reactive changes (Fig. 4.2). The medullary cavity should be assessed for the presence of impacted trabeculation and linear uni-cortically based sclerotic bands. Other findings include transverse or longitudinal breaks in the cortex (Fig. 4.3) as well as trabecular angulation and distraction which may be a manifestation of progression (Fig. 4.4). Altered cortical morphology which may be either

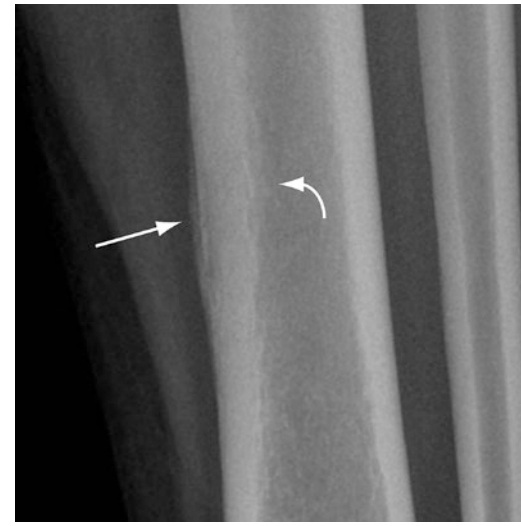


Fig. 4.2 Chronic radiographic findings of stress response. Frontal radiograph of the mid-tibia shows mature periosteal (arrow) and endosteal reactive changes (curved arrow) associated with focal areas of osteopenia in the cortex

focal thickening or thinning is usually an indication of a chronic condition (Fig. 4.5).

It is important to realize that the location and orientation of developing stress fractures influence the radiographic appearance so that fractures at the ends of tubular bones tend to depict linear areas of sclerosis (Fig. 4.6), whereas fractures in the shaft of a tubular bone may be simply a lucent cortical break or focal periostitis [36]. Longitudinal stress fractures have the appearance of a thickened cortex with

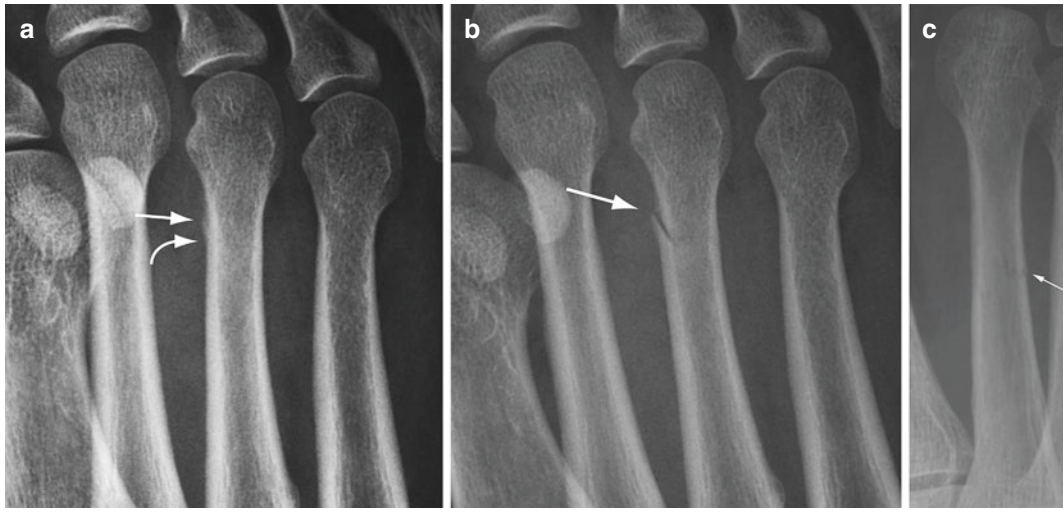


Fig. 4.3 Radiography of early stress fracture. (**a**) Oblique radiograph of the forefoot shows periostitis of the medial cortex of the third metatarsal shaft (arrow) and a subtle lucency (curved arrow) representing the start of a break in the cortex. (**b**) Follow-up image in 3 weeks shows completion of the cortical fracture with oblique lucency (arrow). (**c**) AP radiograph of the foot shows a subtle linear lucency in the lateral cortex of the second metatarsal shaft representing an early nondisplaced stress fracture (arrow)

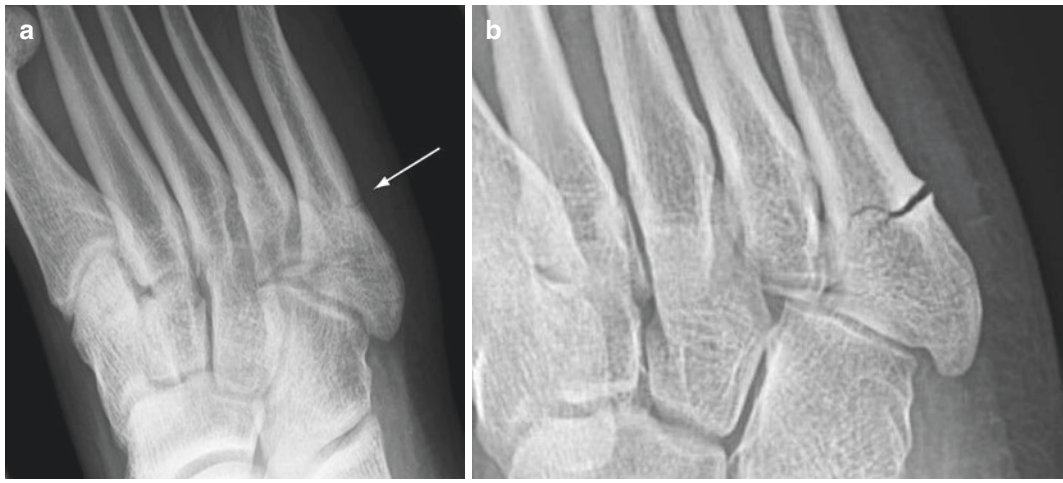


Fig. 4.4 Stress fracture progression. (**a**) Oblique forefoot radiograph in one patient shows a classic Jones stress fracture involving the lateral cortex of the proximal fifth metatarsal shaft (arrow). The fracture was isolated to the cortex. (**b**) Another patient with a Jones stress fracture shows extension of the fracture into the medullary cavity after the athlete felt a "pop" while running

a vertically oriented lucency in the cortex (Fig. 4.7). In bones composed largely of cancellous bone, such as the tarsus and femoral neck, the first sign of a stress fracture may be simply focal linear sclerosis (Fig. 4.8). In these cases, initial findings are subtle blurring of the trabeculae secondary to microfractures. As healing of the microfractures progresses, linear sclerosis appears oriented perpendicular to the course of the trabeculae with extension to one cortical surface. Detectable radiographic changes usually become conspicuous weeks to months after the onset of symptoms and the timing and nature of the changes varies with

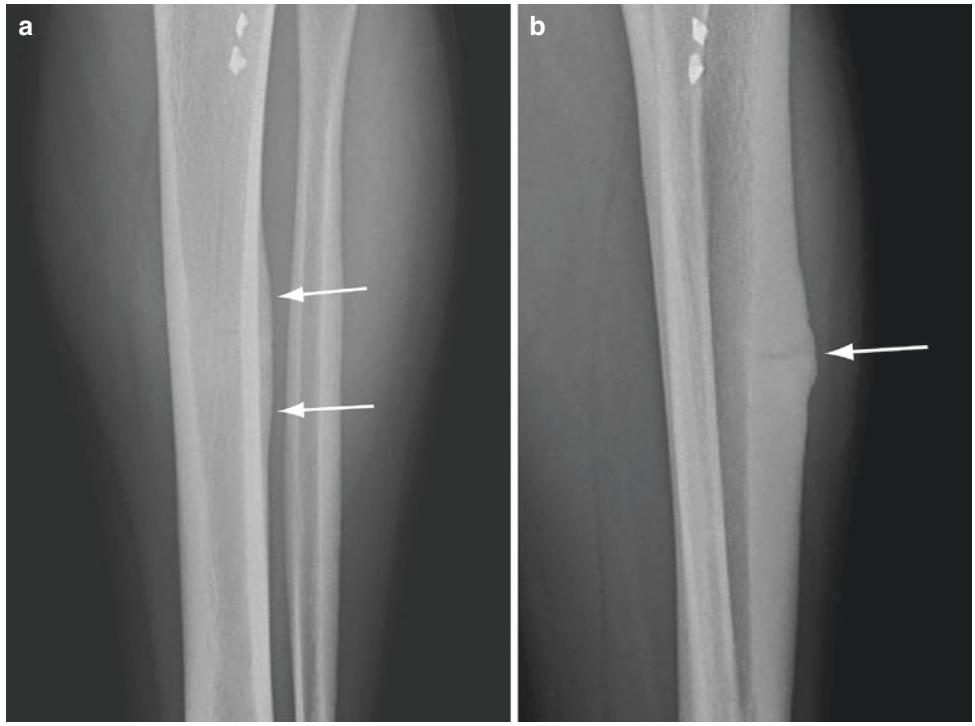


Fig. 4.5 Striated stress fracture. (**a**) Frontal radiograph of the tibia shows periosteal elevation along the anterolateral cortex of the mid-tibia (arrows). (**b**) Lateral view shows a transverse lucency through the cortex with more pronounced periosteal reaction directly adjacent to the fracture (arrow)

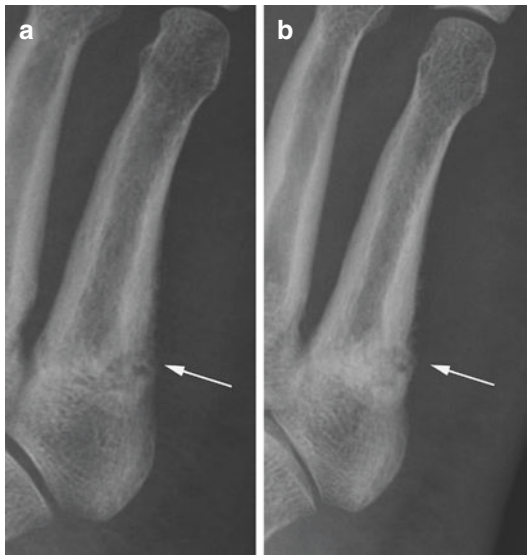


Fig. 4.6 Stress fracture at the base of a tubular bone. (**a**) Frontal radiograph of the fifth metatarsal shows a linear lucency near the base of the fifth metatarsal representing a stress fracture. (**b**) Follow up radiograph shows blurring of the fracture line with progressive surrounding linear sclerosis representing healing (arrows)

the level of activity. However, it is noteworthy that imaging findings may not be necessarily sequential (Fig. 4.9).

The sensitivity of radiographs for detecting early stress fractures is as low as 15% and follow-up radiographs may demonstrate findings in only 50–54% of cases [7, 37]. Development of subsequent radiographic findings is often determined by whether there is cessation of the inciting stress affecting the bone. There are many classifications available for grading the radiographic features of stress fractures but currently none have been ubiquitously utilized [38, 39].

Tomosynthesis, or digital radiography, has recently been shown to be superior to conventional radiography in detection of occult fractures and it may have an application in the evaluation of stress fractures [40]. This imaging technique can depict both cortical as well as trabecular changes, so its performance is considered only slightly worse than that of CT but with a lower radiation exposure [41].

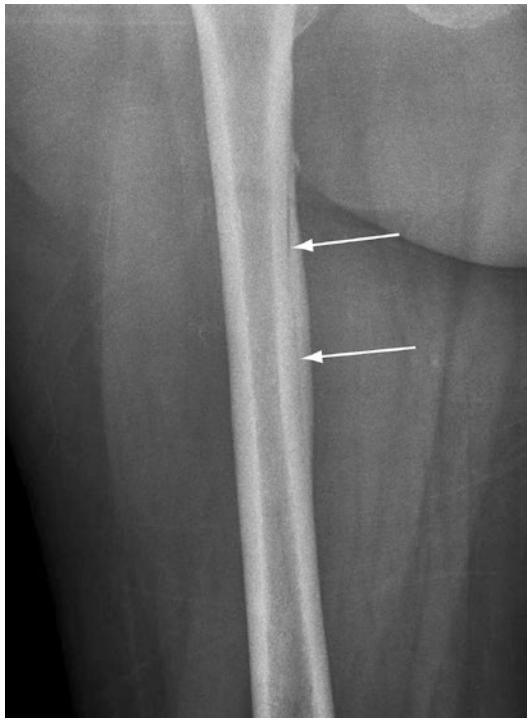
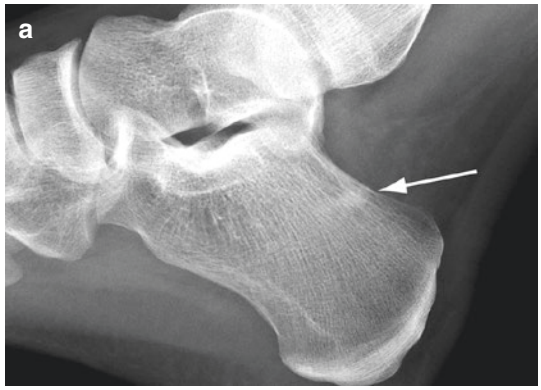


Fig. 4.7 Radiography of longitudinal stress fracture. Frontal radiograph of the femur shows a linear lucency (arrows) within the medial femoral cortex oriented along the longitudinal axis of the bone

The differential diagnosis for stress fracture on radiography is limited, particularly as the specificity of the study increases in the chronic phase of the fracture. Chronic osteomyelitis may present with periosteal and endosteal reactive changes resulting in cortical thickening but clinically, these two entities are not at all similar. Occasionally, a stress fracture may mimic a tumor [42]. Osteoid osteoma may result in cortical thickening and reactive bone formation and is often encountered in a similar patient population as stress fracture. The presence of a central lucent nidus as well as a less linear pattern of sclerosis and clinical history can aid in differentiation.

Radionuclide Scintigraphy

Bone scintigraphy had for many years been regarded as the gold standard for evaluating stress-induced injuries and although recently supplanted by MRI, it continues to be widely utilized in many situations. It measures bone response to injury by depicting areas of increased osseous metabolism through the localization of



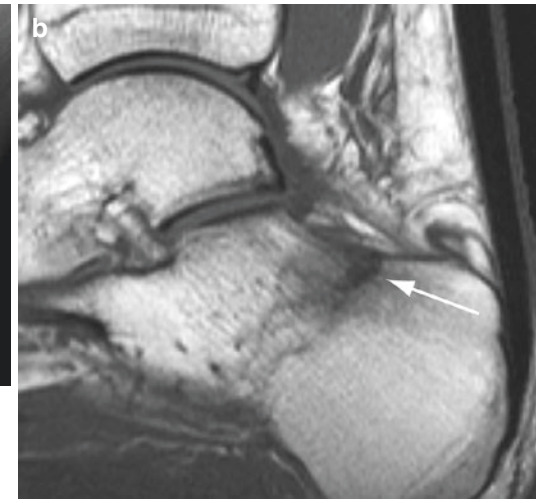


Fig. 4.8 Stress fracture in cancellous bone. (**a**) Lateral radiograph of the calcaneus demonstrates a linear area of sclerosis perpendicular to the trabeculation in the superior calcaneus (arrow). Sagittal T1-weighted (**b**) and STIR (**c**) MR images show a uni-cortical, low signal fracture line (arrows) surrounded by intense bone marrow edema

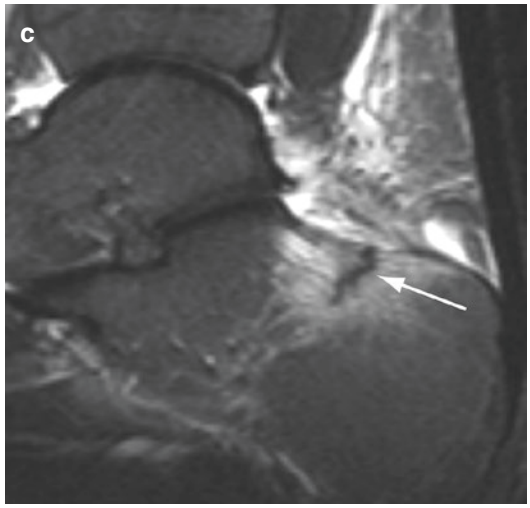


Fig. 4.8 (Continued)

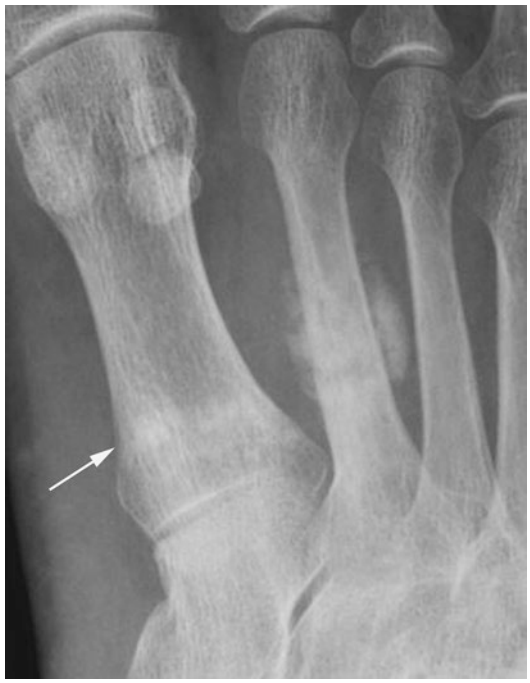


Fig. 4.9 Healing stress fractures. AP radiograph of the foot demonstrates stress fractures of the first metatarsal base and second metatarsal shaft. The first metatarsal fracture appears as thick linear sclerosis without a definite fracture line visualized (arrow). The second metatarsal fracture has a visible fracture line with surrounding sclerosis and overlying periosteal reaction

radionuclide tracers, particularly Tc-99m-MDP. The degree of uptake depends on the rate of bone turnover and local blood flow, and abnormal uptake may be seen within 6–72 hours of injury [7, 37, 43]. Whole body bone scans can be performed with relatively low cost and have the advantage of being able to image the entire skeletal system at once, which is useful in cases when more than one area is symptomatic. The sensitivity of bone scintigraphy is nearly 100% [7].

The specificity of bone scintigraphy, however, is limited by any process that increases blood flow and has osteogenic activity such as arthritis, infection, malignancy, infarction, or a metabolic condition. The specificity can be improved by performing a three-phase study [25]. The first phase includes a dynamic flow study with images obtained at 1-second intervals for 60 seconds after the injection of radiopharmaceutical and is followed by a static "blood pool" image (second phase) obtained a few minutes later. These phases depict vascularity and soft tissue involvement, respectively. The third phase is the standard 2–4 hour delayed images depicting the osteoblastic response. An acute stress fracture will be positive in all three phases while a chronic stress fracture tends to show activity only on the delayed images [7]. Another limitation of scintigraphy in patients with stress fractures is that the scintigraphic abnormality may take 4–6 months to resolve, rendering the modality inadequate for sequential follow-up studies [44]. Several grading schemes are available to characterize the severity of a stress fracture according to its scintigraphic features.

The characteristic scintigraphic appearance of a stress fracture in delayed static imaging is intense, fusiform cortical uptake along the long axis of the bone at the level of the fracture (Fig. 4.10) [45]. However, there can be a wide spectrum of findings representative of the pathophysiologic continuum of the process and variations in the orientation of the fracture such as in a longitudinal fracture (Fig. 4.11). A stress reaction is manifested by an area of less intense radionuclide uptake along the cortex corresponding to areas of remodeling bone during the period in which radiographs are typically normal.

Athletes who are involved in rigorous training regimens may present with multiple symp-

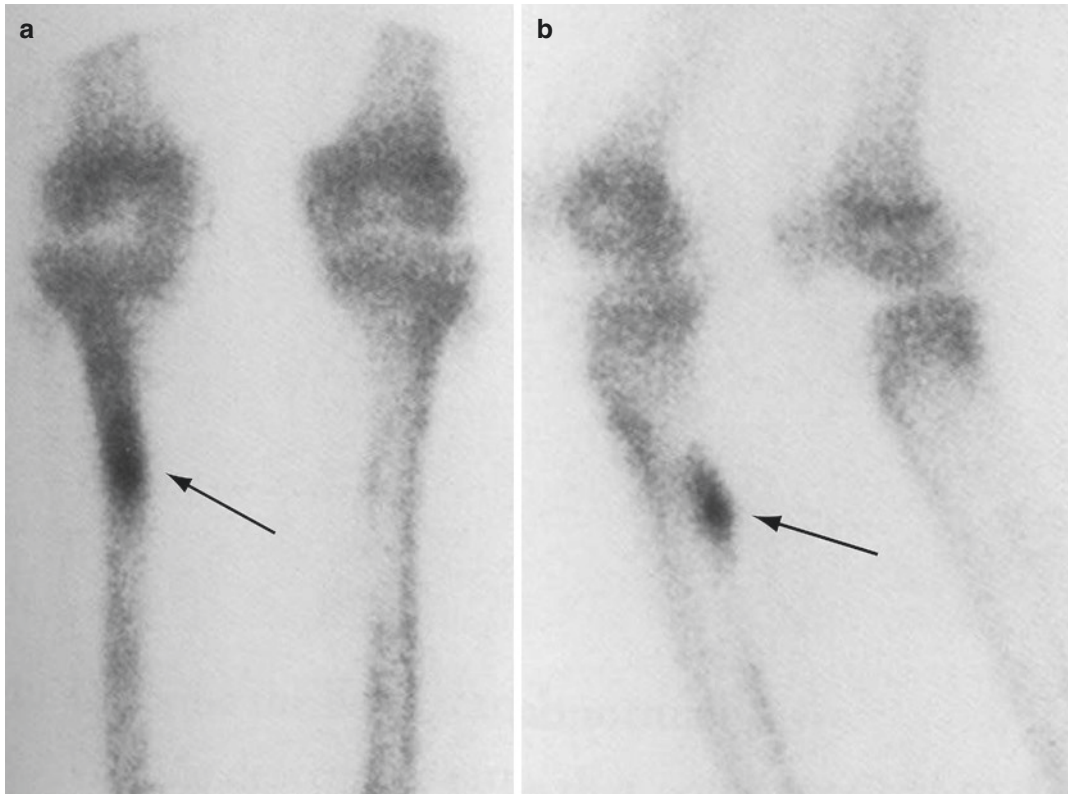


Fig. 4.10 Typical scintigraphic findings of a stress fracture. Delayed static images of the tibia from a whole body bone scan utilizing Tc-99m-MDP in the frontal (**a**) and oblique (**b**) projections show a characteristic appearance of a stress fracture in the tibia depicted as a fusiform region of radionuclide uptake oriented along the long axis of the bone (arrows)

tomatic regions of bone that show abnormal radionuclide uptake, and these findings have been shown to represent both stress reactions and frank stress fractures. However, some patients also demonstrate abnormal uptake in regions of bone that are not symptomatic. This finding likely represents the earliest manifestation of bone remodeling [46]. Areas of abnormal uptake have been reported in as high as 46% of asymptomatic subjects in one series [47]. With continued activity, these areas may progress to symptomatic stress injuries.

The application of planar scintigraphy in combination with single photon emission computed tomography (SPECT) has been advocated for increasing the accuracy of grading stress fractures. In a study evaluating patients with known femoral neck stress fractures diagnosed with MR imaging, the sensitivity of planar scintigraphy alone was reported to be 50%, while the sensitivity for planar scintigraphy in combination with SPECT increased to 92% [48]. Similarly, the accuracy for scintigraphy alone was 12.5% but increased to 70% when SPECT was added. SPECT has also been shown to improve the diagnostic accuracy of stress fractures at the pars interarticularis region of the spine, a process that is commonly observed in adolescent athletes with back pain. SPECT has been shown to provide a more detailed anatomic depiction of the region in comparison to MRI and has a higher sensitivity in comparison to planar scintigraphy alone [49–51]. However, SPECT is limited in the spine owing to a high rate of false positives and false negatives [52].

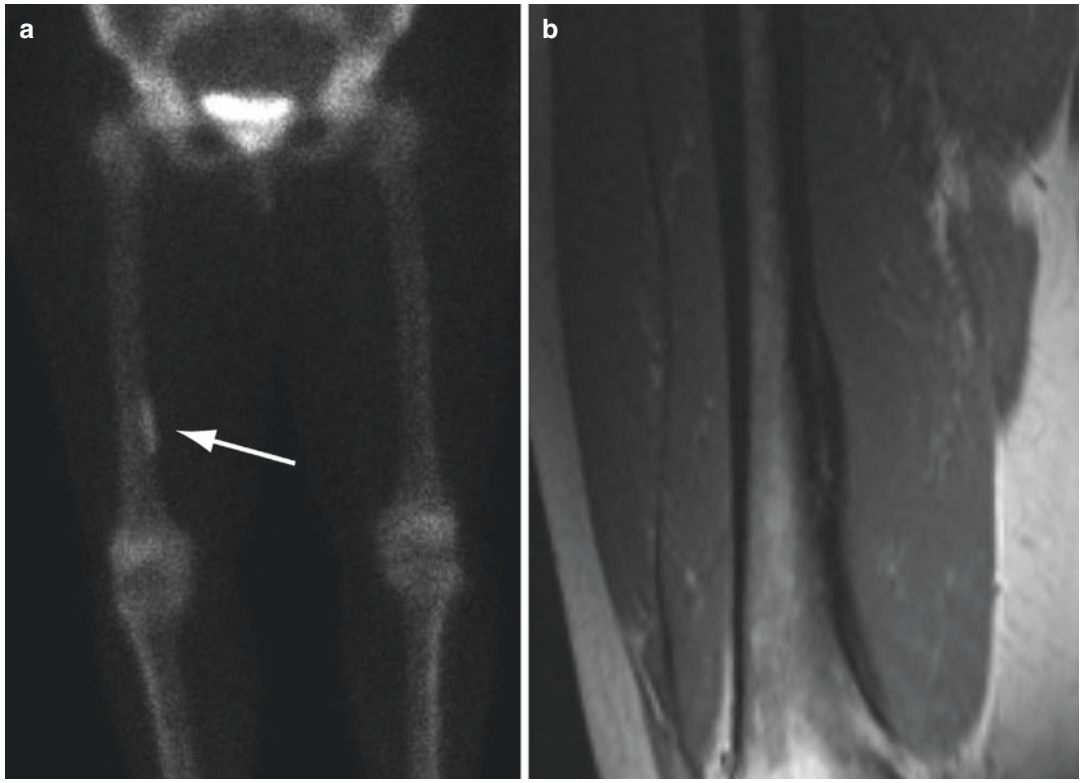


Fig. 4.11 Longitudinal stress fracture on Scintigraphy and MRI. (**a**) Delayed frontal static bone scan image utilizing Tc-99m-MDP shows a thin, linear area of increased activity in the medial cortex of the distal right femoral shaft (arrow). (**b**) Coronal T1-weighted MR image shows a longitudinal stress fracture depicted as a linear area of intermediate signal intensity within the thickened cortex aligned to the longitudinal axis of the bone

Ultrasound

Sonography has a very limited role in the evaluation of stress fractures and is not recommended as a stand-alone modality [53]. However, studies have shown that this modality may occasionally be used to assess the superficial surface of the cortex in bones that are located close to the skin such as in the foot, ankle, and tibia [54]. Cortical irregularities such as periostitis and callus formation can be depicted as well as muscular edema around the bone, and compression of the probe is useful in confirming pain. Color Doppler imaging can demonstrate areas of hyperperfusion at and near the stress fracture.

Recent studies have demonstrated a sensitivity of 82% and a specificity of 67–76%, but predictive values offer a wide range with studies reporting a 59–99% positive predictive value and a 14–92% negative predictive value [27, 55].

Computed Tomography

The role of CT in the assessment of stress-related injuries continues to be relatively limited despite advances in technology. CT is less sensitive than both MRI and nuclear scintigraphy in depicting the early changes of bone remodeling from repetitive stress [7, 30, 32]. However, the ability to produce thin-section, multiplanar-reconstructed images in order to provide high resolution and detailed depiction of cortical bone does relegate CT to an important adjunctive role when the

imaging features in other modalities are equivocal [56]. CT is clearly superior to both sonography and conventional radiography. The earliest finding of a stress injury on CT is focal cortical osteopenia, but this is not a common observation because CT is typically not a first line study (Fig. 4.12). CT manifestations of stress injuries include thickening of the cortex, periosteal reactive changes, intramedullary sclerosis, and longitudinal and transverse lucent fracture lines. The main limitation of CT is that these findings may not develop until the patient has been symptomatic for several weeks. However, high resolution CT is currently the most sensitive modality for detecting subtle cystic changes in the cortex that characterize cortical resorption cavities (Fig. 4.13). Once a fracture line develops in the cortex, the defect is easily demonstrated by conventional axial images as well as by multiplanar reformatted or 3D volume-rendered images (Fig. 4.14) [57].

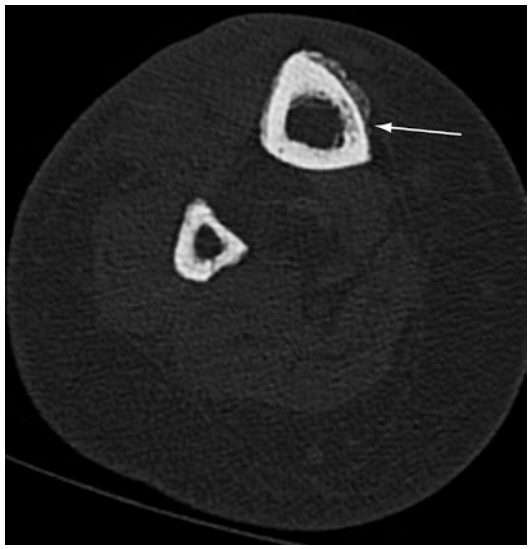


Fig. 4.12 Computed tomography, early stress response. Axial CT image of the calf shows focal osteopenia of the tibial cortex in the location of stress induced changes (arrow). Periostitis is also present

CT is advantageous in certain situations compared with other imaging modalities. It is useful in differentiating healing from progression (Figs. 4.15 and 4.16). Certain location-specific conditions are better suited for CT. Stress fractures that affect the tarsal navicular are often difficult to diagnose because the symptoms associated with this condition are often vague and there may be a paucity of specific physical findings [58]. Additionally, the overall density of the navicular can, in part, obscure the linear focus of sclerosis that accompanies a stress fracture on radiography. In these cases, CT is useful in elucidating the imaging characteristics of the stress fracture such as the extent of abnormality, orientation, and whether avascular necrosis is present.

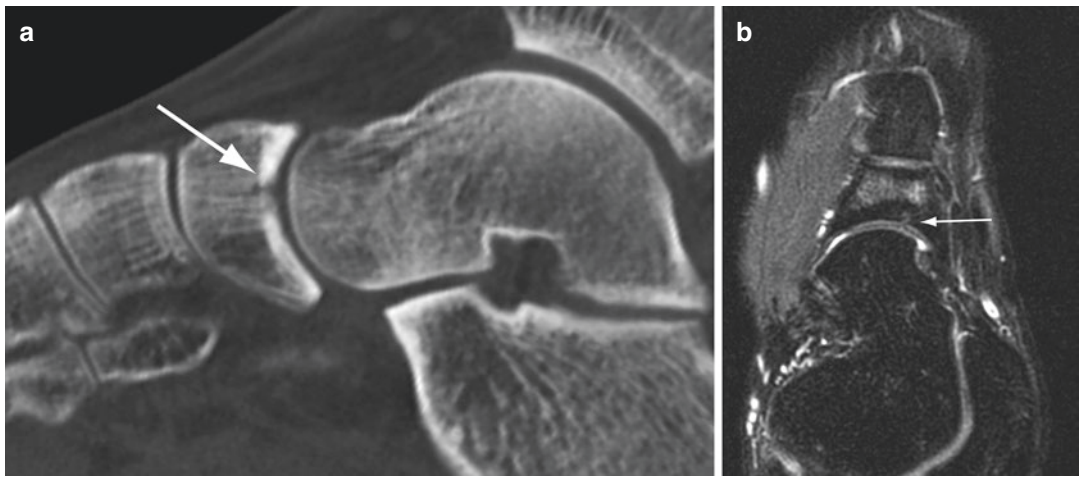


Fig. 4.13 Cortical resorption cavity. (**a**) Sagittal CT image of the foot shows focal osteopenia in the cortex at the point of the fracture (arrow) indicating a developing cortical resorption cavity. (**b**) Axial STIR MR image shows the cystic defect in the cortex (arrow) and bone marrow edema in the medullary space

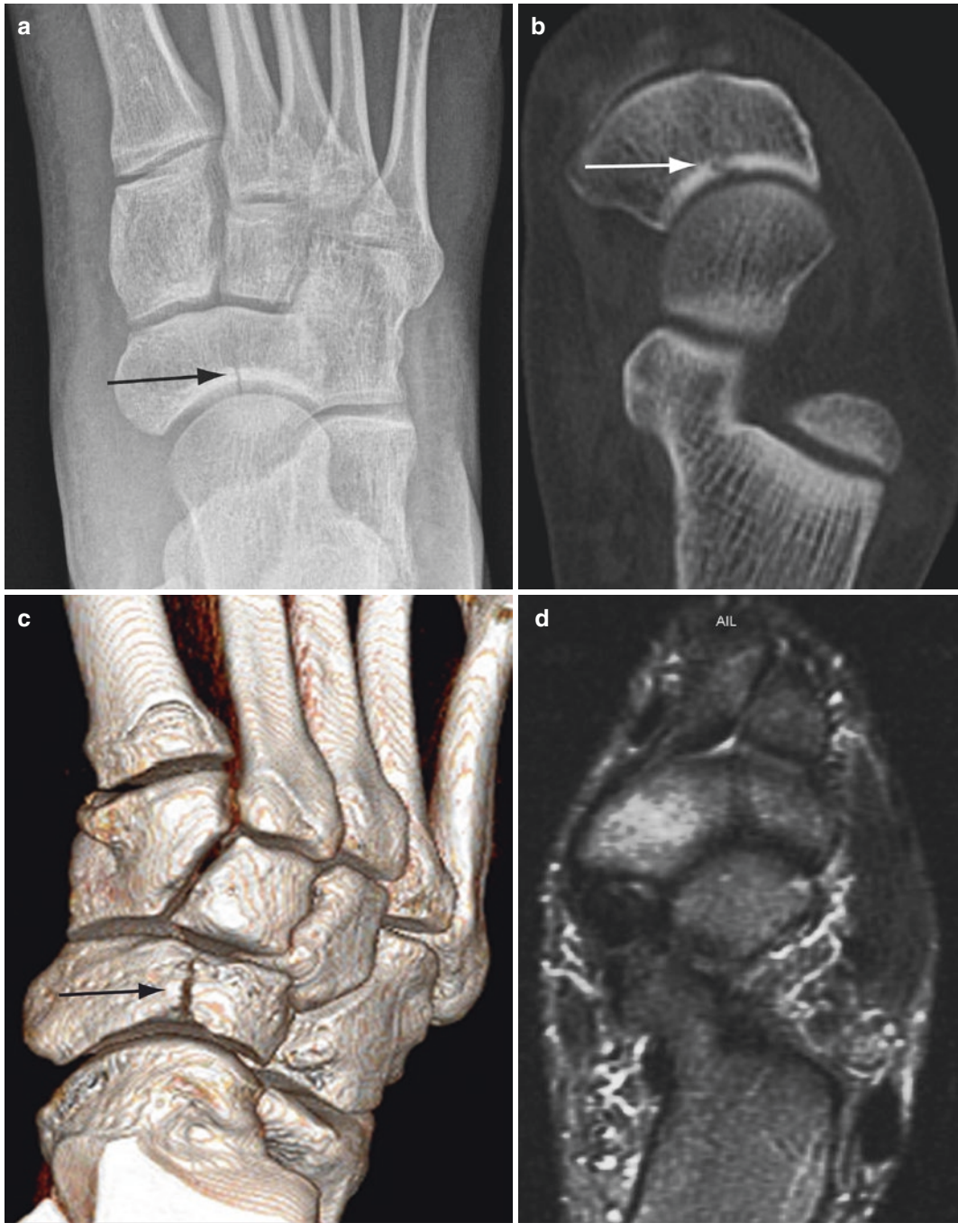


Fig. 4.14 Subacute navicular stress fracture. (**a**) Frontal foot radiograph of a college basketball player shows a vertical lucency (arrow) in the lateral aspect of the navicular. (**b**) Axial CT image confirms the stress fracture (arrow) as well as normal bone density throughout the tarsal bone. (**c**) 3D volume rendered CT image depicts the entire stress fracture (arrow) in one image. (**d**) T2-weighted fat saturated MR image demonstrates bone marrow edema in the medial and lateral bone fragments

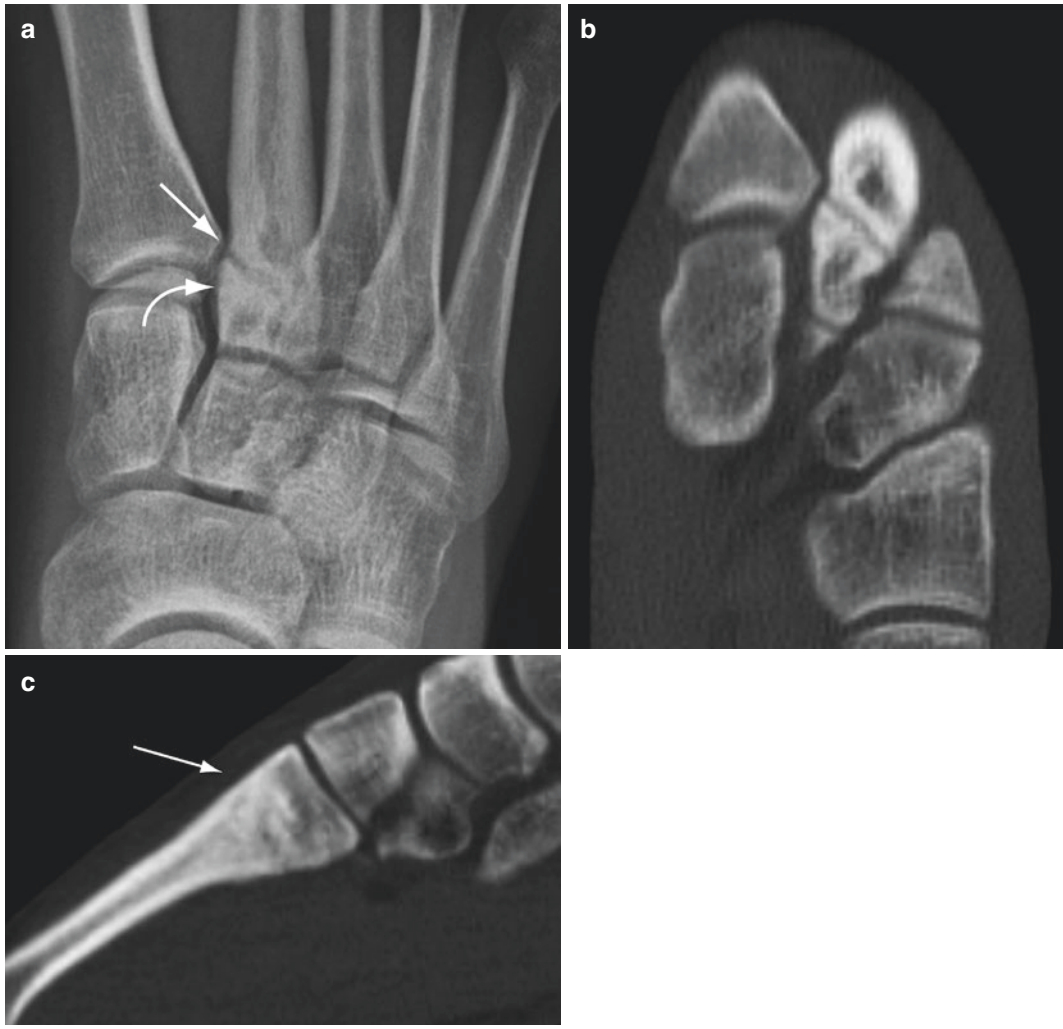


Fig. 4.15 Chronic stress fracture. (**a**) Frontal radiograph of the foot demonstrates a transverse lucency near the base of the second metatarsal bone (arrow). Sclerosis adjacent to the fracture is evident (curved arrow). Axial (**b**) and sagittal (**c**) CT images more optimally characterize the stress fracture and also show that the dorsal cortex is intact (arrow)

A similar challenge may occur in patients with pars interarticularis fractures. Fracture lines are often difficult to visualize utilizing other modalities such as MRI but are clearly illustrated on CT [52, 59]. Occasionally, cortical thickening may be a nonspecific finding. For instance, the radiographic manifestations of an osteoid osteoma may mimic those of a stress fracture because both conditions can result in a thickened cortex and variable periosteal reactive changes. By utilizing thin section CT imaging, these entities can be reliably differentiated by the identification of the lucent nidus that is the classic feature of osteoid osteoma within the region of cortical thickening and sclerosis [60]. One important advantage of CT over MRI lies in its ability to penetrate high-attenuation cortical bone. Although MRI remains the single best method for evaluating early stress injuries, it is relatively insensitive to changes that occur only within the cortex. Therefore, the subset of cortical stress injuries that are characterized by osteopenia, resorption cavities, and striations are better suited for evaluation by CT [61]. Longitudinal stress fractures of the tibia

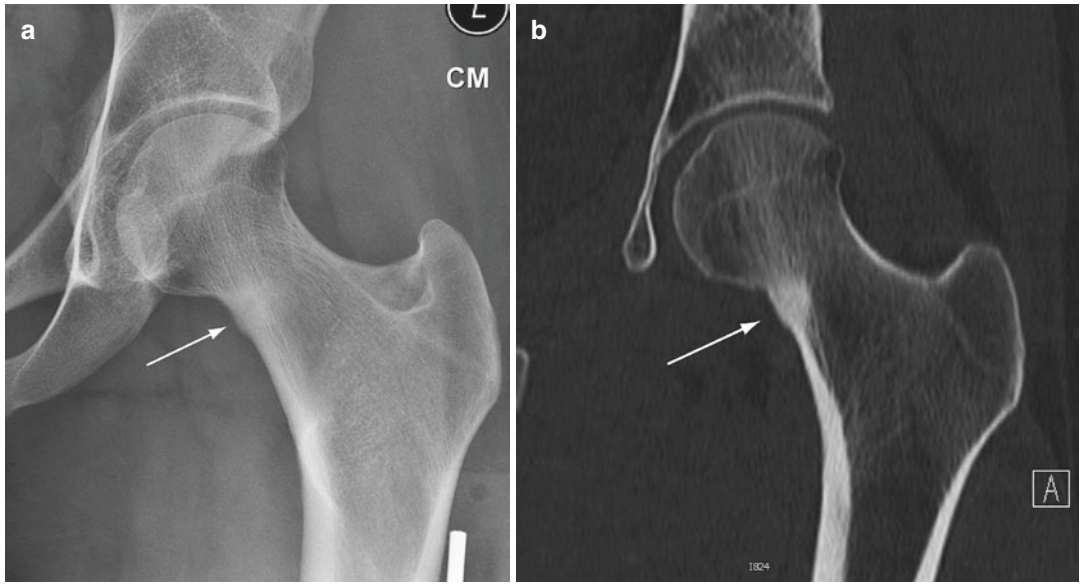


Fig. 4.16 Computed tomography of healing stress fracture. (**a**) Frontal radiograph of the hip shows a region of sclerosis on the compressive side of the femoral neck with focal periosteal reactive changes (arrow). (**b**) Coronal CT image shows that the fracture line has nearly filled in and is no longer evident (arrow)

caused by repetitive torsional loading in runners also are best evaluated with CT. The longitudinal orientation and extension of the fracture negates the effectiveness of radiographs and though MRI is capable of depicting the abnormality, CT has been reported to be more sensitive in identifying the fracture line itself [62].

Peripheral quantitative computed tomography (pQCT) is a CT technique that has demonstrated potential in the evaluation of stress fractures by the acquisition of high resolution images of the extremities at lower radiation doses than with conventional CT. pQCT allows a detailed evaluation of the structure and mineralization of bone at the location of the stress fracture. As such, it may have application in monitoring the stress fracture throughout the healing phase [63, 64].

Magnetic Resonance Imaging

MRI is currently the gold standard for diagnosing and classifying stress-induced injuries. Several important features of this imaging modality have contributed to its emergence as a superior tool for assessing these conditions including unparalleled contrast, outstanding spatial resolution particularly with higher strength magnets, and the capability to image in an infinite number of geometric planes [65]. Additionally, it does not utilize ionizing radiation, which is ideal in the athletic population which tends to be younger [66]. An MRI study typically can be obtained in a shorter period of time compared with a scintigraphic examination and is extremely sensitive to the often-subtle changes seen in patients with early stress fractures. Numerous studies have shown that MRI outperforms radiography, CT, and radionuclide scanning in diagnosing and evaluating stress injuries [29, 30, 32, 67, 68].

MRI examinations are optimized by utilizing dedicated coils in order to increase the signal to noise ratio and decrease artifacts. Higher strength magnets, such as 3-Tesla systems which are becoming more commonplace, offer higher spatial and contrast resolution, shorter scanning times, and improved conspicuity of bone marrow edema than conventional 1.5-Tesla systems [69]. The sensitivity is comparable for both 1.5T and 3T MR systems and 1.5T MR imaging is typically adequate for diagnosis and characterization

of stress fractures [70, 71]. Typical sequences performed include short tau inversion recovery (STIR), which is commonly used in screening since it has the highest sensitivity to edema, and fast spin-echo sequences with fat-saturation, which are excellent in preserving high spatial resolution. A T1-weighted sequence is generally performed to further characterize the inherent signal intensity of lipoid marrow. Intravenous gadolinium is not frequently administered in the evaluation of stress fracture. However, dynamic enhancement has been reported in patients with higher grade stress reactions and stress fractures caused by increased tissue perfusion. Post-contrast MRI may be useful in cases where the pre-contrast MR images show a callus, fracture, or muscle edema, or in situations where there is a concomitant malignancy or infection [72].

MRI is an effective diagnostic technique in patients who show strong clinical manifestations of a stress fracture but have normal initial radiographs [73, 74]. Like scintigraphy, MRI depicts changes in the bone and periosteum weeks before any radiographic abnormality develops. The early stages of a stress fracture are characterized by focal hyperemia and bone marrow edema that correlate with the development of microfractures and osseous resorption. Endosteal reactive changes, periostitis, and peri-osseous edema are important early observations on STIR or T2-weighted spin-echo images and are characteristic of stress reactions (Fig. 4.17) [68, 75]. Edema appears bright in signal intensity on these sequences. Focal periosteal elevation develops as the process becomes more severe (Fig. 4.18). As the injury progresses, marrow edema appears on T1-weighted images as areas of low signal intensity (Fig. 4.19). As breakdown of the cortical bone occurs, a frank stress fracture forms either transversely or longitudinally (Figs. 4.20 and 4.21) [67]. The most common patterns of a fatigue stress fracture on MRI are a linear, unicortically-based abnormality of low signal intensity surrounded by a larger, ill-defined region of marrow edema, or a linear cortical abnormality with adjacent muscular or soft tissue edema [76–78]. Callus formation indicates a more chronic stress fracture. The MRI features in the continuum of a developing stress fracture parallel those that are observed on bone scintigraphy.

Reportedly, MRI has comparable sensitivity to nuclear scintigraphy. Specificity, accuracy, positive predictive value, and negative predictive value are all superior at 100%, 90%, 100%, and 62%,

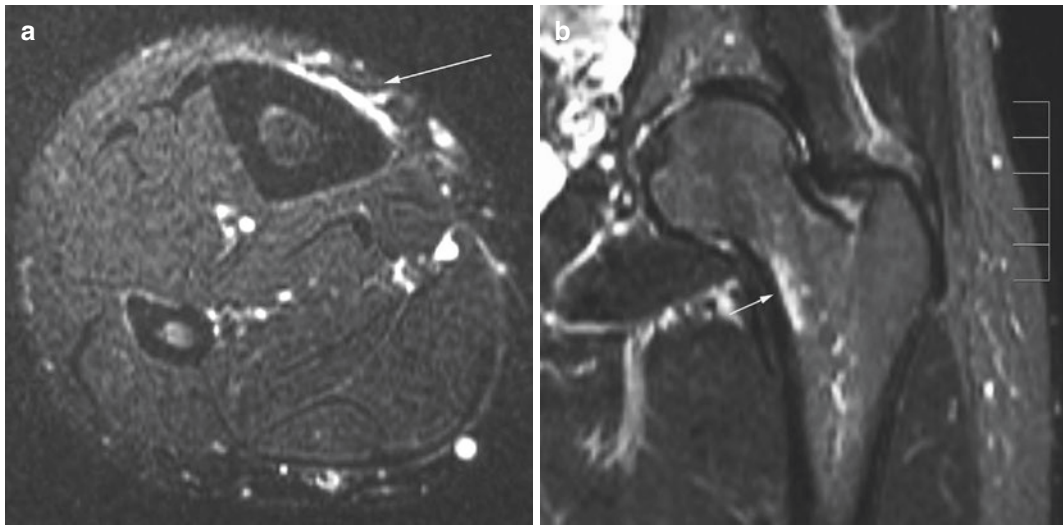
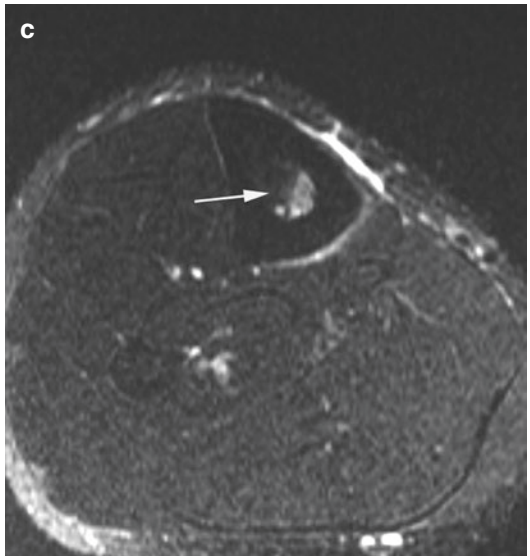


Fig. 4.17 Stress responses on MR imaging. Fluid sensitive MR images in three different athletes. (**a**) Periostitis along the medial cortex of the tibia manifests as linear high signal intensity along the outer cortex (arrow). (**b**) Endosteal reaction with marrow edema along the endosteal surface of the compression side femoral neck. (**c**) A patient with a more severe stress response shows both periosteal and endosteal reactive changes (arrow)



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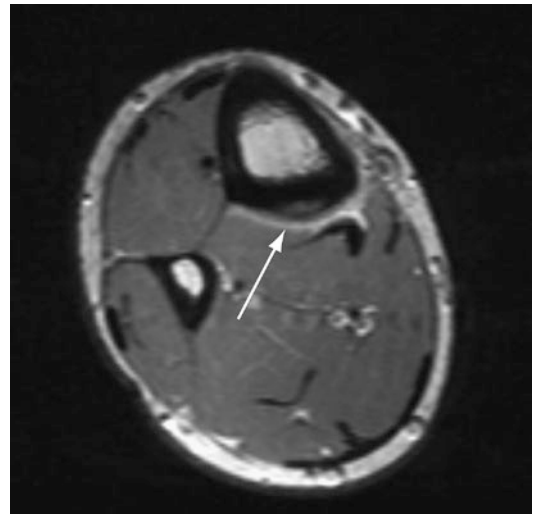
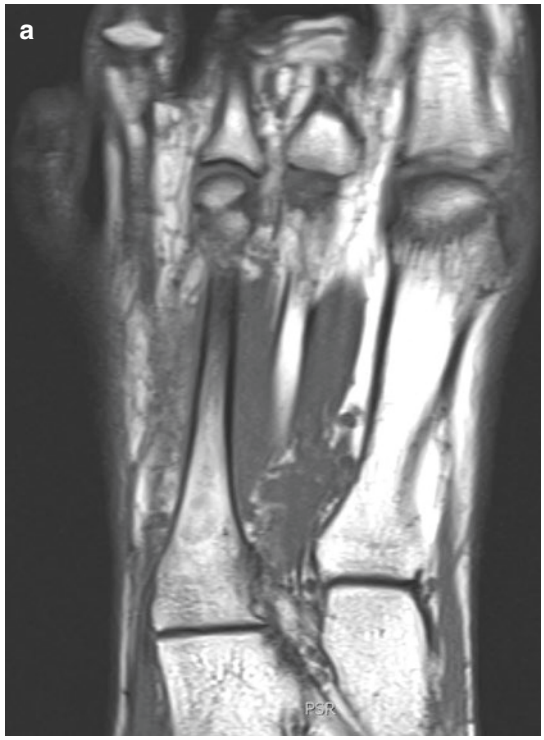


Fig. 4.18 MR of chronic stress reaction. Axial proton-density MR image shows periosteal elevation in the posterior cortex of the tibia (arrow) and adjacent inflammation



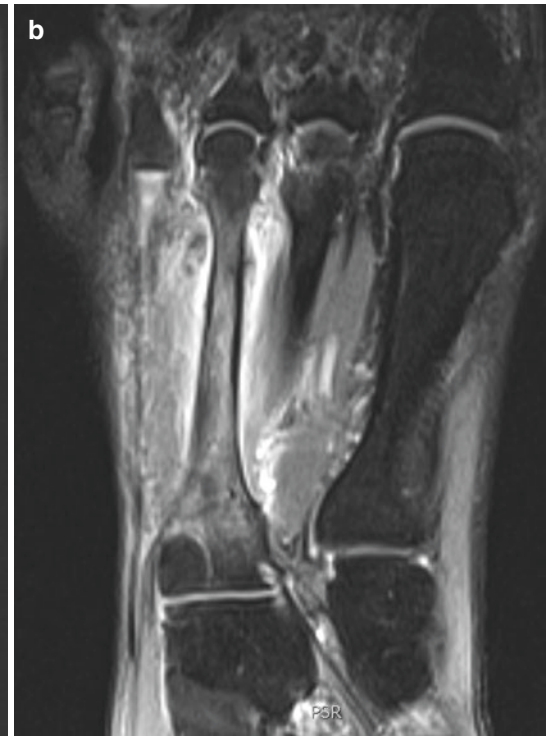


Fig. 4.19 MR features of developing stress fracture. (**a**) Axial T1-weighted MR image shows low signal intensity bone marrow in the third metatarsal bone from edema. (**b**) Corresponding T2-weighted image shows adjacent periosteal inflammation shown as linear high signal intensity fluid along both the superficial and deep cortical surfaces. (**c**) There is rupture of the medial periosteum (arrow) and edema in the adjacent interosseous muscle

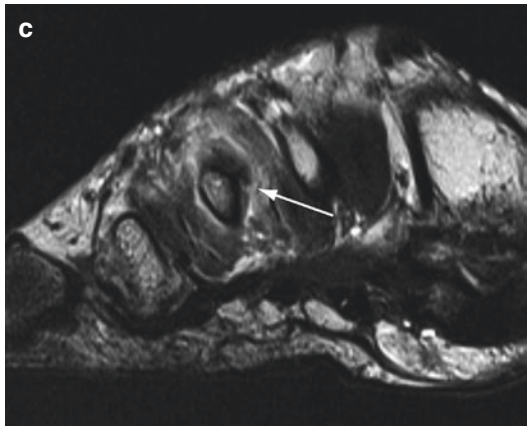


Fig. 4.19 (Continued)

respectively [30]. Additionally, MRI has a distinct advantage by depicting the surrounding soft tissue structures thus permitting concomitant evaluation of muscular, tendinous, or ligamentous structures. In the athletic population, injuries to any of these structures may mimic the symptoms of a stress fracture and reduce the specificity of nuclear scintigraphic studies. Another advantage of MRI that should be underscored is its ability to evaluate regions of the skeleton that are challenging to assess with other imaging modalities. For instance, insufficiency fractures of the pelvis, proximal femur, proximal tibia, and superior acetabulum in elderly patients are often difficult to visualize on CT studies but are unequivocally demonstrated on

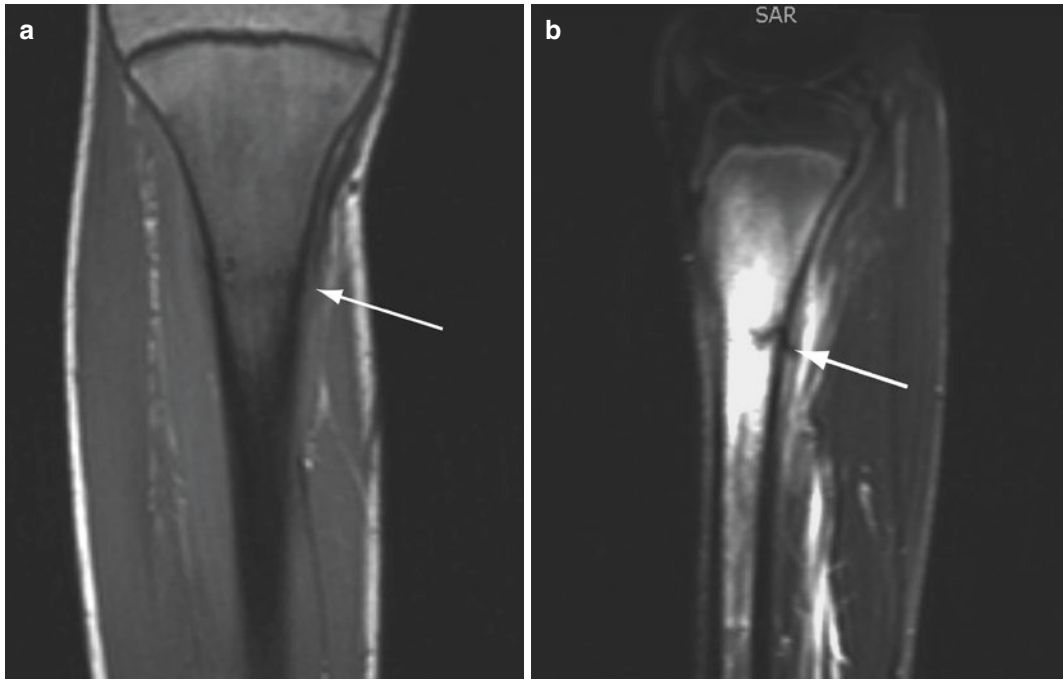


Fig. 4.20 Typical stress fracture on MR imaging. Coronal T1-weighted (**a**) and sagittal STIR (**b**) images of the tibia show marrow edema and periostitis as well as edema in the adjacent posterior soft tissues. The transverse stress fracture is low in signal on both sequences (arrows) and surrounded by a larger region of marrow edema. (**c**) Axial fluid sensitive image demonstrates extensive periosteal elevation (white arrow) and periosseous soft tissue edema (curved arrow)

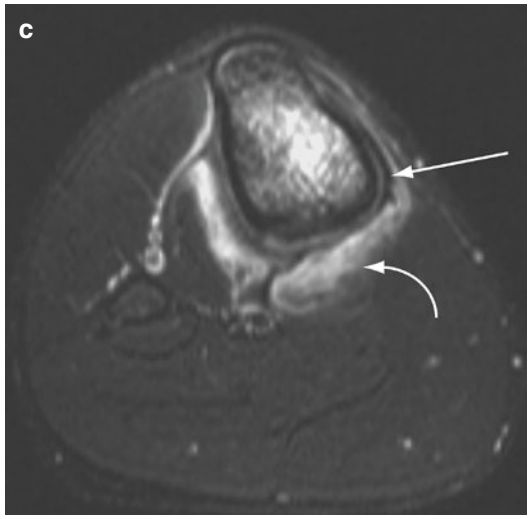


Fig. 4.20 (Continued)

MR images (Figs. 4.22 and 4.23) [79, 80]. Femoral neck stress fractures that are optimally shown on MRI may be occult by radiography or scintigraphy. Any delay in diagnosis of these stress fractures increases the potential of the patient developing a complication. Lastly, the anatomic detail of stress fractures afforded by MRI allows distinction between different types of stress fractures, such as compressive and tensile type stress fractures of the femoral neck, the latter which requires operative fixation (Fig. 4.24) [73, 81].

The majority of proposed grading systems have been for stress injuries of the tibia [44, 82]. Many of the classifications attempt to correlate clinical and imaging findings to those on nuclear scintigraphy but an exact correlation has not been reported to date. Owing to superior spatial

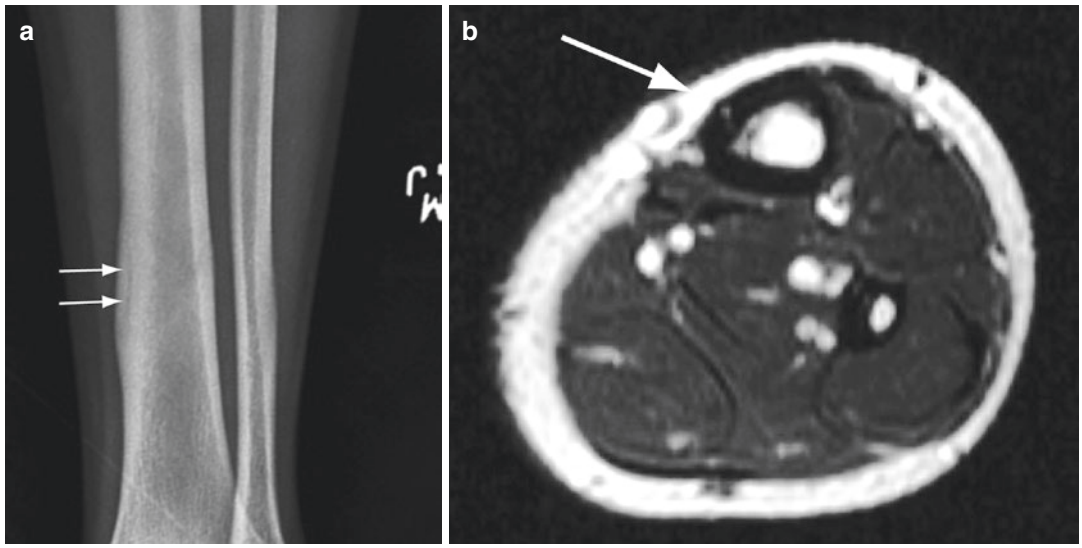


Fig. 4.21 Longitudinal stress fracture. (**a**) Frontal radiograph of the tibia show a linear lucency within the thickened medial cortex (arrows) in the distal shaft. (**b**) Axial T2-weighted MR imaging shows the vertically oriented break in the cortex (arrow)

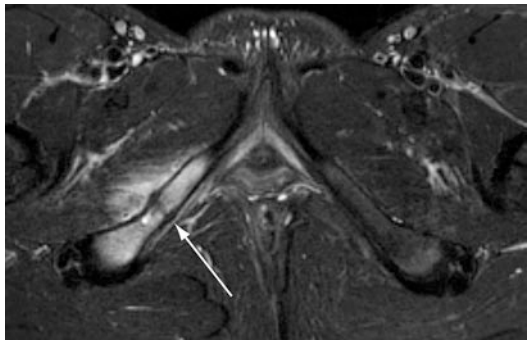


Fig. 4.22 Pubic stress fracture. Axial STIR MR image demonstrates a stress fracture of the right inferior pubic ramus (arrow) with intense surrounding marrow edema and periosseous edema. The abnormality was radiographically occult due to the oblique orientation of the pubic bone

and contrast resolution, grading systems that are based on MR findings have shown superior accuracy over other classifications, thus improving the prescription of appropriate clinical management. According to the commonly used MRI classification system proposed by Fredericson, a grade 1 stress injury includes periosteal edema only, a grade 2 injury includes bone marrow edema visible on fluid sensitive imaging only, a grade 3 injury includes bone marrow edema visible on both fat and fluid sensitive imaging, and a grade 4 injury includes intracortical signal abnormality indicating a stress fracture [82, 83]. Grade 4 stress injuries may also be subdivided into grade 4a, in which a fracture line is not vis-

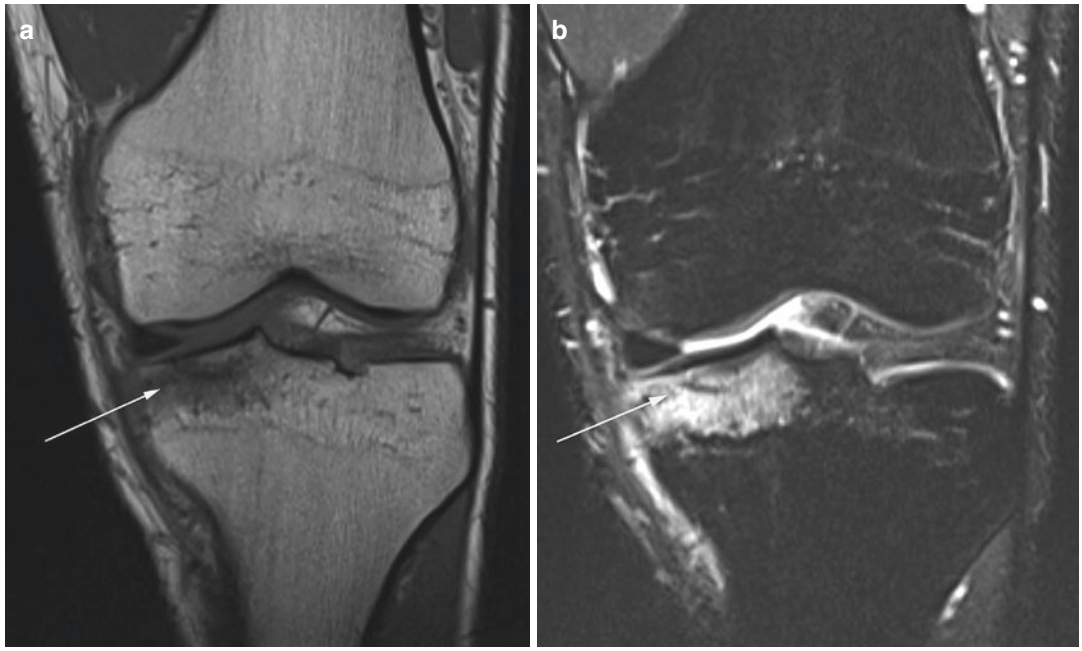


Fig. 4.23 Medial tibial plateau stress fracture. Coronal T1 (**a**) and coronal STIR (**b**) MR images demonstrate a subchondral fracture of the medial tibial plateau, seen on both sequences as subchondral curvilinear hypointense signal (arrows). Marrow edema surrounds the fracture and is hypointense on T1 and hyperintense on STIR. This fracture was radiographically occult

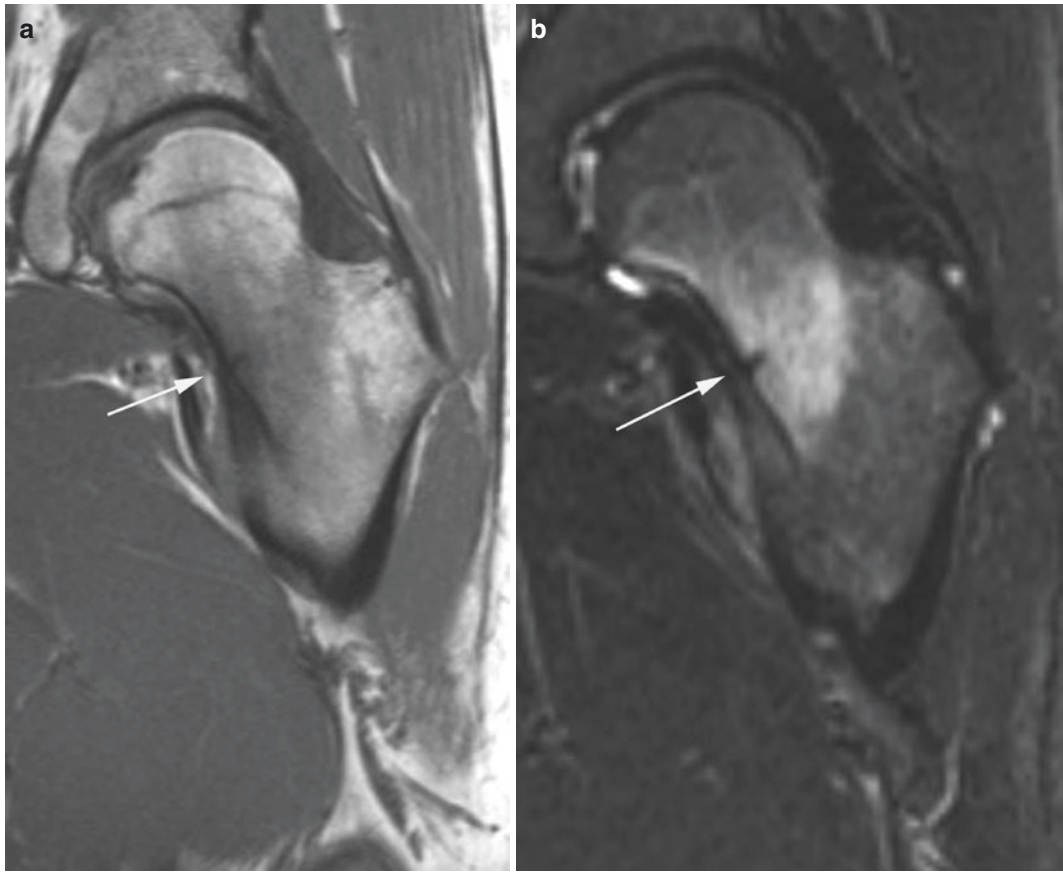


Fig. 4.24 Femoral neck fracture. Coronal T1 (**a**) and coronal STIR (**b**) MR images demonstrate a compression side stress fracture of the left femoral neck, seen as linear hypointense signal with surrounding marrow edema which is hypointense on T1 and hyperintense on STIR (arrows)

ible, and grade 4b, in which a fracture line is visible [83, 84]. Because the MR or scintigraphic grades often have no influence on an athlete's ability to return to active participation, some investigators have suggested simplifying the grading systems to reflect findings that have a strong clinical correlation such as the presence of a cortical fracture [30, 61, 83]. For instance, unless a fracture line is present, patients with MR grades ranging from grade 2 to 4a who show variable severity of periostitis and bone marrow edema may be theoretically combined into one grade since the time that the athlete is not permitted to play is similar among these grades, while the development of a fracture, a grade 4b abnormality, requires a prolonged period away from athletic participation and thus would constitute a more severe grade [83].

The appearance of stress fracture on MRI can occasionally overlap with those of benign and malignant processes [78]. The linear orientation of a stress fracture helps to differentiate it from the more fusiform cortical thickening that may be observed in a patient with a neoplastic process, or the serpiginous intramedullary appearance that is characteristic of osseous infarctions. In-phase and out-of-phase imaging utilizes the differences in the interaction of water and lipid protons in the magnetic field to assess for the presence of fat and water in areas of bone marrow. Stress fractures and other non-neoplastic processes preserve the fat content of normal

marrow, whereas neoplastic processes tend to result in replacement of marrow fat [85]. Other advanced imaging techniques such as chemical shift imaging, diffusion-weighted imaging, and MR spectroscopy are also available for further tissue characterization when required.

The primary limitation of MRI is cost, as it is one of the most expensive imaging techniques available. Utilization must be performed precisely and accurately. False-negative examinations may occur in the setting of technical error such as heterogeneous fat-saturation and partial volume effects, interpretive error, or protocol error by inappropriately selecting the wrong MR sequences. The sensitivity of MRI to edema may result in an errant positive finding if a patient is asymptomatic, so it is important to interpret an examination with proper history and with available correlation to pertinent physical findings [86–88].

Conclusion

Radiography remains the initial imaging examination in a patient suspected of having a stress fracture. A number of options are available for further evaluation depending on the phase of the injury but most experts agree that MRI is the gold standard owing to its superior spatial and contrast resolution, high sensitivity and specificity to both early and late findings, and the lack of ionizing radiation. When available, MRI should be the next imaging modality utilized in the evaluation of a stress injury.

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Classification of Stress Fractures

5

Christopher C. Kaeding and Timothy L. Miller

Defining a Stress Fracture

Stress fractures of bone, also known as fatigue fractures or fatigue failure of bone, are common and troublesome injuries in athletes and non-athletes alike. Typically occurring in individuals who perform repetitive tasks, these fractures result from an overuse mechanism in bone [1–6]. With every strain episode of bone, regardless of its magnitude, microdamage occurs in the bone in the form of microcracks. These microcracks occur in areas of stress concentration. The initiation sites of these microcracks tend to occur at areas of discontinuity in the bone, such as Haversian canals and lacunae. In healthy homeostatic bone, the microdamage elicits a reparative response and the bone is repaired restoring it to its initial structural state. In hypertrophying bone, the microcracks result in a positive adaptive response that results in the overall bone being stronger than its original state. In both of these conditions, the microdamage does not accumulate. When the creation or propagation of microcracks occurs more quickly than the bone can repair them, fatigue failure of the bone occurs.

Stress fractures begin as an increased number or size of microcracks that are not repaired. These microcracks can coalesce or propagate to create a frank fracture line. This fracture line can progress from an incomplete crack to a complete fracture to a displaced fracture and possibly to a nonunion (Fig. 5.1). This progression of microdamage in the bone is dependent on the biologic healing potential of the bone. The more capable the bone is to heal the microcracks, the less likely the microdamage is to progress. As is apparent from this review of the pathophysiology of fatigue failure of bone, stress fractures represent a continuum of structural damage and are not a single consistent entity. They have a spectrum of severity with variations in treatment and prognosis.

A distinction should be drawn between a stress fracture and an insufficiency fracture as these are not the same injury and occur via different mechanisms. Though not mutually exclusive, a stress fracture occurs when an essentially normal bone breaks after being subjected to repetitive tensile, compressive, or torsional stress, none

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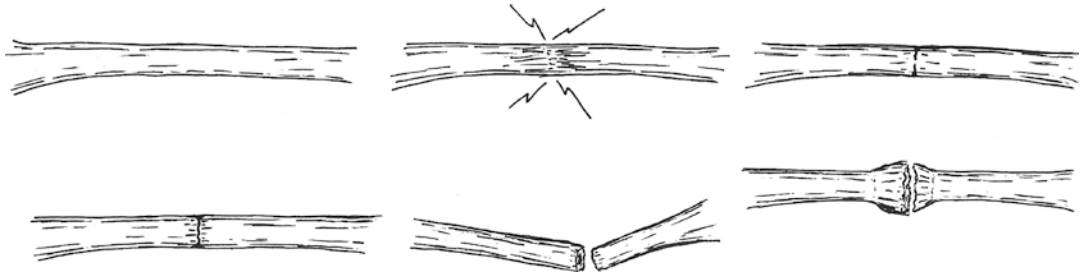

Fig. 5.1 Diagram illustrating the spectrum of severity of bony stress injury including normal bone, stress reaction with no fracture, incomplete fracture, complete fracture without displacement, displaced fracture, and nonunion

of which, individually, would be large enough to cause a bone to fail in a person without underlying bone disease. Insufficiency fractures occur when the mechanical strength of a bone is reduced to the point that a stress, which would not be sufficient to fracture a healthy bone, breaks the weakened bone. Insufficiency fractures, their causes, and treatment strategies will be covered in a separate chapter of this textbook.

Features of a Quality Classification System

The reliability of a classification system requires comparison to the gold standard [7]. Furthermore, the validity of a classification system is dependent upon the accuracy with which the system describes the true pathologic process. Audige's quality criteria from 2004 reflected the importance of clearly described categories and inclusion/exclusion criteria for determining inter- and intra-observer reliabilities [7].

According to Garbuz et al., a classification system should help orthopedic surgeons characterize a problem, suggest a potential prognosis, offer guidance in determining optimal treatment, characterize the nature of a problem, and influence treatment decision-making, ultimately improving outcomes [8]. The same authors further asserted that a classification system should form a basis for uniform reporting of treatments [8].

Stratifying patients with stress fractures into prognostic and treatment groups has historically been difficult given the lack of a single widely applicable standard classification system. Textbooks and review articles have cited techniques for describing stress fractures at a particular location, but have rarely been validated as a method for determination of stress fracture severity, risk, and prognosis [9–16]. An understanding of the basic science of fatigue failure of bone correlates well with our clinical experience that structural failure occurs along a spectrum from micro-fractures to complete structural failure.

Because stress fractures have various degrees of structural failure and healing potential, it is important that we develop standardized categorization and descriptive instruments. Descriptive systems should identify the clinically relevant attributes of the injury in a reproducible fashion and should do so in a simple, inexpensive, safe, and widely applicable manner. For a comprehensive description of stress fractures, these characteristics should be incorporated into a system that describes not only the extent of the structural damage but also the healing potential.

High-Risk vs. Low-Risk Stress Fractures

Unlike most traumatic fractures, in the case of stress injuries of bone, the size and extent of the fracture line vary greatly, and the healing potential varies by location. Some locations typically heal very readily. Other locations, such as the junction of the metaphysis and the diaphysis of the proximal fifth metatarsal, tend to have an increased risk of delayed union, nonunion, and refracture. Boden et al. described high-risk and

Table 5.1 Anatomic sites for high-risk stress fractures [17]

Femoral neck (tension side)
Patella (tension side)
Anterior tibial cortex
Medial malleolus
Talar neck
Dorsal tarsal navicular cortex
Fifth metatarsal proximal metaphysis
Sesamoids of the great toe

low-risk stress fractures by their location [17, 18]. Those locations that have a tendency toward delayed union, nonunion, or refracture are classified as high-risk stress fractures. The varied healing potentials may be related to biologic and/or biomechanical factors of the different anatomic sites.

An important distinction regarding stress fractures is whether they are high- or low-risk fractures (Table 5.1). This classification system has been proposed many times in the literature [17–22]. Such a system provides a reproducible way for medical personnel to determine the course of treatment and the timeframe of recovery before the athlete can return to play. Stress fractures are considered to be high-risk fractures if they have any of the following characteristics. First, these fractures have a predilection to progress to complete fracture (fifth metatarsal), delayed union (anterior cortex tibia), or nonunion (tarsal navicular). Second, a delay in diagnosis and treatment can either prolong an athlete's non-weight-bearing status and his or her restriction from sport, or change a nonsurgical treatment to one requiring operative fixation with or without bone graft.

These high-risk sites possess a common biomechanical characteristic [3–5, 17, 18]. The initiation of their associated fracture lines typically occurs on the tension side of the bone or in a watershed (relatively avascular) area of the vascular supply (e.g., superior side of the femoral neck, anterior cortex of the tibial shaft, lateral aspect of the proximal fifth metatarsal, and the dorsal side of the tarsal navicular) [5]. Because bone is less resistant to tensile than compressive forces, this likely puts the bone at these locations at increased risk for microcrack initiation. Why these "high-risk" locations have an increased risk of impaired healing is likely a result of additional influences beyond the biomechanical factors.

The biomechanical factors of being on the tensile side of the bone explain the increased requirement for a healing response, but biologic factors may come into play as well. For example, the proximal junction of the fifth metatarsal diaphysis/metaphysis is a vascular watershed area with suboptimal blood supply to support fracture healing. Locations of high-risk stress fractures may be the combination of increased micro-failure due to biomechanical conditions coupled with impaired biologic healing capacity. Table 5.1 lists the locations of commonly described high-risk stress fractures.

A common example of a poor natural history of a high-risk stress fracture is neglect of an early proximal fifth metatarsal stress failure that results in either an acute fracture or, should it heal, a subsequent refracture. Recognition of this fracture as a high-risk location and early intramedullary screw fixation will often lead to timely healing, and the athlete can resume his or her career with a markedly decreased risk of re-injury.

When compared with high-risk fractures, low-risk fractures have an overall favorable natural history. In contrast to high-risk fractures, which tend to be on the tension side of bone, low-risk fractures tend to occur on the compression side of bone and typically heal readily. Low-risk fractures are less likely to develop a delayed or nonunion, recur, or have a significant complication should it progress to complete fracture. Low-risk stress fractures can typically be treated with activity modification and rarely require surgical intervention. Low-risk fractures include the femoral shaft, medial tibia, ribs, ulna shaft, and first through fourth metatarsals. Anatomic location of the fatigue bone failure is the distinguishing characteristic between high- and low-risk fractures. Determining whether the stress fracture is in a high-risk versus a low-risk location is key to optimal care as it impacts both treatment and prognosis discussions. This characteristic makes judging a stress fracture to be either high risk or low risk

Table 5.2 Key Characteristics of High-Risk Stress Fractures

Occur where tensile forces are concentrated
Natural history is concerning for delayed union or nonunion
Often require aggressive treatment including surgery or strict non-weight-bearing
Low-risk fractures
Occur on the compression side of bone
Natural history favorable for healing
Usually respond to nonsurgical treatment with rest and gradual return to causative activity.

Adapted from Kaeding et al. [5]

an important element in the "classification" of the injury. Table 5.2 describes the key elements of high-risk versus low-risk stress fractures.

The goal in treating athletes is to make an expeditious diagnosis of a stress fracture because those classified as low-risk fractures can participate in modified sports activity, whereas athletes classified as high-risk should be aggressively managed with non-weight-bearing activity or surgery [3–5, 19, 20]. This obviously important clinical implication of the fracture being identified as either high or low risk makes it one of the most important classifications of fatigue failure of bone the clinician can make.

Current and Historical Classification Systems

A recent literature review by Miller et al. revealed 26 stress fracture classification systems [21]. Table 5.3 lists the classification systems reviewed [17, 18, 23–44]. The goal of this review was to determine what classification and grading systems have been referenced in the literature for stress fractures. At the outset of this review, the authors of this study asked two questions: (1) "What classification systems are used in the evaluation and treatment of stress fractures?" (2) "What are the features of each classification system?" It is clear from their review that many classification systems have been developed and applied to stress fractures since Breithaupt first categorized the injury in 1855 [1]. In 42 articles and citations, 27 classification systems were described or referenced.

These systems were reviewed and analyzed for features such as being generalizable, having been evaluated for intra-observer and inter-observer reliabilities, whether the biologic healing potential was incorporated, and what type of evaluation was required to determine the classification. For example, some systems used a biopsy, some used only a bone scan, and others required multiple imaging techniques or mandated a specific study with computed tomography (CT) or magnetic resonance imaging (MRI). Mandating an imaging modality is fraught with issues affecting safety, expense, ease of use, and availability of the classification system for certain locations. This greatly impairs the generalizability of the classification system. If the biologic healing potential component is not included, the description of the stress fracture is incomplete. As previously discussed, not all locations in the skeleton have equal capacity to heal bony injuries. If the classification has not been statistically analyzed for intra-observer or inter-observer reliability, the validity of the evaluation is open to question.

The four most commonly referenced classification systems are those of Zwas et al., Blickenstaff-Morris et al., Devas et al., and Arendt et al. [23, 24, 27, 44] However, since the early 1990s when MRI became commonly available, the classification system of Arendt et al. has been the most commonly referenced system. The reason for this change in frequency of reference is likely due to the increased specificity of MRI over X-ray and bone scintigraphy for diagnosing stress fractures. Arendt's system includes both MRI and bone scan and is generally considered an academic, radiological method for classification. It is commonly applied for research purposes.

With regard to validation of classification systems, 18 of the 27 referenced systems were correlated with patients' clinical outcomes. Only one of the systems, the one described by Arendt et al., was analyzed for inter- and intra-observer reliabilities [23]. The reason for this low number is likely due in large part to the time and scientific environment during which most of the classification systems were first published. Given that the majority of classification systems referenced

Table 5.3 Stress fracture classification systems

Systems cited	Generalizable	Site	Imaging	Clinical parameters	Other	Clinical correlation	Publication
Arendt	+		XR, BS, MRI			+	1997
Blickenstaff-Morris	–	Fem neck	XR			+	1966
Boden	+		XR	Location, natural Hx		+	2001
Brukner	+		XR			–	1999
Chisin	+		BS			+	1987
Devas	–	Fem neck	XR			+	1965
Edwards	–	Tibia	XR, BS, MRI	Pain and duration		+	2008
Elton	+		XR			–	1968
Ernst	–	Fem neck	XR			+	1964
Floyd	+		BS, XR	Pain		–	1987
Fredericson	–	Tibia	MRI			+	1995
Fullerton-Snowdy	–	Fem neck	XR and BS			+	1988
Gaeta	–	Tibia	CT			+	2005
Griffiths	+		MRI			+	1995
Johnson	–	Fem neck	XR		Path	–	1969
Jones	+		BS			–	1988
Kiuru	+		MRI			–	2001
McBryde	+		XR			–	1975
Naval med Ctr-SD	–	Fem neck	XR and MRI			+	1996
Romani	+		U/S	Pain		+	2000
Roub	+		BS, XR		Path	–	1979
Savoca	+		XR	Location		–	1971
Saxena	–	Navicular	CT			+	2000
Torg	–	5th met	XR			+	1984
Wilson	+		XR			+	1969
Yao	+		MRI			+	1998
Zwas	+		BS			+	1987

Adapted from Miller et al. [21]

were originally described before 1989, and therefore prior to the age of evidence based medicine, it is likely that validation of systems was not a major consideration for many of the original authors.

In conclusion, many classification systems currently exist for stress fractures employing various imaging modalities, but few include clinical parameters. Though many are generalizable, no general classification system that includes both radiographic and clinical parameters has been validated with inter-and intra-observer reliability analyses and clinical correlation. A gold standard classification system for grading stress fractures has not historically been available.

Kaeding–Miller Classification System [22]

As discussed previously, fatigue failure of bone occurs across a spectrum of structural failure and in areas with variable healing potential. A generalizable system to describe these injuries for clinicians has not historically been available. To be clinically relevant, any comprehensive description of stress fractures must correlate with the prognosis and must affect treatment decision-making. In order to do this, incorporating a description of both the extent of the fracture and its healing potential is required. Only describing the extent of the fracture is inadequate, as not knowing if the

"incomplete" fracture is in a low- or high-risk location precludes prognosis and treatment recommendations. If we only mention the location, with its unique healing potential, we are limited by not knowing if the "fracture" is simply an increased number of microcracks or a complete structural failure. We refer to the extent of the fracture as the fracture grade. For an accurate discussion or study of stress fractures, the location and fracture grade and the diagnostic study employed must be described.

Kaeding and Miller undertook a study to develop a new classification system to determine fracture grade, which, when coupled with location, would provide a comprehensive description of a specific stress fracture [22]. The authors sought to design a system possessing the characteristics of being reproducible, generalizable, easily applied, and clinically relevant. This classification system (Table 5.4) uses three descriptors: (1) fracture grade, (2) fracture location, and (3) imaging modality used. With this information, we hypothesized that the clinically relevant characteristics of a stress fracture could be described in a reproducible, easily applied, and generalizable manner.

A key step in the development of this system was to develop a simple reproducible manner to describe fracture grade that, when coupled with location (reflecting healing potential/risk), would provide a user-friendly, clinically relevant description of a stress fracture. We believe that the proposed stress fracture classification system achieves these goals. In describing stress fractures, the size and extent of the actual fracture vary greatly, and the biologic healing potential varies among fracture locations. The variation in healing potential may be related to the specific fracture having developed characteristics of a nonunion or because the natural history of stress fracture location is favorable or unfavorable for healing. These two concepts, fracture grade and location, have important implications on treatment options and prognosis. Garbuz et al. stated that a classification system should be compared with the gold-standard classification system [8]. Unfortunately, no gold-standard classification system exists for describing stress fractures.

Table 5.4 Kaeding–Miller stress fracture classification system

Grade	Pain	Radiographic findings (CT, MRI, bone scan, or X-ray)
I	–	Imaging evidence of stress fracture *No* fracture line
II	+	Imaging evidence of stress fracture *No* fracture line
III	+	Non-displaced fracture line
IV	+	Displaced fracture (>2 mm)
V	+	Nonunion

Adapted from Kaeding et al. [22]

A classification system that is complex, difficult to remember, or difficult to apply is not likely to be of considerable benefit to the clinician. The Kaeding–Miller classification system is simple and easy to use, but still captures key clinical features while being widely applicable and reproducible [22]. Questions regarding prognosis and optimal treatment of a stress fracture cannot be answered without knowing its location, the extent of the structural damage, and the presence or absence of nonunion. The authors feel that each of these parameters is clinically relevant and necessary to accurately describe a stress fracture. Boden et al.'s description of high-risk and low-risk stress fractures was a crucial contribution to the understanding and care of stress fractures, but adding fracture grade to this concept advances our description of a stress fracture [17, 18]. Knowing that a fracture is at a high-risk location is important, but knowing whether it is a grade 2 or grade 4 fracture at a high-risk location is of even greater benefit for treatment and prognosis. A low-grade stress fracture at a low-risk site has a better prognosis for time to recovery than a higher-grade injury at the same low-risk site. Therefore, the management of bony stress injuries should be based on the location and grade of the injury. These two criteria provide the physician with important information when evaluating a patient with a stress fracture, communicating with colleagues and patients, and formulating a treatment plan.

Very early fatigue failure of bone may be asymptomatic. Several authors have reported that athletes may have asymptomatic fatigue fractures. Matheson et al. studied 320 athletes with positive bone scans for stress fractures and concluded that 37% of the lesions were asymptom-

atic [45]. Nussbaum et al. studied bone scans in ballet dancers and found that 3 of 10 stress fractures and 13 of 19 stress reactions were asymptomatic [46]. Bergman et al. reported MRI evidence of tibial stress reaction in 43% of asymptomatic distance runners [47]. Groshar et al. studied military recruits during active training with bone scintigraphy and found that 26% of stress fractures were asymptomatic [48]. Gaeta et al. found that 17% of distance runners had painless tibial stress reactions on high-resolution CT imaging [32].

As the microcracks propagate and coalesce, these fatigue failures of bone can progress from being asymptomatic to clinically painful. One large study by Arendt and Griffiths demonstrated that stress fractures with greater structural damage took longer to heal than lower grade injuries [19, 23]. This study demonstrated that the grade of injury has prognostic implications.

Kaeding and Miller have proposed a comprehensive descriptive system for stress fractures [22]. This includes a grading scale for classifying the extent of structural failure. Examples of each grade are shown in Figs. 5.2, 5.3, 5.4, 5.5, and 5.6. Grade 1 injuries are asymptomatic, usually incidental findings on imaging studies (Fig. 5.2). Grade 2 injuries have imaging evidence of fatigue failure of bone, but no fracture line (Fig. 5.3). Grade 3 injuries have a fracture line with no dis-

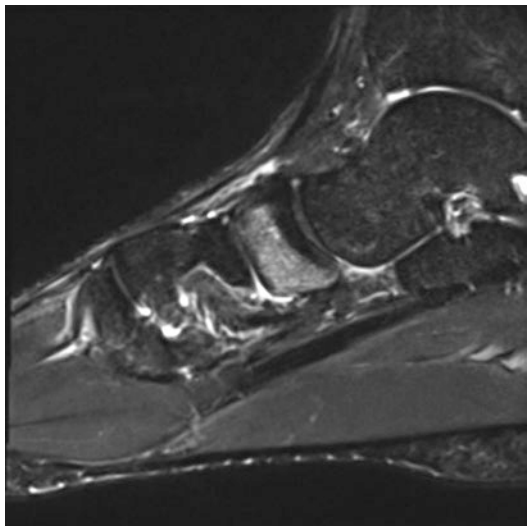

Fig. 5.2 T2 axial cut MRI demonstrating a grade 1 stress fracture of the navicular in a 22-year-old male collegiate distance runner. The patient presented with pain over the third metatarsal shaft with no pain or tenderness at the navicular. A grade 2 stress fracture was evident at the third metatarsal

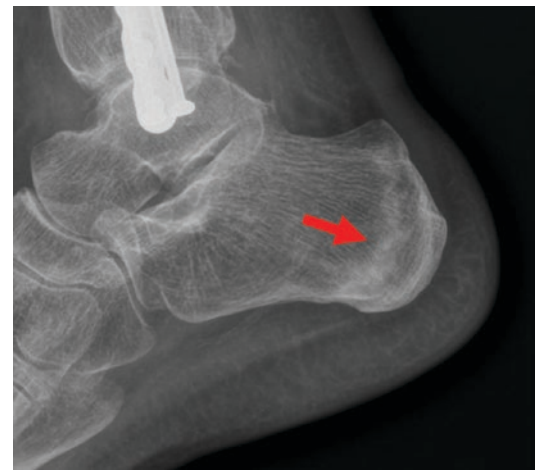

Fig. 5.3 Lateral plain radiograph of the foot in a 20-year-old female lacrosse player with progressive heel pain demonstrating a grade 2 stress fracture of the posterior calcaneal body (arrow). The patient has undergone previous open reduction internal fixation of the fibula and medial malleolus

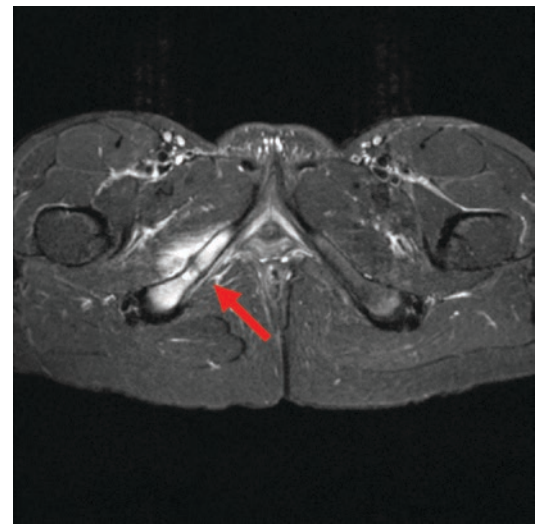

Fig. 5.4 Axial cut T2 MRI of the pelvis demonstrating a grade 3 (complete nondisplaced) stress fracture of the right inferior pubic ramus in a 19 -year-old female collegiate distance runner with worsening right anterior pelvic pain over a 4 week period and absent menses for 5 months

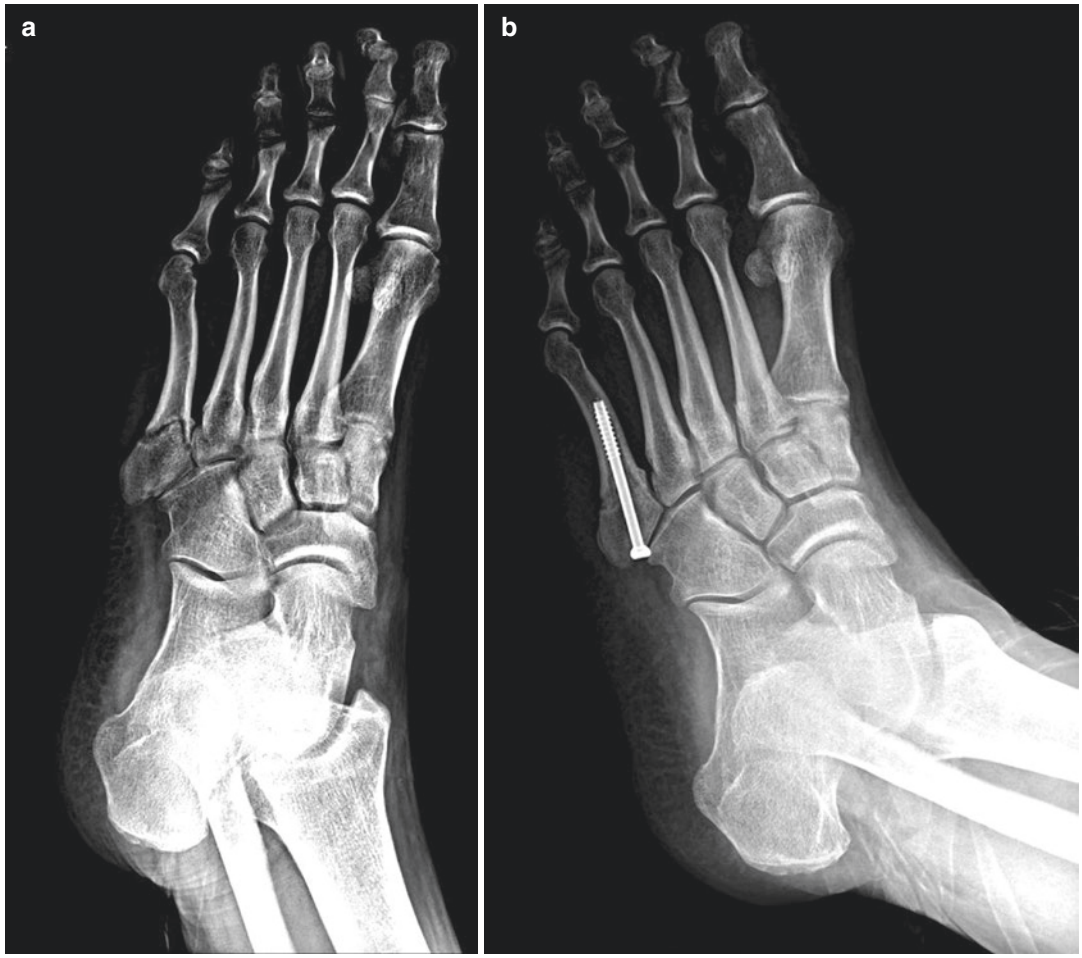

Fig. 5.5 (**a**, **b**) Oblique foot radiograph demonstrating grade 4 (displaced) fifth metatarsal stress fracture in a 21-year-old male college basketball player with worsening lateral foot pain for 5 weeks, acutely worsened with an ankle inversion injury. (**a**) Intramedullary screw fixation was required for fracture stabilization and healing of the fracture (**b**)

placement (Fig. 5.4). Grade 4 fractures are displaced (Fig. 5.5) and grade 5 stress fractures are chronic having gone onto non-union (Fig. 5.6). This system is summarized in Table 5.4. This classification system has been shown to have high inter- and intra-observer reliabilities. [22] Coupling this fracture grade with the location of the fracture provides a more comprehensive description of the injury that takes into account both the extent of structural failure and healing potential of the injury.

When reporting the stress fracture grade in this system, the imaging modality used should be reported. For example, a CT scan revealing a non-displaced fracture line in a tarsal navicular in a healthy collegiate basketball player would be reported as a grade 3 tarsal navicular stress fracture on CT scan. The requirement to provide imaging modality gives a comprehensive description of the injury, which is both clinically relevant and useful in research to ensure comparable groups. We did not mandate a specific imaging modality for the system. To mandate that a bone scan, CT scan, or MRI be performed would result in the system being less easily applied. For example, if a fifth metatarsal proximal metaphyseal-diaphyseal fracture shows evidence of a nonunion on radiographs, mandating a bone scan or MRI

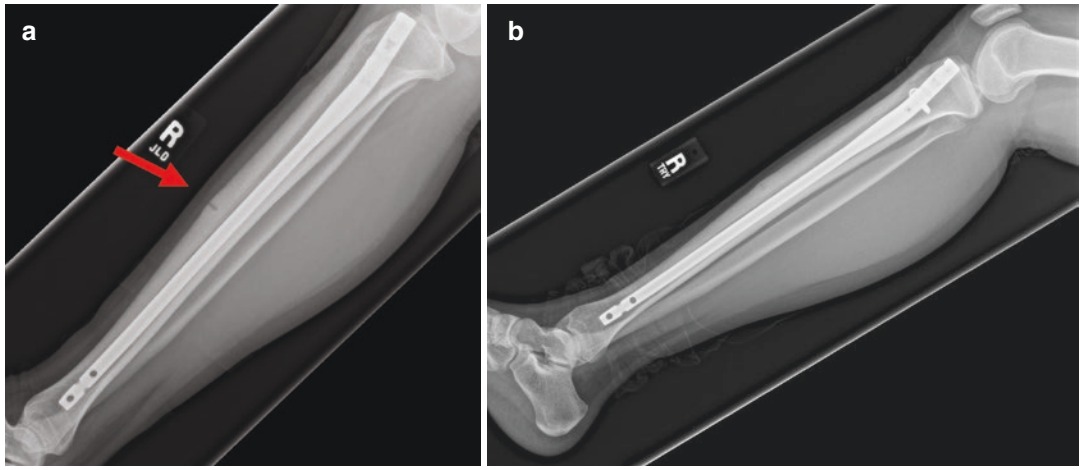

Fig. 5.6 (**a**, **b**) Plain radiographs of a 19-year-old female professional ballet dancer with chronic anterior tibial pain and grade 5 (nonunion/"dreaded black line") stress fracture of the anterior tibial cortex (arrow). (**a**) Final treatment required operative fixation with an intramedullary rod. (**b**) Cortical thickening with fracture healing is evident 4 months post-surgery

adds little to the clinical description. If the clinician does not pursue imaging beyond the radiograph, the system is not, in a practical sense, generalizable to all sites. A downside to not requiring single or multiple imaging modalities is that the fracture grade in the new classification system may change depending on which modality is used. Because of this aspect of the grading system, it is required that the imaging modality used is mentioned when grading the stress fracture.

Three key features of the Kaeding–Miller system are as follows: (1) it is generalizable (i.e., applicable to any location in any bone), (2) it has been validated with intra- and inter-observer reliabilities, and (3) it has been shown to be predictive of outcome and thus is associated with prognosis. It is of great benefit to have a single, reproducible, and easily used grading system that describes the clinically relevant parameters throughout the body. The more concise and reproducible the classification system is, the more accurate the communication between clinicians and patients who are being counseled will be. The responses of the clinicians who evaluated the system and their ability to reproduce the classification system were evidence that evaluators found the system easy to apply and understand. Almost perfect intra-observer agreement was found among 15 evaluators of the classification system which included orthopedists, primary care sports medicine specialists, and physician assistants. Substantial to almost perfect inter-observer reliability was observed for the classification grades among the same evaluators. A total of 14 of the 15 evaluators (93.3%) reported that the system was easy to remember, would facilitate communication regarding stress fractures among medical colleagues, and would be used in their practice in the future. Additionally, the same group of evaluators was able to reproduce the system from memory with 97.3% accuracy [22]. In two published studies, the Kaeding–Miller classification system was shown to be predictive of healing times [49–51]. The two studies looked at the return to sport time in Division 1 athletes after they had been diagnosed with a stress fracture. It was shown in both studies that the higher the Kaeding–Miller grade, the longer it took for the athlete to return to sport [49].

Conclusion

Stress fractures of the axial and appendicular skeleton are troublesome overuse injuries for athletes and non-athletes alike and especially in military personnel. This type of fracture repre-

sents a fatigue failure of bone, occurring within a continuum of severity of structural injury with healing potential varying by location. Though many stress fracture classification systems exist in the literature, there is only one comprehensive classification system for stress fractures incorporating both clinical and radiographic characteristics of the injury that is applicable to all bones. Though many are generalizable, only the Kaeding–Miller classification system has been validated for inter-and intra-observer reliabilities and been shown to be predictive of treatment outcomes.

We have described an easy-to-use, easily-remembered, and readily applied classification system that incorporates clinically relevant parameters and is generalizable to all stress fractures. It has been validated for intra-observer and inter-observer reliabilities and been shown to be predictive of prognosis. This classification system describes the clinically relevant characteristics of a stress fracture in a reproducible manner, enhancing the description, communication, and research of stress fractures. A gold-standard classification system for grading stress fractures is yet to be determined. As with any system, an ideal technique of classifying stress fractures is reproducible, generalizable, easy to use, and clinically relevant, with four key descriptors: fracture location, risk assessment, fracture grade, and imaging modality used to make the diagnosis.

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Insufficiency Fractures

6

Carmen E. Quatman, Mitchell Gray, and Laura S. Phieffer

Repetitive loading of bone over time without the presence of a high-energy trauma can lead to stress fractures or microtrauma bone across all patient demographics. The common types of atraumatic stress fractures include fatigue, pathologic, and insufficiency fractures [1]. It is important to distinguish between causes of stress fracture in athletes because each type requires unique steps in diagnosis and management. Often in the athlete, the more common stress fracture encountered is related to fatigue from repetitive stress from overuse of healthy bone, often due to increased training, altered gait mechanics, or changes in equipment (i.e., footwear). Fatigue fractures will be discussed in a separate chapter and pathologic fractures (tumor/infections) are critically important to be recognized as the cause of a fracture but are beyond the focus of this book [2–5]. It is important to remember that while rare, athletes can develop malignancies, and benign tumors of bone are not infrequent in younger patients. Insufficiency fractures are more commonly thought to occur in older adults, but they may occur also in younger athletes who have systemic bone diseases such as Paget's, osteomalacia, or osteogenesis imperfect, or who have malnutrition and vitamin or calcium deficiencies. They may even result from hormonal deficiencies or alterations. Most insufficiency fractures require further workup for causes of abnormal bone healing/resorption and eating habits, as alterations in metabolic factors can significantly increase risk for fractures and delay bone healing.

Causes of Insufficiency Fractures

Insufficiency fractures can occur throughout the skeleton but are common in the tibia, femoral head and neck, sacrum, pelvis, and feet. The majority of stress fractures in athletes occur in cortical bone. In contrast, insufficiency fractures are often the result of the breakdown of cancellous bone. The race between bone growth and bone absorption through osteoblasts and osteoclasts is an important concept for stress fracture pathophysiology and goals of treatment. Systemic bone disorders such as osteopenia/osteoporosis, osteomalacia, Paget's disease of bone, or a history of radiation therapy lead to weakened bone and, ultimately, potential fracture.

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Osteopenia/Osteoporosis

Decreased bone density (osteopenia) is commonly associated with insufficiency fractures. Although osteopenia/osteoporosis is often thought to be associated with age, it may occur in young athletes and result in an insufficiency fracture. There are numerous causes of osteopenia/osteoporosis that can be either primary or secondary, many of which are potentially reversible or have bone-sustaining treatment options. Primary causes of low bone density include age-related post-menopausal, juvenile, or spontaneous occurrences as well as osteogenesis imperfecta. In contrast, secondary causes may include rheumatoid arthritis, sex hormone deficiency, steroid therapy, hyperparathyroidism, endocrine disorders, renal osteodystrophy, or nutritional deficiencies [6]. Athletes that have any of these conditions are vulnerable to insufficiency fracture, and diagnosis of an insufficiency fracture should alert providers to seek out appropriate nutritional and endocrinology evaluations to potentially identify and treat any underlying secondary causes of osteopenia/osteoporosis in order to provide the best healing potential as well as to reduce the risk of future fractures.

Screening for osteoporosis/osteopenia can help prevent insufficiency fractures. Adult patients should be screened for osteoporosis by evaluating history/risk factors at age 50 or older (Table 6.1), with dual energy X-ray absorptiometry (DXA) testing to screen for osteoporosis for all women ≥65 years of age and all men ≥70 years. Younger adults who exhibit one major or two minor risk factors for osteoporosis should also undergo DXA testing [7, 8]. In addition, transgender athletes who are taking hormonal medications may be at high risk for bone density changes and insufficiency fracture. Optimizing nutrition and working with an endocrinologist may be important for transgender athletes [9].

Bone density evaluation by DXA is commonly interpreted in terms of T-score or Z-score. To evaluate bone density in children and adolescents, the Z-score is used. Which it is based on standard deviations from an age-, gender-, and race-matched reference values. In contrast, for adults above the age of 29, it is more appropriate to use the T-score, which refers to how many standard deviations a patient's bone density lies from the average bone density of a 20–29 year old female or male for comparison. Evaluation of the femoral neck, total hip, or lumbar spine with a T-score of ≤ -2.5 indicates osteoporosis [10]. In addition, patients can have a clinical diagnosis of osteoporosis if they have a hip fracture or other fracture from a low energy mechanism, despite a normal T-score since bone density does not necessarily account for bone quality. Assessing bone quality is challenging, and accurate assessment often requires a biopsy or an invasive procedure, which is not necessarily cost-effective or risk free for determining bone quality [11].

The best time for optimizing bone health is before the age of 25 years. Once skeletal maturity and bone development reach the peak, the process of bone development declines. Instead, bones have an unavoidable increase in oxidative stress and accumulation of free radicals with aging that impact the strength and integrity of the bone architecture. Encouraging healthy habits early in life, particularly nutrition and weight-bearing activities, is important for giving an optimal starting point for bone health [12]. Although

Table 6.1 Risk factors for osteoporosis

Major risk factors	Minor risk factors
Vertebral compression fracture	Rheumatoid arthritis
Fragility fracture after age 40	Past history of hyperthyroidism
Family history of osteoporotic fracture	Chonic anticonvulsant therapy
Systemic glucocorticoid therapy >3 months	Low dietary calcium intake
Malabsorption syndrome	Smoking
Primary hyperparathyroidism	Excessive alcohol intake
Propensity to fall	Excessive caffeine intake
Osteopenia apparent on X-ray film	Weight <57 kg
Hypogonadism	Weight loss >10% of weight at age 25
Early menopause (before age 45)	Chronic heparin therapy

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activities such as swimming and biking encourage cardiovascular fitness, weight-bearing exercises such as racquet sports, jogging, walking, dancing, and other high-impact activities have more influence on bone density.

While sports and activities can help prevent decline in bone density, they do not appear to have a significant impact on bone mineral density improvements later in life [12]. Instead, at this stage, healthy lifestyle habits that incorporate weight-bearing exercise can have the best benefit on bone mass [13]. Older athletes may have to modify activities related to osteoarthritis, balance, or health conditions; however, healthy weight-bearing activities may continue to reduce the risk of insufficiency fractures. In addition, dietary and sometimes even pharmacological steps to prevent osteopenia need to be utilized to prevent insufficiency fractures. Nutritional deficiencies are not uncommon in athletes and may be related to environment, genetics, or eating disorders. Calcium and vitamin D are important dietary components associated with higher bone mineral densities. Vitamin D and calcium are essential for bone health and deficiencies in either can lead to weakened bone and increase susceptibility to stress fractures [14]. Likewise, consumption of calcium and vitamin D can lower the incidence of stress fracture [15]. Recommended doses for vitamin D and calcium vary by sex and age. In general, premenopausal women and younger men should target 1000 mg calcium and 600 IU vitamin D (D3 supplement) daily, and postmenopausal women and older men should target 1200 mg calcium and 800 IU vitamin D daily [16, 17]. The prevalence of vitamin D deficiency is relatively high, and the risk is increased for athletes in higher latitudes, during winter and early spring seasons, and for those involved in indoor sports [17]. For athletes and older adults who experience insufficiency fractures, supplementation of vitamin D3 and calcium may be necessary to help with healing of the fracture as well as further prevention of stress fractures in the future.

Bone health medications have high potential to make an impact on osteoporosis management. However, there are many barriers for utilization of these medications in order to have a public health impact. Often, osteopenia/osteoporosis is unnoticed and undertreated in older patients. In the United States, more than 1.5 million osteoporosis-related fractures occur per year in 30% of men and 50% of women over the age of 50 [18, 19]. Patients who experience an insufficiency fracture are at high risk of future fracture, and this should trigger motivation for potential treatment and prevention strategies. It should also alert health care providers to consider bone health medications. Although there are many medications available (Table 6.2) and well established literature that supports the effectiveness of these medications for prevention of osteoporotic frac-

Table 6.2 Guideline of bone health medications

Generic Drug	Brand	Administration	Duration	After completion
Alendronate Sodium	Fosamax	1/week orally	4–5 years	Drug holiday (2–3 years)
Ibandronate Sodium	Boniva	1–2/month orally	4–5 years	Drug holiday (2–3 years)
Risdedronate Sodium	Actonel	1/day up to 1/week orally	4–5 years	Drug holiday (2–3 years)
Zoledronic Acid	Reclast	1/year Infusion	3 years	Drug holiday (2–3 years)
Calcitonin-Salmon	Fortical/ Miacalcin	SubQ, IM, nasal	4–5 years	Drug holiday (2–3 years)
Denosumab	Prolia	2/year infusion, renal disease pts	3 years	Drug holiday (2–3 years)
Raloxifene	Evista	1/day orally	4–5 years	Drug holiday (2–3 years)
Teriparatide	Forteo	1 SubQ injection daily	2 years (lifetime)	Follow with Prolia or Bisphosphonate
Abaloparatide	Tymlos	1 SubQ injection daily	18 months–2 years (lifetime)	Follow with Prolia or Bisphosphonate

tures, cost, compliance, and tolerance of the medications remain major barriers for widespread use. Pharmacologic treatment guidance is beyond the scope of this chapter; however, Table 6.2 provides some general thoughts related to prescribing practices. It is important to remember that the medications have time-dependent parameters, and bisphosphonates or denosumab require drug holidays to avoid unintended side effects from prolonged use. Anabolic medications such as teriparatide and abaloparatide have great properties of bone building; however, they have only been studied for 18–24 months of utilization and require further treatment with a bisphosphonate or equivalent to help prevent rapid breakdown of the bone mass gains from the medication.

The Female Athlete Triad and Decreased Bone Mineral Density

Female athletes, particularly adolescent and young adults, are at risk for insufficiency fractures if they exhibit some or all of the signs of the female athlete triad. The triad is defined as (1) low energy availability with or without disordered eating, (2) menstrual dysfunction, and (3) low bone mineral density [20]. There are numerous risk factors for the triad (Table 6.3) and female athletes that take part in sports that favor low body mass and small build such as ice skating, gymnastics, dancing, and running may be at a particularly high risk. Athletes that exhibit more triad risk factors are associated with higher risk for stress fractures and lower bone mineral density [21, 22].

Table 6.3 Risk factors for female athlete triad

Menstrual irregularities
Criticism of eating habits by coach, family, or peers
Pressure to lose weight
Depression
Dieting
Obsessive personality
Early sport-specific training
Overtraining
Recurrent injuries, particularly fractures
History of fracture
Low body mass index

Decreased energy availability directly influences female menstruation leading to amenorrhea and suppression of bone production in at-risk athletes. Hypogonadal states influenced by chronic undernutrition disrupt bone mineralization leading to decreased bone mineral density [23]. A 2018 study of collegiate athletes found that oligomenorrhea and amenorrhea were the most influential independent predictors of bone mineral density for total body density on DXA analysis [24]. Eating disorders are a form of chronic malnutrition and serve as a secondary clinical risk factor for bone mineral loss and fracture [23].

Screening for female triad risk factors in collegiate and high school athletes is important for early detection and prompt treatment to avoid future insufficiency fractures. Specific medical questionnaires can be utilized to evaluate the presence of one or more risk factors for a particular athlete, prompting further investigation of any risk factor. Diagnosis of female athlete triad components should involve a multidisciplinary approach, including a physician, a sports dietitian, and a mental health professional as appropriate [23]. Age should be considered when assessing the susceptibility of stress fracture, with athletes in their teenage years at higher risk of developing fracture compared to those in their twenties with triad risk factors [25]. The energy availability of an athlete can influence overall health and should be adequately tracked with the help of a sports nutritionist. Markers such as dietary intake, fat mass, thyroid hormone levels, and resting metabolic rate should be monitored in athletes suspected of reduced energy availability. Reproductive cycle regularity is also directly linked to musculoskeletal and bone health. A prolonged disturbance in hormone levels in women can disrupt bone strength and predispose these athletes to fracture. Diagnosis of amenorrhea and oligomenorrhea should include the athlete's primary care physician, and a workup should rule out pregnancy, endocrine causes, and other systemic pathology.

Bone mineral density (BMD) assessment is critical to determining the fracture potential for athletes. Similar to the diagnosis of osteoporosis in adults, DXA scanning is indicated in adolescent athletes with ≥1 major risk factor or ≥2 minor risk factors as outlined in Table 6.4.

These criteria take into account triad-specific risk factors outlined by the 2014 Female Athlete Triad Coalition, and a score of >2 should indicate the need for DXA imaging [26]. The preferred measurement sites for BMD include the posterior-anterior spine and total body less head; however, the proximal femur, 33% radius ($\frac{1}{3}$ radius), and lateral distal femur can be used as indicated. Z scores are preferentially used for adolescents when BMD values are compared to reference data. The diagnosis of osteoporosis in an adolescent should not be made solely on densitometric findings, but instead alert the medical team to look for other signs of bone pathology such as compression fractures. Clinical diagnosis of osteoporosis can be made with densitometric findings and presence of one or more vertebral compression fractures. If compression fractures are not present, a diagnosis can be made based on ≥2-long bone fractures before 10 years old or >3-long bone fractures before 19 years old with a Z score < −2.0. To avoid insufficiency fractures in patients who do not fit these criteria explicitly, consideration of skeletal fragility should be addressed in patients with Z scores > −2.0 with significant risk factors. For athletes in need of follow-up imaging, DXA scans are indicated at an interval of every 6–12 months [27] (Table 6.5).

Table 6.4 DXA scan eligibility criteria for athletes

Risk factors for DXA scan eligibility in athletes		
Risk factors	Low risk factors (score = 1)	High risk factors (score = 2)
Nutrition status	Current or rior history of disordered eating for ≥6 months	DSM-V diagnosis of eating disorder
Delayed menarche	Menarche between 15–16 years old	Menarche at ≥16 years
Oligomennorea status	Current or prior history of 6–8 menses over 12 months	Current <6 menses over 12 months
BMI status	BMI between 17.5–18.5 or recent weight loss of 5–10% in 1 month or 85–90% expected weight	BMI≤17.5 or recent weight loss of ≥10% of total weight or <85% expected weight
Prior fracture	One prior stress fracture	Two prior stress fractures or one high risk fracture or one low-energy nontraumatic fracture
Bone mineral density	Z-score between −1.0 and −2.0 (1 year from baseline DXA)	Prior Z-score of less than −2.0 (1 year from baseline DXA)

Osteomalacia and Rickets: Role in Fracture Risk

Osteomalacia is a disorder of disrupted bone mineralization which can progress to weakness, joint pain with increased weight-bearing, and fracture. The disease can also present in childhood as rickets and can affect the cartilaginous growth plates in addition to affecting the structural integrity of bone. Calcium deficiency due to insufficient intake or activation of vitamin D and phosphate deficiency due to renal wasting are the primary causes of osteomalacia. Risk factors for osteomalacia include a diet low in calcium, dark skin, malabsorption, and decreased exposure to light causing vitamin D deficiency [28, 29]. Although osteomalacia is uncommon in athletes, it should be considered in adult patients who present with an insufficiency fracture or bone pain and osteopenia. Healthy serum 25-hydroxyvitamin D levels are debated in the literature; however, levels below 30 ng/ml would be considered insufficient for athletes and levels below 20 ng/ml would be considered deficient regardless of the population [17]. Depending on the etiology, treatment involves correction of the deficient state and adequate supplementation of vitamin D. It has been documented that athletes are particularly susceptible to low vitamin D levels, especially at higher latitudes and those who play indoor sports [17].

Table 6.5 Triad cumulative risk assessment

Risk factors	Low risk = 0 points each	Magnitude of risk	
		Moderate risk = 1 point each	High risk = 2 points each
Low EA with or without DE/ID	☐ No dietary restriction	☐ Some dietary restriction‡ current/past history of DE	☐ Meets DSM V criteria for ED*
Low BMI	☐ BMI ≥ 18.5 or ≥90% EW** or weight stable	☐ BMI 17.5 <18.5 or <90% EW or 5 to <10% weight loss/month	☐ BMI ≤17.5 or <85% EW or ≥10% weight loss/month
Delayed menarche	☐ Menarche <15 years	☐ Menarche 15 to <16 years	☐ Menarche ≥16 years
Oligomenorrhea and/or amenorrhea	☐ >9 menses in 12 months*	☐ 6–9 menses in 12 months*	☐ <6 menses in 12 months*
Low BMD	☐ Z-score ≥ −1.0	☐ Z-score-1.0*** < −2.0	☐ Z-score ≤ −2.0
Stress reaction/fracture	☐ None	☐ 1	☐ ≥2; ≥1 high risk or of trabecular bone sites†
Cumulative risk (total each column, then and for total score)	_____ points +	_____ points +	_____ points = _____ total score

∗ Currently experiencing or has a history.
∗∗ ≥ 90% EW; absolute BMI cutoffs should not be used for adolescents.
∗∗∗ Weight-bearing sport.
† High-risk skeletal sites associated with low BMD and delay in RTP in athletes with one or more components of the triad include stress reaction/fracture of trabecular sites (femoral neck, sacrum, and pelvis).
‡ Some dietary restriction as evidenced by self-report or low/inadequate energy intake on diet logs. EW, expected weight.
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Adequate supplementation of vitamin D is indicated in at-risk athletes and guidelines for correct supplementation can be referenced in the osteopenia/osteoporosis section of this chapter.

Paget's Disease of Bone

Paget's disease of bone is typically found in adults over the age of 50 and is characterized by excessive bone remodeling causing replacement with poor quality bone. The disease can present as constant bone pain in one or more skeletal sites that worsens at night. Osteoarthritis, nerve compression from bony outgrowth, and stress fracture are other ways that the disease may present. Involvement of the spine can lead to radiculopathy from nerve compression while involvement of the skull can lead to hearing loss secondary to nerve impingement. Causes of Paget's disease of bone can include genetic factors as well as environmental factors such as viral illness. Serum testing usually reveals normal calcium, phosphate, and parathyroid hormone, but the alkaline phosphatase level can be elevated due to increased osteoblast activity. Abnormal radiographic analysis can also increase suspicion for Paget's disease of bone with findings such as osteolytic areas in the early stages and mixed areas of lysis and sclerosis in later findings [30]. Plain X-rays focused on the abdomen or skull have demonstrated a 79% and 93% chance of detecting bone lesions, respectively [31]. Bisphosphonates are commonly used to control bone pain and prevent spinal cord dysfunction in these patients. Paget's disease of bone should be ruled out in middle-aged athletes with significant risk factors when presenting with an insufficiency fracture.

Hyperparathyroidism and Brown Tumors

Advanced hyperparathyroid states can lead to weakening and loss of bone causing osteitis fibrosa cystica, known as brown tumors. Hyperparathyroidism can be due to genetic factors, pathologic conditions such as parathyroid adenoma or glandular hyperplasia, secondary to chronic renal disease, lithium therapy, or thia-

zide diuretic use. Hyperparathyroidism is classically asymptomatic in the U.S. and is typically discovered through elevated calcium levels on routine screening.

Chronically elevated parathyroid hormone (PTH) levels are uncommon today, but can lead to abdominal pain from gastrointestinal complications, chronic kidney disease, kidney stones, and skeletal involvement. Bone resorption of the subperiosteal layer with fibrous replacement and excessive osteolysis contributes to the fragility of bone in this disease. DXA studies have shown a decrease in cortical bone density with preservation of density at sites with increased trabecular bone such as the spine [32]. However, further investigation with quantitative CT analysis implicates trabecular bone loss and disruption of microarchitecture in the fracture risk for these patients [33]. Parathyroidectomy is indicated in certain patients with hyperparathyroidism, leading to an increase in bone mineral density on DXA scans in the first postoperative year [34]. Although the development of bone pathology is rare in the United States today, brown tumors should be ruled out as a cause of insufficiency fracture in athletes with concurrent endocrine abnormalities.

Radiation-Induced Fracture

Ionizing radiation is commonly used in cancer treatment due to its ability to induce DNA damage and decrease cancer growth. However, in patients treated with radiation therapy, there is an increased risk of skeletal instability and fracture. Radiation causes direct damage to osteoblasts, leading to structural weakness of the surrounding bone and fracture under normal stress conditions. Whole pelvis radiation is well documented in the literature, and patients who undergo radiation therapy for anal, cervical, rectal, and prostate cancers have an increased risk of pelvic fracture [35, 36]. The risk of fracture is associated with the dose per fraction and length of radiation treatment. Symptomatic patients who undergo low dose radiation treatment for 5-6 weeks or a short course of high dose radiation should be thoroughly evaluated [37]. Because the majority of fractures are documented in the first 5 years following radiation therapy, athletes with a recent history of radiation treatment should be monitored for insufficiency fracture [38].

Presentation and Diagnosis of Insufficiency Fracture

Insufficiency fractures often present after a slow, increasing onset of pain to a localized area of bone such as the spine, pelvis, or lower limb and are not usually associated with a specific trauma. In some cases, if the pain is severe enough, a patient may present with an inability to weight-bear on the limb. Physical examination of the area of pain (as well as the uninjured limb for comparison) including inspection of the area for swelling, bruising, warmth, palpation of the area of localized tenderness, neurovascular exam, and often a gait exam can help in the evaluation of a patient for insufficiency fracture.

Studies evaluating site-specific fracture risk with low bone mineral density measures do not always show a clear correlation. It is therefore essential to rely both on patient history and on imaging results to determine the overall risk of insufficiency fracture in a young athlete [39]. Diagnostic imaging should start with plain radiographs. If plain radiographs are non-diagnostic but the concern remains for possible insufficiency fracture, MRI, CT, or bone scintigraphy may aid in diagnosis. On plain radiographs and CT imaging, findings of bone resorption along a fracture line, callus, and osteolysis may be present with insufficiency fractures (Fig. 6.1a–e). MRIs will demonstrate hypointense signal on T1-weighted images and hyperintense signal on T2-weight images, with possible visualization of a fracture line. Bone scintigraphy may be useful to evaluate changes in radionucleotide uptake patterns, revealing evidence of increased osteoblastic activity that may signify an insufficiency fracture.

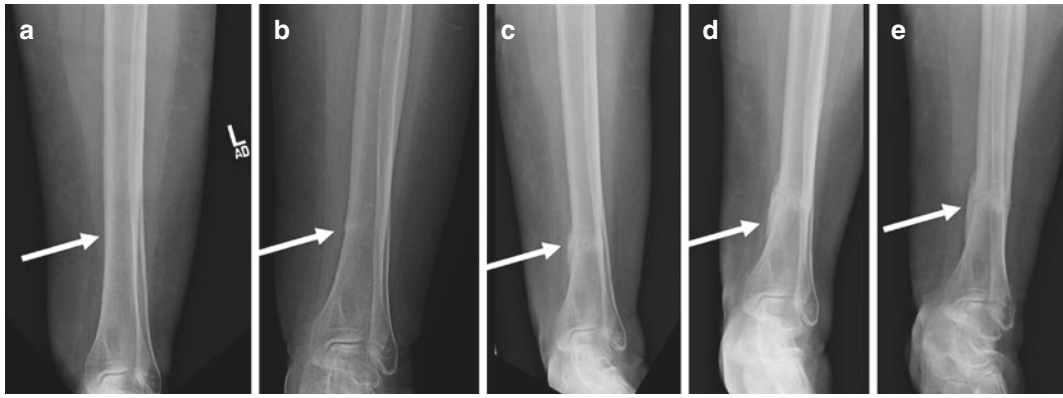

Fig. 6.1 (**a**–**e**) Insufficiency fracture transition over a 5-month timespan

Spine

Vertebral insufficiency fractures are the most common type of insufficiency fracture; however, many patients remain asymptomatic and go undiagnosed. Fracture of the spine is well documented in patients who are elderly with osteoporosis; however, evidence is lacking in the literature for athletes specifically. A common presentation includes mid to lower back pain, and the majority of patients do not describe a traumatic episode that led to their symptoms. Weight-bearing anterior-posterior and lateral radiographs should be performed on initial evaluation and can be used to follow a fracture over time. Development of one insufficiency fracture increases the risk of developing future fractures and also contributes to kyphotic deformity of the spine [40]. For this reason, treatment should include correcting the underlying cause of insufficiency and addressing the instability that could lead to further clinical consequences. In athletes with back pain and risk factors for bone instability, clinicians should have increased clinical suspicion for insufficiency fracture. Non-operative management should be the first line of treatment in athletes with a trial of modified activity, bracing, physical therapy as needed, and management of nutrition/medical disorders that may have led to insufficiency fracture. Surgical procedures such as vertebroplasty (injection of polymethylmethacrylate cement under fluoroscopy) could be considered in older patients with recalcitrant pain.

Pelvis and Sacrum

Pelvic fractures account for around 7% of all fractures and are commonly caused by insufficiency in the older adult population [41]. These fractures are of particular importance because like hip fractures, there is a significant risk of morbidity and mortality. As in most other forms of insufficiency fracture, incidence is higher with age, female gender, and other risk factors for bone instability. Radiation therapy is a well-documented cause of insufficiency fracture and details can be found in the previous section on this topic [35]. In older females or athletes who have risk factors for osteoporosis, acetabular fracture should be considered given the clinical picture.

A thorough history and physical exam should be performed when suspecting pelvic fracture because the location and description of pain can overlap with other pathologies. Patients often describe lower back, hip, buttock, or groin pain that is commonly misdiagnosed as degenerative disc disease or spinal stenosis. Inability to bear weight is common as well as difficulty initiating flexion of the leg. Only a third of patients report minor trauma and the majority report no insidious onset of symptoms [42].

Plain radiographs are commonly obtained when working up hip or lower back pain, yet sensitivity is limited. With negative plain films and a strong clinical suspicion of pelvic fracture, an MRI should be ordered and is a highly sensitive test for this fracture type [43].

Pelvic and sacrum insufficiency fractures are inherently stable, and non-operative management is preferred in athletes with confirmed pelvic fractures that have no neurovascular compromise or concern of instability. Surgical management is avoided except in the presence of open fractures, joint instability, or failure to improve with conservative treatment. Early and progressive mobilization is essential in these patients to improve functional outcomes [42]. Treatment should consist of addressing any underlying medical/nutritional issues and a period of modified activity with gradual return to full activity. Limited weight-bearing with progression to full weight-bearing over 2–6 weeks, followed by avoidance of high-impact exercises until no pain with low impact activities, is recommended for pelvic insufficiency fractures [44]. Integration of the athlete's primary care physician and physical therapist is important to closely monitor improvement of mobility.

Resolution of pain and symptoms from sacral insufficiency fractures can be prolonged. Surgical management, particularly in younger athletes, should be considered only after a trial of conservative management. Similar to treatments for vertebral compression fractures, sacroplasty (injection of polymethylmethacrylate cement under fluoroscopic guidance into the fracture) could provide symptom relief and lead to earlier mobilization. Due to the complication risk profile of neurologic injury should cement extravasation occur, sacroplasty is reserved for patients as a last resort and a detailed risk-benefit discussion with the patient is warranted.

Hip and Femur

Fractures of the hip account for 17% of all reported fractures [41]. The most common types of femur insufficiency fractures are intracapsular and intertrochanteric fractures with other less common types of femur fractures including subtrochanteric, femoral shaft, and lower femur fractures [45]. Female gender, increased age, history of osteoporosis, and recent transplantation are factors predicting greater incidence of overall insufficiency fracture. Patients with suspected insufficiency hip fractures may not present with a history of trauma or fall. These patients typically have trouble bearing weight and describe pain in the groin, thigh, or buttocks region. Plain film radiographs (anterior-posterior and lateral) are indicated followed by MRI if the fracture is not appropriately characterized with plain films alone.

Especially in older athletes with concerning risk factors, addressing the underlying cause of insufficiency after the occurrence of fracture will help prevent future fractures and may aid in the recovery of the patient after surgery. Early fixation surgery has shown to reduce mortality and major complications in patients with a hip/femur fracture. Early mobilization after surgery for patients with hip or proximal femur fracture is indicated to decrease infection rate and improve functional measures [46]. Besides optimizing bone health, which is discussed previously in this chapter, recent studies of osteoporotic hip fractures have suggested that CT scan may be used to predict fracture risk [47].

Tibia

While stress fractures commonly involve the tibia and are due to repeated overuse and recent increase in strenuous activity, insufficiency fracture of the tibia should be considered in patients with a history of bone fragility. Fracture due to insufficiency typically occurs at the distal tibia with inability to bear weight, localized swelling, tenderness, and bruising (Fig. 6.1) [48]. Evaluation of neurovascular involvement as well as acute compartment syndrome should be performed in the case of a large displacement fracture. Plain radiographs have a high sensitivity for larger fractures, although smaller tibial fractures often seen due to stress or insufficiency are better identified using MRI [49]. Nonsurgical management such as a period of non-weight-bearing, bracing, or casting is attempted for the majority of insufficiency fractures. Failure to improve with conservative treatment will lead to the need for surgical treatment for some patients. Surgical fixation with intramedullary nail-

ing, plating, and/or bone grafting of cortical defects of insufficiency fractures could prevent further progression of fracture, help with increased mobility, and improve return to play time [50–56].

Foot

Common insufficiency fractures of the foot are reported in the calcaneus, talus, navicular and metatarsals. They are particularly common in patients with diabetes mellitus, peripheral neuropathy, and osteoporotic patients, yet they may occur in younger athletes with decreased bone density. Plain radiographs may demonstrate insufficiency fractures, but an MRI is often necessary for early diagnosis. Calcaneus and most metatarsal insufficiency fractures may be amendable to conservative nonoperative management with modified activity, trial of non-weight-bearing, splinting, bracing, or casting. However, bases of the second metatarsal and fifth metatarsal fractures have higher risk for nonunion and refracture. Surgery may be necessary to address these more high-risk fractures. Similarly, talus fractures are at high risk for nonunion and may need surgical management to return to activity.

Upper Extremity

Upper extremity insufficiency fractures are uncommon; however, they have been reported in the ribs, humerus, ulna, radius, scaphoid, metacarpals, and shoulder girdle [57]. Sports such as rowing, swimming, throwing sports, weightlifting, and gymnastics are high-risk sports for upper extremity injuries [57, 58]. Modified activities and treatment of any underlying medical or nutritional needs are cornerstones to the treatment for upper extremity insufficiency fractures.

Conclusion

Athletes of any age are at risk for stress fractures, yet older athletes and athletes with metabolic disorders may be more prone to stress fractures and in particular insufficiency fractures. Decreased bone density at any age predisposes athletes to insufficiency fracture and should be evaluated in patients who are at high risk for stress fracture such as patients who exhibit signs of the female athlete triad or frequent fractures. History and diagnostic imaging are important for diagnosis. Often treatment for insufficiency fracture requires a multi-disciplinary approach for management and further fracture prevention. Correction or at minimum sustained management of underlying medical and nutritional pathologies as well as activity modification and possible surgical interventions may be necessary to optimize an athlete's ability to return to full activity safely.

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Part II

Maximizing Healing Potential for Stress Fractures

The Holistic Approach to Stress Fracture Treatment

7

Timothy L. Miller

Introduction

Stress fractures of bone, also known as fatigue fractures or march fractures, are common and troublesome injuries in athletes and military personnel. Many biologic and mechanical factors influence the body's ability to remodel bone and impact an individual's risk for developing a bony stress injury. These factors include sex, age, race, hormonal status, nutrition, tobacco use, neuromuscular function, and genetic factors. Other predisposing factors to consider include mental health status, abnormal bony alignment, improper technique/biomechanics, poor running form, decreased vascularity to specific bones, improper or worn-out footwear, and hard training surfaces.

With single-sport specialization and year-round training, stress fractures and other overuse injuries occur commonly and may be season or even career-ending. An understanding of modern strategies for evaluating and treating stress fractures is paramount for maintaining athletic participation and optimal athletic performance. This begins with assessing an athlete's risk of developing a stress fracture and the likelihood of a stress fracture at a particular site to progress. The goal for all clinicians treating stress fractures should be to reestablish bone homeostasis by balancing training and bone remodeling. Stimulating and supporting bone healing in athletes optimize bone health, expedite recovery, and decrease the risk of nonunion or catastrophic injury.

The Economic Impact of Stress Fractures

Global Economic Impact

Stress fractures are an extremely common injury in endurance sports and military training. They are increasingly common as athletes specialize in one sport or activity at earlier stages in their growth and development. The growing economic impact from stress injuries of bone is threefold: (1) cost of evaluation and treatment, (2) time lost from military service, and (3) limited team or club success [1–3]. These factors have effects that include rising medical care budgets for teams and universities and attrition of military personnel [4–6]. The lack of team success due to athletes being held out due to stress fractures has become increasingly evident in organizations such and the NBA (National Basketball Association) and in European professional soccer [7, 8].

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Individual Economic Impact

Individual economic costs for athletes accrue with the loss of playing time placing them at risk of losing scholarships or professional contract opportunities [9, 10]. These injuries may also place the individual athlete at risk of a shortened career due to medical disqualification or lack of effectiveness while competing [11–13]. Most notably, however, is the risk of a catastrophic injury taking place leading to permanent disability if a stress fracture is inadequately or improperly treated. Additionally, for the physicians and sports medicine clinicians who treat these athletes, the medicolegal implications of a missed or undertreated stress fracture cannot be overstated [14, 15].

Using a Holistic Approach

Stress fractures are the result of the loss of the normal balance between the creation and repair of microcracks in bone. Treatment principles include taking a systemic and site-specific approach to individuals presenting with this injury. In order to decrease the creation of microcracks, one must evaluate the patient's training regimen, biomechanics, and equipment. Maximizing the patient's biologic capacity to maintain bone homeostasis requires an assessment of the athlete's general health. This includes nutritional status, hormonal status, medication use, and mental health. Figure 7.1 demonstrates a treatment algorithm that should be considered when managing stress fractures in athletes.

Stress fractures are a unique problem in that they occur along a continuum of severity which can impact treatment and prognosis [16, 17]. Not only does the extent of these injuries vary, but the clinical behavior of these injuries varies by location and causative activity [18–20]. No two stress fractures behave in an identical manner. Treatment strategies must therefore be individualized to the patient, the sport, the anatomic site, and the severity of the fracture. The key team members required for treating stress fractures in

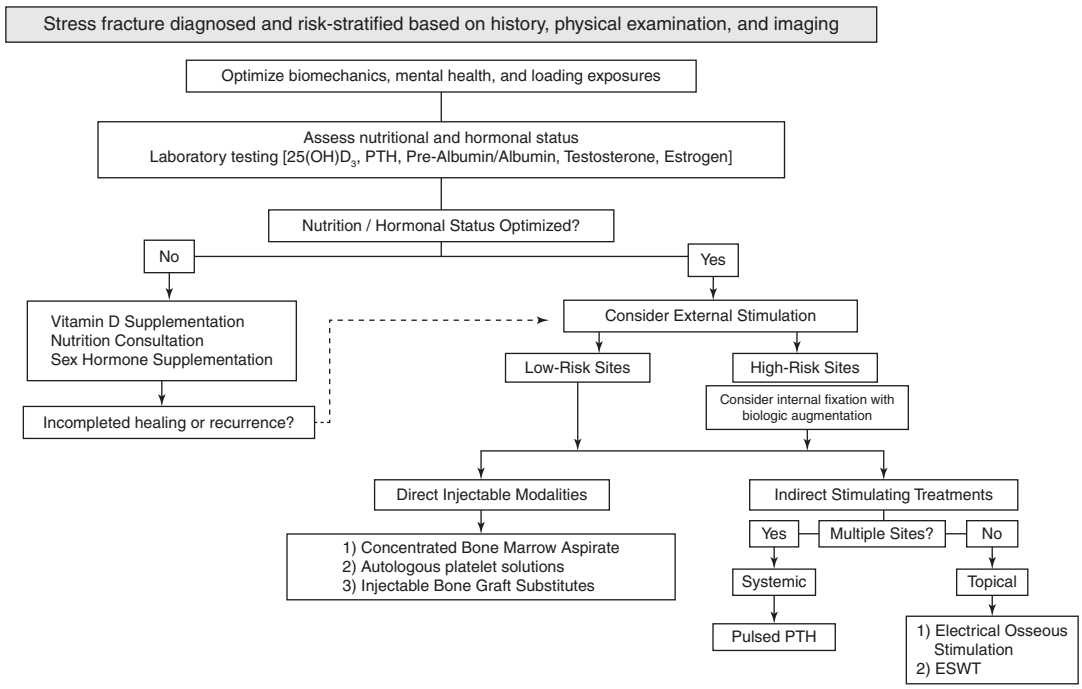

Fig. 7.1 Recommended treatment algorithm for stress injuries of bone. (Adapted with permission from Miller et al. [57])

Table 7.1 Key treatment team members for a holistic approach to bony stress injuries

Orthopedist
Nutritionist
Psychologist
Biomechanist/physical therapist
Endocrinologist
Coaching staff/athletic trainer

athletes are summarized in Table 7.1. The goal of treating bony stress injuries is to decrease the repetitive stress at the fracture site enough to allow the body to restore the dynamic balance between damage and repair.

Biomechanical Modifications

Biomechanical factors including limb length, limb alignment, and running form play a major role in an athlete's predisposition to developing overuse injuries. Running gait analysis or swim stroke analysis has demonstrated an ability to improve running efficiency and energy conservation but also decrease the risk of stress fractures [21, 22]. Physical therapists and biomechanists are able to correct biomechanical imbalances and compensatory mechanisms that may put athletes at risk of developing a stress fracture. This can be as simple as changing from heel striking to midfoot striking in order to more evenly distribute ground reaction forces during running [22].

Nutritional Optimization

Inadequate caloric intake may play a role in amenorrhea, which has been linked to an increased incidence of stress fractures [23, 24]. Dietary intake and disordered eating patterns have been linked to amenorrhea in a number of studies. A concept that has been developed supporting the link between dietary intake and amenorrhea is the so-called energy drain hypothesis [23]. If caloric intake is too low, production of hormones such as estrogen and progesterone is moved lower on the body's list of priorities. These hormones may not be produced in amounts high enough to allow menstruation to occur [24]. Oligomenorrheic or amenorrheic female athletes are at increased risk secondary to decreased estrogen levels and increased osteoclastic activity [25]. A recent study of female track and field/cross-country runners indicated an increased risk of developing stress fractures if body mass index (BMI) was less than 19 [26, 27]. The authors of this case series found that female athletes with BMI of 19 or lower took significantly longer to return to unrestricted training and competition than those with a BMI above 19. The authors further suggested that decreased muscle mass is a risk factor for stress injuries and poor healing [26, 27].

Hormonal Balance

Endocrine and malabsorption conditions can impair the delicate balance between bone formation and resorption, thus predisposing athletes to bony stress injuries. Stress fractures are associated with lower fat intake, lower calorie intake, eating disorders, and body weight of less than 75% of ideal body weight [23, 24]. The female athlete triad (menstrual irregularity, inadequate caloric intake, and decreased bone mineral density) has been associated with increased susceptibility to stress fractures. This increased risk is most commonly seen among female distance runners and military recruits and is increased compared with males performing the same activities [28]. High-intensity training may suppress serum estrogen levels leading to absent or irregular menses and exacerbating the risk of developing bony stress injuries [29].

Table 7.2 lists the key serum laboratory values to obtain when treating male and female athletes with recurrent stress fractures. These tests are crucial for assessing nutritional and hormonal status and healing potential. In female athletes, serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol levels are recommended to determine if an underlying endocrine condition or energy imbal-

Table 7.2 Serum laboratory values recommended to assess nutritional and hormonal status

Vitamin D-25(OH)D_3
Calcium and phosphate
Alkaline phosphatase
Prealbumin
Parathyroid hormone
Luteinizing hormone
Follicle-stimulating hormone
Estradiol
Testosterone
Serum CTX and NTX
Specific tests for GI malabsorption

ance is contributing to decreased bone mineral density or recurrent injury [21].

Mental and Emotional Fitness

Mental skills training has been shown to have a positive effect on endurance athlete performance and likely has an impact on the risk of developing overuse injuries such as stress fractures. For individuals with recurrent bony stress injuries, particularly those with signs or symptoms of an eating disorder, evaluation and treatment by a trained sports psychologist are a crucial adjunct to healing and preventing recurrent injuries. Anxiety and depression have been shown to increase the risk of disordered eating and the likelihood of developing exercise addiction [25, 30, 31]. Additionally, athletes may use exercise as an outlet for other underlying mental health concerns or to justify their eating habits for the sake of athletic success.

Sports psychology specialists assist the athlete with the development of skills such as motivation, goal setting, energy management, self-talk, focus, imagery, performance routines, mindfulness, and exercise balance. Though initial referral to a sports psychologist may be met with stigma and resistance by the athlete, this option is often required for identifying the signs of *unbalanced exercise*. Signs of unbalanced exercise include – but are not limited to – exercising without fueling the body, exercising to allow oneself to eat, and exercising to compensate for calories consumed [31].

Risk Factors for Developing Stress Fractures

Prevention is the ideal treatment of stress injuries of bone, and most other overuse injuries though this may not always be possible or practical. An assessment of the athlete's risk should be made at pre-participation evaluations, especially in those with a history of previous stress fractures and women with light or absent menses [21, 28]. Correction of amenorrhea in females as well as caloric and vitamin D_3 supplementation are recommended in addition to general nutritional optimization. Unfit or unconditioned athletes including military recruits, particularly female athletes with menstrual irregularities, are at peak risk for stress injuries to bone and increased utilization of healthcare resources [24]. Table 7.3 demonstrates the groups of athletes shown to be at highest risk for developing stress fractures.

Biomechanical

Muscle fatigue may be a collaborative culprit in the development of stress fractures in overtrained athletes. Since the late twentieth century, it has been widely accepted that neuromuscular conditioning plays a significant role in enhancing the shock-absorbing and energy dissipating-function of muscles to the ground reaction forces occurring during impact loading. This neuromuscular tone is able to decrease the amount of energy being directly absorbed by the bones and joints [22, 32]. Thus, as muscles fatigue they are less able to dissipate the external forces, allowing for

Table 7.3 Individuals with highest risk for developing stress fractures

Female athlete triad
Relative energy deficiency in sport
Male athlete tetrad
Overtraining syndrome
Unfit female military recruits
Vitamin D insufficiency
Female collegiate athletes participating in gymnastics, distance running, rowing, and lacrosse

more rapid accumulation of microtrauma to the bone [16, 18, 22].

If biomechanical abnormalities are encountered, proper footwear and the use of appropriately designed orthotic devices should be considered as an initial corrective measure. However, running gait analysis and appropriate running form or technique changes may be necessary to decrease the risk of future injuries. Anatomic risk factors include cavovarus foot deformity which has been demonstrated by multiple authors to increase the risk of metatarsal and midfoot stress fractures [33]. Furthermore, corrections should be made to gait in minimalist or barefoot runners in order to decrease the rate of midfoot and tibial stress fractures as well as plantar fascial rupture [34]. Athletes with a history of a lower-extremity stress fractures should be encouraged to vary the surfaces on which they train and not exclusively train on hard surfaces such as asphalt sidewalks or hard tracks. Training on softer surfaces including grass or cinder trails is encouraged to allow recovery of the bones and soft tissues between workouts and develop strength within musculotendinous units.

Vitamin D Insufficiency

It is recommended that most athletes receive at least 800–1000 international units (or perhaps as much as 5000 IU) of vitamin D_3 daily. This level of supplementation is safe and has a high therapeutic index. Serum 25(OH)D_3 level is the study of choice for identifying vitamin D deficiency [35]. In those individuals with low vitamin D or low bone mineral density, the therapeutic goal for supplementation should range from at least 50 nmol/L (20 ng/mL) to as high as 90–100 nmol/L (36–40 ng/mL) based on the Food and Nutrition Board recommendations [35]. In general a serum level as the ideal level for the athlete is 40–50 ng/mL. To achieve this goal, as much 50,000 IU per week may be prescribed for individuals with severe hypovitaminosis D. Although higher dietary intake of vitamin D_3 may provide some protective effect against fractures, the exact role of vitamin D in fracture prevention remains controversial.

Recent studies have evaluated the potential association between serum vitamin D_3 levels and stress fractures. A prospective study of Finnish military recruits found that the average serum vitamin D_3 concentration was significantly lower in the group that had sustained a stress fracture [36]. Another randomized, double-blind, placebo-controlled study examined whether calcium and vitamin D_3 intervention could reduce the incidence of stress fractures in female recruits during basic training [36]. This level 1 study suggested that calcium and vitamin D_3 supplementation may have prevented a significant percentage of their recruits from sustaining a stress fracture and led to a significant decrease in morbidity and financial burden [36].

Relative Energy Deficiency in Sport and the Female Athlete Triad

Athletes of both sexes may be at risk for impaired bone health resulting from nutritional and hormonal abnormalities. “RED-S” (relative energy deficiency in sport) is a comprehensive term used to define the pathology secondary to an inadequate caloric intake during athletic training and has largely replaced the traditional description of the female athletic triad of disordered eating, decreased bone mineral density, and amenorrhea [23, 24, 28]. The female athletes at greatest risk for developing RED-S and subsequently bony stress injuries commonly participate in sports emphasizing leanness. These include gymnastics, dance, distance running, cycling, and rowing [37]. Recognizing RED-S is especially important due to the long-term detrimental effects on reproductive and skeletal health [38].

The Male Endurance Athlete Tetrad

Recent literature suggests that male runners may also be predisposed to decreased bone mineral density [39]. This has been shown to be most common in the lumbar spine and radius. The

cause of this decreased density is most likely multifactorial. Tenforde et al. have suggested that hypogonadotropic hypogonadism (characterized in males by low serum testosterone levels with concomitant clinical symptoms, such as low bone mineral density, reduced energy and stamina, oligospermia, and decreased libido) is analogous to the hypothalamic amenorrhea that occurs in women [37]. Decreased energy availability may be the key factor for low bone mineral density, and decreased testosterone levels have been shown to be present in males who participate in prolonged endurance events. To prevent severe or irreversible effects of low bone mineral density, it is necessary to be as diligent in assessing the dietary behaviors of male endurance athletes as well as female athletes.

Pathophysiology

Healthy bone is constantly in balanced homeostasis between microtraumatic crack creation and repair. The key modifiable risk factors in the development of overuse injuries of bone relate to the pre-participation condition of the bone and the frequency, duration, and intensity of the causative activity [40]. Without preconditioning and acclimation to a particular activity, athletes are at significantly increased risk for the development of overuse and fatigue-related injuries of bone [41, 42]. Repeated episodes of bone strain can result in the accumulation of enough microdamage to become a clinically symptomatic stress reaction or stress fracture [16, 18]. Fatigue failure of bone has three stages: crack initiation, crack propagation, and complete fracture [16, 18].

Crack initiation typically occurs at sites of stress concentration during bone loading [18]. Stress concentration occurs at sites of differential bone consistency such as the lacunae or canaliculi [18]. Initiation of the microcrack alone is not sufficient to cause a symptomatic fracture. It is the first step in bone remodeling and may serve to increase bone density and strength. Crack propagation occurs if loading continues at a frequency or intensity above the level at which new bone can be laid down and microcracks repaired. Propagation, or extension of a microcrack, typically occurs along the cement lines of the bone and is considered pathologic [16, 18]. Continued loading and crack propagation allow for the coalescence of multiple cracks to the point of becoming a clinically symptomatic stress fracture [16, 18]. If the loading episodes are not modified or the reparative response is not increased, crack propagation will likely continue until a complete fracture occurs [16, 43].

Clinical Presentation

Pain that is initially present only during activity is common in athletes presenting with a stress fracture. Symptom onset is usually insidious, and typically patients cannot recall a specific injury or trauma to the affected area. If activity level is not decreased or modified, symptoms persist or worsen. Those who continue to train without modification of their activities may develop pain with normal daily activity and potentially sustain a complete fracture.

Physical examination reveals reproducible point tenderness with direct palpation of the affected bone site. Physical examination tests commonly used for assessing for stress fractures include the fulcrum test for the femur and tibia (Fig. 7.2a, b) where a three-point bending moment is applied to a long bone and the single-leg stance and hop tests (Fig. 7.3a, b) for evaluating pelvic and lower extremity stress injuries [21, 44]. The tuning fork test for identifying sites of bone stress fractures has not been shown to be adequately sensitive or specific and should not be relied upon routinely to make the diagnosis [44, 45].

Radiologic Evaluation

Radiographs

Radiographs are most often negative early in the course of a stress fracture but may become positive after 4–6 weeks [46, 47]. As healing progresses, radiographic findings may be subtle and

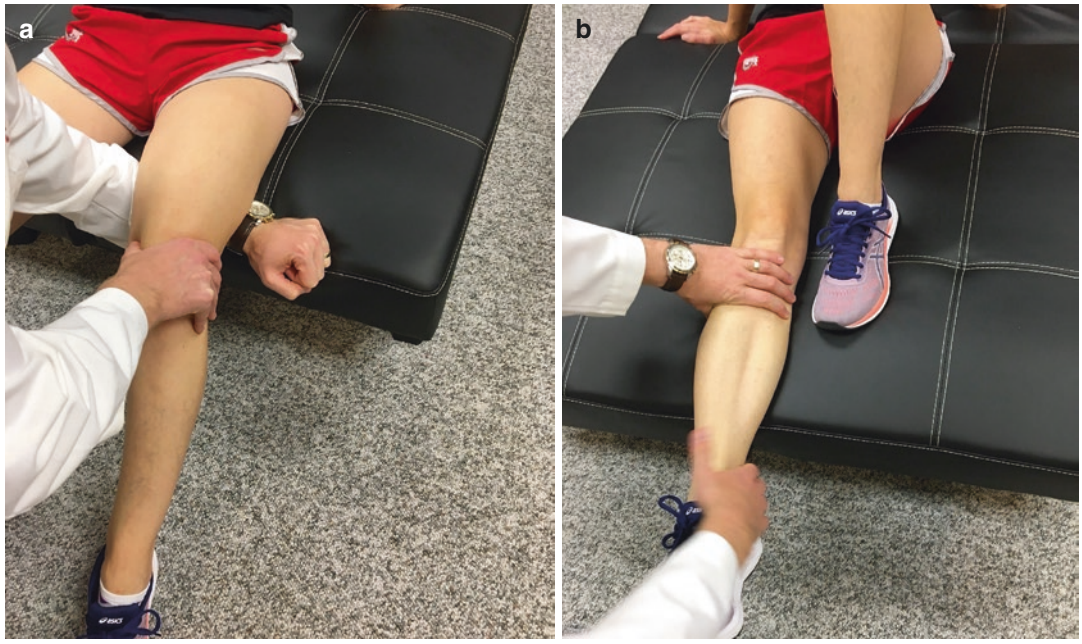

Fig. 7.2 Fulcrum test of the long bones. (**a**) Femoral shaft fulcrum test. (**b**) Tibial shaft fulcrum test

Fig. 7.3 (**a**, **b**) Female runner demonstrates single-leg stance and hop tests

easily overlooked if the images are not thoroughly evaluated. Two-thirds of radiographs are initially normal, but about half ultimately provide evidence of a bony stress injury as healing progresses (Fig. 7.4) [48]. This makes plain radiographs specific but not sensitive for identifying and categorizing stress fractures.

Bone Scintigraphy

Bone scintigraphy has been shown to be nearly 100% sensitive for bony stress injuries, although it has a lower specificity than MRI [49]. It is especially useful for identifying rib, pelvic, femoral, pelvic, tibial, and tarsal stress fractures. Medial tibial stress syndrome has been shown to have a more diffuse distribution along the medial border of the tibia as opposed to a focal "hotspot" indicating a stress fracture [50]. One of the greatest values of bone scintigraphy is its ability to diagnose multiple stress injuries. Bone scans will often demonstrate increased uptake in the affected bone 1–2 weeks before radiographic changes occur (Fig. 7.5). However, given that uptake on bone scan requires 12–18 months to normalize, bone scintigraphy is less helpful for guiding return to sports participation [49].

Computed Tomography (CT Scan)

In the setting of a chronic stress fracture, computed tomography scan is beneficial for demonstrating evidence of healing by clearly showing the presence or absence of a nonunion [51, 52]. CT scan delineates bone well and is of high utility for determining if the fracture is complete or incomplete [51, 52]. It is of particular use in the case of tarsal navicular stress fractures (Fig. 7.6) and for stress injuries of the pars interarticularis [53]. Due to the high-radiation dose given off by CT scan, however, it is not a common first-line option for diagnosing stress fractures but is useful for preoperative planning.

Magnetic Resonance Imaging (MRI)

Currently, magnetic resonance imaging (MRI) is the gold standard for diagnosing and classifying bony stress injuries. It has demonstrated superior

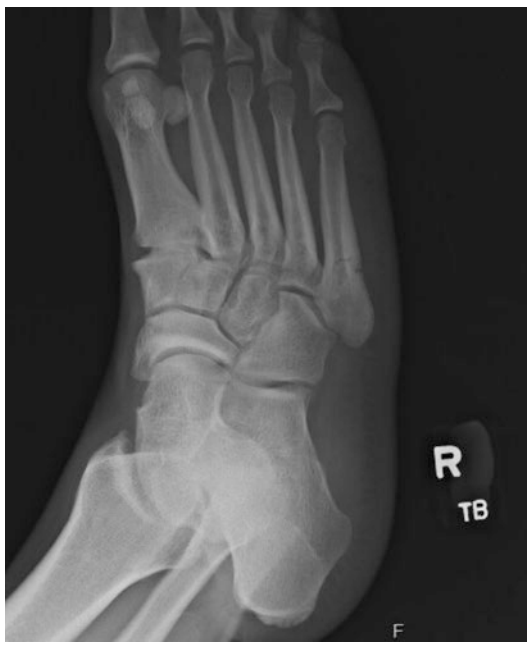

Fig. 7.4 Oblique radiograph of the hip in a 31-year-old female marathon runner with proximal fifth metatarsal acute on chronic stress fracture

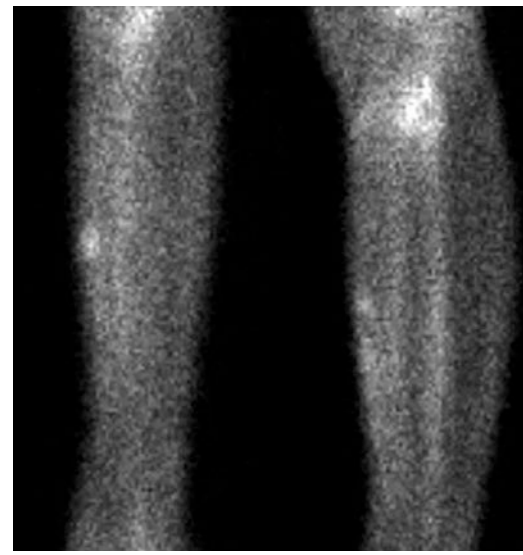

Fig. 7.5 Bone scan of bilateral tibias of a 20-year-old male collegiate distance runner with multiple areas of bony stress injuries of the left tibia and right tibia and fibula

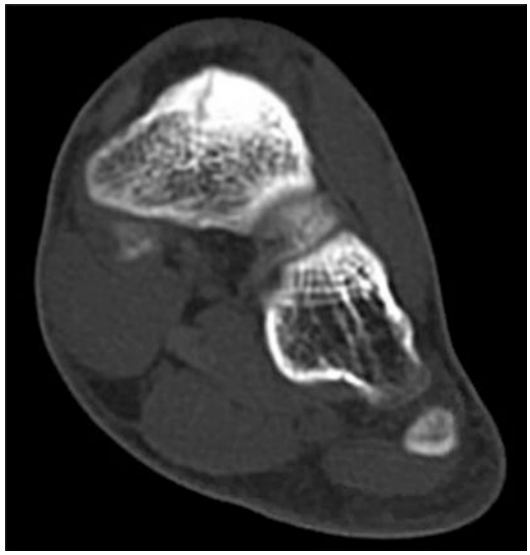

Fig. 7.6 Coronal CT scan demonstrating delayed union of a dorsal navicular stress fracture

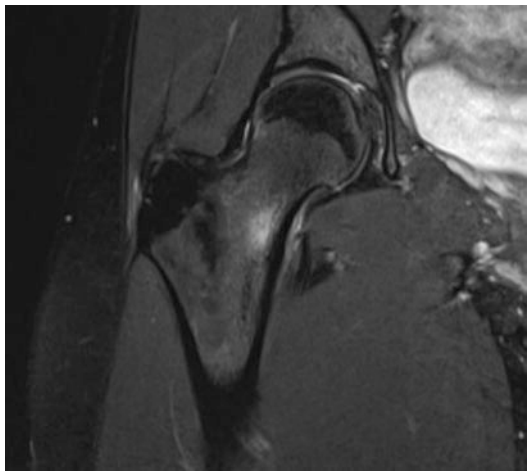

Fig. 7.7 Coronal T2 MRI demonstrating compression side femoral neck stress fracture

sensitivity and specificity to bone scan and CT for associated soft tissue abnormalities and may delineate injury earlier than bone scan [49]. Typical MRI findings on T2 sequences include a band of low signal corresponding to the fracture line, surrounded by diffuse high-signal intensity representing marrow edema (Fig. 7.7) [54]. Its sensitivity is similar to that of a bone scan, and it is much more precise in delineating the anatomic location, acuity, and extent of a bony injury [49]. It has additionally shown prognostic ability regarding time to healing a stress fracture and time to return to sport [26, 27].

Classification/Grading

Stress fractures are categorized in a variety of ways [55]. They are most commonly grouped based on the size of the fracture line, the anatomic site of injury, the biologic healing potential of the injury location, the natural history of the particular fracture, or a combination of these features [17–21, 54]. Multiple authors have advocated classifying stress fractures as either "high risk" or "low risk" [17, 19, 20]. High-risk sites have at least one of the following characteristics: relative avascularity with or without retrograde blood supply and high tensile forces [20, 55]. These characteristics increase the risk of delayed or nonunion, refracture, and significant long-term consequences if a complete fracture occurs including avascular necrosis. In addition to determining the risk level of a stress fracture, the extent of the fatigue failure or "grade" of the injury is necessary to describe and treat it [17, 20, 54, 55].

Risk Assessment

Low-, Intermediate-, and High-Risk Stress Fractures

Low-risk stress fractures include the distal femur, the medial tibial shaft, the ribs, the ulnar shaft, and the first through fourth metatarsals, all of which have a consistent blood supply and favorable natural history. These sites tend to be on the compressive side of the bone and respond well to activity modification and relative rest. Low-risk stress fractures are less likely to recur, develop nonunion, or have a significant complication should they progress to complete fracture [19].

Intermediate risk stress fractures are those that occur near a high-risk site but have a favorable healing potential and biomechanical forces acting on them. Anatomic sites included in this

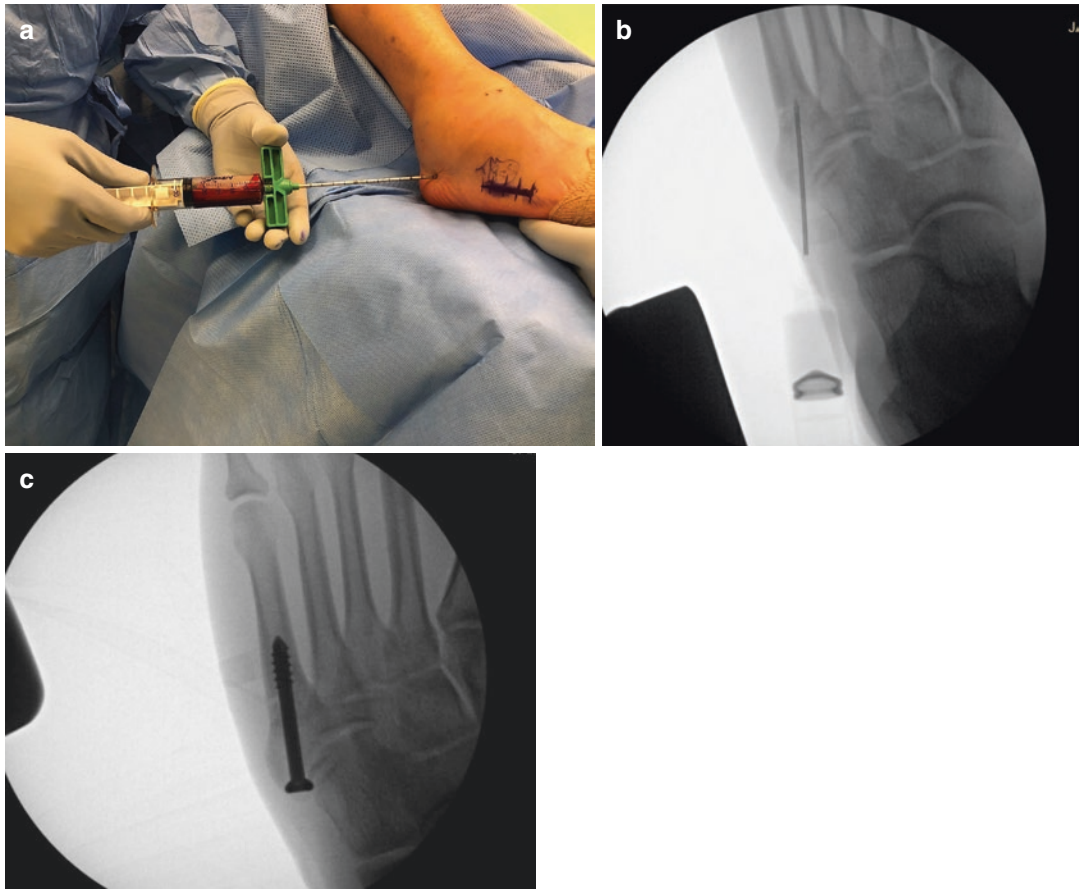

Fig. 7.8 (**a**) Intraoperative photograph of calcaneal bone marrow aspirate harvest. (**b**) Fluoroscopic radiographic image of the right foot undergoing injection of concentrated bone marrow aspirate injection for fifth metatarsal stress fracture. (**c**) Intraoperative fluoroscopy image of a 20-year-old male college soccer player after undergoing internal fixation of a fifth metatarsal stress fracture

group are the inferior surface of the femoral neck, the proximal femoral shaft, the inferior pubic ramus, and the pars intra-articularis. Rarely do these sites require surgical stabilization to allow healing, but given their biomechanical features and proximity to high-risk sites, they have an increased potential to extend into a high-risk site if the fracture propagates.

Table 7.4 presents a list of anatomic locations considered high risk for stress fracture propagation. A delay in treatment for a high-risk site may prolong the patient's period of complete rest and potentially alter the treatment strategy to include surgical fixation with or without bone grafting. Due to their location on the tension side of their respective bones, these fractures possess biomechanical properties that predispose them to propagation of the fracture line. In comparison to low-risk stress fractures, high-risk injuries are not likely to heal without complete rest and surgi-

Table 7.4 Anatomic sites for high-risk stress fractures [20]

Femoral neck (tension side)
Patella (tension side)
Anterior tibial cortex
Medial malleolus
Talar neck
Dorsal tarsal navicular cortex
Fifth metatarsal proximal metaphysis
Sesamoids of great toe

cal stabilization. With less aggressive treatment, high-risk stress fractures tend to progress to nonunion or complete fracture, require operative management, and recur in the same location [16, 44, 56].

Treatment of High-Risk Stress Fractures

Undertreatment of a high-risk stress fracture puts the athlete at risk of a catastrophic fracture and significant long-term complications. The immediate goal of treatment of a high-risk stress fracture is to avoid propagation of the fracture. Typically, this requires either limited resistance, protected weight-bearing, or surgical stabilization. Ideally, while the fracture is healing, a balance is maintained between the athlete maintaining fitness and minimizing the risk of fracture progression.

The presence of a visible fracture line on a plain radiograph in a high-risk stress fracture should prompt serious consideration for surgical stabilization. A low threshold for surgical fixation should be maintained for high-risk stress fractures for several reasons. These include expediting healing, allowing earlier return to activity, and minimizing the risk of delayed union and refracture. In the case of a tension-sided femoral neck stress fracture, urgent surgical intervention may be necessary to prevent a catastrophic fracture [20, 21, 44]. Depending on injury severity, patients with stress injuries in high-risk locations require immediate immobilization and/or restriction from weight-bearing activities with close monitoring. If an incomplete fracture is present on plain radiographs with evidence of fracture on MRI or CT in a high-risk location, immobilization and strict non-weight-bearing is indicated. Worsening symptoms or radiographic evidence of fracture progression despite nonoperative treatment is an indication for operative treatment. All complete fractures at high-risk sites should receive serious consideration for surgical treatment.

Treatment of Low-Risk Stress Fractures

Low-risk stress fractures may be treated most often with relative rest and activity modification. Decision-making should be based in part on symptom severity. Those who experience enough pain to limit function should be treated with relative or complete rest [19, 21]. The decision to continue activity despite the presence of a low-risk stress fracture and titrate the volume of activity to a low but functional pain level can be made after a discussion with the athlete. A key point in this discussion is the possibility of progression to a complete fracture with this approach. If the goal is not to continue activity but to completely heal the fracture, then rest to a pain-free level is required before return to athletic participation. Unless otherwise contraindicated, a patient may be permitted to maintain fitness by cross-training during this time with low-impact alternatives such as cycling, swimming, elliptical, antigravity treadmill, or aquatic running.

Return to Sports Participation

For most athletes, return to sport should only be allowed after proper treatment and complete healing of a stress fracture. Given the heterogeneity of these injuries, however, the time to return is difficult to predict. Multiple recent studies have indicated that the expected time to return to athletic activity following a stress fracture including competitive distance running is 11–14 weeks with female athletes requiring greater time to return to sports [18, 21, 26, 27].

The majority of early stress reactions at high-risk sites heal with nonoperative management [44]. The key difference between a low-grade stress fracture at a high-risk location versus a low-risk location is that with the low-risk site, the athlete may be allowed to continue to train, whereas the high-risk injury requires complete healing prior to return to unrestricted activity [18]. Regardless of the grade and location, the risk of continued participation should be discussed with each athlete, and the management of each fracture should be tailored

to the athlete's goals and risk tolerance. Cross-training while resting from the inciting activity allows maintenance of cardiovascular fitness while decreasing tensile, bending, and rotational forces at the healing fracture site [18, 21, 44]. Return to participation should be a joint decision between the physician, athletic trainer, coach, and athlete.

Biologic Healing Enhancement

Once the risk and severity levels have been determined for a bony stress injury in an athlete, healing optimization must be initiated, and biologic healing enhancement options may be considered. *Osteobiologics* is a term used to describe materials that have been identified and developed to promote bone healing [57]. These options can be divided into direct injectable modalities and indirect systemic stimulating treatments. Many of these options are in the experimental phase, and not every option is ideal for every bone or even each area of the affected bone. Nearly all options may be used in combination or as an adjunct to internal fixation with hardware with each option possessing its own risks and benefits.

Autologous Platelet-Rich Technologies

Platelet-rich blood products are considered to be *osteopromotive* materials when used in bone-healing applications. The efficacy of platelet-rich plasma (PRP) and autologous conditioned plasma (ACP) has demonstrated enhanced bone healing in both animal and human models [57]. Based on current literature, PRP may be most useful for stress fractures when combined with a synthetic osteoconductive scaffold, reducing the need for allogenic products and autogenous bone graft harvest [57]. More comparative studies with standardized protocols for preparation and administration of platelet-rich materials employing randomization and control groups are required to confirm efficacy in human stress fractures.

Concentrated Bone Marrow Aspirate

Concentrated bone marrow aspirate is a biologic treatment that utilizes an individual's own mesenchymal stromal cells to stimulate bone healing. The local application of bone marrow aspirate concentrate (BMAC) for the treatment of delayed healing is an increasingly popular alternative to autogenous bone grafting and may help to reduce donor site morbidity [58–60]. Osteoblastic progenitor cells are available in the bone marrow aspirate of the iliac crest, proximal tibial metaphysis, and calcaneus (Fig. 7.8a–c) with the iliac crest providing the highest yield of osteoblastic progenitor cells for these purposes [61]. A recent review of in vivo studies on the use of BMAC for the treatment of segmental bone defects in animal long bones indicated significantly increased torsional stiffness in BMAC-treated defects and earlier bone healing on histologic evaluation when BMAC was applied [62].

Injectable Bone Graft Substitutes

Injectable bone graft substitutes include a combination of concentrated bone marrow aspirate and demineralized bone matrix injected into a fracture site. The technique employs the osteogenic properties of BMAC combined with the osteoconductive potential of bone matrix to stimulate healing and fracture callus formation and may be used for metaphyseal stress injuries and subchondral insufficiency fracture [63]. An example of this option, Intraosseous BioPlasty™ (Arthrex, Naples, FL), involves percutaneously performing a core decompression of the affected metaphyseal bone site and injecting a mixture of bone marrow concentrate, calcium chloride clot, and demineralized bone matrix (Fig. 7.9a–c) [64]. It is currently used in the distal femur and proximal tibia for subchondral insufficiency fractures as well as the proximal femur at the femoral head and peritrochanteric area [65, 66].

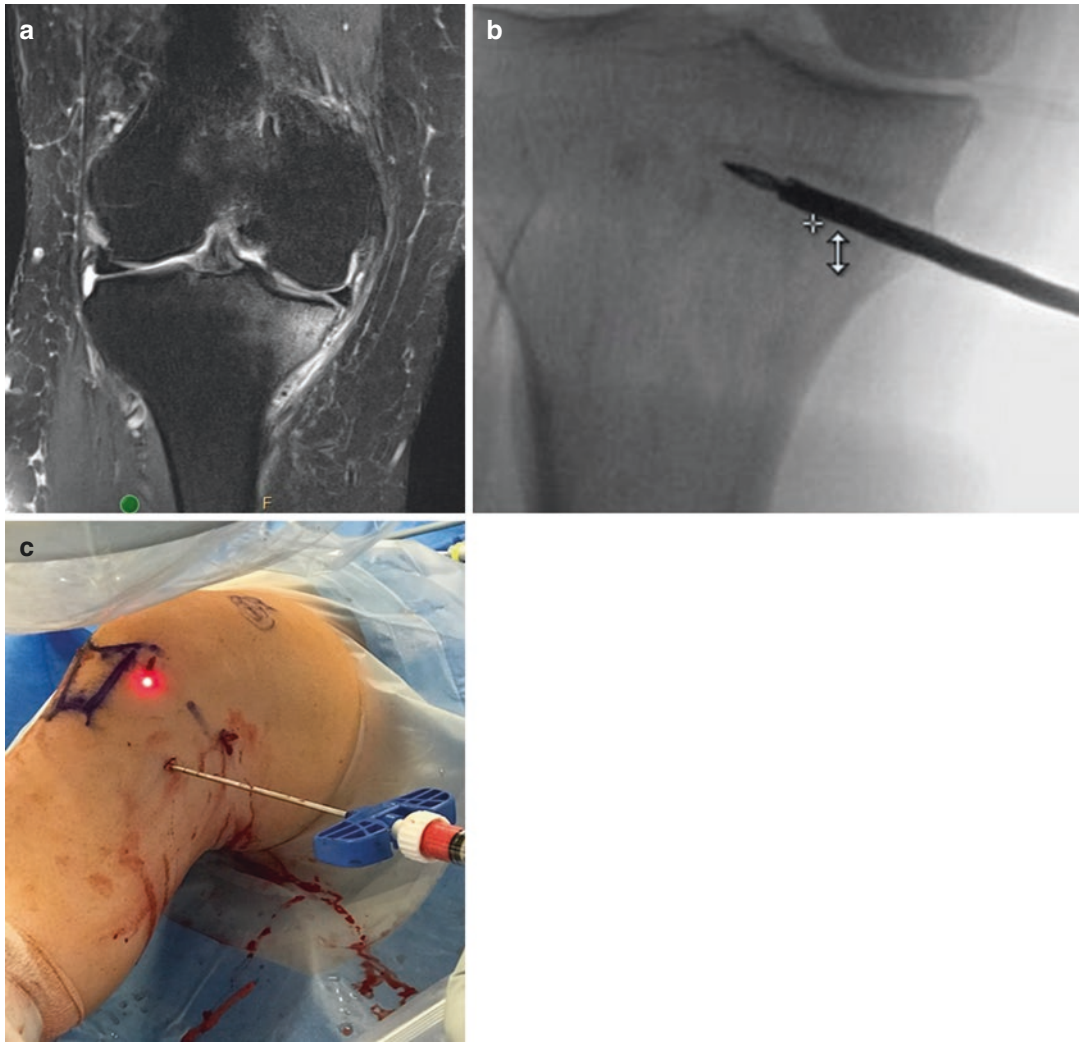

Fig. 7.9 (**a**) T2 coronal MRI demonstrating subchondral insufficiency fracture of the medial tibial plateau. (**b**) Intraoperative fluoroscopic radiograph during intraosseous bioplasty of the medial tibial plateau. (**c**) Intraosseous bioplasty performed at the medial proximal tibia. A mixture of demineralized bone matrix and concentrated bone marrow aspirate is injected after core decompression has been completed

Electrical Osseous Stimulation

Electrical osseous stimulation is one of a number of noninvasive systemic stimulating options for enhancing bone healing. Pulsed electromagnetic fields (PEMF) and low-intensity pulsed ultrasound (LIPUS) are FDA-approved, noninvasive tools that increase the production of regulatory mediators required for physiological bone healing [67]. PEMF creates a magnetic field and a secondary electric impulse activating a series of enzyme reactions that upregulate growth factors. These growth factors include calmodulin, bone morphogenic proteins, and transforming growth factor-β. These factors stimulate osteocyte proliferation and fracture healing. LIPUS appears to have a direct effect on ion channels for stimulating bone cell activity via mechanoreceptors [67]. Bone stimulators have been shown to be the most effective for delayed unions of the tibial shaft and fifth metatarsal shaft [68].

Parathyroid Hormone Stimulation

Pulsed parathyroid hormone (PTH) is an FDA-approved option for the treatment of osteoporosis, though its off-label use has included poorly healing fractures and nonunions. PTH acts within the pathway of bone morphogenic proteins (BMPs). It is a regulator of calcium and phosphate homeostasis and induces an anabolic response in bone when applied at intermittent low doses [69]. Injection is the standard route for administration though topical gel forms can be used locally to increase ease use and patient compliance. The effect of pulsed PTH (teriparatide) on fracture healing has been evaluated with mixed results. Its use has been shown to achieve the primary endpoint of accelerated fracture healing with improved early fracture callus formation compared to placebo [70]. Preclinical animal studies have indicated that supraphysiologic doses of parathyroid hormone can increase fracture site strength and callus quantity along with producing greater mineralization at the fracture site [70, 71].

Summary

Stress fractures are common injuries particularly in endurance athletes and military recruits as well as those who choose single-sport specialization. The diagnosis can be made if a high index of suspicion is maintained and the proper imaging studies are obtained. A holistic approach to treatment should account for nutritional, hormonal, psychologic, and biomechanical factors and requires a team approach to heal preventing recurrence. Treatment should be individualized to the athlete and should consider the injury site (low versus high risk), grade of severity, and timing of the injury within the competitive season. High-risk stress fractures are primarily loaded in tension and/or have relative hypovascularity and commonly require surgical intervention to prevent propagation and displacement. Low-risk fractures are most often those loaded in compression, have a better prognosis, and are unlikely to progress to complete fracture. The recommended treatment of these injuries is based upon injury severity, the biomechanical environment, and general medical risk factors. In addition to the traditional treatment strategies of rest, immobilization, and surgical stabilization, recent modalities for enhancing healing potential may be utilized as adjuncts to help expedite bone healing and promote earlier return to bone homeostasis.

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8 Biomechanics and Stress Fractures: Utility of Running Gait Analysis

Stacey A. Meardon

Introduction

Bone stress injury in active populations creates a challenge to the medical community. First, diagnosis needs to be made, then appropriate treatment needs to be provided, and finally changes need to be made to prevent recurrence. In order to prevent or treat bone stress injury, we need to understand and address the underlying causes. While sometimes the cause of bone stress injury may be obvious, such as a marked increase in training or poor nutrition affecting bone quality, many times a combination of factors interact to increase susceptibility to bone stress injury (Fig. 8.1). The recursive nature of bone stress injury suggests that intrinsic risk factors interact with extrinsic factors to either increase or decrease one's susceptibility for injury. This interaction combined with actual participation in the inciting activity may result in injury [1]. Examination of this pathway reveals potential targets for intervention.

This chapter will focus on the biomechanics of stress fracture and the utility of running gait analysis. Running is fundamental to activities and active lifestyles commonly associated with bone stress injury. During running as well as other physical activities, bone loading occurs when the foot hits the ground and contributes to both bone stress injury (BSI) and bone strength development. Knowledge of factors that influence bone loading during activity will enable the design of interventions to minimize cumulative bone damage and maximize osteogenic potential.

Activity-Related Loading

Activity-related loads and resultant damage formation are key signals for bone modeling and remodeling [2, 3]. Positive bone adaptation (e.g., osteogenesis) occurs in response to dynamic, novel loads of sufficient magnitude with rest between loading bouts [4]. However, negative adaption may occur when repetitive loads and cumulative bouts of physical activity are not balanced with adequate recovery.

During physical activity, bone and joints undergo loading. The forces applied to bone (stresses) cause bone deformation (strain). Stress is defined as the force per unit area and is dependent on the applied force and the area over which force is applied (Eq. 8.1). Strain is defined as the change in length with respect to original length (Eq. 8.2) and is dependent on stress and the material's ability to resist elastic deformation (Eqs. 8.3 and 8.4)

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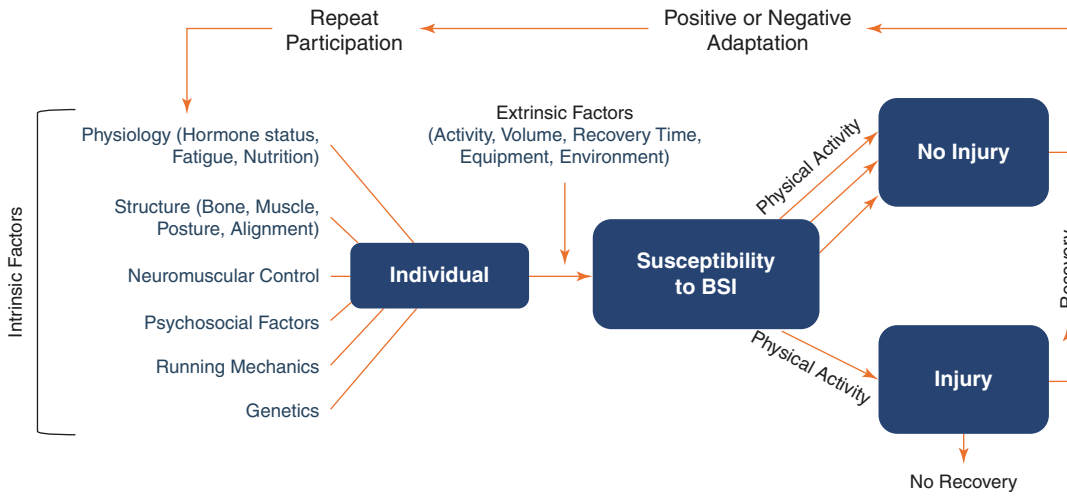


Fig. 8.1 Intrinsic risk factors interact with extrinsic to influence bone stress injury (BSI) risk. (Modified with permission from Meeuwisse et al. [1])

$$\text{Stress} = \frac{\text{Force}}{\text{Area}} = \frac{\text{F}}{\text{A}} \tag{8.1}$$

$$\text{Strain} = \frac{\text{Change in length}}{\text{Original Length}} = \frac{\Delta L}{\text{L}} \tag{8.2}$$

$$\text{Normal strain}(\varepsilon) = \frac{\text{Normal Stress}}{\text{Modulus of Elasticity}} = \frac{\sigma}{\text{E}} \tag{8.3}$$

$$\text{Shear strain}(\tau) = \frac{\text{Shear Stress}}{\text{Modulus of Rigidity}} = \frac{\tau}{\text{G}} \tag{8.4}$$

Normal, or axial, stresses occur when loads act longitudinally to push the bone together (compression) or pull the bone apart (tension) (Fig. 8.2). Tangential forces acting perpendicular to the long axis of bone results in shear stress. In contrast to these uniaxial loads, the forces applied to the human body during real-life activity are often multidimensional resulting in bone undergoing combined loading and varying degrees of bending and torsion. A bending moment (M_b) acting a distance (y) from the neutral axis of bone creates a normal stress (σ) that is proportional to bone's resistance bending (I):

$$\text{Normal stress}(\sigma) = \frac{M_b * y}{I} \tag{8.5}$$

The area moment of inertia (I) depends on the distribution of bone mass relative to the neutral axis making it dependent on the cross-sectional shape of the bone. Torsion creates shear stresses (τ) that are proportional to an applied torque (T) acting about the longitudinal axis at a distance (r) and bone's resistance to torsional loading (J):

$$\text{Shear stress}(\tau) = \frac{T * r}{J} \tag{8.6}$$

The polar moment of inertia (J) reflects the distribution of the mass about the longitudinal axis of bone. Thus, in real-life scenarios applied forces and torques as well as bone ability to resist those forces dictate the loading environment.

Bone strain signals a cascade of events associated with bone modeling and remodeling that can act to maintain bone homeostasis and increase bone geometry and mass [2, 4]. In vivo bone strain during human activities such as walking, running, and jumping is reported to range from −14 to −2104 microstrain (με) for compression, 394–2200 με for tension, and 700–5027 με for shear [5–12]. Given the tensile yield point of bone approximates 6200 and 7300 με for trabecular and cortical bone [13], respectively, a seemingly large safety margin for damage exists. However, high-magnitude repetitive activity-related bone stress and strain

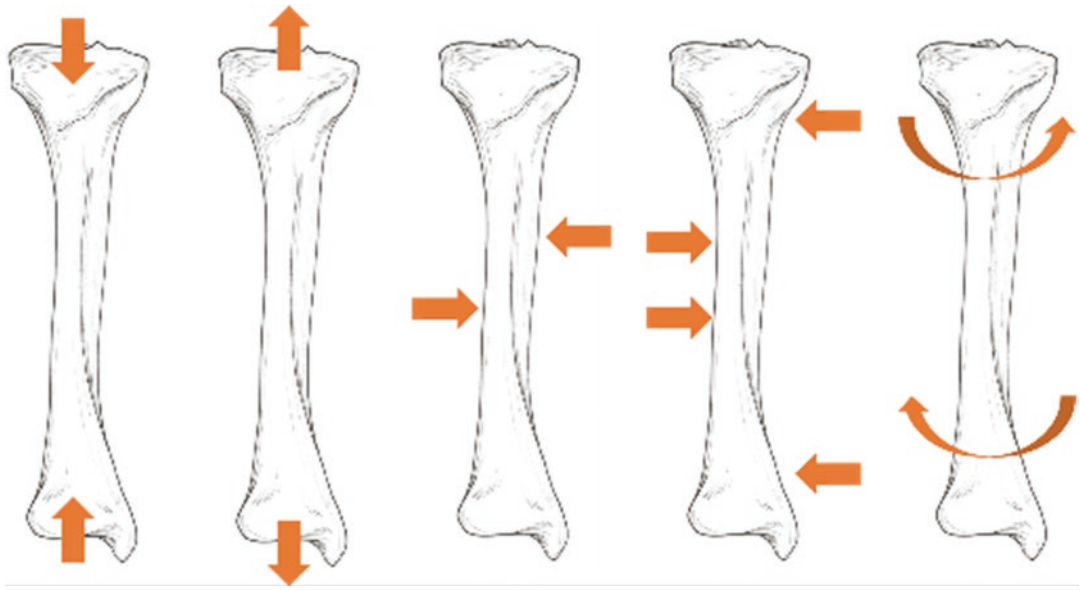


Fig. 8.2 Different types of loading that result in bone deformation. From left to right: compression, tension, shear, bending, and torsion

within bone without adequate rest leads to micro-damage accumulation [6, 7]. An imbalance between damage accumulation and repair can lead to a loss of bone stiffness and strength and ultimately failure [14–16]. A theoretical model illustrating positive and negative adaptation in response to bone loading is presented in Fig. 8.3 [17]. Understanding factors that contribute to high-magnitude bone loading as well as the damage potential associated with cumulative bouts of physical activity will help clinicians, researchers, and individuals susceptible to fracture better prevent and manage bone stress injury.

From a mechanical perspective, bone stress and strain are proximate causes of injury. The ability to quantify these loads is critical to unraveling the complex etiology of BSI. While stress and strain are closely related, fracture of bone is thought to be strain-controlled [18, 19]. Surgical attachment of strain gauges directly to bone [5] is often considered the gold standard measurement of bone loading but is invasive and not feasible on a large-scale basis. Computer-based musculoskeletal models incorporating bone structure derived from bone imaging and applied loads experienced during activity can provide reasonable estimates of bone stress and strain during physical activity (Fig. 8.4).

Models to estimate bone loads range from simplistic, such as modeling the tibia as hollow ellipse [20–22], to computationally complex using subject-specific bone images and finite element analysis [23–25]. Model choice is dependent upon the study question. For larger-scale studies seeking to quantify bone loading in specific regions of the tibia, a cross-sectional subject-specific finite element model combined with three-dimensional forces and moments (due to muscles and ground reaction forces) offers a compromise. This type of model integrates subject-specific bone properties and cross-sectional geometry with traditional musculoskeletal modeling approaches to efficiently estimate bone stress throughout exercise activity, rather than at one point in time [26–28]. Bone stress at common sites of injury has been successfully modeled in young adults using computer-based musculoskeletal models and comprehensively integrates bone structure, muscle forces, and external loads [21, 22, 24, 25, 29–32]. Integrating model-based estimates of bone stress/strain with the measurement of intrinsic and extrinsic factors that influence

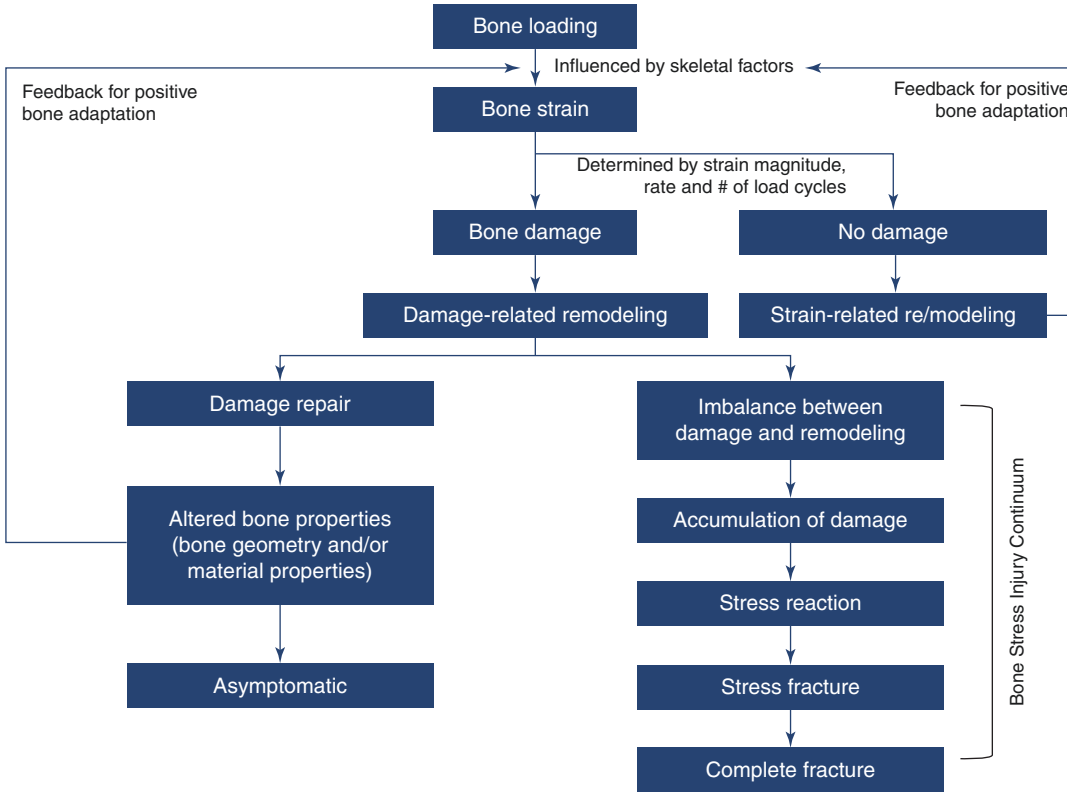


Fig. 8.3 A theoretical model of positive and negative adaptation associated with bone loading. (Modified with permission from warden et al. [17])

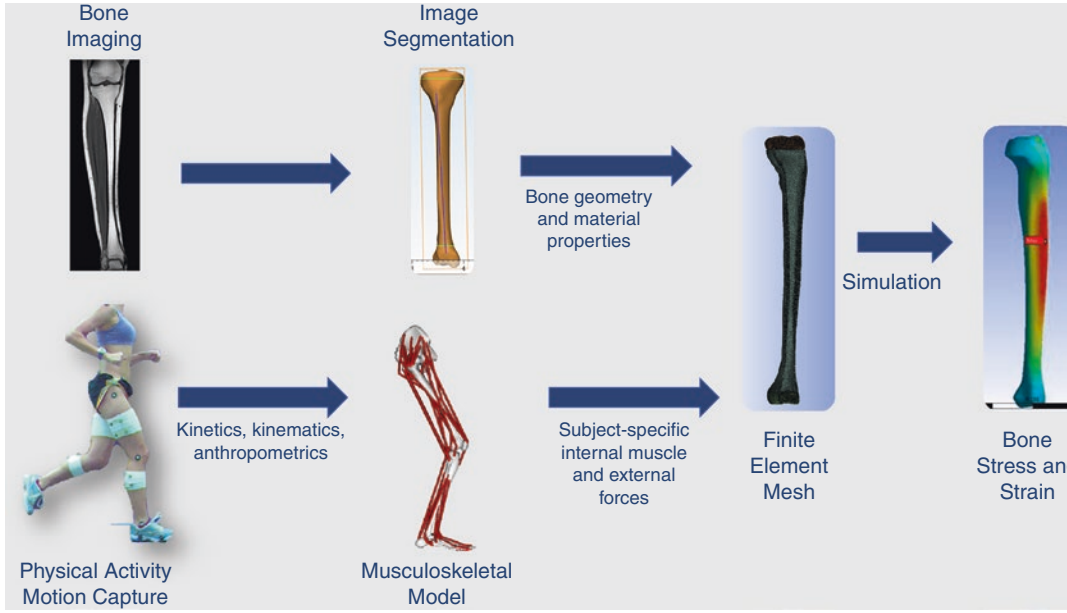


Fig. 8.4 The general workflow to the estimate tibia stress and strain begins with inputs from imaging and 3D motion capture. Data is generally input to a series of models using multiple software programs

injury likelihood has vast potential to advance our approach to managing BSI.

Biomechanical Risk Factors

Individual susceptibility to BSI is postulated to influence response to bone loading [1, 33]. Beyond quantifying tissue load, knowledge of the key factors that may directly or indirectly contribute to high levels of bone stress/strain is essential for injury prevention and recovery. Physical activity characteristics (e.g., magnitude, frequency, duration, and intensity) are necessary factors of BSI development [34, 35]. However, non-training characteristics may act to modify the effect of physical activity on injury risk by augmenting or mediating load magnitude [36]. Factors such as underlying bone structure [37, 38], forces and motions during physical activity [39–44], muscle characteristics [45, 46], fitness and neuromuscular performance [45, 47–49], physical activity history [50], and nutritional status [45] may directly or indirectly influence bone's ability to withstand applied loads.

Given the repetitive nature of sport and exercise, even small increases in loading may be magnified over thousands of consecutive foot strikes. This section will focus on skeletal characteristics and running mechanics associated with stress fracture. Included in the category of gait mechanics are skeletal alignment, foot type, and running kinematic and kinetics. It is important to note that this area is a constantly evolving area of research. Well-designed prospective studies are needed to confirm key factors or a combination of factors that interact to influence bone loading and stress injury risk.

Bone Characteristics

One of the key factors that drive bone's response to loading is its ability to resist applied loads. Underlying bone material properties including density, size, and shape determine the response of bone to loading. Mechanical strength and load capacity are influenced by bone mass and geometry, nutrition, endocrine status, and loading history. Bone mineralization and porosity (apparent density) influence bone's ability to resist deformation, absorb stress, and absorb energy prior to failure. Carter and Hayes (1976) found that the strength of skeletal tissue was approximately equal to the square of the apparent density for a given strain rate [51]. Consequently, a small reduction in bone mineral density can result in a large decrease in strength and contribute to BSI risk. Individuals undergoing large bone strains during exercise may require greater-than-normal bone mass to resist applied loads [52]. Unfortunately, the role of bone mass, as measured by bone mineral density (BMD), in stress fracture is unclear. Studies in athletic populations, including on prospective study, generally report lower BMD in individuals who developed stress fracture [45, 53–55]. However, the effect of BMD on BSI risk in military populations is less consistent [45]. While it is probable that the determinants of BSI differs in these populations [56], cumulative BSI scores that include BMD as a risk factor predict injury better than BMD alone [55, 57, 58]. This suggests that the importance of bone mass on injury risk is likely dependent on the presence of other determinants such as energy availability, disordered eating, hormonal status, body mass index, and prior injury.

Small differences in bone mass can strengthen bone by altering bone shape and size. Increasing bone strength through targeted exercises in animals increases the maximum force that bone can support before failure by 64% and increases energy to failure of 94%; underlying these changes were modest gains in bone mass (5.4–5.9%) and structural changes that corresponded to a 13–31% greater area moment of inertia [59]. Examination of simplified stress and strain equations (Eqs. 8.1, 8.2, 8.3, 8.4, 8.5, and 8.6) highlights the importance of both the geometry and material properties of bone. Bone geometry varies significantly across individuals (Fig. 8.5) and should be considered in the examination of risk factors for BSI. Individuals with smaller bone geometric characteristics will most likely undergo greater bone loading when faced with similar or more demanding loading conditions

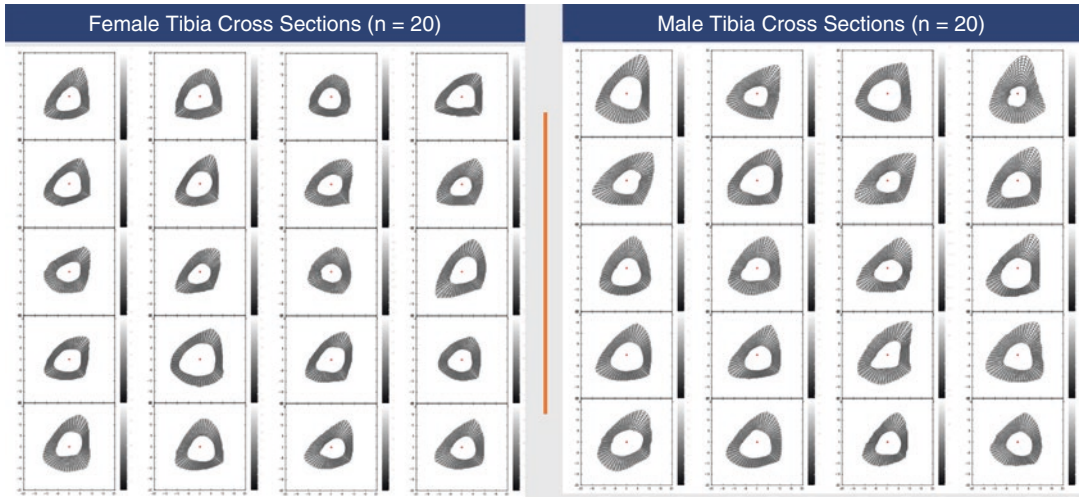
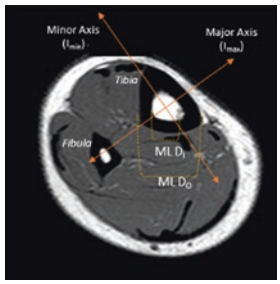


Fig. 8.5 Cross-sectional finite element meshes created from magnetic resonance imaging of the distal 1/3 tibia of 40 healthy recreational runners



Structural Characteristic	Definition
Cortical Cross-sectional Area (CSA)	Area of bone bounded by inner and outer perimeters of the cortex
Inner Diameter (D_i)	Distance between inner borders of the cortex in plane of interest
Outer Diameter (D_o)	Distance between outer borders of the cortex in plane of interest
Area Moment of Inertia (I_{min})	Extent to which bone is distributed away from the minor axis
Area Moment of Inertia (I_{max})	Extent to which bone is distributed away from the major axis
Section Modulus (Z)	$Z = I_{max}/r$ where is the distance from the centroid to the most radial fiber of the cortex

Fig. 8.6 (left) A transverse cross-section of the tibia taken from an MR image slice corresponding to the distal 1/3 tibia. (right) Structural characteristics reported to be associated with stress fracture risk and definitions. ML Medial–lateral

than individuals with larger bone geometry. Smaller bone geometry has been linked with a BSI in epidemiological literature, including prospective studies and one systematic review. Individuals with stress fracture are reported to display smaller cross-sectional area, area moment of inertia, inner and outer diameters, and a lower section modulus (proportional to the bone's resistance to the area moment of inertia and the radius of the bone) [37, 38, 45, 46, 60, 61] (Fig. 8.6).

Advances of imaging technology and software (e.g., magnetic resonance imaging, computerized tomography (CT), peripheral quantitative CT, cone beam CT) allow for the estimation of bone strength that could inform risk profiles. However, these image types may not be widely accessible and can be costly. Consequently, clinical correlates of bone strength may be worth considering in the history-taking and physical examination of individuals with BSI. In addition to the factors related to bone mass mentioned above, bone strength has been positively related to muscle volume [46], maximal vertical jump [47], and biopositive bone-loading history [62]. Along these lines, muscle girth, fitness, and a history of ball sports are reported to be protective against BSI [45, 63, 64].

Skeletal Alignment

Skeletal malalignment may predispose active populations engaged in running and high-impact activities to musculoskeletal overuse injury (Fig. 8.7). Lower-extremity malalignment has the potential to

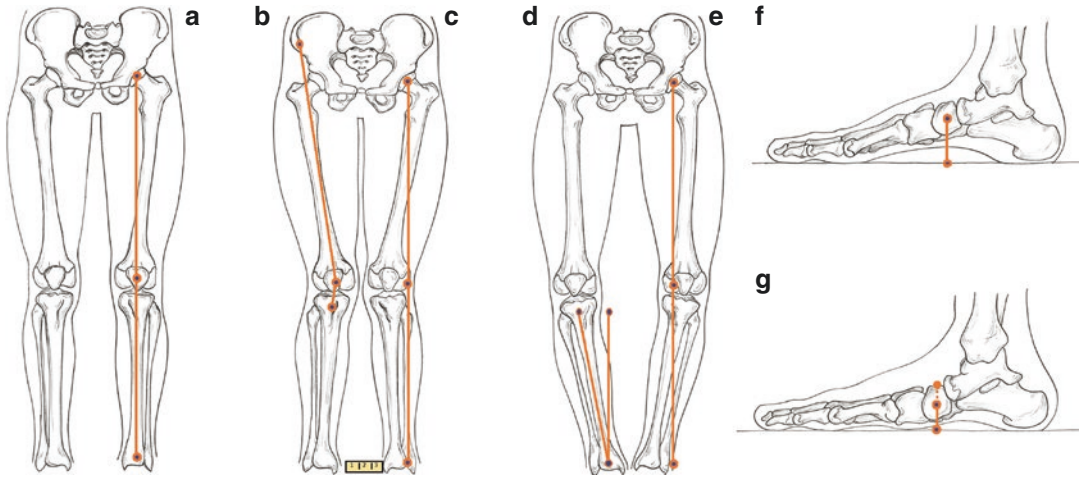


Fig. 8.7 Skeletal alignment to consider in the evaluation of bone stress injury. From left to right: (**a**) neutral alignment, (**b**) quadriceps (q-angle), (**c**) genu valgus, (**d**) tibial varum, (**e**) genu varus, and (**f**–**g**) navicular drop

increase bending moments and alter shock attenuation during high-impact activities, predisposing an individual to injury by creating areas of stress concentration in bone. However, strong evidence consistently linking alignment to BSI is lacking. In a study of 320 athletes with stress fracture, varus alignment of the knee, tibia, subtalar, and forefoot static alignments were associated with BSI [65]. Quadriceps angle (q-angle), tibial varum, and leg length discrepancy have also been linked with BSI, but not consistently [66–71]. A Q-angle greater than 15° has been associated with greater odds of stress fracture and shin injury development in high school cross-country runners and military recruits [72, 73]. However, smaller q-angles have also been associated with BSI [74]. Several researchers have found correlations between odds of stress fracture and the development of shin pain and leg length, with >0.5–1.5 cm difference increasing risk of injury [52, 75, 76]. Arch height and mobility have also been associated with stress fracture risk. Low-arch height has been found to be protective for overall stress fracture in a prospective study of military recruits [77]. However, in another prospective study of 295 recruits, low-arch height was associated with metatarsal stress fractures, and high-arch height was associated with femoral and tibial stress fracture [78]. Studies of the relationship between foot type and stress fracture in civilian populations are also contradictory. In a prospective study of track and field athletes, no relationship between foot type and stress fracture risk was found [52]. But, in a study of 320 athletes, a pronated foot type was associated with tibial and tarsal stress fractures, and a high-arched foot type was associated with metatarsal and femoral stress fractures [65]. Recently foot mobility, in particular a positive navicular drop test >5 cm (Fig. 8.7), has been linked with BSI [66, 79, 80]. The navicular drop test measures the difference between navicular height in sitting and standing subtalar neutral, with a greater difference indicating a more mobile arch [81].

Evaluation of the body of literature in this area suggests that the contribution of skeletal malalignment to BSI risk is an evolving area of scientific research. It is possible that extremes in skeletal alignment magnify tissue loads in dynamic situations. Until the role of skeletal alignment in injury risk is clarified, skeletal alignment should be examined and considered in the examination process with training modification and biomechanical intervention implemented as appropriate.

Movement Patterns

While static measures may predispose an individual to stress injury, some researchers argue

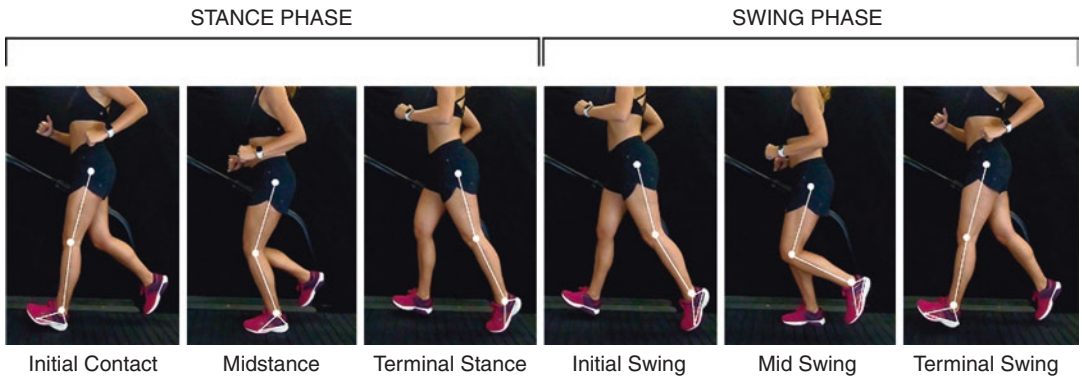


Fig. 8.8 The running gait cycle

that dynamic alignment is more relevant to injury. Three-dimensional video analysis is used in biomechanics laboratories to quantify movement patterns (i.e., kinematics) during physical activities and to gain insight into injurious mechanics. Running, a motor skill that is inherent to active lifestyle, sport, and military performance, is most commonly studied with respect to bone stress injury. Like walking, the running stride is characterized by a swing and stance phase. However, unlike walking, the swing and stance phase are separated by a period of no ground contact (i.e., flight) (Fig. 8.8). The stance phase is typically characterized by two phases: absorption occurring from initial contact to midstance and propulsion occurring from midstance to terminal stance. Compared to walking, running generally requires greater range of motion and greater dynamic postural control. Initial contact following the flight phase of running is characterized by a single leg-landing requiring the runner to attenuate a high magnitude of force. The runner is also required to control forward motion of the body over a changing base of support while maintaining upright posture and lower-extremity alignment. These requirements are further challenged by the inherent narrow base of support associated with running. Inability to control the body, either temporally or spatially, could result in the application of atypical load magnitude and distribution to the skeleton.

Typical three-dimensional running mechanics during overground running in rearfoot strikers are displayed in Figs. 8.9 and 8.10. Studies examining running mechanics associated with overuse injury are abundant; however, studies of BSI mechanics are less copious [82]. Research specific to BSI have predominantly studied individuals with tibia and metatarsal stress fracture. Individuals with BSI are reported to display greater hip adduction, knee abduction, knee internal rotation, and greater eversion than uninjured counterparts [41, 83–85] as well as both greater dynamic pes planus and pes cavus [86–89]. BSI is also associated with less knee flexion [90] and a greater frontal plane lower-leg angle with respect to the ground reaction force vector during running [43]. In addition to peak angular changes, individuals with BSI are reported to display altered timing with shorter times to maximum pronation [91]. While it is doubtful that one specific motion is causative in nature, it is probable that a combination of movement patterns act in concert to contribute to an elevated loading environment and, when combined with other determinants, increase risk for BSI [41, 88].

Load Characteristics

Mechanical loads due to external impact forces and muscle forces cause bone deformation during physical activity. However, direct measurement of bone strain is often not feasible or practical due to methodological constraints and ethical issues in most countries. Thus, surrogate kinetic measures from force platform data and accelerometry have been studied in relation to

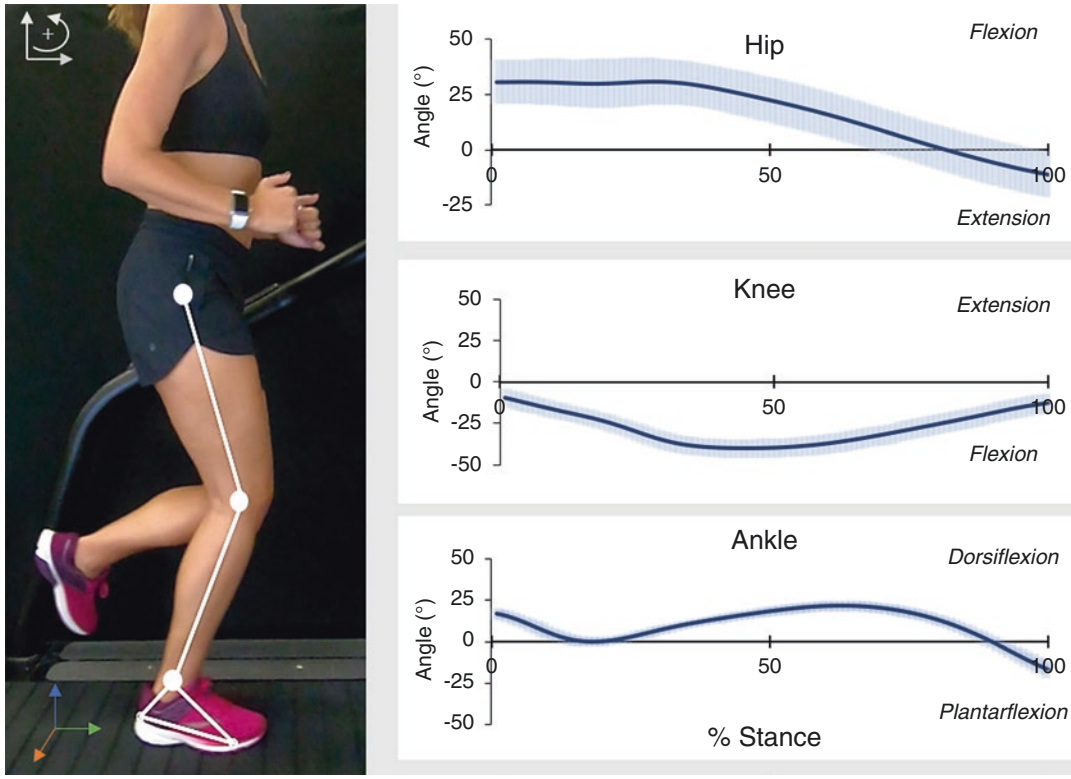


Fig. 8.9 Average sagittal plane three-dimensional running kinematics across the stance phase. Data taken from sample of 40 young healthy adults running overground at 3.5 m/s. Angles were derived using a coordinate system in which lateral, anterior, and vertical are positive and expressed as distal segment moving relative to the proximal segment

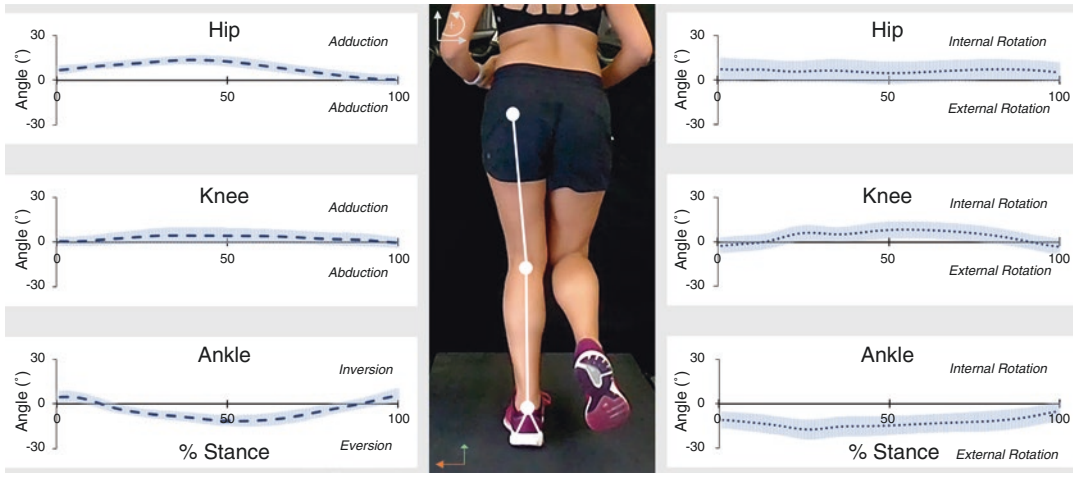


Fig. 8.10 Average frontal (left) and transverse (right) plane three-dimensional running kinematics across the stance phase. Data taken from sample of 40 young healthy adults running overground at 3.5 m/s. Angles were derived using a coordinate system in which lateral, anterior, and vertical are positive and expressed as distal segment moving relative to the proximal segment

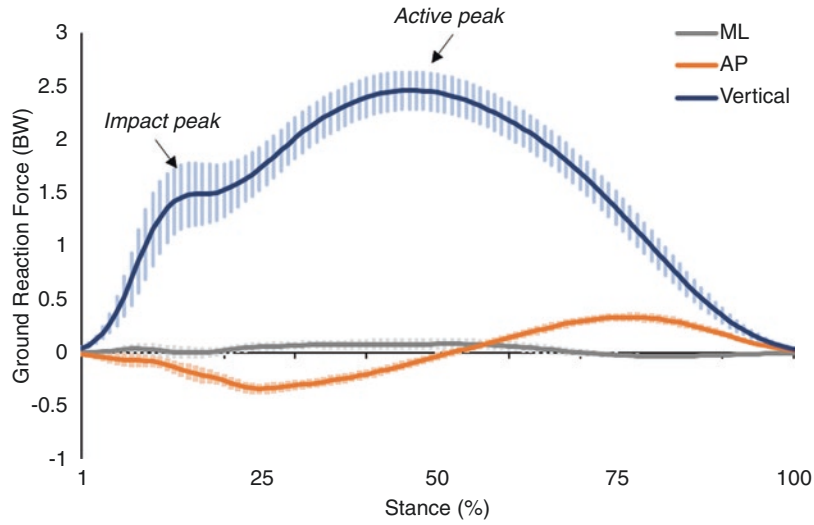


Fig. 8.11 Three-dimensional ground reaction during overground running at 3.5 m/s ($N = 40$ healthy rearfoot striking adults). AP anterior–posterior, ML medial–lateral

stress fracture. This area of research has been insightful with some evidence suggesting that the occurrence of stress fracture is associated with greater external loads.

In running, ground reaction forces (GRF), as measured by a force platform embedded in the floor or an instrumented treadmill, are typically 2.5–3 × body weight in the vertical direction [92, 93] (Fig. 8.11). The GRF provides a measure of the magnitude and rate of external loads acting on the center of mass during physical activity and an indirect estimate on lower-extremity loading. Horizontal anterior–posterior (i.e., braking) forces are reported to be higher in stress fracture groups in some [94, 95] but not all studies [84, 94, 96–98]. However, a meta-analysis and systematic review results suggest that runners with and without stress fracture do not differ in impact or peak vertical ground reaction force magnitude [42, 99]. The free moment, a moment of force about a vertical axis, is the amount of torque occurring due to friction between the foot and the ground during the stance phase of motion. Studies of BSI have used the free moment as an indicator of torsional load acting on the lower leg. Two studies in female runners observed a greater free moment in individuals with stress fracture [41, 100], but a study of male military personal found no group differences [43].

Related to external forces is the rate of loading. The vertical ground reaction force profile (Fig. 8.11) is often characterized by two peaks, an impact peak and an active peak. The high-frequency impact peak is associated with deceleration of the contact limb, whereas the lower-frequency active peak is associated with the deceleration of the body and occurs when the COM reaches its lowest vertical position during stance [101]. From initial contact to the impact peak, a rapid increase in ground reaction force is commonly observed in rearfoot strikers and is reported to be associated with injury but inconsistently so [102]. A meta-analysis and a systematic review indicate that, based on the existing body of literature, instantaneous vertical ground reaction force load rates tend to be higher in individuals with stress fracture [42, 99]. However, a recent study found that the impact peak loading profiles have little effect on the mechanical fatigue of bone when compared to the active or raw vertical ground reaction force profile [103]. Fatigue testing of cortical bone using active peak and raw vertical ground reaction force load profiles resulted in a 5–7× shorter fatigue life when compared to high impact load profiles alone. Thus, the precise nature in which loading rates contribute to injury is unclear. While it has been postulated that adopting forefoot strike would ameliorate this potentially detrimental rapid increase in load, Gruber and colleagues illustrated the presence of impact peaks in both rearfoot and forefoot strikers using wavelet analysis.

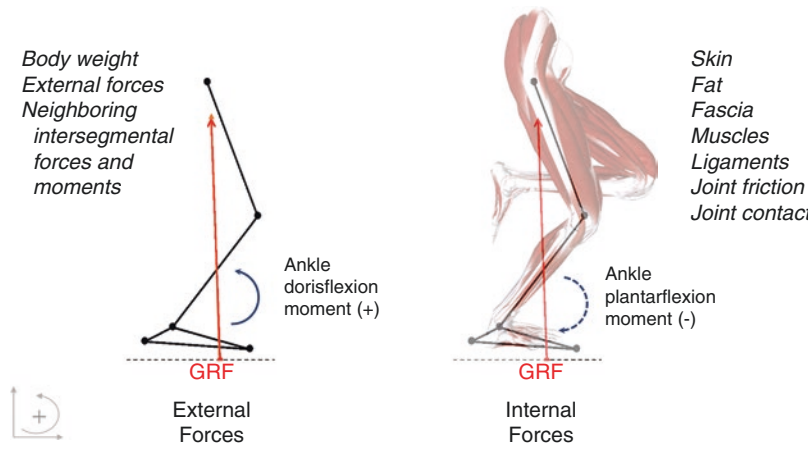


Fig. 8.12 Types of forces and contributing factors acting on the lower leg during human movement and physical activity

Their analysis suggested that forefoot running is associated with a high-frequency impact peak that occurs later in the support period [104]. Thus, adoption of a forefoot strike does not fully eliminate the impact peak.

External loads, such as the ground reaction force, are only a portion of the loading environment (Fig. 8.12). Using computational modeling, Winter et al. found that internal forces at common running injury sites ranged from 6 to 14 BW for compression and 0.4 to 0.7 BW for shear [105]. This suggests that in running, internal forces due to skin, fat, fascia, muscles, ligaments, friction, and joint contact comprise the majority of the loading environment. In vivo bone strain studies provide a criterion measure of bone loading that accounts for both internal and external forces. Human studies using bone strain gauges surgically mounted on bone suggest that injury potential increases under loaded conditions, higher gait speeds, and during uphill, downhill, and zigzag running [5]. Bone strain is also reported to be greater with fatigue [6, 7] and in overground running compared to treadmill running [10]. Unfortunately, the sample sizes in these studies are generally small and primarily male, limiting generalizability.

In recent years, researchers have estimated internal loads through musculoskeletal modeling. Given the challenge of prospective study, estimates of bone stress and strain can provide insight to factors that may influence injury likelihood. Using a combination of subject-specific imaging and motion capture, Meardon and Derrick (2014) found that runners with lower-leg stress fracture display greater tibia stress than their noninjured counterparts. Furthermore, a combination of tibia geometry and the sagittal plane moment acting on tibia predicted bone stress at common injury sites [22]. Peak bone stress is reported to occur during the midstance phase of running [20–22], suggesting that midstance mechanics may be most relevant to injury etiology. In a series of two studies, Edwards and colleagues examined the effect of step length, running mileage, and running speed on the probability of tibia stress fracture [29, 106]. They found that running with a longer step length and faster running speed increased tibia stress fracture probability 6–10%. Examining the influence of step width, Meardon and Derrick reported that a crossover running style increases tibia stress and wider step widths generally reduce tibia stresses [21]. In agreement with in vivo bone strain data, Rice and colleagues report greater bone stress in fatiguing conditions [20]. Contrary to studies of tibia stress fracture, step length modification does not influence the probability of metatarsal stress fracture [24]. However, metatarsal fracture probability was greater in minimalistic shoes compared to traditional cushioned running shoes [24]. In summary, data from modeling studies suggest that bone geometry, bending moments, midstance mechanics, step length, running mileage, running speed, step width, fatigue, and footwear influence injury risk.

Treatment Implications and Utility of Running Analysis

Bone Strength

Based on current knowledge of biomechanical factors associated with BSI, interventions from a biomechanical perspective should focus on improving bone strength and correcting faulty mechanics contributing to a high-magnitude bone strain. Energy availability, nutrition, and hormonal status should be addressed to optimize bone strength. Bone strength can also be improved with short bouts of high-intensity weight bearing or weight-loading exercise separated by periods of rest [2, 4]. High-intensity weight bearing or loading activities include activities such as plyometrics and weight training. Current guidelines suggest that children and adolescents should perform bone-strengthening exercise (e.g., gymnastics, plyometrics, and jumping, moderate-intensity resistance training; sports that involve running and jumping) 3× per week for 10–20 minutes [107, 108]. Bone-strengthening activities can also benefit adults. Current recommendations suggest that adults should participate in a combination of weight-bearing endurance activities 3–5×/week and activities that involve jumping and resistance exercise that targets all major muscle groups >2 days per week [108].

Load Progression

It is important to remember that while high-intensity impact loading can be beneficial to bone, it can contribute to injury if not safely dosed. Unfortunately, the window for optimizing bone health with injury remains elusive. Osteogenic potential of bone saturates after 20–50 load cycles [2]; however, the point at which loading becomes detrimental is not well defined. Gradual load progression with rest and recovery between exercise bouts is recommended to ensure adequate bone adaptation. Research from our laboratory suggests that peak distal tibia forces increase somewhat predictably across a continuum of sport activities (Fig. 8.13). This data suggests that

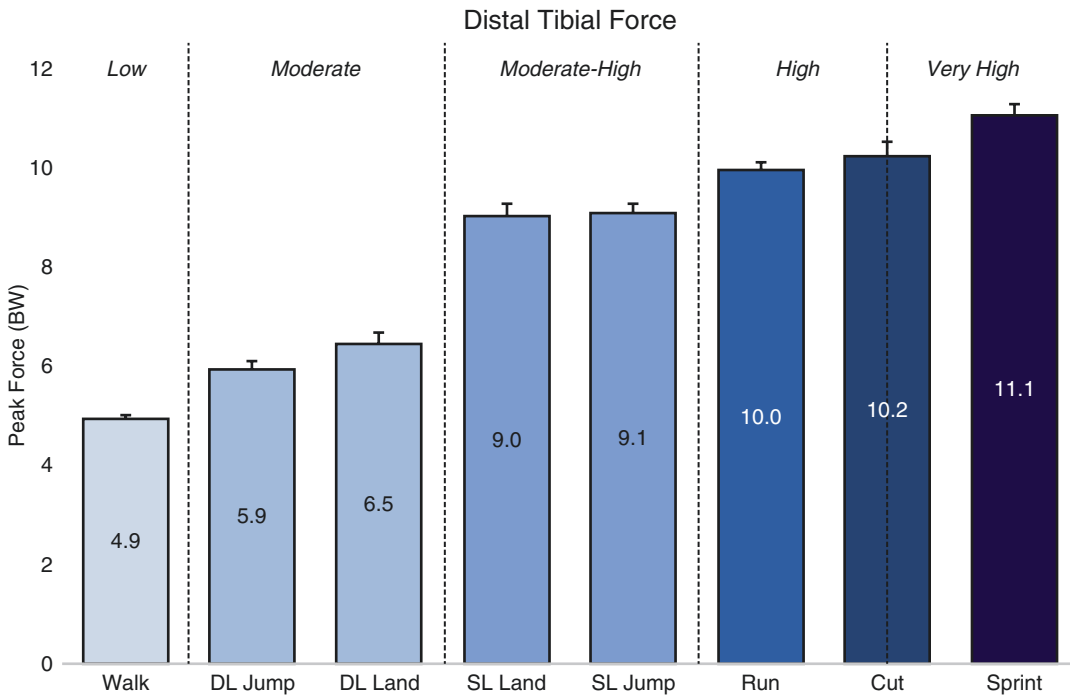


Fig. 8.13 Average distal tibia contact forces during a continuum of sport-related task (N = 40 healthy young adult male) and females

submaximal double- and single-leg jump and land activities could be incorporated into to training programs in order to condition lower-leg bones prior to initiating running and cutting activities commonly associated with BSI. Additionally, running volume and intensity should be titrated to enable adequate adaption of bone. Various programs have been developed for load progression when beginning an exercise program as well as following injury [109]. However, consensus on the best approach is lacking.

Acute to chronic workload ratios provide a metric reflecting acute training loads relative to chronic workload and can be beneficial to monitoring training workload [35, 110]. They provide insight related to preparedness for current activity. Acute to chronic workloads exceeding 1.3 have been linked with injury, and acute to chronic workloads exceeding 1.5 may represent high risk of injury [35, 110]. Acute workload is typically calculated over 1 week and can incorporate both intensity and duration or load cycles. Chronic workloads are generally calculated over 4 weeks. Additionally, given the natural time lag between osteoclastic and osteoblastic activity during bone remodeling and adaptation, a periodized approach to establishing workload could be beneficial [111]. Metrics of load intensity and number of load cycles should be incorporated in load progression guidelines. While laboratory-based measures may be prohibitive, today's wearable devices can provide inference on biomechanical patterns and training load. Real-time feedback from such devices offers a timely and cost-effective way in which to monitor load progression and adherence in the real world [112].

Faulty Running Mechanics Evaluation and Intervention

Despite the limited evidence supporting running biomechanics as a direct causal factor of injury, experts recommend running gait retraining for some lower-limb injuries, including tibial BSI [113]. A number of running-related biomechanical variables discussed earlier in this chapter (step rate, step width, hip adduction, strike pattern, and ground reaction force loading variables) can be changed with gait retraining [114]. Studies reporting altered tissue stress and strain in response to gait modification provide biomechanical rationale and support for gait retraining, although long-term efficacy studies in injured populations are lacking. Moreover, caution should be used when implementing gait modification programs; acute gait modification has been reported to shift loads to other joints [115].

Advanced research methods, such as an instrumented treadmill and 3D motion analysis systems, are commonly used to quantify kinematics associated with BSI. However, faulty biomechanics may be inferred from 2D video analysis. Along with a thorough history and physical examination, clinical running analysis can be an integral part of the BSI evaluation process. In the clinic, video-based 2D analysis while running on a treadmill is more practical than overground running analysis and can provide insight into overground running biomechanics. Souza (2016) and Pipken et al. (2016) provide comprehensive overviews of running gait analysis [116, 117]. Key factors to consider when performing clinical running gait analysis include speed, camera setting, camera position, and anatomical markers:

- Gait analysis should be performed at speeds and under conditions that provoke symptoms in order to understand mechanics associated with injury. Gait velocity can affect lower-extremity kinematics and should be held constant across analysis time points.
- Ideal camera settings (resolution, shutter speed, and capture rate) with adequate lighting are instrumental to good video-based movement analysis. Cameras with 1080 pi and high frame rates (≥120 frames per second) provide good images for evaluation of running kinematics.
- Video should be taken from at least two orthogonal views, posterior and lateral, with zoomed-in views of the foot and ankle to aid in interpretation. Some clinicians may include an anterior view as well.
- Because reliability of gait analysis using a single camera video varies [117, 118], efforts need to be made to ensure reproducibility. These include consistent camera position and angle, use of markers for anatomical landmarks, and

consistent raters. Additionally, examination of multiple footsteps is recommended. Adequate training and use of post-processing software for angular analysis improve reliability [118]. Identifying the following landmarks with markers prior to analysis can be helpful: seventh cervical vertebrae spinous process, anterior and posterior superior iliac spines, lateral knee joint line, lateral malleolus, top and bottom of the heel counter, and the fifth metatarsal.

- Patients should don tight-fitting clothing to ensure visibility of body segments.
- Video analysis should follow a 6–10-minute acclimation period.

Once video is obtained, whole body and lower-extremity segment and joint motions should be systematically analyzed (e.g., distal-to-proximal approach) in both real time and slow motion. Freeware can be used to measure angles and distances, although frame-by-frame analysis at key points in the gait cycle may be sufficient for detection of faulty mechanics [117]. Pipken et al. (2016) provide some general ranges of typical motion that can be beneficial in identifying faulty mechanics [117]. However, key cut points for injurious running mechanics do not yet exist. Furthermore, gait mechanics in isolation may be benign. Findings need to be examined within the context of the complete gait analysis, history, and physical examination. Presented below are sagittal and frontal plane mechanics associated with injury that are easily attainable from 2D video. When appropriate, typical ranges are provided.

2D Running Gait Analysis Lateral View (Fig. 8.14)

Foot Inclination Angle The angle of the foot relative to the ground at initial contact is considered the foot inclination angle. In rearfoot strik-

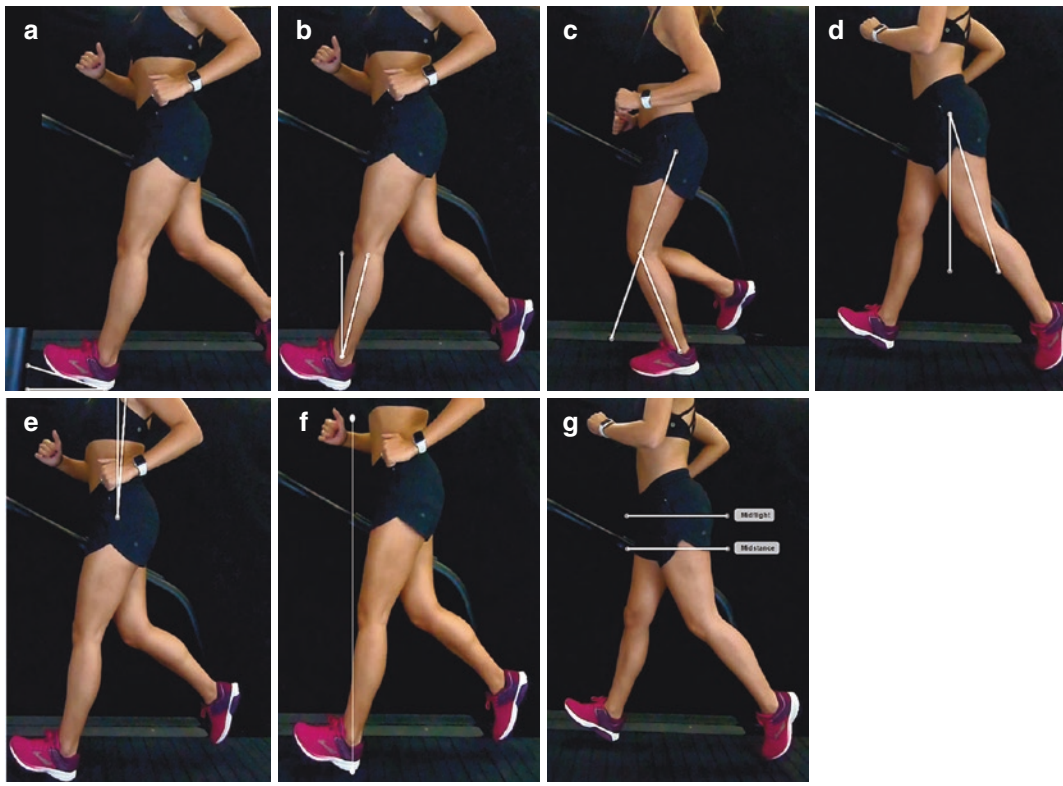


Fig. 8.14 Lateral view bone stress injury-related metrics obtained from 2D running analysis: (**a**) foot inclination angle at initial contact, (**b**) tibia inclination angle at initial contact, (**c**) knee flexion in midstance, (**d**) hip extension in terminal stance, (**e**) trunk lean (slight posterior displayed here), (**f**) overreaching assessed by lateral malleolar position relative to pelvis (anterior to the pelvis may indicate overreaching), and (**g**) vertical oscillation

ers, this angle is positive and is typically associated to ankle dorsiflexion. Higher foot inclination angles are associated with greater peak vertical ground reaction force and braking impulse [119].

Tibia Inclination Angle The angle of the tibia relative to true vertical at initial contact represents the tibia inclination angle. Within 5° of vertical is typical [117] and allows for attenuation of impact forces through knee flexion. An extended tibia during at initial contact is often seen in combination with a high foot inclination angle and overstriding. An extended tibia is evident when the knee marker is posterior to the lateral malleolar marker.

Knee Flexion Peak knee flexion during stance is associated with knee stiffness and may be an indicator of shock absorption [100]. Typical knee flexion angle in midstance values range from 40° to 45° [116, 117].

Hip Extension Reduced hip extension in terminal stance is a common observation. Typical hip extension measured three-dimensionally averages ~10–15°. Reduced hip extension during gait analysis could be secondary to tight hip musculature, but it can also be a consequence of running form. For example, a runner with a short stride and high cadence may display reduced hip extension. Regarding BSI, compensations associated with reduced hip extension in terminal stance may be of greater concern than the absolute degree of hip extension. Compensations can include increased lumbar extension, bounding (which can increase contact velocity and impact forces), overstriding, and a higher-than-expected cadence (which may increase energy expenditure and number of steps per unit distance).

Trunk Lean Approximately 5–10° of trunk lean is typical during running [117]. Upright posture during running is associated with greater knee loads; slight trunk lean reduces knee joint loads with little cost to the hip or ankle [120]. However, excess trunk lean may increase hip and ankle loads and have implications for BSI. Both increased and decreased trunk lean may also be related to lumbopelvic pain [117].

Overreaching Overstriding occurs when the foot lands further in front of the runner's center of mass than expected. This differs from a long stride length and is characterized by overreaching with greater hip extension and knee extension at initial contact. It can be measured as the distance from the lateral malleolus at initial contact to the center of the pelvis. In 2D analysis, increased ankle inclination angle, reduced tibia inclination angle, and less knee flexion at initial contact are often observed with overstriding. Three-dimensional analysis indicates that overstriding increases knee loads and braking impulses [119] and is associated with greater impact forces. Importantly, overstriding may be associated with increased probability of BSI [29]. Like the other measures presented here, the precise cut point or ranges associated with injury have not been defined. However, overreaching may be suspected if a vertical line from the lateral malleolus at initial contact falls anterior to the pelvis [116].

Center of Mass Excursion The difference between the height of the center of mass at its highest point in flight and its lowest point in stance represents center of mass excursion. Greater center of mass excursion is associated with a greater peak knee extensor moment, peak vertical ground reaction force, and braking impulse [119]. Increasing center of mass excursion also contributes to greater contact velocity and increased energy expenditure [121]. Excursion can be estimated from 2D video by identifying a point on the pelvis to act as a surrogate for the center of mass and comparing that point at midstance and mid-swing.

2D Running Gait Analysis Sagittal View (Fig. 8.15)

Step Width The mediolateral distance of the foot to the center of mass has been reported to affect bone stress experienced during running [22]. Specifically, a narrow or crossover pattern increases bone stress. Step width can be measured a variety of ways both three-dimensionally

Fig. 8.15 Posterior view bone stress injury-related metrics obtained from 2D running analysis: (**a**) step width, (**b**) rearfoot eversion angle, (**c**) foot progression angle, (**d**) heel whip, (**e**) knee center position, and (**f**) lateral pelvic drop

and two-dimensionally. In 2D, step width can be examined at initial contact and midstance. A general rule is that the left and right feet should not overlap each other during consecutive ground contacts [116]. A crossover pattern occurs when there is overlap or when the center of the heel or ankle joint center falls medial to the center of the pelvis. In 2D, this can be examined by measuring the distance from the center of the heel or ankle to the sacrum. Greater than 25% of the heel width medial to a vertical line from the mid-sacrum to the ground can be considered crossover.

Rearfoot Eversion Rearfoot eversion is a component of foot pronation, which has been associated with BSI. It can be examined in 2D video analysis by bisecting the heel and measuring the frontal plane ankle relative to the lower leg. In addition to magnitude of rearfoot eversion, rate of rearfoot eversion is relevant to injury. Rate of rearfoot eversion can be quantified by subtracting the rearfoot frontal plane angle at initial contact from the corresponding midstance angle and dividing it over the change in time. Change in time can be determined from the camera sampling rate and the number of frames between time point at which angles were extracting. For example, at 120 frames per seconds, three frames equate to 25 milliseconds.

Foot Progression Angle The degree of toe in or toe out has the potential to alter tissue loads in the lower extremity and may be associated with atypical rotation at the ankle knee and hip. Examining the position of the forefoot relative to the rearfoot in 2D analysis can provide insight into foot progression angle.

Heel Whip Another rotation-related metric is the degree of heel whip during the swing phase of walking or running. The angle of the plantar aspect of the foot at initial swing relative to the angle at maximal rotation during swing reflects the degree of heel whip. A lateral heel whip may be related to internal rotation at the knee and hip rotation. Conversely, a medial heel whip may reflect external rotation of the knee and hip. The clinical relevance of this variable is unknown; Souza (2016) reports that 50% of runners demonstrate 5° heel rotation in either medial or lateral direction [116].

Knee Center Position Dynamic knee valgus (associated with hip adduction, hip internal rotation, knee abduction, and external rotation) and varus (associated with hip abduction, hip external rotation, knee adduction, and internal rotation) affect apparent knee separation during running. During stance, the distance between the medial aspect of the knees can provide insight into the knee center position. Alternatively, knee center position relative to a line connecting the ankle and hip centers can be examined. Lack of knee separation reflects a medial knee position and

dynamic knee valgus. Conversely, a large knee window and lateral knee position reflects dynamic knee varus. Both patterns have been associated with BSI.

Lateral Pelvic Drop Contralateral pelvic drop during the stance phase of running may reflect lumbopelvic muscle control and is often used as a surrogate for hip adduction, despite poor correlation with 3D measures [122]. A 3–7° angle of a line connecting the posterior superior iliac spines and true horizontal is considered typical [117].

Trunk Side Lean Trunk side lean can be characterized by a line from the spinous process of the seventh cervical vertebrae to the relative to true vertical. Excessive lean can be associated with low back pain. Unilateral trunk side lean may be also associated with attempts to offload limb weight-bearing.

Additional Variables

Additional variables often examined along with the kinematics listed above include sound, treadmill vibrations, and cadence. High-noise and treadmill vibrations during ground contact may indicate high-impact forces. Additionally, asymmetrical loading can be readily detected by attending to the auditory rhythmicity of sequential ground contacts. Finally, cadence or step rate should be examined. Running velocity is proportionate to the product of step rate and step length. Thus, as step length increases at a given velocity, step rate decreases. Cadence manipulation has shown promise as a gait modification because of the effect it has on several biomechanical variables associated with injury [113]. However, no optimal cadence has been defined for injury management; 180 steps per minute are commonly cited and have evolved from running economy studies [123].

Gait Retraining

The evidence linking faulty running mechanics to bone loads and BSI supports interventions to alter injury-related mechanics. A systematic review of the literature concluded that in conjunction with traditional therapeutic inventions, running gait modification should be considered during rehabilitation following running-related musculoskeletal injury [124]. The literature reviewed ranged from moderate to high quality and used real-time feedback to reduce ground reaction force variables and improve faulty running kinematics. In addition to verbal feedback, methods used to provide feedback included mirrors, metronomes, in-shoe sensors with auditory feedback, real-time feedback related to gait biomechanics, and video-based feedback. Training programs generally ranged from 1 to 12 session (15–30 minutes) over 2–6 weeks. No one method was considered superior. In the clinic, the use of a mirror or video-based feedback in combination with verbal cues may be most feasible. Due to the potential for load redistribution, gait modification should be undertaken judiciously. Load modification and progression will need to be considered in order to minimize the potential for injury at sites.

Incorporating key principles of motor learning will aid improving motor performance and learning. Motor performance is generally improved with external feedback that focuses learners' attention to the effect of body movement on the environment rather than internal feedback that focuses learners' attention on how the body is moving [125]. Thus, it is recommended that clinicians give external cues during the learning process (e.g., "keep the waistband level" vs. "don't let your hip drop"). Trial-and-error feedback lead to greater retention than errorless feedback [126]. Thus, practice does not need to be perfect. Rather, it is important to set up an environment that allows for discovery learning. Augmented feedback can be provided to enhance learning. Initial high-frequency feedback with a gradual fading of feedback over the course of learning reduces reliance and increases automaticity and retention [127]. Self-controlled feedback in which individuals ask for feedback is also supported in the literature [128].

Conclusion

Bone stress injury is common in active populations and can be a significant barrier to a healthy lifestyle. Optimal training regimens that maxi-

mize osteogenic potential while minimizing fatigue damage are ideal but remain elusive. Examining evidence related to the complex etiology of bone stress injury reveals factors associated with injury. Bone stress and strain are proximate causes of injury proportionate to bone loads experienced during activity and the ability of bone to resist those loads. Interventions to improve bone strength, such as targeted high-intensity activity separated by rest and muscle strength, as well as interventions to optimize bone loads are critical for both prevention and management of bone stress injury. Poor load management, both in terms of magnitude and frequency of loading, can contribute to bone stress injury and recurrence. Monitoring acute to chronic workloads can help manage training loads to ensure individuals are adequately prepared during activity. Additionally, evaluating training load within the context of bone-specific loads can aid in the prescription of load progression. Given the evidence linking faulty running mechanics to bone loads and bone stress injury, running gait retraining using sound motor learning and load progression principles may help reduce injurious loads. Because each individual with bone stress injury has a unique presentation, clinicians need to fully examine the web of determinants that interact to influence bone-specific load and load capacity and direct personalized interventions accordingly.

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9 Nutritional Optimization for Athletes with Stress Fractures

Sakiko Minagawa and Jackie Buell

The occurrence of stress fractures is likely multifactorial and can include inadequate nutrition over an extended period of time. It is of critical importance to assess the dietary needs of an athlete compared with habitual intake when stress fractures occur. Correcting inadequate energy and micronutrient intake may decrease the risk of future stress fractures.

The Relationship of Energy Intake and Balance to Bone Health

In 1993, the American College of Sports Medicine (ACSM) first described the female athlete triad (triad) as a syndrome of three disorders: disordered eating (DE), amenorrhea, and osteoporosis [1]. The triad was redefined in 2007 as a spectrum from healthy to clinically unhealthy of three interrelated components of energy availability (EA), menstrual function, and bone health [2]. Optimal health is characterized by optimal EA, eumenorrhea, and optimal bone health. The other end of the spectra are characterized by low EA with or without an eating disorder, functional hypothalamic amenorrhea (FHA), and osteoporosis. The visualization of the triad paradigm as published in color by De Souza et al. is particularly helpful to understanding the concept of these spectra [3].

In 2014, the International Olympic Committee (IOC) furthered the female athlete triad when they introduced the term "relative energy deficiency in sport" (RED-S), a more comprehensive term that emphasizes low EA as the primary factor resulting in impaired physiologic functions and includes the risks for all athletes [4]. RED-S proposes that low EA can negatively affect many more body systems, beyond the musculoskeletal and endocrine systems presented in the triad model. Additionally, athletes in a prolonged state of low EA are at an increased risk for illness and nutrient deficiencies that may negatively affect athletic performance. Again, visualization of the paradigm in IOC position statement is helpful in understanding the body systems that are theoretically impacted [4], and a more recent affirmation of the model is available [5].

In the RED-S paradigm, the central RED-S is still defined by EA [4, 5]. EA requires specific assessment of the athlete and is operationally defined as dietary energy intake (EI) in kcal minus exercise energy expenditure (EEE) in kcal, divided by kg of fat-free mass (FFM) [6].

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Energy Availability (kcal/kg FFM/day) = [Energy Intake (kcal) – Exercise Energy Expenditure (kcal)]/Fat Free Mass (kg)

Practically speaking, it is the amount of energy intake available to maintain the body after exercise is accounted for, and it is adjusted for body size using lean mass.

Energy availability is an energy balance estimate that specifically accounts for exercise. Research has suggested that EA is optimal at or above 45 (kcal/kg FFM/day) for normal physiological function. Studies show estradiol and progesterone suppression as well as luteinizing hormone (LH) pulsatility disruption which result in menstrual dysfunction (MD) occurring at EA levels below 30 kcal/kg FFM/day [6]. While the triad has long been targeted to females, progression of the paradigm to RED-S includes males and suppression of testosterone. These hormonal suppressions will impact bone modeling and remodeling through time.

Nutritional Assessment

Energy

The estimated energy requirement (EER) is the average dietary energy intake necessary to maintain energy balance in a healthy person based on their sex, age, anthropometrics, and physical activity level [7]. Basal metabolic rate (BMR) is often defined as the basis of energy needs and is difficult to measure within the pure definition of energy used for physiologic existence. More often this component is more generically referred to as resting metabolic rate (RMR) [8]. The RMR is the starting point to estimate energy needs, and it should be adjusted with physical activity and stress factors for injured athletes to ensure adequate support for healing [8].

Energy requirements can be measured and estimated in many ways, including indirect and direct calorimetry and predictive equations [8]. Direct calorimetry measures total heat lost from the body, while indirect calorimetry measures the volume of oxygen (O_2) inspired and/or volume of carbon dioxide (CO_2) expired. The gold standard for energy balance studies is the doubly labeled water (DLW) method and is considered to be free-living indirect calorimetry [7]. DLW relies on expensive and complex methods and is not widely available to most clinicians. In addition, DLW is not able to partition calories due to exercise. While "direct" calorimetry seems to connote some level of certainty, there are very few direct chambers available, and measurement of free-living exercise is not possible inside these chambers; thus, practitioners will rely on indirect calorimetry machines or predictive equations. The choice of method often depends on the resources available and the relative importance of accuracy.

Indirect machines range in accuracy and cost. Large and more expensive units are often called metabolic carts and vary in software and ability according to the manufacturer. These units often measure oxygen (O_2) utilization as well as carbon dioxide (CO_2) production. Not only does this allow for an estimate of calories consumed, but it allows for partitioning the nonprotein sources of fuel (carbohydrates versus fats). Similarly, smaller tabletop models are also available and often only measure oxygen utilization thus caloric expenditure. It is generally assumed that metabolic carts are more accurate than the less expensive tabletop machines. When calorimetry machines are used to estimate RMR, the accuracy of the measurement should be preserved by following the suggested protocols prior to measurement [9]. While indirect machines are widely available and frequently used, it should be noted that oxygen-only machines must assume a given respiratory quotient and the actual value may be different depending on the actual diet consumed [10]. Indirect calorimetry is helpful in estimation of energy needs and is limited by access to the tools.

More commonly, predictive equations are employed to estimate the RMR component of caloric needs due to ease of access. Because these are regressions formulated from specific populations, the practitioner should select the most accurate equation to fit the specific athlete and his/her goals. Common equations for athletes older than 18 years old include the Harris-

Benedict, Mifflin St. Jeor, Owen, and Cunningham, and details can be found in many academic resources [8]. The RMR estimate then needs to be adjusted for physical activity level and possibly the thermic effect of food. The RMR calculation only covers estimated resting needs and should not be considered an adequate intake for athletes. The DRIs should be used for athletes younger than 18 years old to facilitate growth alongside the exercise volume [7]. Energy intake and expenditure should be evaluated by a trained professional to rule out inadequate intake as this can be an ongoing source of reinjury and frustration. Athletes with active stress fractures need enough energy to heal, and this is not a time for significant restriction.

Dietary Recommendations

In addition to consuming adequate energy, athletes need to consider the composition of the diet. The evidence-based macronutrient recommendations are provided specific to athletes by the Academy of Nutrition and Dietetics, Dietitians of Canada, and American College of Sports Medicine (Academy/DC/ACSM) [11]. In addition, the International Olympic Committee (IOC) [12] and the International Society of Sports Nutrition (ISSN) [13] have presented nutritional position statements for athletes. While slightly dated in reference to the Academy/DC/ACSM guidelines, Potgieter has presented these guidelines in one document that includes tables for easy comparisons [14]. The macronutrient recommendations for carbohydrate and protein are provided per kilogram (kg) body weight for athletes and not as a percentage of calories in the diet.

Protein

Protein is the building block for bone and important to the healing of injuries. The suggested protein intake for athletes is higher than the RDA of 0.8 g/kg/day [7] and depends on the nature of the sport or activity, length of training, and goals of the athlete. According to the Academy/DC/ACSM position statement, athletes' protein needs range from 1.2 to 2.0 g/kg/day [11]. However, studies suggest protein intakes greater than 1.6 and up to 2.2 g/kg/day do not elevate gains in LBM in trained weight-stable athletes [15]. On the other hand, protein intakes of 2.0 g/kg/day or higher may be necessary when the athlete is in energy restriction (trying to lose weight) or injured [16]. More specifically, systematic review by Helms et al. concluded that protein intakes of 1.8–2.7 g/kg/day was optimal in reducing fat mass while preserving lean body mass (LBM) [17]. A noteworthy limitation was that the review only included six studies. Table 9.1 summarizes the range of protein intakes considered optimal according to athlete goal. Protein needs for athletes are highly debated, and not all professional groups agree on these points.

Table 9.1 Summary of protein needs for athletes

	Protein: daily needs (grams per kg of body weight)
RDA [7]	0.8
Athletes maintain or gain LBM [11]	1.2–2.0
Athletes during energy restriction [17] or if injured [16]	1.8–2.7

Additionally, recent evidence suggests protein intake recommendations be met across the day and is often described as "protein-centric" meals. Paddon-Jones and Rasmussen first suggested this as a method of mitigating the loss of lean mass with aging [18], and the science has progressed to include optimizing muscle recovery for athletes, especially relative to strength training. Early estimates of the dose of protein per meal suggested 20–30 grams per meal, while later evidence supports 40 grams per meal may still reap even more lean mass benefits. It is important to realize that the science on protein timing and dose still spawns much debate from high-level researchers [19, 20]. Another current protein intervention for athletes desiring to gain lean mass alongside strength training is to consume 40 g of casein protein prior to bedtime [21]. These strategies may or may not apply to athletes

with stress fractures, but familiarity with the trends benefits questions athletes might have for the practitioner.

Protein is known to influence bone metabolism. Since the 1920s, it was common to believe that high-protein intake, especially from animal sources rich in sulfur amino acids, was associated with increased renal acid load and urinary calcium excretion, purportedly from bone [22, 23]. Later studies with stable isotopes disproved this phenomena demonstrating that the higher calcium excretion was accompanied by a higher intestinal absorption [24]. A review commissioned in 2016 by the National Osteoporosis Foundation (NOF) recounted the history including the more recent studies to conclude that (animal) protein in a diet with adequate calcium and fruits and vegetables promotes higher bone mass [25]. Athletes trying to heal or prevent stress fractures should strive to follow the protein recommendations for athletes within a balanced diet.

Carbohydrate

Carbohydrates are recognized as the primary fuel for high-intensity exercise. While the casual athlete might train well and feel healthy at 3–5 g/kg, a higher-intensity, more serious athlete might require more depending on the volume and intensity of training. Table 9.2 provides guidance for various levels of participation.

Another potentially important aspect of carbohydrate nutrition for athletes with stress fractures is the specific fiber content of the diet. In the 1990s, high-fiber intake due to phytates and lignins was purported to decrease bone density accrual in young women through decreasing estrogens [26, 27]. More specifically, Goldin has shown this to be more the case in wheat bran and not oat or corn brans [28]. Others suggest this may be more the case when the fat in the diet is lower [29]. More recent studies suggest soluble corn fiber may increase calcium absorption acting as a prebiotic for the intestinal microbiome [30]. Nonetheless, recent studies still demonstrate a significant association for lower BMD at the spine with higher-fiber diets [31]. Athletes with stress fractures should likely be sure they are not overconsuming whole-grain wheat products.

Table 9.2 Summary of carbohydrate needs for athletes

Intensity		Carbohydrates: daily needs (grams per kg of body weight)
Light	Low-intensity or skill-based activities	3–5
Moderate	Moderate exercise (~1 hour)	5–7
High	Endurance program (1–3 hours/day moderate- to high-intensity exercise)	6–10
Very high	Extreme commitment (>4–5 hours/day moderate- to high-intensity exercise)	8–12

Adapted from Position of the Academy of Nutrition and Dietetics, Dietitians of Canada, and the American College of Sports Medicine: Nutrition and Athletic Performance [11]

Epidemiology studies have held similar suggestions. In the Framingham Offspring study, Dai et al. observed the role of dietary fiber between genders as well as the types of fiber (total fiber, fiber from cereal, fruits, vegetables, nuts, and legumes) [32]. Higher total dietary fiber intake as well as fruit fiber was associated with slowed femoral neck bone loss in men but not in women. On the other hand, fiber from vegetables was associated with slowed spinal bone loss in women but not in men. It is plausible that the "fiber from fruits and vegetables" is really a surrogate marker for higher phytonutrients and a more alkaline intake. The key point is that athletes with stress fractures should consume their fruits and vegetables within a balanced diet.

Fat

Dietary fat recommendations are not typically described in grams per kg body weight and should meet the acceptable macronutrient dietary intake range of 20–35% of an isocaloric diet [7]. Among other functions, fat is important to hormonal production and muscle fueling and

thus should not be minimized in the athlete's diet. In practice, many athletes are consuming differing patterns of fat in the diet: low fat, high fat or ketogenic, and everything in between. Encouraging athletes to get fats from a wide variety of foods provides nutrients important to healing. In particular, omega-3 fatty acids may help decrease inflammation [33]. More recently, omega-3s have also been implicated in bone health through various proposed mechanisms in animal and cell models [34], though research in adolescent and young adult human models is scant. Athletes with stress fractures need enough fat in their diet to support hormonal health and should not be recommended a low-fat diet.

Micronutrients

In addition to adequate energy and macronutrient intakes, athletes need to meet the basic micronutrients necessary for a healthy individual as put forth by the National Academy of Medicine (NAM) as the Dietary Reference Intakes (DRI). The DRIs are published and freely available at USDA.gov [33] and are best used in athletes to approximate adequate intake of vitamins and minerals without exceeding the tolerable upper intake levels. The specific role for each "bone nutrient" is provided, and suggested intakes for these micronutrients have been transcribed in Table 9.4 in the Resources section of this chapter as an overview.

Approximately 50% of athletes report micronutrient supplementation. The systematic review of 128 studies by Heffernan et al. assessed the efficacy of minerals and trace element supplementation for athletic performance [35]. Calcium and vitamin D are the best-known dietary components for the development and maintenance of bone health. Additionally, other dietary components such as iron, vitamins A and K, phosphorus, potassium, and magnesium play important roles in bone metabolism. However, the complex interactions between genetics, environment, lifestyle, and nutrition as well as individual versus groups of nutrients make it difficult to obtain consistent findings, especially in athletic populations.

Calcium

Calcium is the most prevalent mineral in the body, primarily as calcium hydroxyapatite in the bones and teeth (*Dietary Reference Intakes for Calcium and Vitamin D*). Sources of calcium come from both food and supplements. Dairy food products such as milk, cheese, and yogurt are the major dietary sources of calcium in the United States. The fortification of foods such as orange juice and plant-based milks is also available in the American diet. Fortification creates a challenge for nutritional databases to accurately estimate calcium intake. Many athletes with stress fractures consume calcium supplements or fortified foods in order to confer a level of healing and protection against future fractures. It should be noted that calcium is "permissive" to bone formation, meaning consumption of the threshold is important, but overconsumption will not necessarily lead to additional bone mineralization. Calcium citrate is more readily absorbed than other forms of calcium [36] and should be consumed in 500–600 mg doses with food. Currently there are no specific calcium intake recommendations for athletes; thus athletes should follow the DRI life stage recommendations [37].

As with many nutritional issues, too much calcium may have adverse or unintended side effects. Consumers should be aware of the young adult upper limit of 2500 mg for calcium per day as set forth in the DRI system. Calcium is a divalent mineral and may compete for absorption with other divalent minerals such as zinc, iron, and copper. There is a significant ongoing debate as to whether calcium supplementation has potential adverse gastrointestinal and cardiovascular effects, and it is still argued by some that supplemental calcium may increase the risk for nephrolithiasis (kidney stones) for some individuals [38]. Calcium supplementation should be considered for athletes with stress fractures within the full picture of the athlete's usual diet, medical history, and health risks.

Vitamin D

Defining and achieving adequate vitamin D status are a consistently hot topic for athletes with a number of purported benefits. Vitamin D receptors (VDRs) have been identified in many body tissues including the muscles, intestines, and bones. Vitamin D acts as a secosteroid in the body and interacts with parathyroid hormone (PTH) and bone to maintain the required level of serum calcium. In addition, vitamin D promotes small intestinal absorption of calcium through the VDR stimulation of calcium transport proteins. Current paradigms propose that vitamin D is an important regulator of parathyroid–kidney–intestinal and bone–kidney–intestinal axes. The axes are important to the regulation of calcium uptake and renal excretion influencing calcium balance.

Vitamin D is different than other micronutrients because the body can manufacture it when the skin is exposed to adequate sunlight. The metabolic pathway that makes vitamin D also requires hydroxylation from the liver then the kidneys to make the hormonally active calcitriol. Of course, vitamin D can also be consumed in the diet from foods which naturally contain it or are fortified with it. Given that it is difficult to consume enough in the diet, athletes with limited exposure to direct sunlight may consider supplementation with vitamin D_3 when serum values fall below optimal levels. Defining the optimal serum, vitamin D is still under debate, but there is general agreement that the RDA is not likely adequate. While many guidelines suggest a serum value of 30 ng/mL as "adequate," current literature suggests that muscle tissue may saturate slightly higher at 40–50 ng/dL of vitamin D. With the surge of vitamin D research comes awareness of inadequate lab values [39] and the influence it may have on athlete health and performance [40].

Studies have observed the role of vitamin D in bone health, but the role of vitamin D in the face of significant physical activity is less clear. A review by Owens et al. describes the role of vitamin D in performance, muscle recovery, and function [41]. More specifically, research in athletes has failed to confirm a direct relationship between bone density and adequate vitamin D status. A study of athletes with stress fractures by Miller et al. demonstrated that 44/51 athletes had a serum vitamin D lower than 40 ng/mL [42].

While calcium and vitamin D are both important factors for bone health, the tightly regulated serum calcium value is not a good indicator for bone support. However, periodic assessment of serum vitamin D should be considered to support long-term bone health. Owens et al. suggest a protocol for vitamin D assessment and discourages the practice of blind supplementation [41]. Athletes with stress fractures should be evaluated for serum values, and treatment should proceed accordingly.

Iron

Iron is a critical element involved in numerous metabolic reactions. Dietary iron is found in two forms: heme and nonheme. While heme iron found in animal products such as poultry, red meat, and seafood is highly bioavailable, nonheme iron found in plant products such as spinach, pumpkin seeds, and lentils is variable in absorption. Absorption of nonheme iron is enhanced by ascorbic acid and inhibited by phytates, calcium, and polyphenols [43]. The RDA for iron differs based on age, sex (see Table 9.4), pregnancy, and/or lactation.

Adequate iron and iron balance are important hematological considerations for athletes. Iron balance means the athlete not only has to consider intake but also iron loss. Potential causes of iron deficiency include limited consumption of iron-rich foods, decreased iron absorption, and heavy menstruation in females. Additionally, endurance athletes are at higher risk for iron deficiency because of increased iron losses through sweat, as well as urine and feces, from intravascular hemolysis and micro-bleeds that occur while running [44]. Iron inadequacy and deficiency can cause muscle weakness, fatigue, impaired body temperature, and impaired cognitive and physical performance.

While most studies have evaluated iron status and supplementation on performance-related outcomes such as VO_{2peak}, lactate levels, fatigue

resistance, and strength (Heffernan), the recent review by Petkus et al. outlines numerous mechanisms whereby poor iron status may interact with the female athlete triad characteristics to indirectly and directly impact bone health [45]. Evidence also suggests iron overload conditions such as hereditary hemochromatosis (HH), thalassemias, and sickle cell disease, are associated with decreased bone mass, osteoporosis, and increased frequency of bone fractures [46]. This is supported in osteoblast cell line model work by Zhou where having too little or too much iron inhibits osteoblastic activity. Thus it appears that having the right amount of iron is important [46]. Iron is essential for cell growth and function, and it plays two direct roles in bone health. First, iron is essential for collagen synthesis, a major component of total bone protein. Additionally, iron plays a role in vitamin D activation. Athletes with low vitamin D or stress fractures should likely have iron status checked within the blood work to ensure iron nutrition is adequate and balanced.

Measurement of iron status usually involves markers of hemoglobin and red blood cell health and iron-binding capacity to look for iron-deficiency anemia (IDA) [47]. Additionally, ferritin can be estimated as a method of looking at iron status prior to reaching the functional deficiency stage, though clinicians should bear in mind that ferritin is an acute phase reactant and will falsely increase with any inflammation. Given the many markers and levels considered adequate, it is common to talk about iron deficiency in three stages: depletion, deficiency, and IDA [47]. Sim et al. provide a suggested iron-testing algorithm within their review of athlete iron considerations [48]. Athletes with a stress fracture should have an iron panel checked along with their other serum testing, and noted deficiencies should be addressed.

Other Important Micronutrients

Other nutrients are known to be involved in bone metabolism. However, these nutrients are not often considered low in the athlete's diet or have not been implicated in low bone mass in athletes. Most of the studies on these nutrients have been carried out in postmenopausal women and have not been considered in athletes with stress fractures.

Vitamin K, a fat-soluble vitamin, is best known for its role in blood clotting and is also an important vitamin for calcium metabolism and bone health. Vitamins K and D are often seen as teammates in bone health. Vitamin K supports osteocalcin activation (gamma carboxylation) to promote calcium storage in the bones and teeth [49]. As well, vitamin K has been purported to slow osteoclastic activity [49]. With the roles affecting bone formation and breakdown, it is intuitive that vitamin K may positively impact remodeling balance. Athletes consume vitamin K1 in dark leafy green and other vegetables, while K2 is found in some animal products and can be synthesized by the bacteria in the human gut. While there is some research on vitamin K and fractures in aging populations [50], research examining treatment with vitamin K supplementation in athletic populations is still limited with mixed results [55, 56]. This is likely due to the complexity and more impactful factors related to bone health such as energy availability, amenorrhea and concurrent estrogen supplementation.

Vitamin A is also a fat-soluble vitamin and is essential for vision, healthy hair, skin, nails, and bones. There are two forms of vitamin A, preformed vitamin A (retinol and retinyl ester) and provitamin A substances such as the carotenoids. There is an upper limit for preformed vitamin A, but excess carotenoids, specifically beta-carotene, will not be converted to vitamin A if the body already has sufficient levels of vitamin A. Retinol is found in animal foods such as eggs, liver, and fatty fish. Beta-carotenes are found in orange and dark leafy greens such as sweet potatoes, carrots, winter squash, and kale and are converted to retinol in the body. Similar to iron, it can be too high or too low. However, most research on this nutrient is in the aging population relative to fracture risks when vitamin A is too high. Vitamin A deficiency is rare in the United States.

Phosphorus is the major anion in the human body, and 85% of the phosphorus in the body is in the bones and teeth. In the skeleton, phosphate is present with calcium in the form of hydroxyapatite crystals. Phosphate homeostasis is tightly regulated

by PTH, calciferol, and peptides known as "phosphatonins" [51]. Phosphate is important for many processes including skeletal development and ongoing bone health. Dietary sources of phosphorus include protein-rich foods such as meat, poultry, fish, nuts and beans, and cereal grains. Additionally, current processed foods are high in phosphates. Phosphate deficiency results in bone pathology and clinical illness such as rickets and osteoporosis. A common Western diet provides adequate phosphorus; thus deficiency is rare. It is more likely that most Americans will get too much phosphorus in the diet which can cause bone, cardiovascular, and renal damage [52]. Education of athletes regarding processed food and soda consumption is crucial for not only optimizing performance but also promoting better general health.

Studies suggest an alkaline diet may reduce bone loss and fractures. *Potassium* and *magnesium*, primarily found in fruits and vegetables, are sources of creating a high-alkaline state for the body. The original Framingham cohort study showed higher magnesium and potassium intake associated with higher BMD [53]. In men, higher potassium intake was associated with higher BMD at all four sites (femoral neck, peritrochanteric, Ward's area, and the radius) and three sites for women (peritrochanteric, Ward's area, and the radius). Greater magnesium intake was associated with greater BMD at the hip for both men and women. About 60% of total body magnesium is banked in bone such that when a person has inadequate magnesium intake, it is detrimental to the skeleton [54]. These micronutrients have not been well-explored in athletes, but it is likely beneficial for athletes with a stress fracture to eat plenty of fruits and vegetables [53].

A well-rounded diet incorporating all of the food groups is important to athletes with stress fractures to promote healing. While many of the nutrients discussed are tightly regulated in the blood, vitamin D and iron levels are not and can be easily tested with a blood draw. Bone density overall can and should be screened using the iDXA for total body, hip, and lumbar spine to screen for low bone mass. The advantage of learning body composition in the total body scan enhances the nutritional assessment of the athlete's needs. While polyphenols or other antioxidant nutrients were not discussed, it is widely believed that antioxidants found in foods will protect bone density. These are the same foods high in potassium and magnesium. Any athlete experiencing injury should work to consume a diet rich in foods with anti-inflammatory properties.

Resources

Practitioners often look for handouts and resources to provide to patients for guidance on nutrient-dense food or education on specific issues. Table 9.3 provides website URLs for fact sheets of interest to the nutritional evaluation or treatment of athletes with stress fractures.

The DRIs for all nutrients are available free at the National Academies Press (nap.edu) by searching "DRI" and the nutrient name. Table 9.4

Table 9.3 Useful fact sheets to share with stress fracture athletes as indicated from dietary assessment

	EA	Pro	Carb	Fat	Ca	D	Fe	Vit K	Vit A	Ph	K	Mg
https://ods.od.nih.gov/factsheets/list-VitaminsMinerals/					x	x	x	x	x	x	x	x
https://www.teamusa.org/nutrition	x		x			x	x					
https://www.sportsrd.org/educational-resources-2/educational-resources/	x											
https://www.scandpg.org/scan/educational-resources/fact-sheets/sn-fact-sheets	x	x				x	x					
https://www.sportsdietitians.com.au/factsheets/		x		x		x	x					
http://www.ncaa.org/sport-science-institute/nutrition	x											
https://www.gssiweb.org/en/sports-science-exchange/All/sports-nutrition	x	x	x			x						

Table 9.4 RDAs for bone nutrients in this chapter as set forth within DRI system

	Source of goal	Female 9–13	Male 9–13	Female 14–18	Male 14–18	Female 19–30	Male 19–30	Female 31–50	Male 31–50	Female 51+	Male 51+
DRI calorie level(s) (nonathletes)		1600	1800	1800	2200, 2800, 3200	2000	2400, 2600, 3000	1800	2200	1600	2000
Calcium, mg	RDA	1300	1300	1300	1300	1000	1000	1000	1000	1200	1,000[a]
Vitamin D, IU	RDA	600	600	600	600	600	600	600	600	600[b]	600[b]
Iron, mg	RDA	8	8	15	11	18	8	18	8	8	8
Vitamin K, mcg	AI	60	60	75	75	90	120	90	120	90	120
Vitamin A, mg RAE	RDA	600	600	700	900	700	900	700	900	700	900
Phosphorus, mg	RDA	1250	1250	1250	1250	700	700	700	700	700	700
Magnesium, mg	RDA	240	240	360	410	310	400	320	420	320	420

Adapted from https://ods.od.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx
[a]Calcium RDA for males ages 71+ years is 1200 mg
[b]Vitamin D RDA for males and females ages 71+ years is 800 IU

Table 9.5 DRI upper limits for bone nutrients in this chapter

	Source of goal	Female 9–13	Male 9–13	Female 14–18	Male 14–18	Female 19–30	Male 19–30	Female 31–50	Male 31–50	Female 51+	Male 51+
Calcium, mg	UL	3000	3000	3000	3000	2500	2500	2500	2500	2000	2000
Vitamin D, IU	UL	4000	4000	4000	4000	4000	4000	4000	4000	4000	4000
Iron, mg	UL	40	40	45	45	45	45	45	45	45	45
Vitamin K, mcg	UL	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Vitamin A, mg RAE	UL	1700	1700	2800	2800	3000	3000	3000	3000	3000	3000
Phosphorus, mg	UL	4	4	4	4	4	4	4	4	3	3
Magnesium, mg	UL	350	350	350	350	350	350	350	350	350	350

Adapted from https://ods.od.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx
ND Not determinable due to lack of data, consume from food only to prevent high levels of intake

provides the life stage DRIs as the recommended daily allowance (RDA) or adequate intake (AI) for nutrients of importance for athletes with stress fractures. Adequate intakes are set when there is not enough data to set an RDA. Our current society seems to have a "more-is-better" attitude when it comes to nutrition, and some nutrients can be damaging to bone if overconsumed. Upper limits (ULs) are set to prevent toxicity or undesired side effects. A table outlining the DRI upper limits for each bone nutrient is provided as in Table 9.5.

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Systemic Treatment Modalities for Stress Fractures

10

Megan Roche, Geoff Abrams, and Michael Fredericson

Introduction

Bone stress injuries (BSIs) occur when bone experiences structural fatigue as a result of repetitive, mechanical loading. BSIs account for 0.7–20% of all sports medicine injuries and are a common concern in athletes participating in sports emphasizing leanness [1]. The 1-year prospective incidence of BSI in competitive cross-country and track and field athletes is as high as 21.1% [2].

BSIs exist along a pathology continuum that can be defined based on findings on magnetic resonance imaging (MRI). The pathology continuum progresses from periosteal edema with varying degrees of bone marrow edema to more advanced injuries showing evidence of a cortical fracture line [3].

Athletes who have delayed healing, incomplete healing, nonunion, or repeat BSI may benefit from systemic treatment modalities to optimize recovery. In this chapter, we examine the impact of teriparatide, extracorporeal shockwave therapy (ESWT), and bone stimulation devices on BSI recovery.

Athletes who have low bone mineral density (BMD) or osteoporosis may also benefit from systemic treatment modalities. However, systemic treatment modalities for athletes with osteoporosis are better covered in other chapters. Systemic treatment modalities that are covered elsewhere include bisphosphonates, hormone replacement therapy, and vitamin D supplementation.

Overall, this chapter covers evidence-based guidelines for systemic treatment modalities in BSI recovery. Within this framework, the mechanism of action, clinical use, and benefits and considerations of systemic treatment modalities are discussed.

Teriparatide (TPTD)

Mechanism of Action

Teriparatide (PTH 1-34, or TPTD) is a recombinant protein of the bioactive portion, or the first 1-34 amino acids, of parathyroid hormone (PTH) [4]. PTH is responsible for regulation of calcium and phosphate homeostasis (Fig. 10.1) by influencing 1,25-dihydroxyvitamin D production, renal tubular calcium reabsorption, and bone resorption. Production of PTH increases in response to low serum calcium levels and enhances the release of calcium from the reservoir in bone. Continuous hypersecretion of PTH leads to bone resorption, whereas intermittent

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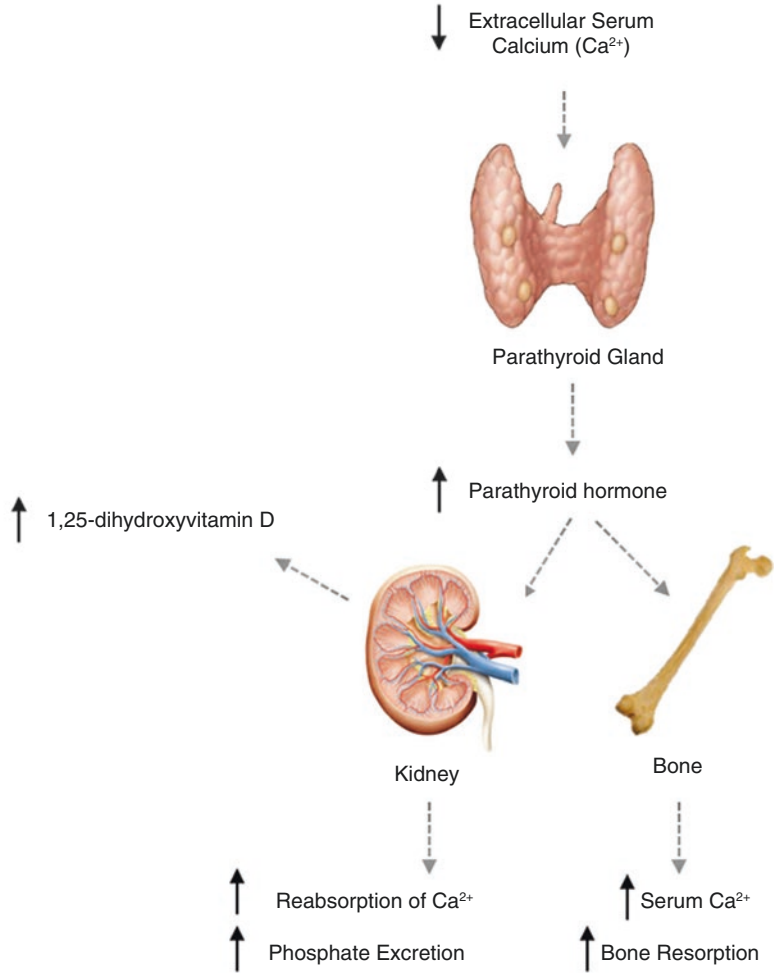

Fig. 10.1 Parathyroid hormone and calcium homeostasis

and low-dose administration of PTH has an anabolic effect on bone [5].

Once a day, subcutaneous administration of PTH yields a transient peak blood level that produces an anabolic effect by increasing the activation of osteoblasts compared to osteoclasts [6]. In this process, PTH exerts its anabolic effect by binding to the PTH1R receptor in osteoblasts. PTH binding activates cAMP-dependent protein kinase PKA, leading to a direct anabolic effect on osteoblasts, an indirect activation of skeletal growth factors, and an indirect inhibition of growth factor antagonists [7]. PTH activates insulin-like growth factor (IGF-1) and inhibits the growth factor antagonist sclerostin.

As a PTH analog, teriparatide stimulates osteoblastic bone formation and regulates growth factors in order to improve bone volume and microarchitecture [8]. In animal models, teriparatide has been shown to enhance fracture healing through increased callus formation [9].

Clinical Use

Teriparatide, also known under the brand name of Forteo, has been FDA-approved since 2002 for the treatment of osteoporosis in postmenopausal women at high risk for fracture, for primary or hypogonadal osteoporosis in men, and for glucocorticoid-induced osteoporosis in men and

women. In addition, teriparatide has been used off-label to treat fracture nonunions and decrease duration of fracture repair [10].

Teriparatide is an injectable medication that is administered once daily, in a 20-microgram dose. The use of teriparatide is clinically limited to 24 months. However, other treatment protocols including once-weekly teriparatide injections and intermittent administration of teriparatide in 3-month intervals are being investigated [11, 12].

Prior to starting treatment, patients should have a dual-energy X-ray absorptiometry (DXA) scan and serum calcium, phosphorus, creatinine, alkaline phosphatase, albumin, and 25-hydroxyvitamin D measurements. Follow-up serum calcium levels should be measured to evaluate risk for hypercalcemia.

Patients should have optimized vitamin D and calcium levels. Dietary and supplemental calcium should be approximately 1000–1200 mg/day, but supplementation should not exceed 1500 mg a day due to concern for hypercalcemia. Vitamin D supplementation should be at least 800 IU per day [13].

Benefits and Considerations

Overall, teriparatide treatment is associated with increased BMD and significantly decreased fracture rates. However, a large majority of the research is derived from studies examining postmenopausal women or men aged 60 and older. In addition to that research, we preferentially highlight a few smaller studies in younger patients to provide context for the use of teriparatide in BSI management in younger athletes.

A 2017 study found that 83% of patients with osteoporosis showed a >3% increase in lumbar spine BMD after 18–24 months of teriparatide treatment [14]. A meta-analysis of randomized controlled trials found a 70% risk reduction in vertebral fractures and a 38% risk reduction in non-vertebral fractures in postmenopausal patients with osteoporosis [15].

Cohen et al. completed a pilot study examining the effects of 18–24 months of teriparatide treatment in 21 premenopausal women with unexplained fragility fractures or idiopathic osteoporosis. On average, treatment caused increased BMD at the spine, total hip, and femoral neck; improved trabecular microarchitecture at the hip; and increased bone strength in the radius and tibia. Of the 21 women, 4 women had no increase in BMD following treatment [16].

The effect of teriparatide on radiographic healing and functional recovery time is still uncertain for osteoporotic patients with a fracture and premenopausal/young male patients with lower-extremity stress fracture.

Studies have investigated teriparatide's impact on radiographic healing and functional recovery times compared to controls in osteoporotic patients. A 2017 systematic review found that the use of teriparatide had positive effects on radiographic healing in six studies with no effect in three studies. Further, teriparatide was associated with decreased pain and increased mobilization in six studies with no effect in three studies [17].

A 2016 pilot, placebo-controlled study investigated 8 weeks of teriparatide treatment in premenopausal women with lower-extremity stress fracture. 87% Of the teriparatide-treated group showed improved healing on MRI compared to 57% in the placebo group. However, due to small sample size ($N = 13$), the results did not reach statistical significance ($p = 0.18$) [18]. More comprehensive randomized controlled trials are needed to understand teriparatide's effect on fracture healing in patients.

Teriparatide may be considered as an off-label use for premenopausal athletes with osteoporosis or male and female athletes with delayed BSI healing or nonunion. Male athletes with osteoporosis are covered under FDA approval. The treatment designer should consider teriparatide side effects as well as the benefits of using other pharmacologic agents for bone health.

Side Effects

Teriparatide is contraindicated in patients at risk for hypercalcemia. Patients with primary or secondary hyperparathyroidism or other hypercalcemic disorders such as hypercalcemia of malignancy are at risk. Further, teriparatide is

contraindicated in patients with increased baseline risk for osteosarcoma as a result of skeletal radiation therapy, Paget's disease, skeletal malignancy, or open epiphyses (in young patients). Teriparatide should not be given to patients who are pregnant or breastfeeding [19].

Two randomized double-blind, placebo-controlled trials of 1382 patients found at least a 2% increase in adverse events in the Forteo group compared to the placebo group in the following effects: nausea (14% vs 7%), gastritis (7% vs 3%), pneumonia (6% vs 3%), insomnia (5% vs 1%), anxiety (4% vs 1%), and herpes zoster (3% vs 1%), respectively [10].

In theory, there is an increased risk of osteosarcoma in patients treated with teriparatide. The theory is derived from teriparatide's mechanism of action and from results of rats treated with the highest level of teriparatide. However, the Forteo Patient Registry (FPR) was established in 2009 with linkage to US State Cancer Registries to evaluate prospective cases of osteosarcoma. As of 2017, after 242,782 person-years of observation, no incident cases of osteosarcoma were identified in the FPR [20]. More research is needed to investigate the relationship between teriparatide and osteosarcoma. Screening for baseline risk of osteosarcoma is important before prescribing teriparatide.

Extracorporeal Shockwave Therapy

Mechanism of Action

Extracorporeal shockwave therapy (ESWT) involves the application of focused high-energy ultrasound shockwaves, which promote biological healing processes through mechanotransduction. Shockwaves are characterized by high peak pressure, low-tensile amplitude, short rise time, and short duration. The positive phase of the shockwave produces a direct, mechanical effect, whereas the negative phase produces a cavitation effect in biological tissue [21].

In bone, the application of ESWT is thought to cause neovascularization, periosteal stimulation, and osteoinduction (Fig. 10.2). Periosteal stimulation contributes to cell migration and the development of callus at a bone injury site. Osteoinduction involves osteoblast differentiation from mesenchymal stem cells and inactive cells [22].

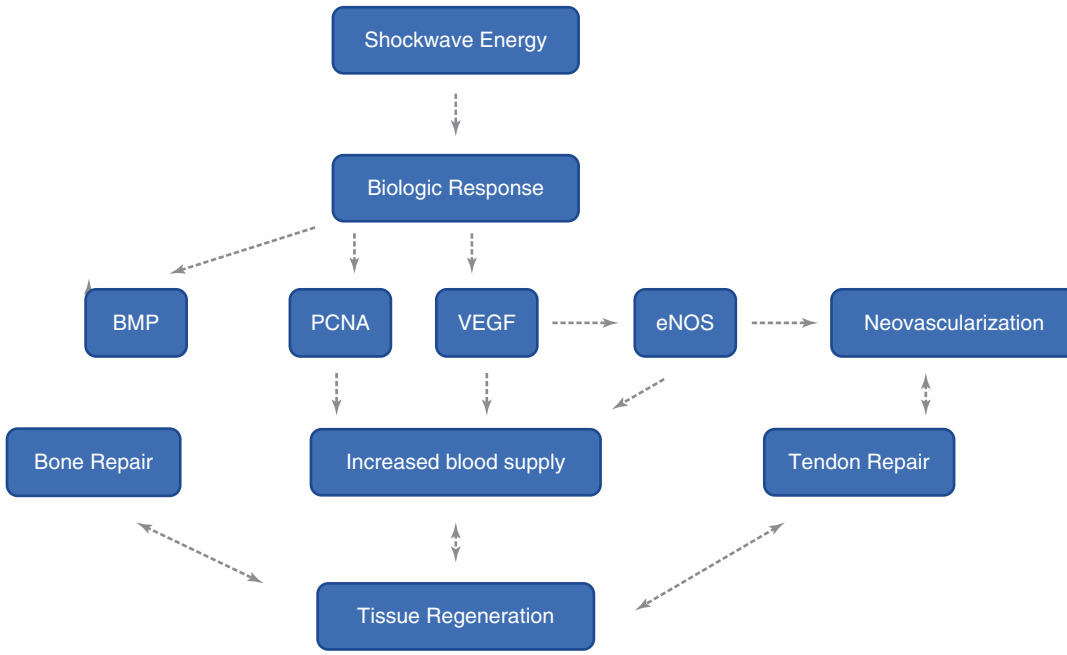

Fig. 10.2 Proposed biologic mechanism of ESWT

Clinical Use

In bone, ESWT has been used in the treatment of delayed unions, avascular necrosis, and osteochondritis dissecans. The International Society for Medical Shockwave Treatments has approved stress fractures as an indication for ESWT. However, to date, there are no randomized control trials that provide level I evidence for ESWT in bone pathology [23].

In research studies, ESWT has been used for management of BSIs refractory to conventional therapy and for BSIs in high-performance athletes. As a result, clinical guidelines for shockwave application and clinical management after ESWT are not yet established for BSIs.

As summarized by Leal et al., the majority of ESWT protocols for BSI use mid- and high-energy shockwave devices, over 1 or 2 sessions, with a maximum of 2000 shockwaves of 0.2–0.5 mJ/mm^2 over the fracture site. Although ESWT is a noninvasive procedure, anesthesia or sedation is recommended for shockwave application over a painful bone site. Fluoroscopic X-ray can guide shockwave approach [22].

A radiographic example from a ballet dancer with a subtle, transverse third metatarsal neck stress fracture, who was treated with rest, nutritional supplementation, and ESWT for 3 weeks, is provided. Figure 10.3a shows the metatarsal neck stress fracture, and Fig. 10.3b shows a follow-up standard radiograph at 3 weeks with abundant callus formation at the fracture site.

At this time, BSI clinical management following ESWT follows traditional BSI guidelines

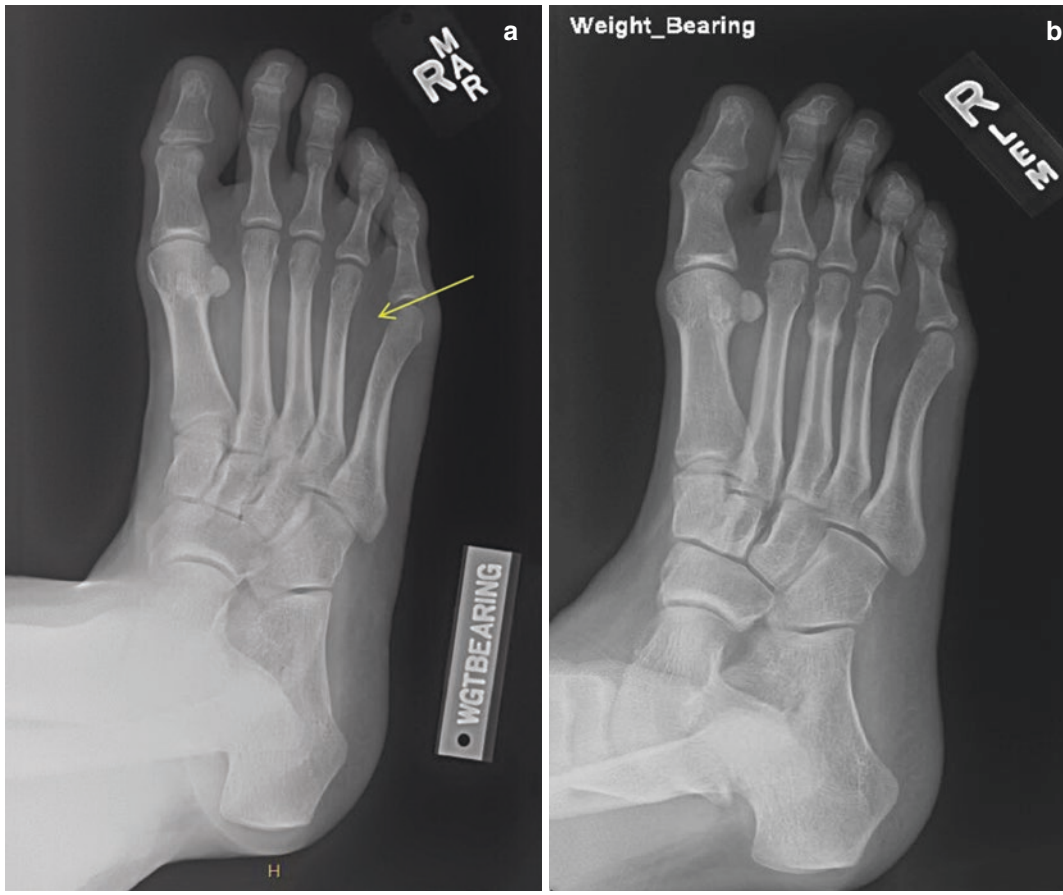

Fig. 10.3 (**a**) This weightbearing standard radiograph is from a 24-year-old ballet performer with 7 weeks of worsening forefoot pain. Standard radiograph revealed a subtle transverse 3rd metatarsal neck stress fracture. (**b**) The patient was treated with rest, nutritional supplementation and ESWT for 3 weeks. A new weightbearing standard radiograph at 3 weeks showed abundant callus formation at the fracture site

with a focus on early load control and specific rehabilitation exercises.

Benefit and Considerations

There are several case reports and studies that highlight encouraging results from ESWT in stress fracture management.

In 2007, Takai et al. used ESWT in five athletes with stress fractures refractory to conventional therapy. Stress fracture sites included the tibia, fifth metatarsal base, inferior pubic ramus, and the medial malleolus. ESWT was performed under anesthesia. In all five cases, radiographic consolidation occurred, with an average duration of bony union of 2.9 months. There were no complications or recurrences, and on average, athletes returned to competition at 4 months [24].

Abello and Leal presented a case report of a favorable outcome following ESWT in an Olympic gymnast with a navicular stress fracture [22]. A retrospective study examined ESWT in ten athletes with refractory stress fractures of the fifth metatarsal and tibia. Athletes received three to four sessions of mid-energy ESWT. At the 8-week follow-up, radiological healing was confirmed, and all athletes were able to gradually return to sports participation [25].

Although research studies show favorable outcomes with no reported complications for ESWT in stress fracture management, the population studied is still small. More research is needed before ESWT can be considered in clinical practice.

Bone Stimulation Devices

Mechanism of Action

In the 1950s, Fukada and Yasuda recognized the piezoelectric property of bone, which enables bone to generate electric charge in response to mechanical stress. By applying an outside electrical charge, the piezoelectric property of bone can be manipulated. A stronger net negative potential is theorized to stimulate bone growth [26].

The exact mechanism of action of electrical stimulation on the cellular and molecular level is still debated. Electromagnetic fields are thought to stimulate the production of osteogenic growth factors and promote the synthesis of extracellular matrix proteins that regulate gene transcription [27].

There are three different clinical approaches for electrical stimulation (E-stim) from bone stimulation devices: direct current (DC), capacitive coupling (CC), and inductive coupling (IC) or pulsed electromagnetic field (PEMF).

All three approaches induce an electric field at the fracture site. In DC E-stim, a cathode is implanted at the fracture site and attached to a subcutaneous or external power source. In CC E-stim, two electrodes are placed on either side of the fracture site and attached to an external power source. In IC E-stim (PEMF), an electromagnetic current carrying coil is placed on the skin overtop of the fracture site, and the magnetic field induces an electrical field [28].

Low-intensity pulsed ultrasound (LIPUS) (Fig. 10.4) is another bone stimulation modality used in fracture healing. Ultrasound applies

A runner using a low-intensity pulsed ultrasound (LIPUS) device for a second metatarsal stress fracture

Fig. 10.4 Bone stimulation device

transcutaneous acoustic energy, producing sound waves that are involved in tissue healing [29]. Overall, the waves induced by LIPUS convert to biochemical signals within cells that increase blood flow, angiogenesis, protein synthesis, calcium uptake, and osteogenic gene expression [30].

Clinical Use

Bone stimulation devices are primarily used in cases of delayed union and nonunion. According to a review of more than 100 studies of E-stim treatment, the most commonly used clinical approach was the noninvasive PEMF treatment [31]. There are level I studies to support PEMF treatment in nonunion. Three level I randomized controlled trials support the use of PEMF stimulation for tibia nonunion [32]. However, given that the level I studies have small sample sizes (16–23 patients in the PEMF treatment arm) as well as the overall quantity of level IV studies, PEMF stimulation carries a "B" grade of recommendation for the treatment of delayed union and nonunion [28].

The efficacy of LIPUS on bone healing outcomes remains uncertain. A systematic review and meta-analysis of 26 randomized controlled trials consisting of patients with any kind of fresh fracture or osteotomy found that LIPUS did not improve functional recovery outcomes, pain reduction, or radiographic healing compared with a sham device [33]. Further, a prospective, randomized, double-blind clinical study in Navy recruits (20 men, 13 women) with tibial stress fractures found that LIPUS did not significantly reduce healing time [34].

Insurance coverage varies for cases of noninvasive bone stimulators in nonunion. Many insurance companies require the fracture to have persisted for 3 months with a gap of less than 1 cm. The patient is expected to comply with immobilization and bone stimulation treatment protocols [35]. For many elite and professional athletes, the concern for cost of a bone stimulation device may be negated by the need for return to training and athletic participation as soon as possible. In this situation, its use may serve a psychological adjunct to assure the athlete that he or she is "doing everything possible" to help the injury heal quickly.

Insurance companies largely consider the treatment of delayed union and bone stress injury as investigational applications of noninvasive bone stimulators and as such do not cover patient treatment.

The doses, regimens, and exposure time of E-stim treatment vary widely, which is a reflection of differences between E-stim treatment, bone site, and fracture type. Treatment protocol can be decided based on selection of E-stim device.

Bone stimulation is considered an adjunctive therapy. Adhering to traditional BSI, delayed union, or nonunion guidelines with a focus on early load management and specific rehabilitation exercises is important for overall BSI outcome.

Benefit and Considerations

Although there are multiple randomized trials that support bone stimulation for delayed union and nonunion, the studies have small population sizes and are limited to predominantly radiographic endpoints [36].

In addition, bone stimulation has not been conclusively shown to enhance healing in stress fractures. A 2007 level I randomized controlled trial assigned 20 men and 24 women with tibial stress fractures to CC E-stim or placebo groups. There was no difference in time to healing between the treatment and placebo groups. Subjects with more severe stress fractures did heal more quickly with CC E-stim, but the result was not significant due to small sample size [37].

Cohort studies looking at bone stimulation in stress fractures have shown some beneficial results but are challenging to evaluate due to the lack of a control group [38]. In general, studies are challenged by patient compliance through inconsistent use and early termination of use.

Few complications of bone stimulation treatment have been reported in the literature. Increased pain, numbness, and tingling may

occur, but are rare, and not always associated with treatment itself.

A cost-benefit analysis should be evaluated prior to considering bone stimulation treatment. Bone stimulators are only covered by insurance under specific circumstances. Given the limited evidence for bone stimulation efficacy, treatment should only be considered in highly motivated patients with more severe BSI, delayed union, or nonunion who are willing to comply with treatment protocol.

Conclusion

BSIs are a common occurrence in athletes and can be season-ending injuries. Athletes with delayed healing, incomplete healing, nonunion, or repeat BSI may receive benefit from systemic treatment modalities. Teriparatide, ESWT, and bone stimulation devices are systemic treatment modalities that have been used in BSI management. Practitioners should consider the relevant evidence as well as the overall goals of the athlete prior to initiating treatment. In most circumstances, systemic treatment modalities are considered adjunctive therapy. The main focus of BSI management should involve early load management, rehabilitation principles, and risk factor optimization.

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11 Orthobiologic Treatment Options for Stress Fractures

Greg Robertson and Nicola Maffulli

Orthobiologics

Orthobiologic agents are a cohort of bioactive substances used to aid healing of musculoskeletal system ailments [1]. The orthobiologic agents relevant to the management of stress fractures are those which facilitate bone healing [2, 3] (Table 11.1). Specifically, these include bone graft, synthetic bone graft alternatives, growth factors, stem-cell based treatments, and cell-directing proteins [1, 2, 4].

Orthobiologic agents in bone healing must exhibit at least one of the three following properties: osteoconduction, osteoinduction, and osteogenesis [1, 2]. The use of these substances should be considered as an integrated part the four pillars of the "diamond concept" of bone healing [5]. The four pillars comprise osteogenesis (cell-containing substrates), osteoconduction (scaffold materials), osteoinduction (growth factor-containing substrates), and mechanical stability [5].

Osteogenic agents contain a population of native osteogenic cells to coordinate bone formation [1, 2]. Osteoinductive agents contain relevant growth factors to coordinate mesenchymal stem cell differentiation into osteogenic cells, facilitating bone formation [1, 2]. Osteoconductive agents provide a structural scaffold, on which the host's osteogenic system can integrate to allow bone formation [1, 2].

Orthobiologic agents currently in use for the promotion of bone healing in stress fractures include bone graft, bone marrow aspirate concentrate (BMAC), and bone morphogenic protein

Table 11.1 Orthobiologic agent use for stress fracture surgery

Orthobiologic agents	Recorded use in stress fracture surgery	Highest level of evidence
Autologous bone graft	✓	3
Allogenic bone graft	✓	4
Demineralized bone matrix	✓	4
Calcium ceramics	–	–
Bone marrow aspirate concentrate	✓	4
Bone morphogenetic protein	✓	4
Platelet-rich plasma	–	–
Platelet-derived growth factor	–	–

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Table 11.2 Properties of orthobiologic agents used in stress fracture surgery

Orthobiologic agent	Osteogenic	Osteoinductive	Osteoconductive
Autologous bone graft	✓	✓	✓
Allogenic bone graft	–	✓	✓
Demineralized bone matrix	–	✓	✓
Bone marrow aspirate concentrate	✓	✓	–
Bone morphogenetic protein	–	✓	–

(BMP) [1, 2, 4] (Table 11.2). The agents currently used in orthopedic practice, with a potential future role for the management of stress fractures, include platelet-rich plasma (PRP) and platelet-derived growth factor (PDGF) [1, 2, 4].

Bone Grafting

The two main types of bone graft used for stress fracture management are autologous bone graft and allogenic bone graft [2, 3, 6, 7].

Autologous bone graft is bone graft which has been harvested from the individual's own skeleton [2, 6, 8]. Given the biologically active and compatible nature of this material, it is considered the best form of bone graft to favor fracture healing [1]. Biologically, it confers osteogenicity, osteoinduction, and osteoconduction [2, 6, 8].

Autologous bone graft can be harvested from cancellous and cortical bone [1, 2]. Cancellous autograft is more biologically active than cortical autograft, offering improved contribution toward the healing process [1, 2]. However, cancellous autograft contributes limited structural support to the healing environment; in contrast, cortical autograft can provide significant structural support during healing [1, 2]. For the management of stress fractures, cancellous bone autograft is regularly used, given its superior contributions toward bone healing and the fact that structural support is often not required [2].

Autologous bone graft has been used for the management of stress fracture of the tibial diaphysis [9–16], navicular [17–22], metatarsal [23–27], toe sesamoid [28], lumbar spine [29–34], femoral neck [35–39], and olecranon [40–43]. The site of bone graft harvest varies, depending on the location of the fracture, though normally

Fig. 11.1 Autologous iliac crest cancellous bone graft

comprises either the iliac crest (Fig. 11.1) or local site-specific harvest sites [1, 2, 6]: both have been found to offer a similar quality of graft [44].

Allogenic bone graft is harvested from the skeleton of another individual [2]. This can offer both osteoconductive and osteoinductive properties to facilitate the healing process [1]. Similar to autologous bone graft, allogenic bone grafts can be divided into cortical and cancellous grafts [6]. Of the two, cancellous allogenic bone graft is predominantly used in stress fracture surgery [2]. Within the fracture site, the graft undergoes incorporation into the host bone through osteoconduction [1]. The graft can also provide a limited degree of structural support at the healing site in compression [2].

Cancellous allograft is often prepared intraoperatively from a donor femoral head and may be provided as preprepared allograft cancellous

chips [2]. The allogenic bone can be stored using a fresh frozen technique or a freeze-dried technique [2]. Fresh frozen processing preserves BMPs and improves the osteoinductive properties of the graft [2]. Freeze-dried processing reduces the immunogenicity of the product, limiting the potential for host–graft reactions: however, this is at the expense of depleted BMPs, reduced osteoinductive properties, and reduced structural support [2]. There is potential to augment the osteoinductive properties of allogenic bone graft, through the addition of BMP and BMAC [1]. However, the current literature would suggest that most authors avoid the need for such considerations, by using autograft over allograft, to supplement stress fracture fixation [2].

Site-Specific "Bone Grafting" Use

Tibial Diaphysis

The use of bone grafting in tibial diaphyseal stress fractures surgery has been reported for the management of anterior tibial diaphyseal stress fractures. The described techniques include drilling and grafting of the stress fracture site [9–11], excision of the stress fracture nonunion with grafting [14], biopsy of the fracture site and grafting [12, 13], and plating and bone grafting [13]. All but one of the studies used autologous bone graft [10], with the reported harvest sites including the lateral part of tibia [9] and the iliac crest [10]. Successful union was achieved in all studies [10–13], except Johansson et al. [9], who only recorded a 25% rate uneventful union. No adverse bone graft-related complications were reported [9–13].

Navicular

The use of bone graft in the management of navicular stress fractures has been reported for complete stress fractures [18–20], incomplete stress fractures [19, 20], sagittal plane stress fractures [17], delayed unions (presentation after 3 months of injury) [20], and nonunions [21].

The published techniques include debridement and bone grafting [13, 17, 19, 21], and debridement, with autologous bone graft and internal (screw) fixation [17, 18, 20–22] (Fig. 11.2).

Autologous bone graft was used in all [17, 18, 21, 22] but one study [18]. The bone graft-harvest sites included the calcaneus [18] and the iliac crest [21].

Success rates for those with debridement and grafting ranged from 67% [17] to 80% [19]. Success rates for those with debridement, grafting, and fixation ranged 67% [17] to 100% [20, 21]. Patients treated with allograft had a high rate of nonunion, with rates as high as 67% recorded [18]. Reported complications from bone grafting included incorrect positioning of the graft and sequestra [17].

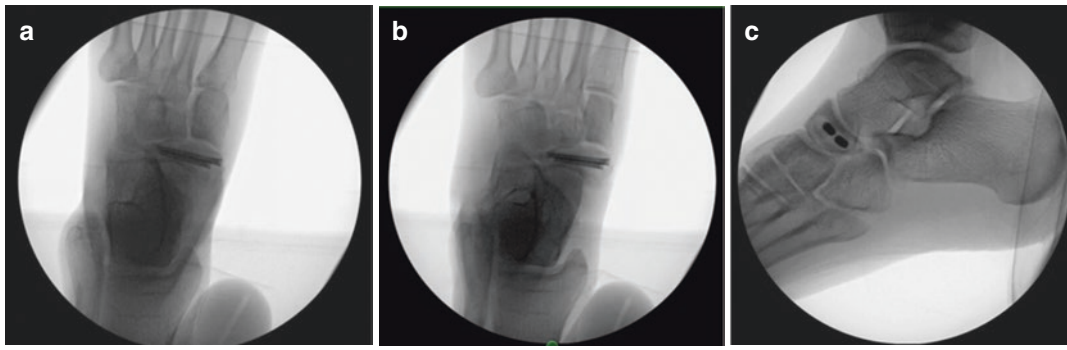

Fig. 11.2 (**a**–**c**): (**a**) Anteroposterior, (**b**) oblique, and (**c**) lateral intraoperative radiographs of a navicular stress fracture treated with screw fixation and autologous bone grafting

Metatarsal

The use of bone graft in the surgical management of metatarsal stress fractures has been widely reported, with study cohorts comprising stress fractures of the fifth metatarsal [23–25], the fourth metatarsal [26], and the second metatarsal [27]. Most studies reported on the treatment of stress fracture nonunions [25–27].

For fifth metatarsal stress fractures, the described techniques include intramedullary screw fixation with autologous cancellous bone graft (from the posterolateral aspect of the os calcis) packed into the fracture site and bone marrow aspirate concentrate injected into the intramedullary canal and fracture site [23]; open intramedullary screw fixation, with packing of autologous cancellous bone graft (from the iliac crest), into the fracture site [24]; and modified tension band wiring, with packing of the fracture site using autologous corticocancellous bone graft (harvested from the anterior process of the calcaneus) [25]. Reported union rates ranged from 92 [25] to 100% [24].

For fourth metatarsal stress fractures, the described techniques included debridement of the fracture, packing of autologous calcaneal bone graft into the fracture site, then stabilization with a dorsolateral mini-fragment bridging or locking plate [26]. All patients achieved successful union [26].

For second metatarsal stress fractures, the described techniques included debridement of the fracture site, packing of the site with autologous corticocancellous bone graft, and fixation with a narrow five-hole low-contact dynamic compression plate [27]. The patient achieved successful union [27].

All of the studies used autologous bone graft [23–27]. The harvest sites included the calcaneus [23, 25, 26] (specifically the posterolateral aspect [23] and the anterior process [25]) and the iliac crest [24].

Only one study, a level 3 investigation, compared the effect of bone grafting in stress fracture surgery [24]. The authors performed a retrospective cohort study, to compare the outcome of percutaneous intramedullary screw fixation of fifth metatarsal stress fractures ($n = 11$) to open intramedullary screw fixation with autologous iliac crest cancellous bone grafting ($n = 7$) in soccer players. The mean time to clinical healing was similar for both groups: 6 weeks (range, 5–8) (percutaneous fixation) vs 7.1 weeks (range, 6–9.2) (open fixation). The mean time to radiographic union was also similar for both groups: 11.3 weeks (range, 9–11.8) (percutaneous fixation) vs 10.1 weeks (range, 8.6–10.5) (open fixation). The mean return time to sport for the two groups was 12 weeks. Regarding complications, refracture was reported in three players from the percutaneous group, occurring 4.5, 5.2, and 6.0 months following return to full-level sport. They all required revision screw fixation with autologous cancellous bone grafting. None of the patients in the open group suffered refracture. There were no cases of nonunion reported in either group. The author concluded that open intramedullary screw fixation with autologous iliac crest cancellous bone grafting could be recommended as primary management of fifth metatarsal stress fracture in elite athletes.

Great Toe Sesamoid

The use of bone grafting as treatment for stress fractures of the great toe sesamoid has been reported by Anderson and McBryde [28]. The authors managed 21 patients with hallux sesamoid nonunions, using curettage of the fracture site followed by packing of the defect with autologous bone graft, harvested from the medial aspect of the first metatarsal head [28]. Union was noted in 19 of the 21 fractures: the 2 patients who suffered nonunions both demonstrated excessive fracture site motion intraoperatively, and both underwent subsequent sesamoidectomy [28]. Sporting follow-up was performed for 20 patients, with 17 of the 20 patients returning to preinjury sports the two nonunion patients returned to preinjury sporting level after sesamoid excision [28].

Spondylolysis

The use of bone graft in the surgical management of stress fractures of the spine is predominantly reported from the management of spondylolysis.

The published techniques include direct posterior-based segmental wire fixation (Scott technique) with autologous iliac bone graft [29]; direct intralaminar lag screw fixation (Buck technique) with autologous cancellous iliac crest bone grafting [30–32]; direct pars repair with variable angle pedicle screw and sub-laminar hook using autologous iliac bone graft [33];, and indirect pars repair with pedicle screw instrumentation and laminar compression hook constructs, using local spinous process autologous bone graft [34].

All studies used autologous bone grafting [29, 30, 32–34], with the recorded harvest sites including the iliac crest [29, 30, 32, 33] and local spinous process autograft [34]. One hundred percent union rates were reported when a direct pars defect repair was undertaken [29–33]; the study which used an indirect repair method reported a union rate of 78% [34]. No bone graft-related complications were reported [29, 30, 32–34].

Femoral Neck

There is limited evidence on the use of bone graft for primary surgical fixation of femoral neck stress fractures (FNSF) [45]. Historically, bone graft has been used in conjunction with trifin nail fixation, with good results [35]. However, the use of bone grafting for the primary management of these injuries is not routine practice, given the standard technique of closed reduction and dynamic hip screw fixation or cannulated screw fixation [46, 47]. Curettage or reaming of the fracture site, though, can be considered to promote an osteogenic reaction, which may aid fracture healing [48].

Bone grafting can, however, be considered for use in the management of FNSFs with delayed presentation [36]. Lee et al. [36] reported three cases of FNSFs in military recruits, with delayed presentation (5 or more days between injury and surgery): each was treated by open reduction and cannulated hip screw fixation, with a quadratus femoris muscle pedicle bone graft [36]. All three patients subsequently developed avascular necrosis of the femoral head, with a poor functional outcome, and all required secondary arthroplasty procedures [36]. The delay was the likely cause of the high rate of avascular necrosis [36].

Bone grafting has been regularly recorded for use in revision procedures, as part of the management of delayed union or nonunion of surgically treated FNSFs. The techniques described included bone grafting with compression screw fixation [37], iliac crest cancellous bone grafting with a compression nail-plate system [38], and cancellous autograft bone grafting with cannulated hip screws [39]: satisfactory union was reported in all studies [37–39].

Upper Limb

The evidence for the use of bone grafting in the management of stress fractures of the upper limb is limited [49]. The published studies focus on olecranon stress fractures [43, 50] and olecranon epiphyseal stress fractures [40–42]. The published techniques include open biopsy and autologous bone graft [40, 41], cannulated cancellous compression screw with local autologous bone graft [42], and titanium screw fixation with iliac crest bone grafting [43]. Bone graft harvesting was performed from the iliac crest [43] and from local upper limb sites [42]. Successful union was reported in all cases, with no bone graft-related complications recorded [40–43, 50].

Bone Marrow Aspirate Concentrate

Autologous bone marrow aspirate concentrate is a developing orthobiologic in the field of stress fracture surgery [23, 51–53].

Harvested from the patient's red marrow within the skeletal system, this product contains mesenchymal stem cells, hematopoietic stem cells, and various osteogenic growth factors including vascular endothelial growth factor

(VEGF), transforming growth factor-β (TGF-β), and platelet-derived growth factor (PDGF) [1, 2, 54]. These factors have been hypothesized to confer a beneficial effect to fracture healing, and, through the intraoperative use of BMAC during stress fracture surgery, surgeons can provide a concentrated medium of these factors to the fracture site [23, 51]. The evidence to support the use of BMAC in stress fracture surgery remains, however, limited [23, 51–53].

The theoretical benefit of BMAC is that bone marrow stem cells and the associated growth factors play a major role in the inflammatory reaction that precedes fracture healing [1, 2]. The mesenchymal stem cells also play a key role in osteogenesis, forming first osteoprogenitor cells, and then osteoblasts, when exposed to the appropriate growth factors and cytokines [1, 2]. Furthermore, osteogenic mechanisms of BMAC are likely due to cytokine-induced promotion of host cells, which, in conjunction with BMAC's endothelial progenitor cells, promote angiogenesis to improve the blood supply to the fracture [1, 2]. As such, it is theorized that the introduction of BMAC into the fracture site will promote fracture healing [3]. However, the exact mechanisms by which bone marrow stem cells influence fracture healing remains to be defined [55, 56]. While data have been suggestive of the benefit of BMAC in the management of nonunion and delayed unions [56–58], there has very limited data specifically focusing on stress fractures [23, 51–53]. To note, while BMAC application has been shown to be beneficial to fracture healing in vitro and in vivo, harvesting techniques remain varied, and the concentration of BMAC is inconsistent throughout studies [53, 55].

The concentration of the bone marrow aspirate seems to exert a positive influence on the concentration of osteogenic components [1, 2]. Pre-concentrate aspirate has a concentration of 612 progenitor cells/cm^3, while post-concentrate aspirate concentration increases to 2579 progenitor cells/cm^3 [57]. Specifically, the number and concentration of progenitor cells significantly affect bone healing in nonunion treatment: increased rates of treatment failure have been found with BMAC samples containing less than 1000 progenitors/ cm^3 and less than 30,000 progenitors in total [57]. The integration of BMAC within a solid "osteoconductive" material such cancellous autologous bone graft or demineralized bone graft can allow more controlled application of the concentrate [1].

The harvest process for BMAC from the iliac crest is as follows [52]:

1. Routine surgical preparation and draping.
2. Palpate the medial and lateral borders of the iliac crest.
3. Define the center of the iliac crest.
4. Make a 1 cm incision over this area (approximately 2 cm posterior to the ipsilateral anterior superior iliac spine).
5. Insert a sharp "BMAC harvesting" trocar through the incision.
6. Ensure the trocar is centered over the iliac crest.
7. Insert the trocar through the outer cortex, to a depth of 3 to 4 cm, remaining between the outer and inner cortices of the iliac crest.
8. The sharp trocar can be replaced with a blunt trocar, once the outer cortex has been breached.
9. A cannula should then be advanced down the blunt trocar into the cancellous bone.
10. The trocar can then be removed.
11. 5–10 mL quantities of bone marrow can then aspirated sequentially, with the cannula gradually withdrawn before each aspiration
12. With no further aspirate obtained, the trocar can be reinserted into cancellous cavity, at a different trajectory, with the process repeated.
13. This can be performed several times to achieve the desired volume.
14. The aspirate is then sterilely decanted, followed by processing and concentrating with a centrifuge.
15. The cell plasma is siphoned from the nucleated cell concentrate, to provide the BMAC.
16. Most authors aspirate around 30–60 mL of bone marrow, which provides between 5 and 10 mL of BMAC (Fig. 11.3).

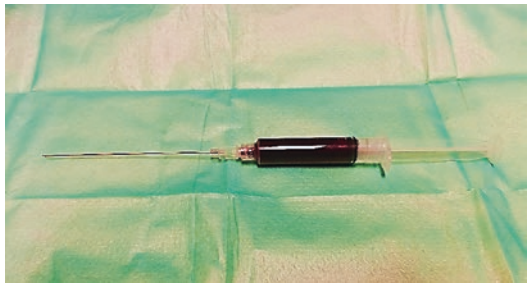

Fig. 11.3 A "bone marrow aspirate concentrate" sample prior to injection

This technique could be applied to any appropriate skeletal site, such as the tibia or calcaneus [52]. However, the iliac crest provides sufficient volumes of marrow aspirate for BMAC production, without the risk of increased fracture in a long bone [52]. Harvesting from the iliac crest does have potential complications, which include intra-abdominal injury, secondary to trocar misplacement [52], and a potential risk of heterotrophic ossification from leakage of the BMAC as the needle is withdrawn [52]. In practice, the reported rate of complications with this technique is very low [59]. Considering the potential risks, this method allows for ease of harvest, low infection risk, and prevents allogenic reactions [60].

With this minimally invasive technique to harvest BMAC, and the hypothesized osteogenic benefits from stem cells and growth factors provision, the potential for BMAC to provide significant benefit to the future of stress fracture surgery remains significant [23, 51–53].

Site-Specific "Bone Marrow Aspirate Concentrate" Use

Metatarsal (Fig. 11.4)

The evidence for the use of BMAC in the surgical management of metatarsal stress fractures is limited, with only one study on fifth metatarsal stress fracture recording its use [23].

Miller et al. [23] performed a retrospective review of prospectively collected data on a series of 37 elite-level professional football players, who underwent intramedullary screw fixation of fifth metatarsal stress fractures. The surgical technique comprised intramedullary screw fixation, with autologous cancellous bone graft (from the posterolateral aspect of the os calcis) packed into the fracture site and bone marrow aspirate concentrate injected into the intramedullary canal and the fracture site [23]. At a minimum follow-up of 24 months, the mean return to play was 10.5 weeks, and mean time to complete radiological union was 12.7 weeks [23]. The union rate for the cohort was 97% [23]. One patient sustained a refracture 10 months postfixation. There were no BMAC-related complications reported [23].

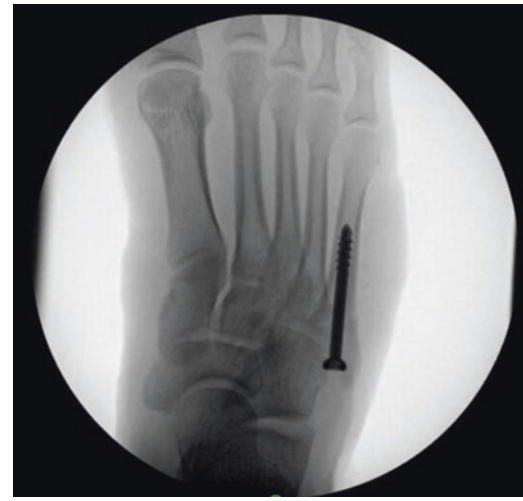

Fig. 11.4 An oblique intraoperative radiograph of a fifth metatarsal stress fracture treated with intramedullary screw fixation and injection of bone marrow aspirate concentrate to the fracture site

Weel et al. [61] are currently conducting a randomized controlled trial to assess the effect of BMAC on surgical fixation of fifth metatarsal stress fractures. One hundred patients with fifth metatarsal stress fractures will undergo surgical fixation with a cannulated intramedullary compression screw and internal bone graft: 50 patients will receive concentrated blood and BMAC, harvested from the iliac crest, to the fracture site, while the other 50 patients will not receive the adjunctive treatment but will undergo a placebo procedure to the iliac crest. This will provide the highest level of evidence to date regarding the effectivity of BMAC in stress fracture surgery.

Medial Malleolus

The use of BMAC in the surgical management of medial malleolar stress fractures has only be reported in one case series [51].

Nguyen et al. [51] reported on 16 professional soccer players with stress fractures of the medial malleolus. The surgical technique comprised open reduction and internal fixation of the fracture with three partially threaded cancellous screws, arthroscopic debridement of impingement spur, and injection of BMAC from the iliac crest into the fracture site [51]. At 12 months follow-up, the study cohort achieved 100% clinical union and 88% radiographic union [51]. All athletes were able to return to sport at a mean of 4.1 months, and all patients returned to professional football at their preinjury level [51]. There were no BMAC-related complications reported [51].

Navicular

There is no current evidence to guide the use of BMAC for navicular stress fractures [53]. Expert advice has advised for it to be used for Saxena Type II (incomplete) and III (complete) fractures [53]. The recommended surgical technique comprises open reduction with screw fixation, with application of iliac crest-harvested BMAC and autologous bone graft to the fracture site [53]. The bone graft can be harvested percutaneously using the trocar used for BMAC aspiration [53].

Cuneiform

The use of BMAC in the surgical management of cuneiform stress fractures has been reported in only one case report [52].

The patient presented with a medial cuneiform stress fracture, without associated Lisfranc ligament injury [52]. The surgical technique comprised fracture site drilling with a 0.062-inch Kirschner wire, cannulated screw fixation across the fracture, and injection of BMAC, harvested from the iliac crest, into the fracture site [52]. Union of the fracture was achieved by 10 weeks postsurgery [52]. No complications were recorded.

Tibial Diaphysis (Fig. 11.5)

The use of BMAC in tibial diaphyseal stress fracture surgery is limited to a single case series of elite dancers with anterior tibial diaphyseal stress fractures [10]. The described technique comprised drilling of the stress fracture followed by packing of the fracture site with iliac crest aspirate in conjunction with either iliac crest bone graft, BMP-7, or demineralized bone matrix putty with cancellous allograft [10]. Successful union was achieved in all five cases [10]. Clinical union was achieved at a mean of 5.2 weeks and radiographic union at 5.3 months [10]. Return to full dance activity was at a mean of 5.5 months postoperatively [10]. No BMAC-related complications were recorded [10].

Gigis et al. [15] reported a case report of a professional soccer player with an anterior tibial stress fracture, treated with fracture site drilling and intramedullary nailing. The authors additionally inserted autologous growth factors through the drilling holes, but this is not formally described as BMAC [15]. Radiographic union was noted 2 months postoperatively, and the player returned to full activity 9 weeks postoperatively [15].

Bone Morphogenetic Protein

Bone morphogenetic proteins are growth factors part of the transforming growth factor beta (TGF-b) superfamily [1–3]. They facilitate bone healing through osteoinductive and osteogenic mechanisms [1–3]. A key group of morphogenetic signaling proteins, BMPs contribute a significant influence over osteoblast differentiation and angiogenesis which serve as important factors in their ability to induce bone formation [1–3]. Synthesized as a liquid, BMPs require integration with a structural scaffold (i.e., calcium ceramic or collagen matrix), to allow it to be retained at the surgical site [1–3].

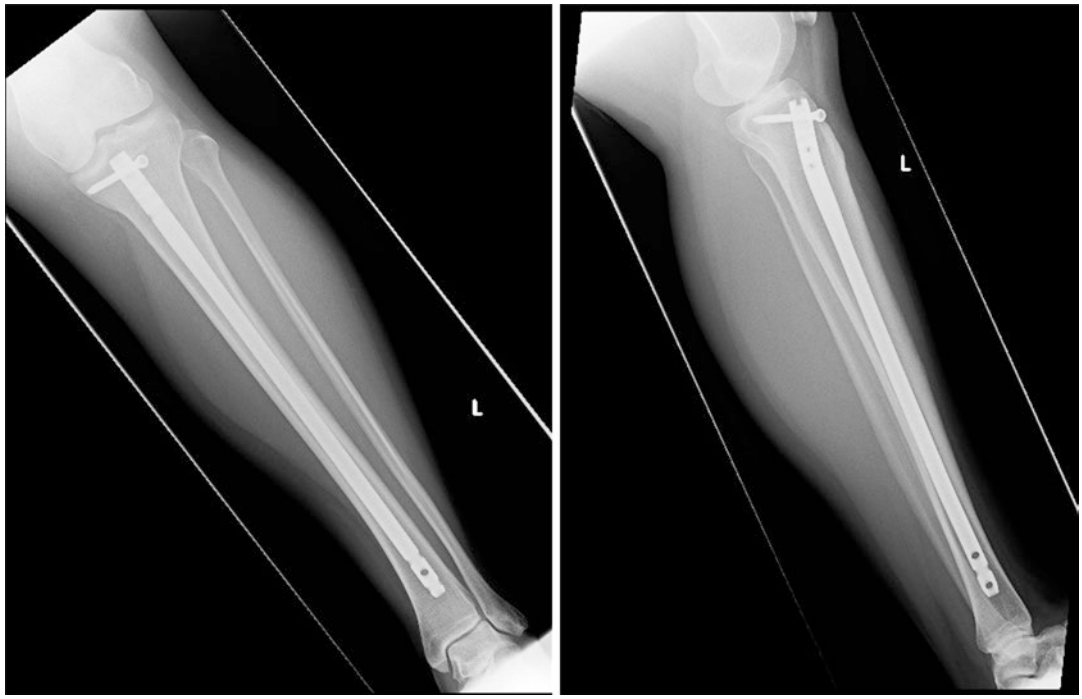

Fig. 11.5 Anteroposterior and lateral follow-up radiographs of an anterior tibial diaphyseal stress fracture treated with fracture site drilling, intramedullary nailing, and injection of bone marrow aspirate concentrate to the fracture site

There are two BMPs approved for clinical use: BMP-2 and BMP-7 [62, 63]. The patented use for these include open tibia fracture surgery, nonunion management, and spinal fusion [62, 63]. The use of this substrate remains limited, despite the positive evidence regarding its effects on bone healing [1]. This is in part due to its high cost, as well as due to its side effect profile [1]. Key side effects include an increased risk of heterotrophic ossification, increased risk of carcinogenicity, renal and liver impairment, and compartment syndrome [2–4, 64]. The benefit of BMP in stress fracture surgery remains to be confirmed [2].

Site-Specific "Bone Morphogenetic Protein" Use

Spondylolysis

There is limited evidence for the use of BMP in spinal stress fracture surgery, with only one study reporting the use of BMP for surgical management of spondylolysis [65]. Gillis et al. [65] reported on seven high-level athletes and one member of the National Guard with symptomatic spondylolysis. The authors described a minimally invasive surgical technique to directly repair the pars defect which comprised debridement of the pars defect, packing of the site with corticocancellous bone graft and BMP, and pedicle screw insertion and subsequent tensioning using a Dynesys® cord, threaded through the head of the screws [65]. Complete union was achieved in six of the eight patients: those six patients were able to return to their preinjury level of sport, while the two patients in whom union did not occur did not return to their preinjury sporting level [65]. There were no BMP-related complications reported [65].

Tibial Diaphysis

The use of BMP in tibial diaphyseal stress fractures surgery is limited to a single case series, which reported on elite dancers with anterior tibial diaphyseal stress fractures [10]. The described technique comprised drilling of the stress fracture, followed by packing of the fracture site with iliac

crest aspirate in conjunction with BMP-7 [10]. Successful union was achieved in both cases [10]. Clinical union was achieved at a mean of 5.5 weeks and radiographic union at 6.5 months [10]. Return to full dance activity was performed by a mean of 6.5 months postoperatively [10]. There were no BMP-related complications reported [10].

Bone Graft Substitute

A number of bone graft substitutes exist, including synthetic calcium salt-based substitutes and demineralized bone matrix (DBM) [1–3, 7, 62].

Calcium phosphate, tricalcium phosphate, calcium sulfate, and coralline hydroxyapatite are all commercially available synthetic bone graft substitutes [3, 7, 62]. In fracture surgery, these compounds are used for their mechanical properties, often serving as fillers for bone loss, while providing osteoconductive potential during the healing process [1, 2]. These substances can be biologically enhanced, through the integration of BMAC, BMPs, or PRP: such compounds then provide the triad of osteoinduction, osteogenesis, and osteoconduction [3, 7, 62].

DBM is a form of bone graft substitute, comprising allogenic bone graft which has been purged of calcium hydroxyapatite [1–3, 7, 62]. However, several organic components including BMPs and other growth factors are retained [1, 2]. As such, this material contributes both osteoconductive and osteoinductive properties toward the healing process [3, 7, 62]. In acute fracture surgery, DBM has similar healing potential to autologous bone grafting [66]. In nonunion surgery, when combined with BMAC, DBM demonstrated improved healing potential over BMPs [67].

The role of bone graft substitutes in stress fracture surgery, however, remains to be defined [2].

Navicular

There is only one report of bone graft substitute use in the management of navicular stress fractures. Saxena et al. [22] retrospectively reviewed the data from 22 navicular stress fractures sustained during athletic activity: nine of these patients underwent surgical fixation, one of which had adjunctive bone graft substitute. The surgical technique involved fracture site debridement, packing of the fracture site with autogenous bone graft (from the ipsilateral calcaneus), or bone graft substitute followed by cancellous screw fixation if further fracture compression was required [22]. For the surgical cohort, the mean return to activity (RTA) time was 3.1 ± 1.2 months (range, 1.5–5 months) [22]. No complications related to bone graft substitute use were reported [22].

Tibial Diaphysis

The use of demineralized bone matrix in tibial diaphyseal stress fractures surgery is limited to two case series, one reporting on elite dancers [10] and the other on world-class female athletes [16]. Both reported on anterior tibial diaphyseal stress fractures [10, 16]. The described surgical techniques included drilling of the stress fracture, followed by packing of the fracture site with iliac crest aspirate, demineralized bone matrix putty, and cancellous allograft [10] along with compression plating of the anterolateral aspect of the tibia with 3.5 mm six-hole Synthes plates with packing of demineralized bone matrix into the fracture site [16].

Successful union and return to preinjury activities were achieved in all cases [10, 16]. Mean return times to full activity ranged 10 weeks [16] to 5.3 months [10].

Future Orthobiologic Treatment Options for Stress Fractures

Platelet-Rich Plasma

Platelet-rich plasma (PRP) has a number of uses in sports medicine, particularly in the field of tendinopathy and ligament healing [1, 4, 68]. However, its use in stress fracture surgery has yet to be validated [2].

PRP is defined as a sample of plasma which has a platelet count greater than peripheral blood

[1, 68]. It is most commonly prepared through venipuncture of the patient's autologous blood, followed by centrifugation, then extraction of the platelet-concentrated plasma [1, 3, 4, 68].

As an orthobiologic, it has osteoinductive capacities, as the platelets (i.e., alpha granules) contain a number of active growth factors, including transforming growth factor B1, platelet-derived growth factor, epidermal growth factor, vascular endothelial growth factor, insulin-like growth factor 1, and fibroblast growth factor [1, 4, 68].

Regarding the use of PRP in fracture surgery, positive results have been found when used in the treatment of long-bone atrophic nonunions. This has been observed both with direct injection to the nonunion site (87% union rate at 4 months) [69] and when combined with exchange intramedullary nailing (93% union rate with PrP vs 80% union rate without PRP) [70]. However, there are no published studies which record the use of PRP specifically in stress fracture surgery [2].

A key factor that still requires addressing in the use of PRP is the significant variability in product preparation and composition between studies [1, 4, 68, 71]. This has been highlighted in a recent systematic review on the topic, and future work should aim at creating standardized protocols for the preparation, analysis, and use of this orthobiologic [71].

Platelet-Derived Growth Factor

Platelet-derived growth factor is a potent growth factor that regulates osteoblast proliferation and plays a significant role in angiogenesis related to fracture healing [1, 2]. Released from degranulating platelets during the commencement of fracture healing, its positive contribution toward fracture healing has been capitalized upon in orthobiologic practice [72, 73].

Recombinant human PDGF (rhPDGF) has been used both in animal studies and in human studies [74, 75]. Improved healing characteristics were demonstrated in tibia fractures in rats with the use of rhPDGF [74]. However, no significant improved healing characteristics were demonstrated with the use of rhPDGF (compared to control) in distal radial fractures in humans [75]. No adverse side effects were recorded though, with the additional use of rhPDGF [75].

There are no published studies of the use of rhPDGF in stress fracture surgery, and so the potential benefit of this orthobiologic in the stress fracture management remains to be confirmed [1, 2].

Conclusion

Orthobiologic agents with relevance to stress fracture surgery are those which can facilitate the fracture healing process. These include bone graft, synthetic bone graft, bone marrow aspirate concentrate, bone morphogenetic proteins, platelet-rich plasma, and platelet-derived growth factor.

There is well-established evidence to support the safe use of autologous bone grafting in the surgical management of stress fractures of the tibial diaphysis, metatarsal, navicular, great toe sesamoid, lumbar spine, and olecranon. There is growing evidence to support the safe use of bone marrow aspirate concentrate in the surgical management of stress fractures of the fifth metatarsal, medial malleolus, and cuneiform. There is also evidence to support the safe use of bone morphogenetic protein in the surgical management of stress fractures of the lumbar spine and tibial diaphysis. However, none of the evidence confirms that the use of orthobiologic agents provides superior healing properties to non-orthobiologic treatment. Additionally, donor site morbidity remains a potential issue related to autologous bone harvesting.

Potential future orthobiologic agents in the field of stress fracture surgery include platelet-rich plasma and platelet-derived growth factor.

Orthobiologic agents appear to be a safe adjunct to stress fracture surgery. However, it is unclear whether their use actually enhances the healing process.

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Part III

Management of Common Stress Fracture Sites

Stress Fractures of the Ribs and Shoulder Girdle

12

Alex C. Dibartola, Gregory L. Cvetanovich, and Timothy L. Miller

Introduction

Stress fractures have been historically regarded as predominantly occurring in the lower extremities secondary to the repetitive impact loading, frequently occurring due to walking, running, or jumping [1–3]. Stress injuries of the ribs and shoulder girdle are much less commonly reported, and subsequently these injuries are often omitted from the differential diagnosis of rib or upper extremity pain. Few case series have described the precipitating activities and common locations of these injuries given their rarity. The two largest series of rib and upper extremity stress fractures include 44 cases described by Sinha and associates [7] and Miller and Kaeding's case series of 70 patients [8]. Sinha et al. described four categories of athletic activities at risk: (1) weight lifters, (2) weight bearers, (3) throwers, and (4) swingers. Miller and Kaeding further categorized the causative activities of rib and upper extremity stress fractures into (1) axial rotators, (2) rowers, (3) overhand throwers, (4) weight bearers, and (5) weight lifters.

While rib and upper extremity stress fractures are uncommon, they can be troublesome injuries for athletes and manual laborers [1–3, 5, 6]. As awareness of overuse injuries of the thorax and shoulder girdle increases, so does the rate of diagnosis of stress fractures of the ribs and upper extremities [8]. Appropriate evaluation of these injuries requires a thorough history and physical examination. Radiographs may be initially negative, requiring bone scintigraphy or MRI to confirm the diagnosis. Nonoperative and operative treatment recommendations are made based on location, injury classification, and causative activity. To prevent diagnosis and treatment delay, clinicians should be aware of the common precipitating mechanisms and locations of these injuries, as well as the indications for operative and nonoperative treatment.

Rib and shoulder stress fractures are diverse in their presentation, appearance, and healing potential. Stress injuries to bone represent a continuum of mechanical failure ranging from simple bone marrow edema (stress reaction) to a small microcrack with minor cortical disruption to a complete fracture with or without displacement to nonunion. Most of the literature related to stress fractures of the ribs and upper extremities are limited to case reports and small case series.

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Risk Factors

In the shoulder girdle and ribs, strain is generated by the rotational torque of swinging or throwing and by the tensile and compressive forces produced from muscle contraction [9]. Additionally, repetitive axial loading of upper extremity may generate forces sufficient to produce microtrauma to bone. Muscle function influences the amount of energy directly absorbed by the bones and joints, thereby affecting their susceptibility to stress injury. As muscles fatigue, they are less able to dissipate externally applied forces. Two key modifiable risk factors for these injuries are pre-participation conditioning and the volume (frequency, duration, and intensity) of the causative activity [10]. Neuromuscular conditioning plays a significant role in enhancing the shock absorbing and energy-dissipating function of muscles and soft tissues [11]. Other predisposing factors include abnormal bony alignment, muscular imbalance, improper technique/biomechanics, and poor blood supply to specific bones [11].

Clinical Presentation

Athletes with atraumatic shoulder or chest wall discomfort associated with repetitive activity should be evaluated with concern for possible stress fracture [5, 11, 12]. Causative mechanisms may involve repetitive and resisted scapular retraction, humeral torsion (e.g., pitcher, javelin), or weight-bearing (e.g., cheerleading, gymnastics). Muscle contraction may generate both compressive and tensile loads on the skeletal structures of the thorax and upper extremity leading to stress injuries of bone [11]. Typically, patients cannot recall a specific injury or trauma to the injury site, and onset is most often insidious as in the lower extremities. Given the relative infrequency of rib and shoulder girdle stress fractures, the potential for concomitant soft tissue overuse injuries is high and must be diagnosed and treated if present.

Athletes with stress fractures of the ribs and shoulder girdle present initially with pain that is present only during the inciting activity [5]. If the activity level is not decreased or modified, symptoms usually persist or worsen. Those individuals who continue to train and compete without modification of activities may develop pain with activities of daily living and may even progress to complete fracture, with or without displacement [11].

Physical Examination

The physical examination performed for a suspected rib or shoulder girdle stress fracture should include evaluation of the neck, chest, heart, lungs, and abdomen to rule out non-musculoskeletal causes of shoulder, rib, and thoracic pain. Examination should begin with a thorough inspection of the skin and soft tissues. Palpation for tenderness, assessment of active and passive range of motion, and strength testing should be performed for all affected bones and joints of the cervical spine, scapulothoracic joint, sternoclavicular joint, acromioclavicular joint, glenohumeral joint, and elbow. Unlike non-musculoskeletal sources of pain, stress fractures often produce reproducible point tenderness at the affected site. Soft tissue or bony swelling also may be present.

In the early stages of the injury, it may be necessary to have the patient perform or recreate the causative activity in order to reproduce the symptoms [11]. Any biomechanical causes of injury, including muscle imbalance or abnormal mechanics of the throwing or rowing motion, should be noted. A thorough neurovascular exam is essential because vague exertional upper extremity pain may be due to peripheral nerve entrapment and/or peripheral vascular disease or other vascular etiologies such as deep vein thrombosis and thoracic outlet syndrome [11].

Differential Diagnosis

The most common differential diagnoses for rib stress fractures include the following [11]:

- Costochondritis
- Intercostal neuralgia
- Intervertebral disk pathology
- Skin infection (i.e. methicillin-resistant *Staphylococcus aureus*)
- Herpes zoster
- Cardiac-related chest pain
- Pneumothorax
- Peripheral vascular disease
- Pleuritis
- Tumors

Causative Activities

Muscle contraction in the upper extremity and thorax produces tensile, compressive, and rotational stress on bone. Throwing and/or swinging motions are the two most common inciting activities to generate these forces [8]. Less common mechanisms of creating bone stress in the shoulder include repetitive axial loading, resisted retraction of the scapula, and weight lifting [8].

Miller and Kaeding reported on 70 cases of upper extremity stress fractures in skeletally mature patients collected over a 10-year period [8]. Analysis of these cases demonstrated no patterns for causative activities. Injury mechanisms for the majority of the patients with rib and upper extremity stress fractures fall into one of the following five categories: (1) upper extremity weight bearers (gymnastics, cheerleading), (2) rowers, (3) axial rotators (golf, tennis, discus), (4) overhead throwers, and (5) weightlifters. The distribution of these injuries is detailed in Table 12.1 [11].

In this case series, rib stress fractures appear to be a common problem among rowers, as all 11 rowers were diagnosed with stress fractures of the ribs. Ten of the eleven developed their stress fractures in the lower ribs. Five of the eleven rowers developed stress fractures in multiple lower ribs. Like rowers, the axial rotator group showed a strong predilection for fractures of the ribs (7/10). Among overhead throwers, injuries were more common about the elbow (9/16).

Weightlifters showed the greatest anatomical variability for location of injury, with injuries occurring as far proximal as the sternum and as far distal as the scaphoid. This group also showed a significantly disproportionate number of rib and shoulder girdle stress fractures (13/24). Notably, they sustained more injuries to the first rib (7/24) than any other group. While a clear mechanism for such injury patterns cannot be determined, a wide variety of repetitive bending, and torsional and axial loading forces applied to the thorax and upper extremity during weight training may contribute to the breadth of injuries this group sustains [11].

Table 12.1 Anatomic distribution of rib and upper extremity stress fractures by causative activity

Weight bearer ($n = 12$)	Rower and axial rotator ($n = 21$)	Overhand throwers ($n = 24$)	Weight lifter ($n = 14$)	Miscellaneous ($n = 7$)
Olecranon (1)	First rib (2)	Clavicle (2) Scapula (1)	Acromion (4)	Phalanx (1)
Ulna shaft (3)	Lower ribs (16)	First rib (3)	First rib (7)	Ulna shaft (3)
Distal radius (2)	Ulnar shaft (1)	Lower ribs (1)	Proximal humerus (1)	Distal humerus (2)
Scaphoid (3)	Radial shaft (1)	Distal humerus (5)	Ulna shaft (2)	Metacarpal (1)
First rib (1)	Metacarpal (1)	Olecranon (4)		
Sternum (1)		Scaphoid (4)		
Distal humerus (1)		Sternum (2)		
		Proximal radius (1)		
		Coracoid (1)		

Adapted from Miller et al. [8]

Stress Fracture of the Ribs

Rib stress fractures have been reported in several sports, including discus, rowing, rugby, golf, weightlifting, volleyball, gymnastics, judo, tennis, table tennis, baseball, basketball, soccer, javelin, backpacking, and wind surfing [5, 8]. Tensile muscular forces (rather than axial compressive forces) are predominantly responsible for rib stress fractures, as ribs are non-weight-bearing bones [9]. The most common sites of fracture include the anterolateral first rib, the posterolateral fourth through ninth ribs, and the posterolateral upper ribs [11, 12].

First Rib

The sports most commonly associated with first rib stress fractures (Fig. 12.1a, b) – such as baseball pitching, swimming, basketball, lacrosse, weightlifting, ballet, javelin, kayaking, and tennis – often involve repetitive overhead positioning of the arm [8, 13–16]. And while these injuries are, like most stress fractures of the upper extremity, thought to be rare, some sports may result in higher rates of first rib stress fractures. Elite rowers for example have an overall career first-rib stress fracture rate of 8–9% [17]. This highlights the importance of sport-specific diagnostic suspicion.

These fractures are almost always reported on the patient's throwing or dominant limb [18]. Patients with first-rib stress fractures present with insidious onset of dull, vague pain in the anterior cervical triangle and mid-clavicular region, with occasional radiation to the sternum and pectoral region [12]. Repetitive scalene muscle contractions elevate the first rib, while actions of the serratus anterior and intercostal muscles depress it [19]. These opposing forces generate bending and torsional forces leading to microtrauma. A recent review by Funakoshi et al. [20, 21] demonstrated that the most common presenting symptoms for athletes with first-rib stress fractures included posterior shoulder or upper thoracic back pain. Cervical spine imaging may more accurately identify first-rib stress fractures when compared to shoulder radiographs [20].

Prisk et al. [22] proposed the "trapezius squeeze test" or "pinch test" for the diagnosis of first-rib stress fractures. This test involves applying pressure to the anterior aspect of the trapezius muscle, causing involuntary contraction of the muscle and eliciting rib pain (Fig. 12.2). This test was found to be reliable for diagnosing first-rib stress fractures on physical examination

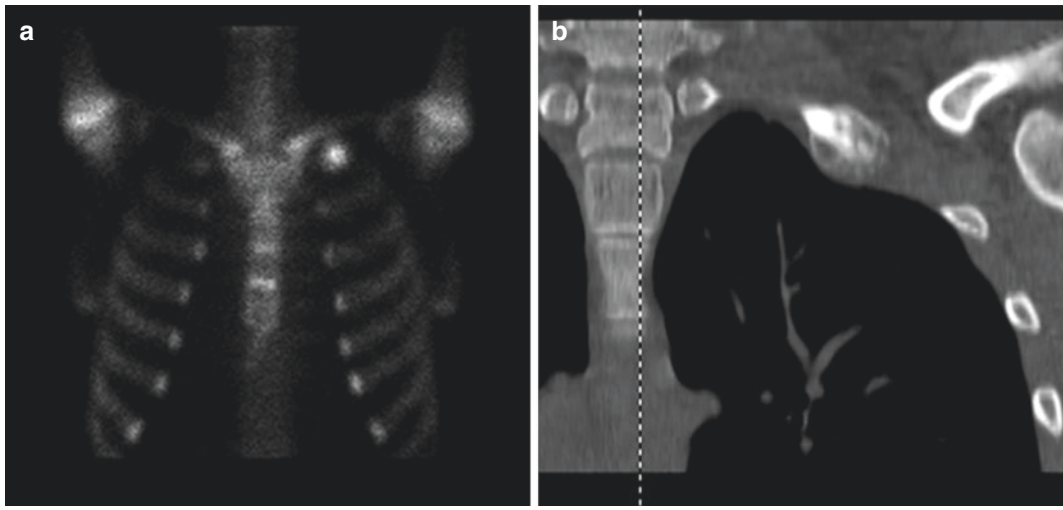

Fig. 12.1 (**a** and **b**) Bone scan (**a**) and coronal CT scan (**b**) images demonstrating left first-rib stress fracture in a male collegiate gymnast. CT scan demonstrates healing with abundant callus formation

in five cases of stress fractures in ballet dancers [11, 22].

These fractures generally heal well with conservative management including appropriate physical therapy, complete rest until deep breathing is pain free, and an eventual gradual return to sport [23]. There are rare reports of complications related to extensive callus formation causing thoracic outlet syndrome [24].

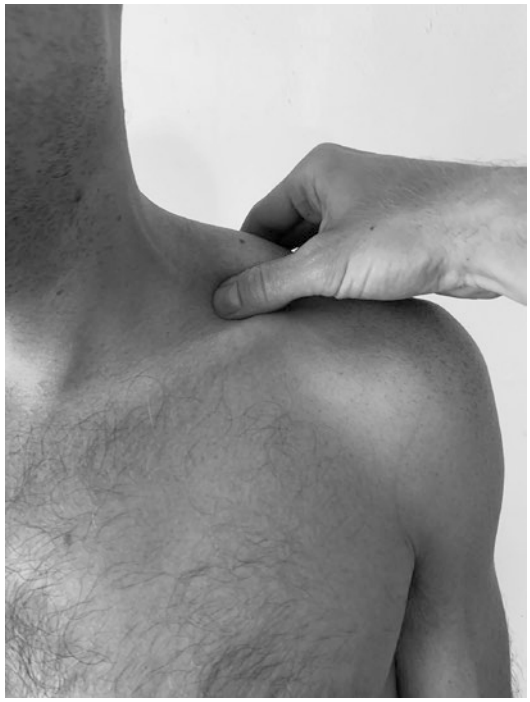

Fig. 12.2 First-rib trapezial squeeze test/"pinch test"

Second Through Twelfth Ribs

Repetitive strain on the torso contributes to middle- and lower-rib stress fractures (Figs. 12.3a, b and 12.4a, b). These are most commonly described in athletes involved in rowing, discus, throwing, and golf [7, 8, 19, 25–27]. Patients with this injury present with increasing lateral chest pain and are diagnosed most commonly by radionuclide scans [25]. Other athletic activities associated with these fractures include tennis, gymnastics, and throwing sports [5, 8]. Among rowers, fractures are found most commonly between the fifth and ninth ribs. Pain is generally greatest at the finish of a stroke and may be exacerbated by coughing, sneezing, or deep inhalation [8, 11, 19]. Among golfers, Lord [28] described 19 cases of rib stress fractures. A total of 16 of the 19 golfers sustained injury in their leading arm's trunk side. The posterolateral aspects of the fourth through sixth ribs were the most commonly injured sites [28]. The authors suggested that the ribs on the leading arm side are most commonly involved because of repetitive contraction of the serratus muscle through all phases of the golf swing on the leading side compared with the trailing side [28].

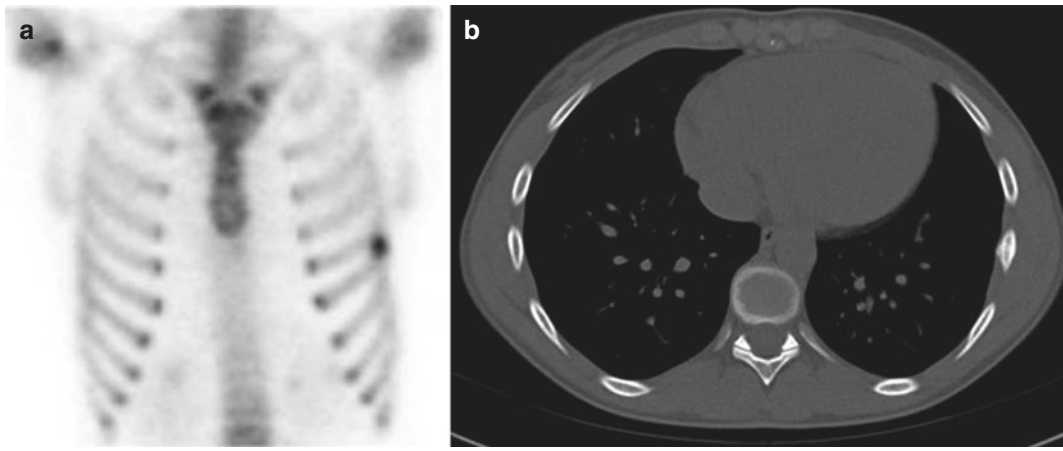

Fig. 12.3 (**a** and **b**) Bone scan (**a**) and axial CT scan (**b**) images demonstrating stress fracture of the left mid-seventh rib. CT scan demonstrates fracture callus present

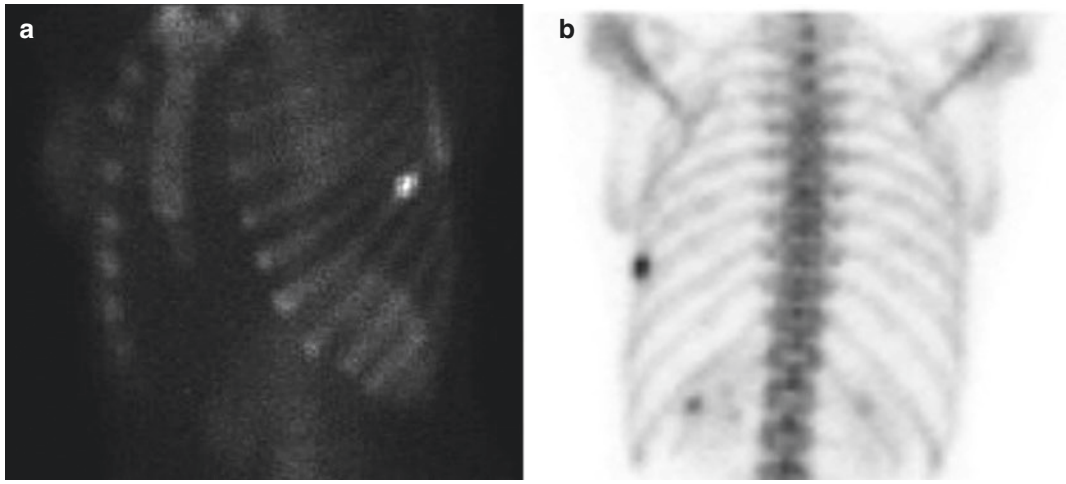

Fig. 12.4 (**a** and **b**) Bone scans of left-sided eighth-rib stress fracture (**a**) and left-sided sixth-rib stress fracture (**b**)

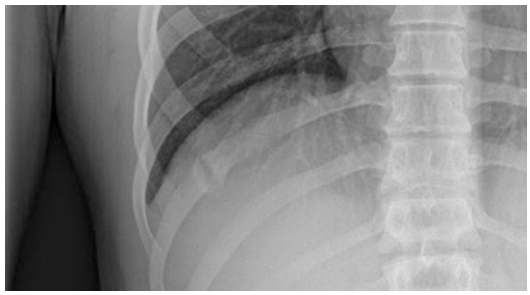

Fig. 12.5 Anteroposterior chest radiographs demonstrating nonunion (grade V) of the right tenth-rib stress fracture in a male collegiate rower

The treatment of rib stress fractures is nearly always nonoperative, with the initial goal being to provide symptomatic relief. In general, rib stress fractures rarely fail to heal with modification or complete discontinuation of the causative activity for 4–6 weeks [5]. Treatment includes relative rest by avoiding overhead lifting, throwing, or rowing sports. Nonunion of rib stress fractures (Fig. 12.5) has been described; however, this is rare and may ultimately be asymptomatic [5, 22].

Sternum

Figure 12.6 shows the coronal and sagittal MRI images of a 28-year-old male competitive weight lifter with a midsternal stress fracture. Stress fractures of the sternum may be diagnosed on radiographs, CT scan, technetium bone scan, or MRI. Athletes with this injury typically present with dull to progressively sharp anterior chest pain. In addition to weight lifting, stress fractures of the sternum have also been described in athletes participating in military training [29], golf [30], cycling [31], and wrestling [32]. In one case described in the literature, the athlete described an audible "pop" [33] while performing core exercises. In nearly all cases of sternal stress fractures described in the literature, athletes were performing intensified repetitive activities of the pectoralis muscles, triceps, or rectus abdominis. Relative rest from the causative activity led to resolution of symptoms within 6–10 weeks.

Scapula

Stress fractures of the scapula in athletes are uncommon [34, 35]. Cases reported in the literature include fractures in the following athletes: gymnast, baseball pitcher, golfer, jogger carrying weights, and professional football player [5, 11, 34, 36]. Others have sustained stress fractures of the scapular spine (high school football quarterback) (Fig. 12.7) and the coracoid process (trap shooter, intensive shoulder rehabilitation pro-

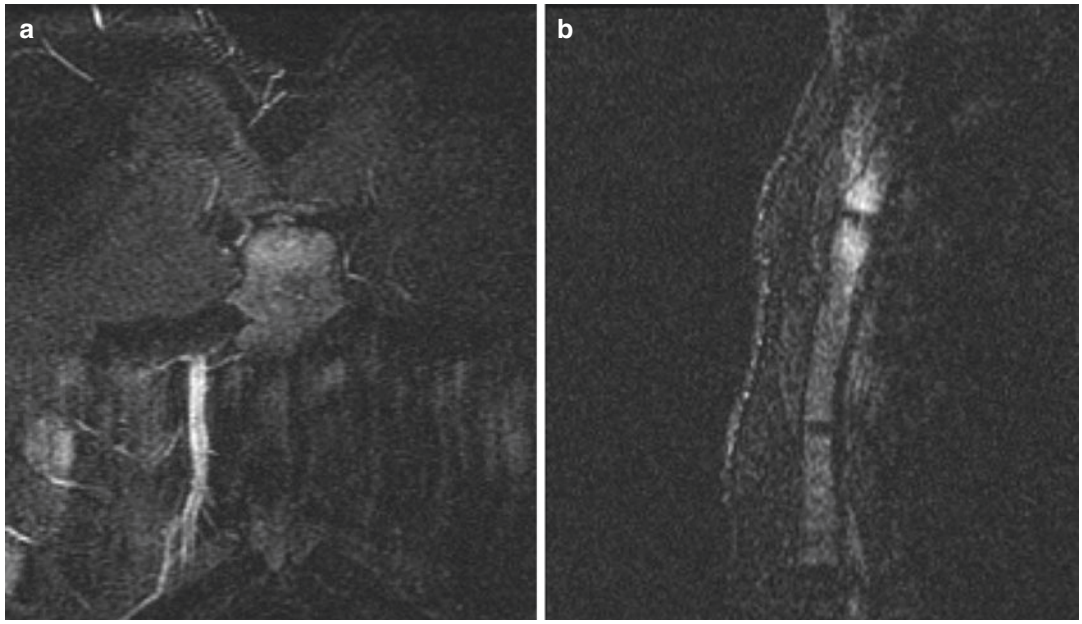

Fig. 12.6 (**a** and **b**) T2 coronal (**a**) and sagittal (**b**) MRI series demonstrating stress fracture of the mid sternum in a competitive weight lifter

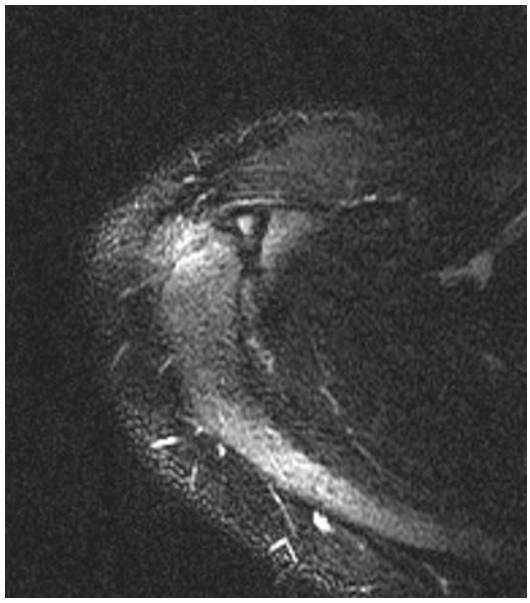

Fig. 12.7 T2 axial MRI demonstrating grade II stress fracture of the medial scapular spine in the dominant right shoulder of a high school quarterback. An unstable os acromiale is also evident

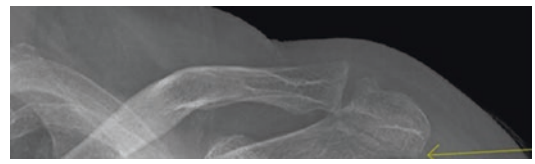

Fig. 12.8 Anteroposterior radiographs of the left shoulder demonstrating grade III stress fracture of the acromion process

gram activities) [37]. Stress fractures can occur about the coracoid, acromion (Fig. 12.8), scapular spine, and scapular body [11, 34, 38]. In addition, while not often associated with high-level athletics, stress fractures also occur about acromion process after total shoulder arthroplasty and may be a source of postoperative pain (Fig. 12.9).

As the scapula has a complex array of muscle attachments and corresponding bone stress patterns, these injuries represent a diagnostic challenge to clinicians. Depending on the specific patient's motion, stress concentration occurs at

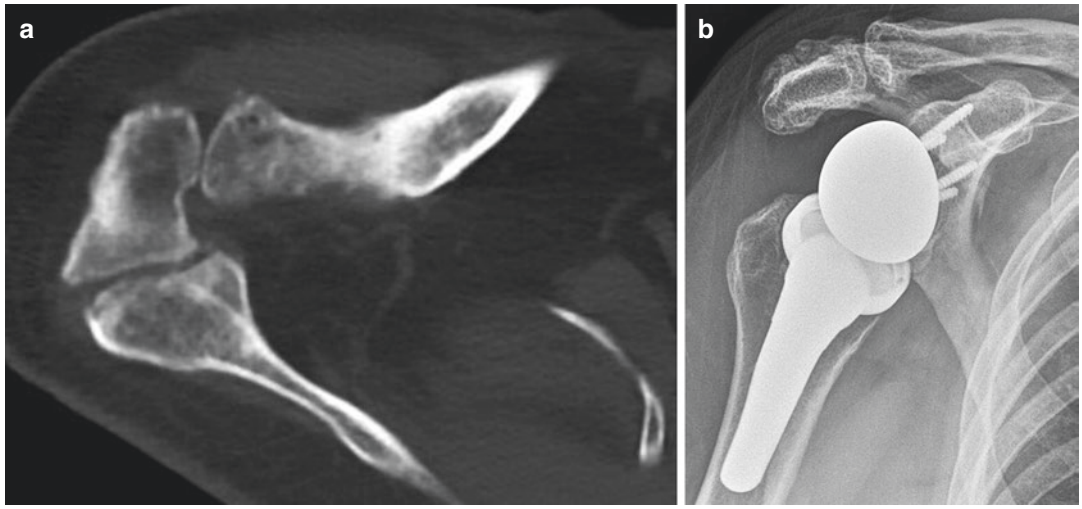

Fig. 12.9 (**a** and **b**) AP XR and coronal CT scan demonstrating a left-sided acromion stress fracture after total shoulder arthroplasty

a variety of locations in the scapula. Likely, the cause of these injuries is overuse or fatigue of one or more of the 17 muscles that control the scapula, leading to stress-related injury [5, 8, 11].

Clavicle

Cases of clavicular stress fractures have been reported among rowing, diving, javelin, weight lifting, gymnastics, and baseball sporting activities [5, 7, 35]. Abnormal bending, shear, and rotational forces can develop across the clavicle if there is any imbalance in muscular contraction between the pectoralis major, deltoid, and sternocleidomastoid muscles [5]. Repetitive bone strain by these forces may exceed the reparative capacity of the bone and lead to a stress fracture. Seyahi et al. [35] described a patient with a clavicular stress fracture presenting as atypical severe arm pain radiating throughout the upper extremity and hemithorax. In the case of clavicular stress fractures, activity modification until pain is resolved, postural training, and scapulothoracic stabilization exercises have yielded symptom resolution [5, 35].

Proximal Humerus

Stress fractures of the proximal humerus have been described most commonly in throwing, overhead, and weight lifting athletes [39–42]. In throwers and overhead athletes such as tennis players, poor conditioning and fatigue of the shoulder girdle musculature allow for increased rotational strain at the cortical surface, predisposing to stress fracture. Bending forces generated by opposition of the deltoid and pectoralis major muscles is the suspected mechanism for the transversely oriented stress fractures in weight lifters [43]. Athletes typically present with increasing insidious onset arm pain, acute on chronic pain, or sustain a "pop" following antecedent activity related pain of the shoulder or arm [5].

If incomplete or non-displaced, proximal humeral stress fractures may be treated nonoperatively in a sling or functional brace until the athlete is pain free with activities of daily living or radiographic healing is evident. Some advocate for physical therapy for analgesia control, stretching, and deltoid and rotator cuff strengthening exercises for the treatment of proximal humerus stress fractures [44]. Treatment for incomplete or

nondisplaced proximal humeral stress fractures should include rest and cessation of the offending activity. Athletes should be counseled that as long as 12 months may be required to become asymptomatic [11]. If there is fracture displacement, open reduction and internal fixation may be necessary to ensure timely healing.

Little Leaguer's Shoulder

Little Leaguer's shoulder is epiphysiolysis of the proximal humerus secondary to repetitive microtrauma and rotational torque sustained during overhead activity [45, 46]. The proximal humeral physis fuses between approximately 14 and 17 years of age among females and between 16 and 18 years among males [47, 48]. As such, little league shoulder injuries are commonly found in baseball pitchers between the ages of 11 and 14, prior to physeal fusion [46]. Factors that contribute to the development of little leaguer's shoulder include excessive throwing, poor throwing technique, and muscular imbalance.

Athletes typically describe diffuse shoulder pain that is worse with throwing, often following an increase in the throwing frequency or intensity [49–51]. On physical examination, patients demonstrate weakness with resisted abduction and internal rotation along with tenderness and swelling over the anterolateral shoulder. Anteroposterior X-rays may reveal widening of the proximal humeral physis (Fig. 12.10). Plain radiographs may also display fragmentation or demineralization of the metaphysis, cyst formation, physeal fragmentation or widening, or periosteal reaction [49–52]. MRI of the shoulder is often required if the diagnosis is unclear (Fig. 12.11).

Treatment requires rest from throwing for 6–12 weeks followed by a progressive throwing program with alterations to the athlete's throwing techniques and biomechanics. The return to throwing progression begins with light tossing and as distance and velocity increases [51]. Potential complications include premature physeal closure with resultant humeral limb length discrepancy or angular deformity. Because the proximal humerus has an excellent remodeling potential, however, these complications are

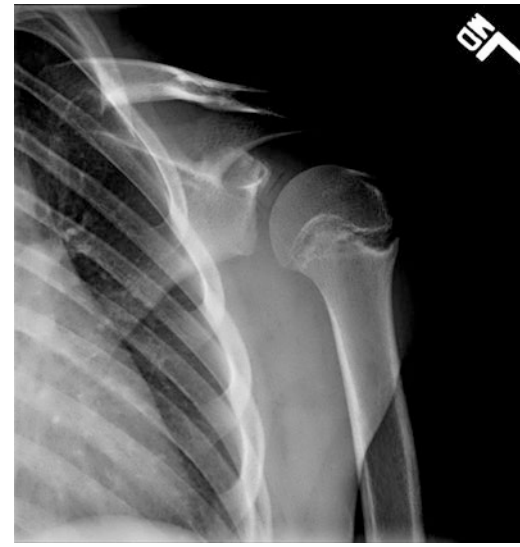

Fig. 12.10 AP radiograph showing lateral widening at the proximal humeral physis in an athlete with little leaguer's shoulder

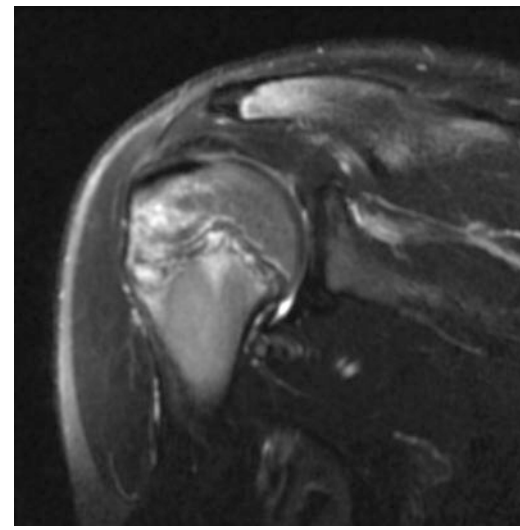

Fig. 12.11 Coronal T2 MRI image demonstrating periphyseal stress fracture of the proximal humerus in an athlete with little leaguer's shoulder

rare. Heyworth et al. recently reviewed this subject and demonstrated a 7% recurrence rate at 7.6 months after diagnosis [53]. Proper throwing mechanics and close monitoring of pitch counts in the skeletally immature athlete are crucial for recovery from little league shoulder and prevention of further injury.

Diagnostic Imaging

X-Ray

Plain radiographs are frequently negative early in the course of rib and upper extremity stress fractures. Although two-thirds of initial X-rays are negative, one-half will be positive around 3 weeks after symptom onset or once healing begins (Figs. 12.3, 12.6, and 12.7a) [54]. Even after healing has taken place, radiographic findings such as cortical thickening and bone edema are subtle and easily overlooked if images are not thoroughly scrutinized [54, 55]. Depending on the severity and chronicity of the injury, plain radiographs may be inconclusive and advanced imaging may be required. In the case of proximal humeral periphyseal stress fractures (Little Leaguer's shoulder), X-rays may initially be mistakenly read as a normal incompletely closed physis in a skeletally immature patient. MRI or bone scan is often necessary to make a diagnosis, with MRI being the preferred modality due to the superior specificity (>85%).

CT

Computed tomography (CT) is useful when the diagnosis of a stress fracture is indeterminate based on plain X-rays. CTs can also delineate a complete fracture from an incomplete fracture. CT is not as commonly used as MRI due to the increased amount of radiation exposure and decreased ability to evaluate surrounding soft tissue structures. CT is useful for demonstrating evidence of healing by showing periosteal reaction and the presence or absence of lucencies, nonunions, or sclerotic fracture lines (Figs. 12.1b and 12.3b) [11].

Bone Scan

Bone scans or bone scintigraphy has been shown to be up to 100% sensitive for stress injuries of bone [55]. Bone scintigraphy allows early diagnosis of stress injuries and it can diagnose bony stress injuries at multiple sites simultaneously (Figs. 12.1a and 12.3a). This is often the case with rib stress fractures. Bone scans will often demonstrate increased uptake and a focused area of increased osteoblastic activity in the affected bone 1–2 two weeks before radiographic changes occur [55]. Uptake on bone scan often requires 12–18 months to normalize, lagging behind the resolution of clinical symptoms [55]. Thus, bone scans are less helpful for guiding return to activity and/or sports participation. In the case of first rib injuries, bone scintigraphy has demonstrated 100% sensitivity for early detection and diagnosis; however, it was shown to have a lower specificity than MRI [56].

MRI

Magnetic resonance imaging (MRI) is the most sensitive and specific imaging study available to evaluate stress injuries of bone [56, 57]. In addition, MRI may detect injuries earlier than bone scan [57]. MRI is now the primary diagnostic tool for stress fractures. Its sensitivity is similar to that of a bone scan; however, it is much more precise in delineating the anatomic location and extent of injury, and it may be able to detect stress injuries up to 2 weeks prior to bone scans [11] [57].

Typical MRI findings on T2 sequences include a band of low signal corresponding to the fracture line, surrounded by diffuse high-signal intensity representing marrow edema (Figs. 12.6a, b, 12.7, and 12.11). Though expensive, it has the additional benefit of identifying soft tissue injuries and is often beneficial in the athletic population. MRI may be even more helpful when used in conjunction with experienced musculoskeletal radiologists familiar with specific imaging protocols [58].

Table 12.2 Kaeding–Miller Stress Fracture Classification System [60] – shown is a combined clinical and radiographic classification system for stress fractures that has shown high intra- and inter-observer reliabilities

Grade	Pain	Radiographic Ffndings (CT, MRI, bone scan, or X-ray)
I	–	Imaging evidence of stress FX No fracture line
II	+	Imaging evidence of stress FX No fracture line
III	+	Non-displaced fracture line
IV	+	Displaced fracture (>2 mm)
V	+	Nonunion

Classification

Stress fractures occur along a spectrum of severity. Not only does the severity of these injuries vary, but the behavior varies by location and causative activity as well. In addition to risk stratification of stress fractures, which is based largely upon anatomic site, the "grade," or degree of cortical failure, at a specific site is also used to describe the injury, determine prognosis, and inform the appropriate treatment options [59]. The continuum throughout which stress fractures occur in the ribs and shoulder include simple bone-marrow edema (stress reaction), small unicortical disruptions, and complete fractures with or without displacement and possibly nonunion. The management of bony stress injuries should be based on the location, grade of the injury, and healing potential of the injured site. A combined clinical and radiographic classification system developed by Kaeding and Miller is shown in Table 12.2 [60]. This system has shown high inter- and intraobserver reliabilities among sports medicine and orthopedic clinicians [60]. A more in-depth discussion of stress fracture classification and grading is presented in a different chapter of this textbook.

General Treatment Principles

The treatment for stress fractures of the ribs and shoulder girdle should be individualized to the patient's functional needs, symptom severity, causative activity, anatomic site, nutritional status, and fracture grade. Rehabilitation and training programs focused on proper mechanics and technique should be included in the treatment protocol after the fracture has been given sufficient time to heal [25, 28, 38, 44]. If the fracture does not heal or symptoms persist beyond 4–6 weeks, the options for treatment include immobilization, restrictive bracing, or even surgical fixation, depending on the site and injury severity [11]. In addition to rest, activity modification, and rehabilitation protocols with integrated physical therapy, metabolic factors including calcium and vitamin D levels should routinely be evaluated and addressed [31]. Furthermore, athletes with stress fractures at low-risk sites (those with adequate blood supply and low shear and tensile forces) and who are without functional limitations may continue their activities as tolerated using symptoms as a guide.

The decision to continue but decrease the causative activity in the presence of a stress fracture must be made in conjunction with the athlete and only after a thorough open discussion of possible injury progression. The activity may be continued with pain as a guide [11]; however, close follow-up of these patients is necessary to ensure compliance with activity restrictions and to prevent fracture progression to a higher grade. This approach is acceptable if the risk and consequence of fracture completion are acceptable to the patient due to the importance of continuing their activity. Unless contraindicated, patients should be encouraged to cross-train to maintain fitness and supplement training as the fracture heals.

Low-grade stress injuries or those without a clear fracture line at a low-risk site have a shorter time to recovery than a higher-grade injury at the same low-risk site [11]. The differences in treatment options between these two levels of severity of injury are duration of treatment, degree of activity modification, and need for immobilization (usually in a sling). The goals of treatment are symptomatic relief and decrease of repetitive stress at the fracture site, thereby restoring the dynamic balance between bony damage and repair [10, 11]. This potentially involves a decrease in the volume of the offending activity, equipment

changes, technique alterations, or cross-training. If pain persists or intensifies, despite activity modification alone, treatment must be advanced to include complete rest, immobilization, or possibly surgical stabilization [11].

Return to Sports Participation

Return to sport decision-making following a stress fracture is multifactorial. Despite advances in the imaging and understanding of stress fracture behavior, the decision to return to activity continues to challenge sports medicine practitioners. Critical to any return-to-play consideration is a thorough understanding of the risk of possible injury progression by all parties (i.e., physician, athlete, coaches, trainers, family). All patients, particularly those with stress fractures at sites with poor healing potential, must understand the risk of noncompliance with the treatment. A treatment plan should be tailored to athletic and personal goals, and the risks and benefits of continued participation thoroughly outlined.

As with most low-risk stress fractures, the point in the competitive season at which a rib or shoulder girdle stress fracture is diagnosed is often a major consideration for return to play. Athletes near the end of a competitive season or in the "off-season" may desire to be healed from their injury prior to returning training or competition. For these individuals, the treatment plan should include strict rest and activity modification to a pain-free level. In contrast, athletes at mid-season with low-risk stress fractures may desire to finish the season and pursue treatment at a later time. A gradual increase in activity can begin once the athlete is pain-free with activities of daily living and when the site is nontender [11].

Prevention of Rib and Shoulder Girdle Stress Fractures

Prevention of rib and shoulder girdle stress fractures is preferred. At the pre-participation physical examination, an evaluation of risk should occur. This is especially important for individuals with a history of previous stress fractures. A history of prior stress fracture should alert the clinician to review that individual's risk factors. In females, correction of menstrual irregularities and poor nutritional status is critical. Team physicians involved with female athletes must also be vigilant for signs of the classic female athlete triad of osteopenia, disordered eating, and amenorrhea or relative energy deficiency in sport (RED-S). Calcium and vitamin D supplementation is often recommended in addition to general nutritional optimization with increased caloric intake. If biomechanical abnormalities are encountered, video analysis with appropriate muscular strengthening, proper equipment, and technique alterations is indicated for prevention of future injuries.

Conclusions

Stress fractures of the ribs, thorax, and shoulder girdle can be a source of pain and missed time from training and competition for athletes participating in a variety of sports. Stress fractures, along with bony tumors, insufficiency fractures, and neuralgias, should be included in the differential diagnosis when patients who regularly participate in repetitive activities present with pain of the ribs and upper extremities. They are common injuries in rowing and throwing athletes and in individuals performing repetitive activities through their upper extremities. The diagnosis requires a high index of suspicion and proper imaging. Treatment of these injuries should be individualized to the athlete's goals, sport, biomechanics, physiology, nutritional status, and injury site and severity. Factors influencing treatment decisions include location (low vs. high risk), fracture grade, activity level, the timing of the competitive season, and the athlete's risk tolerance. With appropriate diagnosis and treatment, return to pre-injury activity level can be expected for the majority of patients with upper extremity stress fractures.

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Upper Extremity Stress Fractures

13

Wendell W. Cole III, Mary K. Mulcahey, and Felix H. Savoie

Epidemiology

Upper extremity (UE) stress fractures are less common than lower extremity stress fractures and account for 2.8% of all stress fractures among high school athletes [1]. In collegiate athletes, UE stress fractures have been reported to be as low as 0.001% (1/671) [5]. Lower extremity stress fractures are typically caused by impact loading from running or walking, while the majority of UE stress fractures are due to repetitive loading in athletes who take part in weight lifting, rowing, gymnastics, and overhead sports, which may lead to missed competitions and loss of training time [1–6]. Athletes who sustain injuries to the shoulder girdle are more likely to be throwers (i.e., baseball pitchers), while those who sustain injuries distal to the elbow are UE weight bearers (i.e., gymnasts) [7] (Fig 13.1).

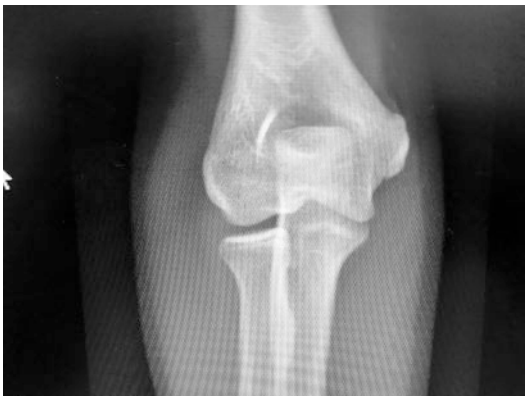

Fig. 13.1 Stress fracture involving the medial epicondyle. Courtesy of Felix H. Savoie III, M.D.

Pathophysiology

Repetitive stress and loading of upper extremity bones at the point of muscular attachments can lead to stress fracture [2]. Increased physical activity at the start of a new season can lead to changes in the degree of bone remodeling due to the stresses of the muscle on the bone. During this period, bone resorption outpaces bone production, causing a lag of several weeks before new lamellar bone is laid down [8]. It is during this period of imbalance that the local bone is weakened and microdamage accumulates. If the activity is continued, there is further weakening of the bone and ultimately mechanical failure, which can then lead to the development of a true macroscopic stress fracture [9, 10]. During training, muscle hypertrophy and fatigue decrease the shock-absorbing effect of muscle and lead to more stress applied to the bone [11, 12]. Early injuries (with no radiographic findings) are often

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can diagnosed as a stress reaction versus a true stress fracture, which may appear as a distinct fracture line on X-ray or MRI [6].

Skeletally immature patients are more susceptible to stress injuries due to the weaker physeal cartilage of the epiphysis and apophysis. Repetitive loading and microtrauma disrupt the endochondral ossification in the physis of long bones and result in the extension of unmineralized cartilage into metaphyseal bone [13]. Causes of stress fractures in children include overuse injuries, underlying bone weakness, endocrine abnormalities, congenital abnormalities, infection, and an inflammatory response, with an increased prevalence in sports [14].

Imaging

X-rays are insensitive for detecting stress injuries and will be positive less than 50% of the time [3, 4]. Most stress fractures can be identified later when periosteal or endosteal new bone formation is apparent on X-ray [9]. Computed tomography (CT) also has a limited role in detecting stress injuries in the early stages [15].

Radionuclide scanning has been shown to be extremely sensitive in detecting early-stage osseous stress injury. The radiopharmaceutical agent is taken up at areas of active bone turnover, which can identify stress fractures before the development of clinical symptoms [9]. If radionuclide scans are not available, magnetic resonance imaging (MRI) scans are just as sensitive, showing marrow edema and periosteal fluid at the site of the stress reaction [9].

First Rib

Stress fractures of the first rib are commonly seen in athletes with repetitive overhead positioning of the arm, such as baseball pitching, basketball, lacrosse, javelin, and weight lifting [9]. First-rib stress fractures occur most commonly near the subclavian groove between the insertions of the scalene anterior and medius muscle where the bone is the thinnest. The first rib is broad and flat with two shallow grooves for the subclavian artery and vein. This groove is the weakest link and the most common area for stress fracture [16]. The scalene muscles pull the rib superiorly, while the serratus anterior and intercostal muscles direct the rib inferiorly [16].

Patients will present with a dull vague pain in the anterior cervical triangle and mid-clavicular region. There will be occasional radiation to the sternum and pectoral region, and the pain is sometimes worse with cough and deep inspiration [16]. The trapezius squeeze test, where pressure is applied to the anterior trapezius, leads to involuntary contraction of the muscle, eliciting rib pain which can help induce the diagnosis [17]. Initial radiographs are often negative and bone scans are recommended.

Initial treatment consists of non-operative measures including activity modification and rest. Careful attention is made to avoid overhead lifting, throwing, rowing sports, and wearing a backpack. Stress fractures of the first-rib stress typically heal within 4–6 weeks. Non-union of first rib stress fractures has been reported, but these fractures are rare and can be asymptomatic [17]. Brachial plexus palsy due to excessive callus formation from a hypertrophic nonunion has also been reported [18].

Coracoid

Coracoid stress fractures in the adolescent athlete is a rare injury associated with repetitive overhead activity during tennis or cricket and is typically seen as an avulsion of the coracoid process [19, 20]. The coracoid process is the attachment site for the coracobrachialis, short head of the biceps, and pectoralis minor tendons along with the coracoacromial, coracohumeral, and coracoclavicular ligaments. The attachments of the coracobrachialis and pectoralis minor muscle cause adduction and protraction of the scapula, which is seen during the follow through motion of throwing [21].

On presentation, patients will complain of shoulder pain and have tenderness to palpation about the coracoid process. Pain can also be elic-

ited by adduction and forward flexion of the arm against resistance [22]. Initial treatment involves activity modification and temporary cessation of the inciting activity. In cases where non-operative treatment is ineffective, internal fixation may be required with a cannulated screw and washer or excision of the fracture fragment and reattachment of the conjoint tendon [19, 20]. Athletes are often able to return to play at approximately 2.5 months post-operatively Abduction weakness and posterior shoulder discomfort have been noted in patients who underwent fracture excision and tendon reattachment [19].

Proximal Humerus

Stress fractures of the proximal humerus are commonly seen in pitchers between the ages of 11 and 13 and have also been noted to occur in other sports such as badminton [23, 24]. Proximal humerus stress fractures are also known as osteochondrosis of the proximal humeral epiphysis, proximal humeral epiphysiolysis, and little league shoulder [25]. These injuries are caused by repetitive rotational stresses seen during throwing activities, which leads to microtrauma and eventual fracture (Fig 13.2) [23]. The cocking phase and the acceleration phase of the throwing motion contribute to stress fractures of the proximal humerus. The significant amount of external rotation torque on the proximal humerus during the final part of the arm-cocking phase leads to the deformation of the proximal humeral epiphysis cartilage and eventually epiphysiolysis [26].

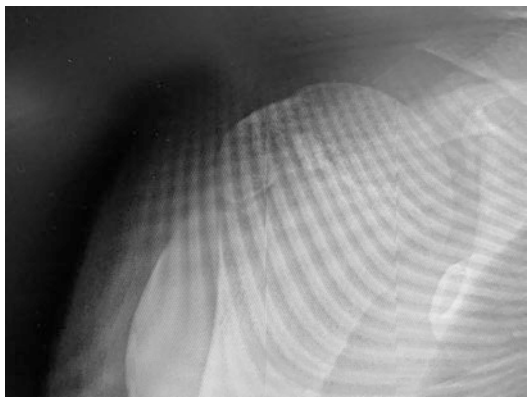


Fig. 13.2 A stress fracture of the proximal humerus greater tuberosity is noted. A significant amount of external rotation torque on the proximal humerus during the final part of the arm-cocking phase leads to deformation of the proximal humeral epiphysis cartilage. (Courtesy of Felix H. Savoie III, M.D.)

Patients will often complain of shoulder pain during pitching and have tenderness to palpation about the lateral aspect of the proximal humerus over the growth plate. Initial radiographs may show widening of the humeral physis, which is associated with sclerosis, fragmentation, and cystic changes of the proximal humeral metaphysis [23].

Initial treatment includes rest and discontinuation of overhead activity. Pitchers should refrain from pitching for 2-3 weeks, and then start a return-to-throw program maintaining a pain-free state for an average of 8-12 weeks. Proximal humeral physeal widening may still be evident in asymptomatic patients [23, 25, 27]. Growth arrest of the proximal humeral physis has been noted to be a potential complication of these stress injuries [23]. Once the athlete has no pain at rest, physical therapy is initiated and should include core muscle and rotator cuff strengthening [26]. Proper pitching mechanics, concentrating on technique over speed, generates lower humeral internal rotation torque, generates lower elbow valgus load, is more efficient for the athlete, and leads to fewer future injuries [28].

Medial Epicondyle

Medial epicondyle stress fractures are common in adolescent athletes who participate in overhead throwing sports (e.g., baseball). In children, the medial epicondyle apophyseal plate is weak and is vulnerable to injury [29]. Adolescent athletes in the beginning phases of learning proper throwing mechanics are exposed to repetitive tensile and compressive and rotational forces (Fig 13.3). The medial aspect of the elbow experiences a significant amount of tensile force due to the valgus stress that occurs during the late cocking and early acceleration phases of throwing [29]. Repetitive throwing leads to contraction

of the forearm flexor-pronator muscles against the growth plate, continued microtrauma, tensile force across the medial elbow, compression across the lateral compartment, and a posterior shear/traction force on the elbow (Fig. 13.4) [23, 30]. This constellation of symptoms in the adolescent thrower is frequently termed the little league elbow [23, 30].

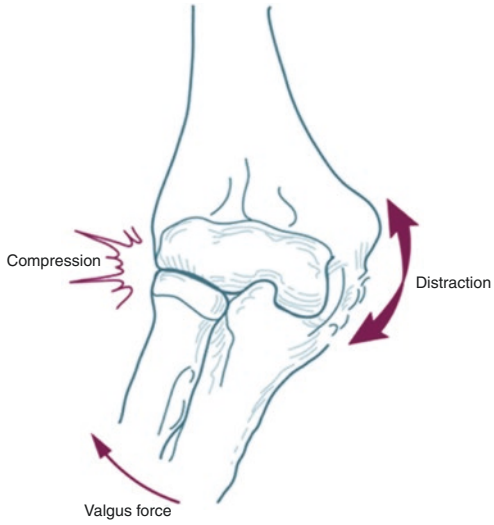
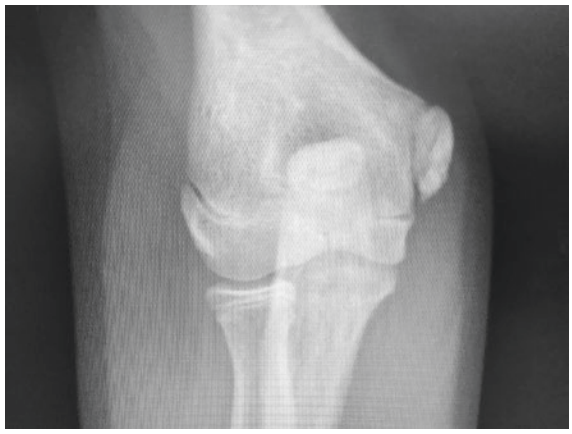
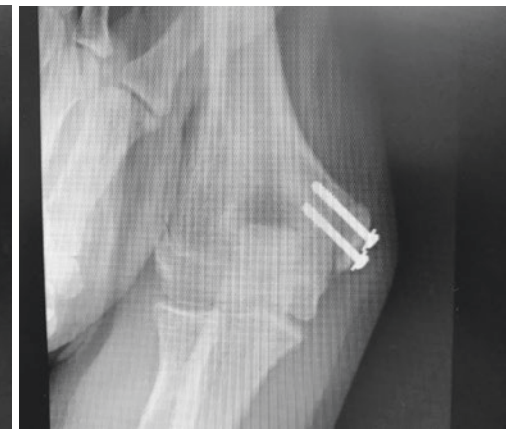


Fig. 13.3 During the throwing motion, significant forces are seen throughout the elbow that include medial-sided tension, lateral-sided compression, and posterior olecranon fossa shearing. Valgus extension overload syndrome occurs secondary to this repetitive microtrauma in the posterior compartment and leads to posteromedial olecranon osteophyte (bone spur) formation as seen on the left side of the figure labeled with the star. These osteophytes then limit terminal extension and cause pain with range of motion. Reprinted with permission from O'Connell RS, Field LD. Handheld osteotomes facilitate arthroscopic treatment of elbow valgus extension overload. Arthroscopy Techniques. 2020;9(3):e387–91, Elsevier

Patients often present with pain over the medial epicondyle, elbow swelling, and a possible flexion contracture of up to 15° [23, 29]. A thorough neurovascular exam should be performed due to the proximity of the ulnar nerve to the medial epicondyle. Radiographic imaging may show subtle widening of the apophysis or fragmentation of the medial epicondyle ossification center. With increased chronicity, accelerated growth and gradual epicondyle deformity can be seen along with changes in the radiocapitellar joint, indicating pathologic compression overload laterally [29]. Bone marrow edema is seen early on MRI, which may aid with the diagnosis.

Treatment of medial epicondyle stress fractures consists of rest, ice, splinting, and activity modification. Dual bracing with a hinged elbow brace to keep the arm within a pain-free range of motion along with a wrist brace in 0° of flexion–extension to prevent tension of the flexor-pronator muscles is key to maintaining elbow motion and overall body condition. Dual bracing may allow the young athlete to continue to participate in sports to a limited extent as long as the activity is pain free. Pitchers should be given a position change to allow for continued participation and





Figs. 13.4 and 13.5 Medial epicondyle stress fracture is noted in a skeletally immature athlete (Fig. 13.4). Medial epicondyle fragment fixation with partially threaded screws and washer construct. (Courtesy of Felix H. Savoie III, M.D.)

decreased stress to the elbow in the protective brace, resuming a return to pitch program once symptoms resolve. If symptoms return, players should rest for the remainder of the season or consider surgical intervention (Fig. 13.5) [23, 29, 31].

Olecranon

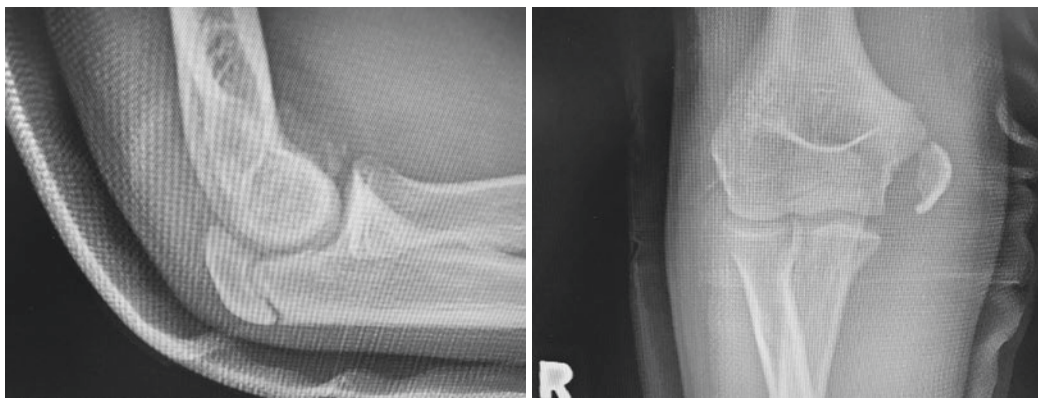
Olecranon stress fractures occur mostly in throwing and overhead athletes, but they have also been seen in gymnasts, javelin throwers, and weight lifters [32, 33]. During the growth period, the normal olecranon epiphysis closes between the ages of 15 and 17 years [34, 35]. Repeated stress and high-intensity training can lead to stress fracture of the olecranon [36].

With repetitive stress, patients may develop stress fractures through the olecranon tip, oblique fractures through the mid-portion of the olecranon, transverse fractures, or osteochondrosis. Osteochondrosis and transverse avulsion fractures occur in skeletally immature patients, while fractures through the tip and oblique fractures occur in older athletes [37–39]. Transverse fractures are caused by repeated contraction of the triceps against the olecranon, which results in transient localized ischemia compromising cartilage mineralization [36]. Osteochondrosis, which may occur due to avascular necrosis, is more common in adolescents and results from similar traction forces that cause transverse fractures by causing fragmentation and disturbing ossification.

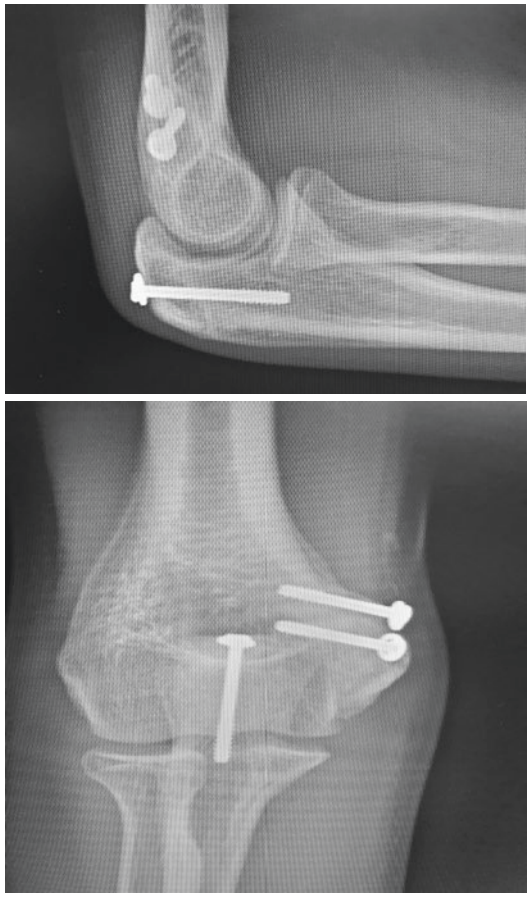
Olecranon stress fracture injuries are mostly due to the pathologic shear forces during the acceleration and deceleration phases of throwing. During the acceleration phase of throwing, impingement of the olecranon in the olecranon fossa combined with a valgus torque results in impaction and posterior-medial olecranon shearing forces [30, 40–41]. Valgus extension overload can cause chondromalacia, loose body formation, and posterior-medial osteophytes contributing to the constellation of signs and symptoms known as pitchers elbow (Figs. 13.6 and 13.7) [42]. If deceleration while throwing is not controlled dynamically by muscle forces, the olecranon abuts the posterior compartment of the elbow close to full extension and contributes to overuse injury [40].

The patient will often complain of pain over the medial aspect of the olecranon that is present in the acceleration and deceleration phases of throwing [42]. On physical examination, patients often have point tenderness at the tip of the elbow, localized swelling, and possible loss of terminal flexion and/or extension [14]. In young adults, there may be pain with elbow extension secondary to impingement of the posterior-medial osteophytes [43].

Radiographs may show widening of the olecranon apophysis. In young adults, initial imaging may show loose bodies or osteophyte formation. Most olecranon stress fractures can be treated conservatively with 3-4 weeks of rest from throwing, a position change, and protective bracing. Acute non-displaced avulsion fractures may be treated



Figs. 13.6 and 13.7 Stress fracture of the olecranon and fracture of the medial epicondyle. (Courtesy of Felix H. Savoie III, M.D.)



Figs. 13.8 and 13.9 Fractures of the olecranon and medial epicondyle after reduction and fixation with partially threaded screws. Courtesy of Felix H. Savoie III, M.D.

with casting [29]. Physical therapy for gentle range of motion exercises, flexibility, and strength may be helpful immediately [29, 44]. Most athletes can return to play after 3-6 weeks of conservative treatment. If the fracture is displaced, the patient may benefit from open reduction and internal fixation. Most fractures are repaired with single or dual screws, although tension band fixation has been used in adolescent patients with open physes (Figs. 13.8 and 13.9) [45].

Ulna Shaft

Ulna shaft stress fractures have been described in tennis players, softball pitchers, weight lifters, bowlers, and baton twirlers [46, 47]. The middle third of the ulna has a triangular shape, which is less resistant to chronic stress, and has the thinnest cortex and smallest cross-sectional area compared with the proximal and distal ulna. Repetitive pronation torsional forces act on the most vulnerable portion of the ulna shaft, which can lead to a stress fracture of the middle third of the ulna [2, 47]. The majority of ulnar diaphyseal stress fractures are due to torsional forces. In tennis players, stress results most commonly from repetitive wrist dorsiflexion with forearm supination during the backswing followed by forearm pronation during the ball strike and follow-through [48, 49]. In fast-pitch softball players, torsional stress occurs during the "windmill pitch" where the wrist is extended and the forearm is supinated during windup and when the forearm is pronated during delivery and follow-through [50]. In addition many softball pitchers are taught to "brush" the leg with the arm as it passes the post-leg, and this repetitive contact can lead to ulnar stress reaction.

Patients will often complain about pain along the ulna during physical activity, and on physical examination they will have tenderness to palpation of the ulnar shaft. Radiographs may show a small crack in the cortex or a subtle periosteal reaction at the site of the fracture [2]. Radionuclide imaging or MRI can be used to confirm the diagnosis. The treatment of choice for ulna stress fracture includes rest from the offending activity for a period of 6-8 weeks [2].

Distal Radius

Injuries to the distal radial physis are most commonly found in young gymnasts due to repetitive loading [14, 23, 51]. Chronic injuries are noted in 25% of non-elite gymnasts, with an increased prevalence in elite gymnasts [52]. Physeal injury may be due to the result of a compromised blood supply to the metaphysis and epiphysis leading to uncalcified chondrocytes [53]. In gymnasts, excessive body weight compressive forces, up to 16 times their body weight, along with rotational shear forces, contribute to the formation of these stress injuries [9, 54, 55].

Patients often complain of dorsal-sided wrist pain, which is exacerbated by activities that increase pressure in the wrist and is relieved with rest. On physical examination, tenderness to palpation at the distal radial physis is noted. Radiographic findings include physeal widening, cystic changes of the metaphysis, and haziness within the physis [2, 9, 56]. Radiographic findings may be present bilaterally due to the nature of the sport [9]. If initial films are negative, additional imaging with a MRI may be warranted.

Treatment should begin with 4-6 weeks of rest, avoidance of compressive loading, and splint immobilization or bracing. If conservative measures fail, surgery may be necessary and can include arthroscopy or a shortening osteotomy of the distal ulna based on the amount of ulnar variance [56].

Long-term complications include symmetrical and asymmetrical growth plate retardation and positive ulnar variance [2].

Conclusion

Stress fractures of the upper extremity are more uncommon as compared to the lower extremity or spine; however, they can be debilitating. Early diagnosis hinges on understanding the etiology and having an index of suspicion. Most upper extremity stress fractures can be managed nonoperatively when diagnosed early in the process, with a rapid return to sport. More chronic injuries may require surgical intervention to achieve a satisfactory outcome.

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14 Stress Fractures of the Lumbar Spine

Arash J. Sayari, Garrett K. Harada, and Gregory D. Lopez

Introduction

Spondylolysis is defined as the unilateral or bilateral anatomic defect in the pars interarticularis of a spinal segment. The pars interarticularis, or pars, is the bony bridge (or isthmus) between the inferior and superior articular facets of a vertebra. Anatomically, this unit can be found between the lamina and pedicle of a vertebra. Spondylolysis has been historically classified into five broad categories, with the isthmic type II defined as a stress fracture of the pars, and is the topic of this review (Table 14.1) [1].

Isthmic spondylolysis, or stress fractures of the lumbar spine, are almost exclusively seen in the lumbar vertebra, most notably at L5 [2]. Given the increased repetitive stresses on the spine in an adolescent athlete, this population becomes at a distinct risk of developing spondylolysis. Frequently, cases are discovered incidentally on imaging in a patient who is otherwise asymptomatic. However, symptomatic patients often report a history of repetitive twisting, back extension, and axial loading, and may report an acute or chronic pain in the lower back, often not associated with neurologic compromise.

Table 14.1 Types of spondylolysis

Type	Classification	Pathophysiology
Type I	Dysplastic	Congenital
Type II[a]	Isthmic	Fracture of the pars interarticularis
Type III	Degenerative	Degeneration of the intervertebral disc causing instability
Type IV	Traumatic	Acute fracture, not involving pars interarticularis
Type V	Pathological	Tumor or infection

[a]Type II is sub-classified into fatigue fracture (type II-A), pars elongation due to healed stress fracture (type II-B), and acute fracture (type II-C)

Overall, spondylolysis is responsible for more than 70% of cases of back pain in adolescent athletes [3]. While uncommon as a whole, spondylolysis in an adolescent athlete is a relevant diagnosis that requires a thorough history and physical examination to corroborate with imaging. Spondylolysis should be high on a clinician's differential as athletes may self-treat with rest for an extended period, resulting in delays in appropriate care [4]. Further, diagnosis and management of isthmic spondylolysis in the adolescent athlete are crucial as it can lead to spondylolisthesis and be associated with disc degeneration, resulting in chronic low back pain with or without neurologic compromise. Treatment algorithms vary based on such factors and require a multidisciplinary approach involving parents, coaches, and trainers such that athletes can safely return to their sport.

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Etiology, Epidemiology, and Biomechanics

The exact cause of spondylolysis continues to be debated. However, the relationship between repetitive mechanical stresses and pars defects is more so accepted. By being bipedal, humans place increased loads through the axial spine, and studies of non-ambulatory patients and newborns demonstrating no cases of pars defects further support the etiology of spondylolysis [2, 5]. Evaluation of 500 6-year-old subjects demonstrated a 4.4% prevalence of unilateral or bilateral pars defects. By the next one to two decades of life, eight more subjects developed defects, increasing the incidence to 6% with a 2:1 male/female ratio. However, at 45-year follow-up, three of those with unilateral defects demonstrated healing, while the slip progression in those with bilateral defects was slow and variable depending on the onset of spondylolysis, and generally slowed with each decade [6]. Similarly, two other studies of 4001 and 1500 patients demonstrated a 4.6% and 3.7% incidence of spondylolysis, respectively, highlighting the influence of a genetic component to the development of spondylolysis [7, 8]. In fact, nearly one-fifth of relatives in one review study demonstrated findings of spondylolysis, and an even higher incidence in siblings [9].

Axial loading and rotational torque including extremes of extension increase the biomechanical loads on the cervical and lumbar spine. Biomechanical stresses imparted on a genetically or congenitally weakened spine structurally weaken the spine and increase the risk for a pars fracture. Furthermore, dysplastic or hypoplasia of facet joints is unable to resist shear forces naturally resisted by the intervertebral disc and posterior elements, thereby increasing risks for spondylolysis and spondylolisthesis. Cyclic loading increases these shear forces, fatiguing and eventually fracturing the pars. Often unilateral, spondylolysis could progress to a bilateral fracture as the intact pars can see up to a 12.6-fold increase in stress compared to an unaffected spine [10]. Specifically, on lumbar extension, the inferior articular facet of the cranial vertebrae collides with the pars of the caudal vertebrae in a "nutcracker" fashion [9]. Furthermore, the pars is weaker in tension, becoming vulnerable in cases of traction on the spine.

Stress fractures of the lumbar spine are also not reserved only for the adolescent athlete. Elite-level and professional adult athletes are also at risk of developing pars defects. A series of 11 adult athletes with back pain demonstrated increased stress to the lower lumbar spine resulting in 9 patients with unilateral defects and 2 with bilateral defects, highlighting the relevance of biomechanical stresses on a developing and mature spine [11].

In general, male athletes are more than twice as likely to develop spondylolysis, which is most likely to occur at the L5 vertebra and is more common in Caucasians. Specifically, contact sports such as American football can illicit forces on the lumbar spine to as high as 8670 N [12]. Similarly, asymmetric loading and increased traction forces in gymnasts increase the forces across the pars [10]. Muscle asymmetry and hand dominance may play a role in hand-intense sports such as cricket and may even be associated with the sidedness of a unilateral stress fracture, though football (soccer) [13], rugby [12], swimming [14], American football [15], weight lifting, wrestling, and other sports have also been cited as a risk factor for developing isthmic spondylolysis [16–18]. Fortunately, the natural history of unilateral spondylolysis is promising and usually does not progress to spondylolisthesis [6].

History and Presentation

The clinician is often faced with an adolescent athlete who has had an incidental finding of spondylolysis and is otherwise asymptomatic. Such cases often involve no inciting event and lack symptoms of back pain or neurologic deficit, and are identified on CT or MRI of the abdomen/pelvis. Some patients may be prompted into endorsing paraspinal fatigue or occasional discomfort, but all should be thoroughly examined for any underlying spinal pathology and signs or symptoms that would warrant further workup or evaluation.

On the other hand, 47–70% of adolescent athletes with back pain will have spondylolysis [3, 19]. Symptomatic pars fractures are associated

with midline back pain with or without paramidline pain over the site of facet joints. A rare portion of these patients will report radiation of pain to the buttock and proximal lower extremities, though radiculopathy, neurologic deficit, bowel or bladder dysfunction, or night pain may increase consideration for an alternative diagnosis. There is often no inciting event, though adolescent athletes may be able to identify sport-specific activities that reproduce symptoms. Back extension and arduous activities worsen an insidious pain. Discogenic or sacroiliac pain from sitting and forward bending are usually absent, while pain at rest may indicate progression to spondylolisthesis or another diagnosis.

Physical Examination

Even symptomatic patients will often appear with no observed abnormalities, maintaining normal posture, gait, and strength, and may or may not have reproducible symptoms during physical examination. Gait should be assessed for any favoring of one side or gait abnormalities. Adolescents with more advanced spondylolisthesis will portray hamstring tightness with hip and knee flexion and a crouched gait during ambulation. Patients can be asked to "toe walk" and "heel walk" to assess for global balance and dorsiflexion and plantarflexion strength, respectively. Direct examination of the back should be made for any hairy patches or discoloration which would highlight an underlying neurological anomaly. Patients may also demonstrate a visible loss of lumbar lordosis with a palpable step-off.

Range of motion should be well documented. Specifically, the Stork test is when the patient has reproducible ipsilateral lumbar pain with single-leg hyperextension (Fig. 14.1). Adam's forward bend test of the spine can expose any obvious deformity, shoulder asymmetry, or scapular bulging indicative of an underlying coronal deformity. The lumbar spine, paraspinal muscles, and sacroiliac joint should be precisely palpated as

Fig. 14.1 (**a**) Frontal and (**b**) side views of a patient demonstrating hyperextension, or stork test. The patient balances on one leg while maintaining a flexed position of the contralateral hip and knee. As the patient extends through the lumbar spine, the test is positive if pain is elicited in the ipsilateral, weight-bearing side

patients may have midline and paraspinal tenderness. Neurosensory examination of dermatomes and myotomes should be normal, as should reflex testing. Finally, straight leg raise testing and other tension signs can be performed to complete the physical examination.

Imaging

Over the past decade, imaging evaluation of spondylolysis continues to evolve given the historical difficulties associated with its diagnosis. Frequently, the identification of positive imaging findings on routine studies is poorly correlated with clinical presentation, and as such, optimal diagnostic algorithms continue to be debated. Nonetheless, symptomatic low back pain in the young athlete warrants the use of imaging studies, and careful consideration for each test's sensitivity and specificity is paramount.

Plain Radiographs

Typical radiographic evaluation of symptomatic low back pain is often initiated with standard anteroposterior (AP) and lateral views of the lumbar spine. While this is often sufficient for adults, diagnostic accuracy of spondylolysis in the adolescent using these techniques has traditionally been poor [20, 21]. This lack of adequate sensitivity, combined with the inherent anatomy of the pars interarticularis, led to the adoption of oblique ("Scotty dog") evaluation. These views, when taken at a 35–45 degrees from midline, better visualize the course of the pars interarticularis and are thought to maximize detection of radiographic defects (Fig. 14.2).

In recent years, however, diagnostic utility of oblique views in spondylolysis has been debated. This suspicion largely arose from the frequency in which two-view studies lacked efficiency in successfully diagnosing spondylolysis, often requiring advanced imaging studies to further evaluate a patient's symptoms. In 2013, Beck et al. compared the sensitivity of standard AP and lateral views to those including oblique images

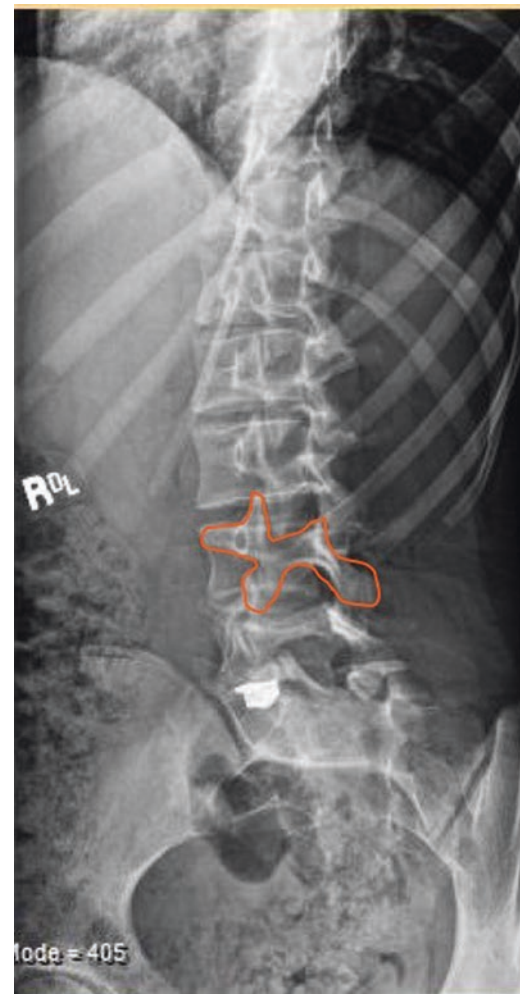

Fig. 14.2 Oblique lumbar radiograph demonstrating a pars defect (arrow) with illustrated shading representing the classic "Scotty dog" appearance. The "neck" represents the pars interarticularis

and found no significant difference in either test's diagnostic utility [21]. This, combined with the risks of additional radiation exposure, has largely led to adoption of other modalities in suspected spondylolysis cases.

Bone Scintigraphy

Single-photon emission computerized tomography (SPECT) is a scintigraphic imaging technique that has now begun to replace the use of oblique radiographs in the setting of suspected

spondylolysis. Like other nuclear medicine imaging techniques, SPECT has the ability to evaluate metabolic activity, and as such, can differentiate between spondylolysis and chronic non-unions of the pars [22]. Some studies suggest that SPECT has greater diagnostic utility when compared to magnetic resonance imaging (MRI) and computed tomography (CT) and can detect roughly 20–25% more lesions with excellent negative predictive value [23–26]. Furthermore, lesions on SPECT are often detectable earlier than CT or MRI, giving it particular utility in acute settings [26].

Though SPECT remains the most sensitive of the imaging techniques in identifying spondylolysis, it includes notable consequences such as poor image resolution, leading to its inability to evaluate neurologic symptoms and for presence of malignancy or infection. Patients with positive findings on SPECT will ultimately undergo CT and/or MRI, and further growing support for adequate diagnostic sensitivity of MRI or CT has led many to forego the use of SPECT in clinical practice [27, 28].

Computed Tomography

Positive lesions identified with SPECT may be evaluated with the use of CT given its superior ability in evaluating osseous anatomy. This is typically performed by limiting image acquisition to thin 1 mm slices of the affected level(s) [24, 29]. Frequently, incomplete and early defects found on SPECT can be missed on CTs [26]. These patients often require a period of conservative management appropriate follow-up to prevent progression of their symptoms [30]. In lesions identified, however, CT is capable of providing enough resolution to assist in staging and can be used to evaluate healing [31]. In 1995, Morita et al. described the progression of spondylolysis based on CT findings, suggesting these features were associated with the severity of the injury [32]. The "early" lesions typically presented with minimal or hairline defects in the pars, while "progressive" injuries were grossly fractured. Later "terminal" lesions, in comparison, presented with sclerotic lesions at the site of injury, and were indicative of more chronic non-unions. This staging has significant implications in the management of the condition as older or more severe injuries often warrant surgical intervention [33].

Though radiation exposure has traditionally been a significant concern with the use of CT, recent evidence suggests that technique of limiting the range of image acquisition may lead to acceptable radiation doses when compared to plain films. In 2015, Fadell et al. evaluated the radiographic exposure and diagnostic utility of CT and two, three, and four views in diagnosis of spondylolysis [34]. In addition to relatively low radiation levels, they found that interpretation of CT had significantly higher interobserver agreement, which suggests that its use may supplant the use of traditional two-view plain films in patients with high suspicion for spondylolysis.

Magnetic Resonance Imaging

Use of MRI in the evaluation of spondylolysis has arguably seen the greatest changes in clinical application. This is largely due to not only concerns over radiation exposure with the aforementioned techniques but also its superiority in the evaluation of neurologic symptoms and ability to diagnose lesions earlier than CT [35]. Therefore, MRI, combined with the development of stronger magnetic fields and various imaging sequences, has begun to emerge as a favorable diagnostic tool in spondylolysis.

In a recent systematic review of the literature, Tofte et al. summarized the findings of all studies making recommendations on imaging techniques for spondylolysis [27]. Given their findings, they note that most of the studies recommend MRI as an early or first-line diagnostic tool, with potential consideration for CT in challenging presentations [31, 35–37]. Similarly, in 2018, Dhouib et al. performed a meta-analysis evaluating the combined sensitivity of MRI in diagnosing pars lesions [28]. They found that MRI was capable of identifying 81%

of spondylolysis cases with a 99% specificity, further providing support for its use in spondylolysis. Despite this, no higher-level prospective studies have investigated the clinical benefit of a particular imaging modality, and firm guidelines regarding the use of MRI have yet to be established.

However, developments are being continuously made to MRI with growing suspicion that new protocols may improve its diagnostic power. In 2019, Finkenstaedt et al. utilized an ultrashort time to echo MR protocol for detection of simulated spondylolysis in human cadavers, noting its superiority to conventional and optimized MR techniques at 3 Tesla [38]. Though such applications may be far from use in the adolescent cohort, MRI will likely continue to evolve and help guide treatment decisions.

Diagnostic Algorithm

Based on the results of their systematic review, Tofte et al. proposed a diagnostic algorithm for evaluation of spondylolysis [27]. First, in patients without evidence of neurologic deficits, but with symptoms consistent concerning for lumbar spondylolysis, two-view plain films are suggested due to low cost and radiation exposure. If plain films are uninformative, CT or MRI should be considered based on the chronicity of symptoms. If the injury is chronic, CT may be used to better evaluate for non-union. Conversely, MRI is more applicable in acute cases or an inciting event in the athlete. MRI should also be more urgently considered in adolescents with neurologic compromise. The authors advocate against the use of CT to evaluate for healing, though this is a consideration in elite athletes or patients whose symptoms fail to improve with appropriate treatment. SPECT is generally not used as part of the treatment algorithm unless a patient is unable to undergo CT or MRI. As with any diagnostic algorithm, these recommendations should be weighed with the symptomatology of the patient, as those with more severe presentations should be more carefully monitored.

Treatment

Given the information from diagnostic studies, the practitioner must carefully weigh the severity of the disease with the patient's athletic goals in designing an appropriate treatment plan. While most instances of spondylolysis may be adequately treated with nonoperative management, a discussion should be had with the patient to optimize return to play and establish expectations. However, much like the literature surrounding imaging in spondylolysis, there is little high-level evidence to support specific strategies in rehabilitation.

Bracing

Use of a lumbosacral (LSO) or thoracolumbosacral orthosis (TLSO) in spondylolysis is believed to help minimize motion of the pars defect, which has been met with varying degrees of support from clinicians (Fig. 14.3). While initial studies demonstrated excellent clinical results with their use, others have demonstrated that bracing may not be a necessary intervention [33, 39]. In a meta-analysis of observational studies investigating clinical outcomes of spondylolysis, Klein et al. note that the use of a brace had no influence on clinical improvement [39]. Moreover, many lesions failed to heal in the observed studies, suggesting that bony fusion is not necessarily required for positive results. Long-term progression of fibrous union of these lesions is poorly understood, though only few progress after adolescence. Generally, bracing may pose utility in the acute phase of spondylolysis by limiting sagittal plane motion.

While both LSOs and TLSOs offer similar utility in limiting lumbar motion, the authors prefer off-the-shelf TLSO with a thigh extension as they provide three-point stability and control pelvic motion. Regardless, orthotic use is left up to patient preference and the clinician's discretion, though implementation is also useful when athletes fail to comply with activity restriction or when they are faced with pressure from coaches, teammates, or family members to return to play.

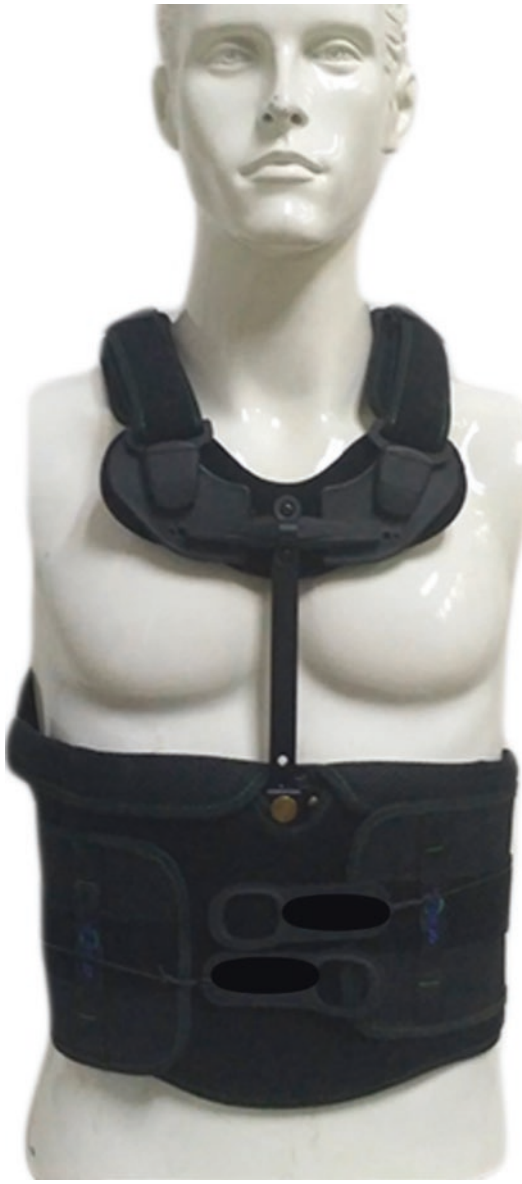

Fig. 14.3 Thoracolumbosacral orthosis

Activity Modification

In any case, the practitioner should emphasize the importance of pain control and limitation of physical activity during recovery. In 2013, El Rassi et al. performed a retrospective analysis of patients with spondylolysis and examined their clinical outcomes based upon adherence to physical activity limitations [40]**.** Notably, those who stopped sports for at least 3 months had significantly greater outcomes when compared to those who continued athletic participation. More recently, in 2016, Panteliadis et al. performed a narrative review on the available literature regarding conservative management of spondylolysis. They reported that athletes who underwent activity limitation returned to sports in 3.7 months, while those undergoing surgery required nearly 8 months [3]**.** No statistical comparisons, however, could be made on their findings due to lack of homogeneous subgroups, illustrating the relative lack of studies validating specific activity restrictions. Until further investigations are completed to establish more precise return to play guidelines, clinicians should attempt a period of rest from play for 3–4 months with close clinical monitoring, adding radiographs when indicated. Rest and gradual return to sport is paramount in avoidance of reinjury and other musculoskeletal injuries from deconditioning.

Low-Intensity Pulsed Ultrasound (LIPUS)

However, with growing favorability of nonoperative management, the practitioner is forced to consider alternative interventions that may promote osseous fusion. Again, though good clinical outcomes do not require bony union, the theoretical risk of instability has led to exploring for alternative treatment regimens [32]. Previous studies have begun to explore the utility of low-intensity pulsed ultrasound (LIPUS) in the promotion of fracture healing, noting significantly shorter times to union in various animal models and clinical trials [41–43].

Similarly In 2017, Arima et al. explored this technique in a small case series, utilizing LIPUS on pediatric patients with progressive-stage spondylolysis and comparing their outcomes relative to an activity-restriction and bracing control group [44]. They noted that time to osseous union was roughly 1 month shorter in the LIPUS cohort, with a fourfold increase in fusion incidence. Though LIPUS has particular appeal given its potential and may reduce time spent in

rehabilitation, there is little evidence to support its consistent use in all adolescent athletes with spondylolysis.

Rehabilitation and Return to Sport

Initial rehabilitation, whether it follows nonoperative or operative management, roughly mimic a three-tiered approach as published by Radcliff et al. [45]. First, particular emphasis is placed on non-impact aerobic activity with neutral spine orientation during the first 3 months. Higher impact activities can then be pursued with eventual reintroduction to sport-specific training at 4–6 months, depending upon the patient's tolerance and symptomatology. Criteria for clearance for return to play include a clinical assessment documenting restoration of normal strength, range of motion, and absence of pain with sport-specific activity [45]. Given the findings of the aforementioned studies, evaluation for osseous fusion may not preclude the adolescent from return to play, and instead clearance should be symptom-based (Fig. 14.4). Imaging techniques should be implemented as needed, primarily if symptoms fail to improve. To date, no guidelines exist regarding specific rehabilitation protocols, nor their effectiveness in improving subsequent outcomes.

Surgical Management

Surgical intervention is reserved for select cases where conservative management has failed to improve symptoms, or when cases of spondylolysis have progressed to a more terminal stage involving incurable pain, progressive spondylolisthesis, or neurologic compromise. In general, 9–15% of patients with symptomatic spondylolysis require surgery [18]. Though the literature on return to play is scarce, surgical intervention may more rapidly allow for an athlete to return to play [46]. Despite this, most authors agree that conservative treatment should be attempted for 6–12 months before recommending surgical intervention, and controversy exists on the most appropriate technique and approach [47, 48].

Direct repair of lumbar stress fractures is reserved for L1 to L4 levels with an intact intervertebral disc, spondylolysis of multiple levels, and low-grade spondylolisthesis. Direct fracture fixation is useful in the adolescent athlete as there is preservation of the motion segment of the spine. Historically, the Buck procedure was described in 1970 as a direct repair of the pars using a 3.5 millimeter (mm) screw in a lag technique to perpendicularly compress across the fracture [49]. More recent studies on adolescent and young adult athletes demonstrated bone healing at a mean of 48 months using a 4.5 mm screw and cancellous bone graft along with decortication of the defect [50]. In addition to cancellous graft, structural fibular grafts can be similarly implemented (Fig. 14.5). In cases of young female athletes with osteopenia or those involving congenital changes to spine such as a dysplastic lamina, screw fixation may be challenging or insufficient. Therefore, variations of pars fracture stabilization have been described. The Scott technique requires 2 mm holes in the bilateral transverse processes and a 4 mm hole in the spinous process, through which 20-gauge wire is passed through in a figure-of-eight technique and compressed over bone graft at the defect site [51]. Other techniques have also been described, though symptomatic hardware may necessitate implant removal after healing [3, 52].

In more advanced cases of high-grade isthmic spondylolisthesis (>30%) in the adolescent, surgical stabilization is the preferred treatment, even in asymptomatic patients [53]. Controversy exists whether slipping vertebrae should be reduced to increase the surface area available for arthrodesis and minimize the risk of pseudarthrosis while improving sagittal parameters. However, aims at minimizing surgical exposures and operative times have been highlighted in cases of in situ fusion. A 1996 review of 59 adolescent patients comparing anterior approach alone in situ fusion versus posterior instrumentation and anterior lumbar fusion shed light on these differences [53]. The non-instrumented anterior lumbar fusion cohort had a higher rate of pseudarthrosis, increased slip, reduced kyphosis, and longer average time to bony union. Therefore, posterior stabilization is a useful adjunct and should be implemented

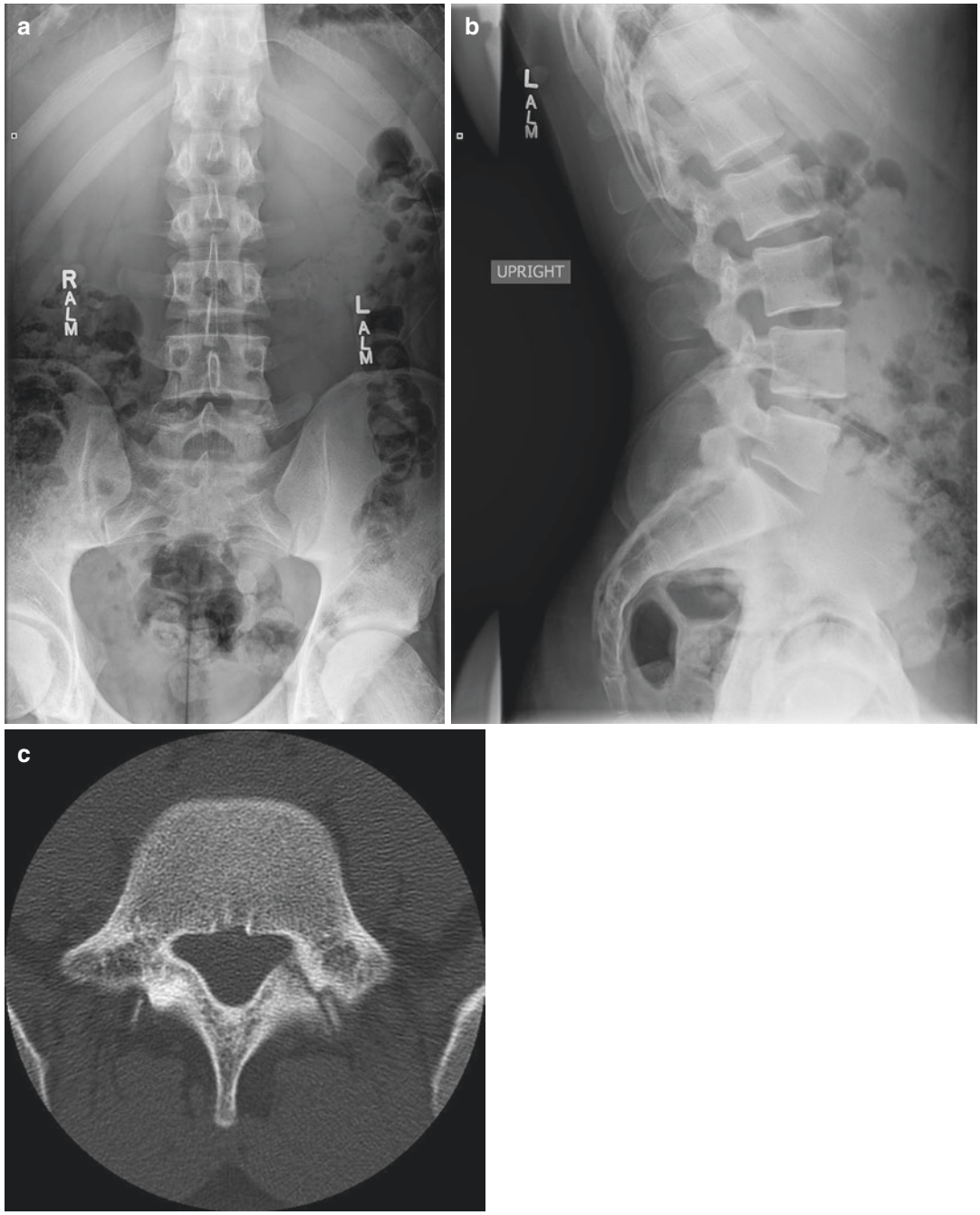

Fig. 14.4 (**a**) AP, (**b**) lateral, and (**c**) axial CT of a 20-year-old collegiate baseball player with 2 months of acute back pain after a swing at bat. He was found to have an acute, left-sided unilateral L5 spondylolysis and was successfully treated with rest and physical therapy

in cases of high-grade isthmic spondylolisthesis undergoing partial reduction. Furthermore, in high-grade spondylolisthesis, the listhetic vertebrae may be difficult to reduce once it becomes wedged between adjacent vertebral bodies and may increase the complication rate when compared to in situ stabilization, which is why reduction remains a controversial topic. Grade IV and

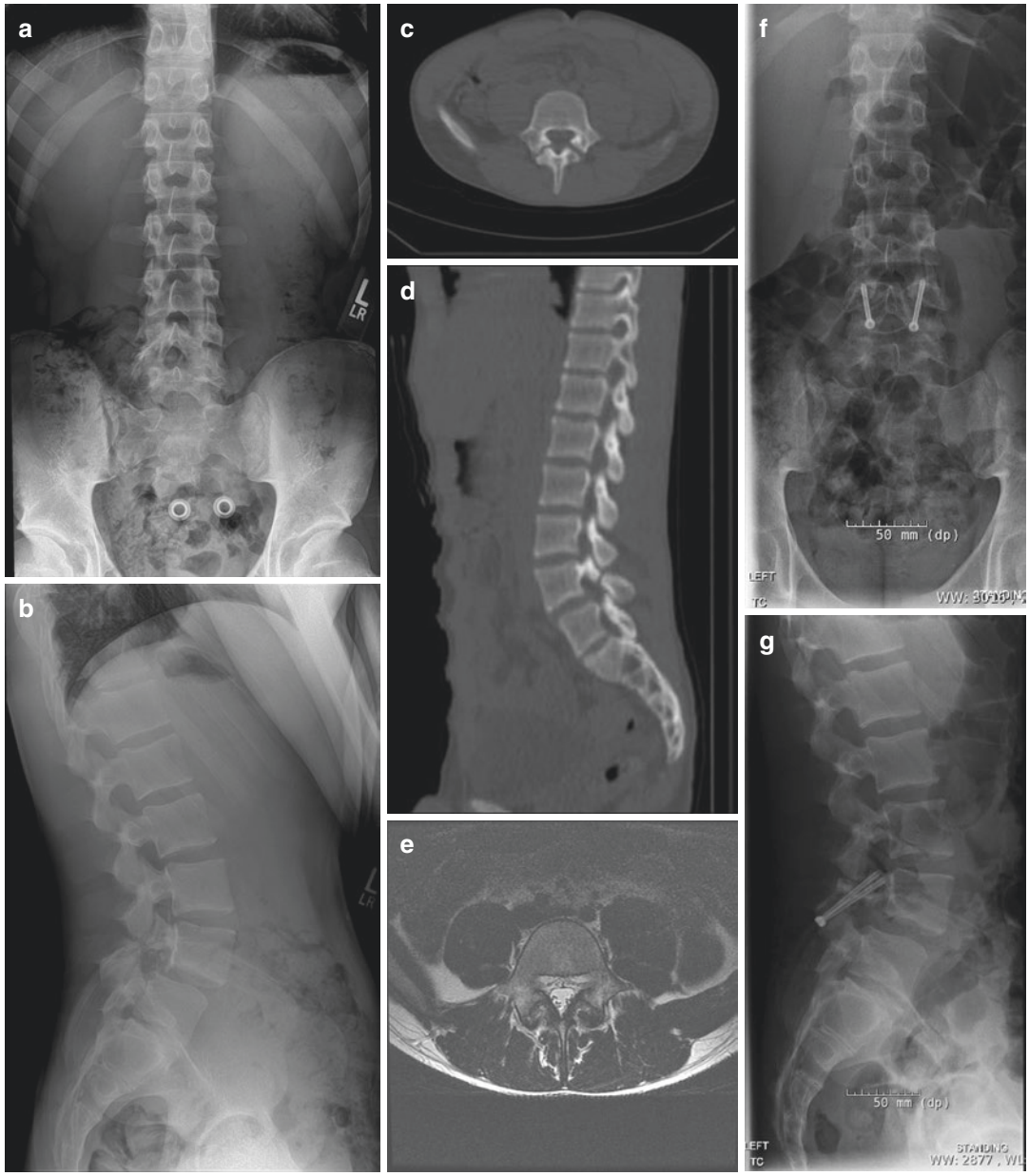

Fig. 14.5 Preoperative (**a**), AP, and (**b**) lateral radiographs of a 17-year-old male collegiate baseball player with back pain, L4 spondylolysis, and L4-L5 grade I isthmic spondylolisthesis who failed 9 months of conservative treatment and rest. (**c**) Axial and (**d**) sagittal CT, and (**e**) axial T2-weighted MRI cuts demonstrated subacute bilateral pars defects without evidence of healing. Fibular strut and cancellous allografts were placed in bilateral pars defects followed by 4.0×40 mm partially threaded cannulated screws to compress across the fracture seen on postoperative (**f**) AP and (**g**) lateral radiographs

grade V slips can leave the vertebra in a more vertical orientation that cannot be reached by an anterior approach, but interbody devices can be placed to increase contact areas and improve fusion rates (Fig. 14.6). Finally, techniques such as fibular dowels placed through the sacrum into the L5 body via a Bohlman technique to optimize local biology and bony fusion have been demonstrated with high success rates, though this technique has become less utilized [54]. On the other hand,

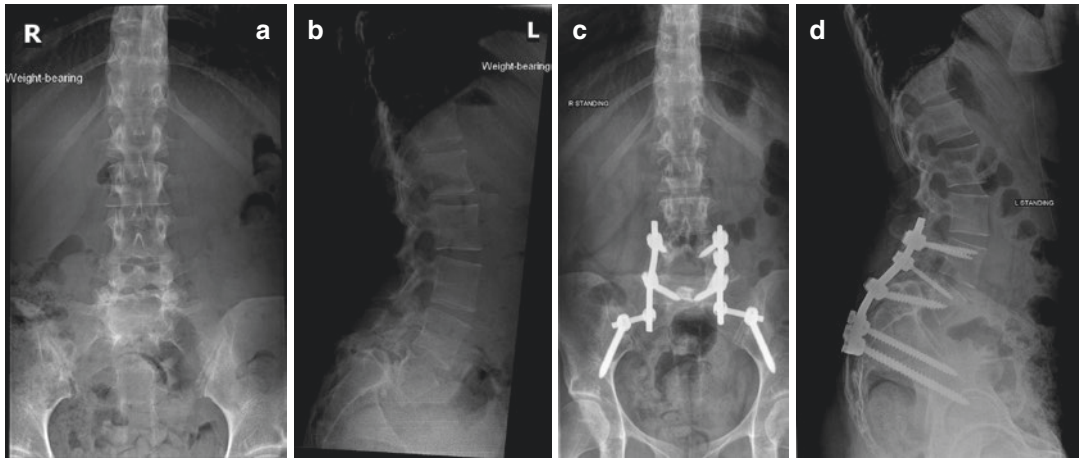

Fig. 14.6 (**a**) AP and (**b**) lateral radiographs of a 14-year-old high-level gymnast demonstrating bilateral L5 spondylolysis and grade III spondylolisthesis. She was treated with in situ L4-S1 decompression, sacral dome osteotomy, L5-S1 transforaminal lumbar interbody fusion, and posterior instrumentation of L4-S1 with allograft and autograft, as seen on the postoperative AP (**c**) and lateral (**d**)

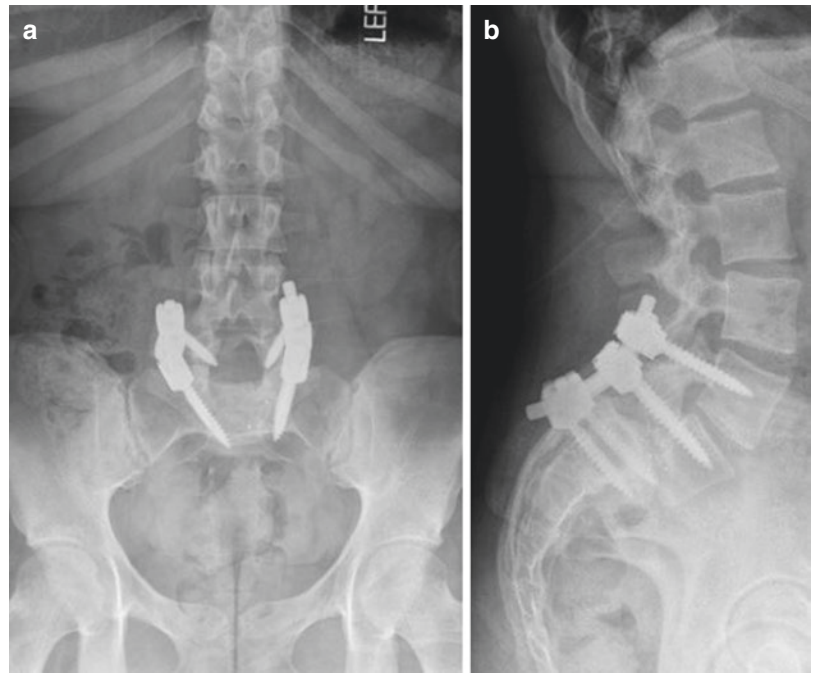

Fig. 14.7 (**a**) AP and (**b**) lateral radiographs of a 29-year-old female, prior athlete, with bilateral L5-S1 grade II isthmic spondylolisthesis and L5 radiculopathy. Given her neuroforaminal stenosis, she was successfully treated with anterior lumbar interbody fusion and percutaneous posterior instrumentation

reduction of high-grade slips improves sagittal balance and increases the contact area between vertebrae and sacrum, improving fusion and decreasing the risk of pseudarthrosis in patients receiving a wide decompression. Surgeons should be aware of the increased difficulty, operative times, and blood loss associated with reduction, as well as the risk of nerve stretch injury, primarily at L5. Therefore, most will err on the side of a wide decompression and partial reduction, followed by instrumented fusion in cases of high-grade slips [52]. In adult athletes who have developed radiculopathy symptoms, Gill laminectomy and foraminotomy are useful though destabilizing, and requires instrumentation for fusion. Alternatively, interbody devices are useful when placed anteri-

orly (Fig. 14.7). Regardless, no consensus exists for the surgical treatment of spondylolysis in the adolescent athlete and surgical nuances are often dictated by surgeon preference and experience.

Pseudarthrosis is the most commonly faced complication after fusion of spondylolysis and spondylolisthesis. However, the risk becomes more evident in cases of high-grade slips and may be more apparent when in situ fusion is attempted in high-grade spondylolisthesis, though the risk is present regardless of the method of attempted fusion. Fortunately, radiographic evidence of pseudarthrosis does not correlate with symptoms. The adolescent with asymptomatic pseudarthrosis can be monitored closely without further intervention. However, progression of spondylolisthesis or deformity, persistent back pain, and neurologic compromise may require revision surgery involving circumferential instrumentation with or without decompression [55].

Neurologic injury is quite rare after surgical stabilization and is generally due to stretch injury during attempted reduction of a high-grade spondylolisthesis. A wide range of neurologic complications has been reported, notably an L5 radiculopathy. In one series, the rate of L5 radiculopathy with motor deficit reached 29%, with improvement being reached by 3 months [56]. Though long-term outcomes are likely unchanged, even transient motor weakness in the athlete can have more devastating effects, demonstrating a useful role for intraoperative neurologic monitoring for early identification of nerve root injury in hopes of minimizing long-term deficits. Bowel, bladder, and sexual dysfunctions are also risks associated with lumbar spine surgery in the adolescent, and progression of such neurologic injuries may necessitate urgent exploration and wide decompression.

Outcomes

Outcomes following surgery or a period of nonoperative management are generally favorable and can be evaluated by the resolution of patient's symptoms and ability to return to pre-injury levels of athletic performance. Some clinicians may also choose to evaluate for osseous union of the pars defect, though studies have demonstrated that most patients do well irrespective of this outcome. Previous investigations suggest that various factors lead to the development of non-union and note association with specific anatomic features, such as bilateral pars defects and/or lesions at L5. In 2004, Miller et al. examined 40 athletes who were 7 to 11 years after initial diagnosis of spondylolysis and found that none of the bilateral defects had healed [57]. However, if caught early, bilateral defects have been shown to heal in some cases [58]. Though nearly all patients reported excellent outcomes in either study, a more recent observation by McCunniff et al. suggests that bilateral pars lesions at L4-L5 may be associated with worse degenerative disc disease than L5-S1 [59]. However, their investigation was limited by use of cadaveric specimens, and it is unclear if the amount of degeneration observed was clinically significant.

Degenerative spondylolisthesis caused secondarily by spondylolysis is a fairly common complication, though it rarely presents symptomatically or with progression after adolescence [2]. However, before skeletal maturity, regular monitoring should be performed to assess for progression of vertebral slippage. Beutler et al. note that a cohort of asymptomatic patients with concurrence of spondylolysis and spondylolisthesis have a clinical course resembling the general population when followed from childhood to age 50 [6]. Of note, spondylolisthesis did not develop in patients with unilateral pars defects and, when present, largely only progressed during the adolescent growth spurt. Though no clear guidelines exist, patients should receive repeat radiographic evaluation every 6–12 months until skeletally mature to evaluate concurrent spondylolisthesis [60]. Those with persistence or progression of symptoms during this interval may subsequently be considered for surgery.

Cervical and Thoracic Stress Fractures

Stress fractures of the upper thoracic and lower cervical spine are far rarer than their lumbar spine counterparts. Such fractures, termed clay shoveler's fractures, are stress injuries to the

posterior elements, namely the spinous process. Though the term was derived from early laborers, athletes with similar biomechanical motions develop similar injuries [61]. Like lumbar stress fractures, studies have evaluated various sports implicated in the development of clay shoveler's fractures and include American football [62], volleyball [63], rock climbing [64], powerlifting [65], baseball, and wrestling [66]. Repetitive shear forces seen during golf have played a role in the development of multi-level injuries.

Diagnosis

A cervical stress fracture should be suspected in athletes with atraumatic acute or chronic neck pain. Classically, patients report a "pop" or knife-like stabbing sensation in the posterior elements of the spine, between the scapulae. Patients will also often report an associated sensation of muscular fatigue and spasms.

On examination, patients will often rest with relative neck flexion and scapular elevation as to compensate for painful upper thoracic and lower cervical spine motion. Furthermore, upper extremity and neck range of motion is limited secondary to pain, though it may be normal in more chronic cases. Patients will often experience reproducible tenderness to palpation over the affected spinous processes. However, neurologic examination of the upper and lower extremity dermatomes, myotomes, and reflexes should be normal, and abnormalities may point toward an alternate diagnosis.

Plain radiography including AP and lateral views of the lower cervical spine may be normal. However, practitioners should seek out abnormalities such as the appearance of a "double spinous process sign," on the AP view, suggesting an avulsion injury [67]. Normal-appearing X-rays may require advanced imaging to avoid delays in diagnosis and management (Fig. 14.8). CT and MRI provide useful clues and sheds light

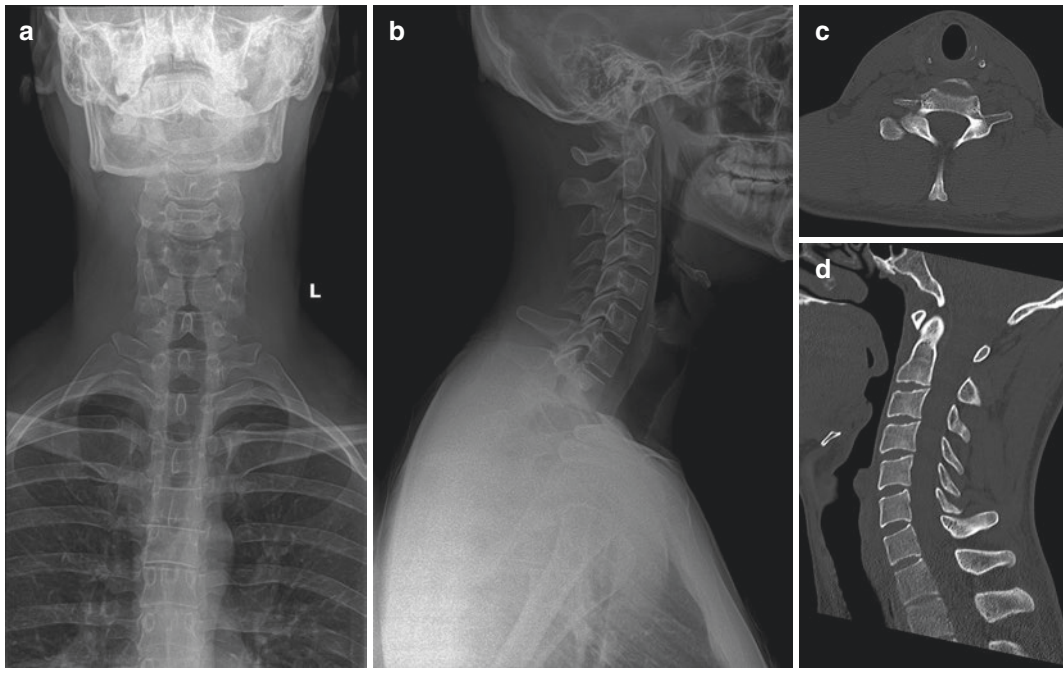

Fig. 14.8 (**a**) AP and (**b**) lateral radiographs of a 25-year-old construction laborer who presented with 2 months of midline, lower neck pain, which were interpreted as normal. However, close examination demonstrates a vertically oriented fracture of the T1 spinous process. (**c**) Axial and (**d**) sagittal CT redemonstrated the fracture without sclerosis. Not pictured, an MRI did not demonstrate significant bony or soft tissue edema, highlighting the subacute nature of the fracture. The patient was successfully treated conservatively with time off work and physical therapy

on the chronicity of such injuries and should be obtained when necessary [68].

Management

Athletes should refrain from play until symptoms resolve, which may take 4–6 weeks, or longer [61]. While physical therapy can aggravate acute symptoms, it may be a useful adjunct in improving strength and mobility and should include pain control modalities and posture training [62]. Also during the acute phase of injury, hard cervical collars limit motion of the upper thoracic and lower cervical spine, providing symptomatic relief by prohibiting motion of the avulsed segments. Case reports have suggested positive outcomes when using hard collars for 3–4 weeks, though appropriate fit is critical [63, 69].

Outcomes are favorable following diagnosis of cervicothoracic stress fractures, and most athletes will return to their prior level of play. However, in the select population who do not improve following conservative treatment, surgical excision of bony fragments may be required. Reports suggest surgical excision at 3–10 months of persistent symptoms, which involves the removal of loose fragments and ossicles that have achieved non-union [70, 71].

Surgical Treatment

In cases of severe or persistent pain, surgical intervention is warranted [61]. Typically, surgery involves excision and removal of bony fragments. In a unique case report, a 38-year-old male presented with a C7 clay shoveler's fracture after playing a Wii video game [72]. He was treated conservatively with bracing and physical therapy for 3 months. However, his pain persisted and surgery was performed. Removal of the bone fragments completely resolved his pain. In a case series by Murphy and Hedequist, three athletes who were initially treated with rest and activity modification for a fracture at T1 continued to have persistent and debilitating pain after 10 months of treatment [71]. They were found to have non-union of the ossicle and were treated with surgical removal of the loose fragments, followed by smoothing of the intact spinous process. This completely resolved their pain symptoms.

Summary

The adolescent athlete with back pain requires close attention as many will have undiagnosed pars fractures that they have attempted to self-treat by resting with inadequate evaluation and management. In cases of high suspicion, X-ray and advanced imaging should be implemented. Fortunately, most unilateral defects will not progress or remain asymptomatic, while bilateral defects and chronic defects often require more frequent follow-up. Initial management involves rest from play with or without physical therapy, though bracing has limited utility. Surgical intervention is generally successful when appropriately indicated, and may involve pars repair or more advanced stabilization and fusion techniques. Similarly, stress injuries to the upper thoracic and lower cervical spine are related to repetitive shear forces that cause avulsion injuries to the spinous processes. Fortunately, these injuries respond well to conservative treatment.

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15 Stress Fractures of the Pelvis and Sacrum

Burak Altintas, Timothy L. Miller, and Mary Lloyd Ireland

Introduction

Pelvic and sacral stress fractures are uncommon overuse injuries accounting for up to 7.1% of all stress fractures in athletes [1, 2]. They are considered low-risk stress fractures and can be broadly categorized into two groups: insufficiency fractures and stress fractures [3, 4]. The pathophysiology differs between these two types of fractures, though the two injuries are not mutually exclusive. Insufficiency fractures result from normal stresses being applied to osteopenic or osteoporotic bone, while stress fractures occur from abnormally intense or overly frequent stresses being applied to normal bone. For this reason, insufficiency fractures are typically observed in elderly patients while the stress injuries are more common in young athletes. Most season-ending stress fractures affect the lower back, lumbar spine, or pelvis (16.5%) after the metatarsals (25.9%) and the tibia (18.0%) [5]. Moreover, the locations with the largest proportion of recurrent stress fractures has been shown to be the lower back, lumbar spine, and pelvis (22.2%) following the metatarsals (29.2%) [5]. They may be difficult to detect and have been reported mainly in long-distance runners and female military recruits [6–8].

Pubic rami stress fractures are more common in women than in men, with pain occurring in the inguinal, perineal, or adductor region [9]. Sacral stress fractures are a rare but treatable cause of low-back and buttock pain, and these injuries have been shown to occur in both female and male athletes as well as in military recruits [3]. They may also occur in young females training in the peripartum period [3]. Given how uncommonly this injury is diagnosed in young athletes and the non-specific symptoms with which the athletes present, orthopedists and sports medicine clinicians must have a high index of suspicion for this injury and for the pathophysiology underlying it. The objective of this chapter is to provide a better understanding of the risk factors and the common presentation of these injuries and to provide rehabilitation and prevention strategies for the athletes in which they occur.

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Predisposing Factors and Training

Repetitive axial loading, resulting from ground reaction forces and muscle contraction, is inherent to these injuries [10]. Pelvic stress fractures affect predominantly females. Bennell et al. found pelvic stress fractures only in female track and field athletes, including mostly middle-distance and long-distance runners [11]. Subjects in this study had an average BMI of 21, were 20 years old on average, and were running 40–53 km per week [10, 11]. Low pre-training aerobic fitness was found to be the only modifiable risk factor associated with stress fractures during boot camp in female military recruits [7]. Risk factors for stress fracture among young female runners include previous stress fractures, lower bone mineral density, and menstrual irregularity [12].

Sacral stress fractures have been occasionally reported during the last trimester of pregnancy and the early postpartum period. Risk factors for sacral stress fractures during pregnancy or in the first weeks after delivery include vaginal delivery of a high-birth-weight infant, increased lumbar lordosis, excessive weight gain, and rapid vaginal delivery. Other probable promoting factors include vitamin D insufficiency, anticoagulant therapy with heparin, and transient osteoporosis associated with pregnancy and lactation [13].

Nutritional risk factors may be present in both male and female athletes who participate in sports that emphasize the need for leanness such as distance running, dance, and gymnastics. In these populations athletes should be screened at the pre-participation physical and upon injury evaluation for signs of the female athlete triad of (1) disordered eating, (2) decreased bone density, and (3) amenorrhea. More recently the term Relative-Energy Deficiency in Sport (RED-S) has been used to more broadly encompass inadequate caloric intake in both male and female athletes who may overtrain without fully replacing the energy stores used during training. This condition has shown a direct correlation with stress fracture risk including pelvic and sacral stress fractures [14].

Table 15.1 Differential diagnosis for pelvic and sacral stress fracture in the running athlete

Insufficiency fracture
Spondylolysis
Apophysitis and avulsion fracture
Tendinopathies
Sports hernia
Osteitis pubis
Athletic pubalgia
Rheumatologic disease (ankylosing spondylitis)
Tumor
Gastrointestinal disorder
Urologic or gynecologic condition

Differential Diagnosis

During the evaluation of pelvic and sacral stress fractures, a broad range of differential diagnoses should be considered. Table 15.1 summarizes the common conditions that are present in a similar manner to pelvic and sacral stress injuries. These include muscle injuries such as adductor strain, piriformis syndrome, lumbar disc disease, spondylolisthesis or spondylolysis, or referred pain from gastrointestinal or genitourinary disorders. Moreover, the possibility of soft tissue and osseous tumors along with infection/osteomyelitis should also be taken into consideration. Systemic diseases affecting bone metabolism such as osteoporosis, rheumatoid arthritis, fibrous dysplasia, Paget's disease, osteogenesis imperfecta, osteomalacia, and hyperparathyroidism are also to be considered. Other rheumatologic conditions such as ankylosing spondylitis should be ruled out, particularly in young males with pain around the sacroiliac joint (Fig. 15.1) [15].

Patient History and Physical Examination

Bony stress injuries occur most often in women, military recruits, long-distance runners, or joggers after increases in duration, frequency, or intensity of impact loading exercise. The most common site for stress fracture of the pelvis is the pubic rami [10]. Patients with stress fractures of the pubic rami present with insidious pain in the inguinal, perineal, or adductor regions that

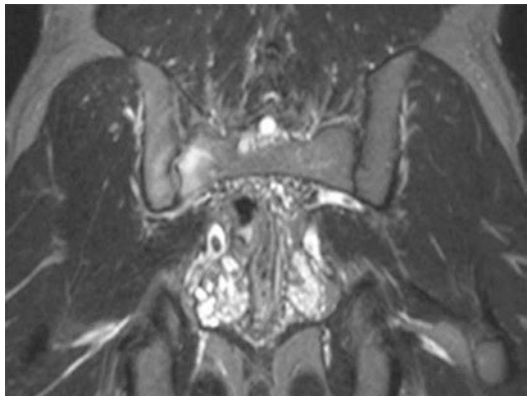

Fig. 15.1 Coronal T2 MRI of a 21-year-old collegiate long jumper with chronic sacral pain initially diagnosed with sacral stress fracture later determined to have seronegative arthropathy from ankylosing spondylitis

is relieved by rest [10]. Given the multitude of differential diagnosis including but not limited to muscle injuries such as adductor strain, piriformis syndrome, lumbar disc disease, spinal stenosis, spondylolisthesis or spondylolysis, as well as tumors and infection, a thorough clinical history and physical examination are essential in the diagnosis of sacral and pelvic stress fractures [3]. It is important to ask about training habits as they contribute to stress fractures and a sudden increase in exercise intensity increases the risk of a stress fracture [16, 17]. The female athlete triad consisting of amenorrhea, disordered eating, and osteopenia/osteoporosis is associated with the development of stress fractures [18]. Thus, careful evaluation of the menstrual history and screening for nutritional deficiencies are key.

Clinical assessment begins with lower limb alignment (varus or valgus) and symmetry in the legs, ankles, and feet, as these may affect biomechanical forces. Sacral stress fractures may produce a positive FABER test (pain with hip flexion, abduction, and external rotation) as well as a positive Flamingo (single leg stance) test, and pain with single leg hop testing (Fig. 15.2) [10, 16]. As with any musculoskeletal examination, a neurovascular assessment should be included. Patients with pelvic stress injuries may demonstrate an antalgic gait, a positive Trendelenburg sign, limited range of motion of the hip, tenderness over the pubic rami, or an inability to stand

Fig. 15.2 Female collegiate runner demonstrated single leg hop test

unsupported on the affected side. These patients usually will have normal hip and spine range of motion but complain of deep groin pain at the extremes of hip motion.

Imaging Evaluation

Radiographs

Appropriate radiographs of the pelvis (anteroposterior, inlet and outlet views, if necessary, iliac and obturator oblique views) should be obtained initially in patients with suspected pelvic or sacral stress fractures. Radiographic imaging may not show any changes or only a subtle cortical radiolucency with possible periosteal reaction [19]. The definitive diagnosis of these injuries with plain radiographs remains challenging as

the presence of bowel gas may limit adequate visualization of the bone anatomy, though more severe or chronic injuries may be identifiable after some healing (Fig. 15.3a).

MRI and Bone Scintigraphy

Further imaging is usually recommended in cases where there is high suspicion for a stress fracture based on the patient's history and examination. The MRI is often the chosen imaging modality due to its higher sensitivity and specificity for detecting stress fractures without the risks of radiation exposure [16]. Signs of stress fractures such as surrounding soft tissue swelling, bone edema, or fracture line can be demonstrated [19]. (Figures 15.1, 15.3b, 15.4, and 15.5) While the MRI is helpful in the diagnosis and staging of stress fractures, MRI changes may not be definitive in the early stages of the injury [20]. While nuclear scintigraphy or bone scans may also be used to evaluate stress fractures, they are not the preferred diagnostic tool given the limited specificity and relatively high radiation exposure given the young age of the patients [19]. Additionally, while whole body bone scans are of high utility for identifying stress fractures at multiple sites, they may remain positive for up to 2 years, despite clinical healing of the injury [19]. This makes them less useful for determining the appropriate time for return to athletic participation.

CT

CT scans are a reasonable alternative to MRI for evaluation of stress reactions/fractures of the pelvis or sacrum. They are especially useful for evaluating healing in the case of a delayed union or for pre-operative planning [21]. However, due to the high radiation dose which they impart onto patients, they are less desirable as a first-line imaging modality than MRI, specifically for young women who may be pregnant or planning to become pregnant [19, 21].

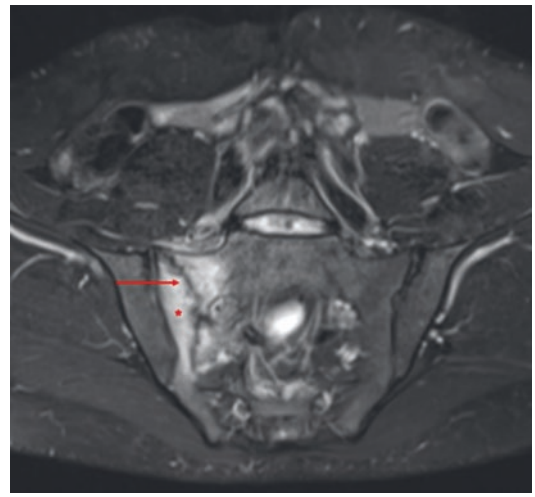

Fig. 15.4 The T2-weighted coronal MRI slide shows a right sacral stress fracture in this 28-year-old male runner. The red arrow shows the fracture line and the asterisk points out the surrounding edema

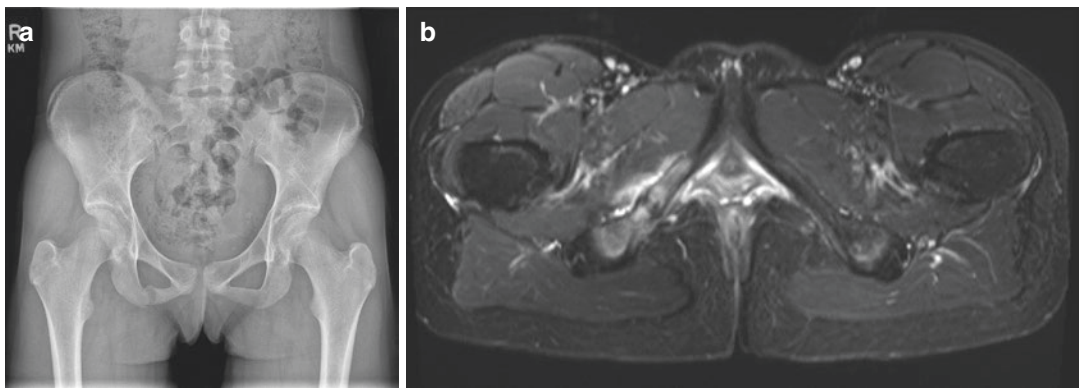

Fig. 15.3 Radiograph (**a**) and axial T2 MRI (**b**) of a 20-year-old female collegiate long-distance runner with Kaeding–Miller grade III stress fracture of the right inferior pubic ramus. She was also diagnosed with rib and tibial stress fractures. Laboratory testing revealed a gastrointestinal malabsorption disorder

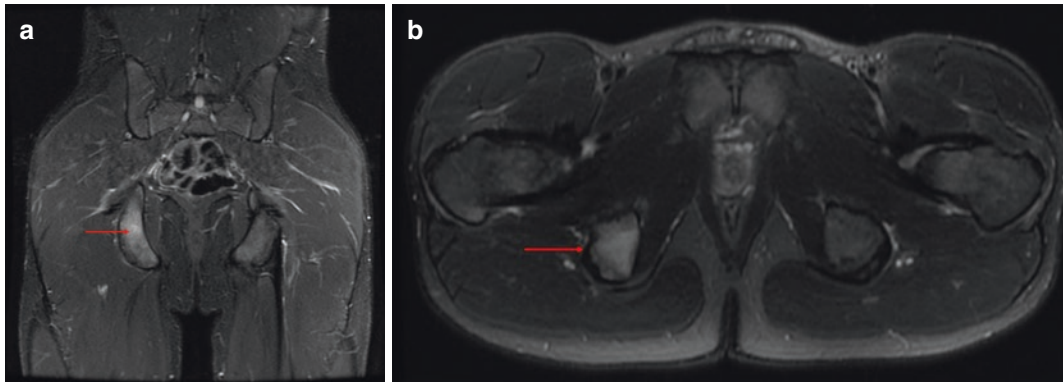

Fig. 15.5 The STIR coronal MRI cut (**a**) and axial T2 fat-suppressed MRI cut (**b**) show a right ischial stress reaction in this 15-year-old male football athlete. The red arrows show the surrounding edema

Bone Density and Body Composition Testing

Tenforde and associates have recently demonstrated a correlation between low bone mineral density and stress fracture risk particularly in areas of trabecular bone [22]. Bone density testing with DEXA (duel energy X-ray absorbtiometry) scanning is therefore recommended for athletes with recurrent, multiple, or chronic stress fractures and for surveillance of athletes with insufficiency fractures from known low bone mineral density [23]. More recently, body composition testing with iDEXA (Intelligent DEXA) to determine an athlete's muscular makeup has been performed to assess an athlete's risk of bone stress injury [24]. The derived data include lean mass/non-lean mass ratio as well as identification of areas of localized muscular atrophy. This data may then be used to design protocols for strengthening areas of muscular weakness and reducing fatigability at specific sites thus decreasing the load and ground reaction forces absorbed by the bones [24].

Laboratory Workup

The causes of pelvic and sacral stress fractures are often multifactorial with a systemic etiology rather than a simple biomechanical or musculoskeletal explanation. Because of this, a systemic workup including serum laboratory testing is recommended for athletes who present with recurrent or multiple stress fractures following imaging evaluation. Table 15.2 describes the serum laboratory testing that is recommended for athletes with recurrent, multiple, or chronic stress injuries. In addition to vitamin D levels, important laboratory values to obtain include serum calcium and phosphate levels, parathyroid hormone (PTH), thyroid-stimulating hormone (TSH), alkaline phosphatase, albumin, and pre-albumin [25]. These tests are crucial for assessing nutritional status and healing potential. In female athletes, serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol levels are recommended to determine if an underlying endocrine condition or energy imbalance is contributing to decreased bone mineral density or recurrent injury [25]. Furthermore, specific tests for rheumatologic conditions such as HLA-B7

Table 15.2 Recommended laboratory tests for athletes diagnosed with pelvic and sacral stress fractures [25]

Comprehensive metabolic panel
Calcium, magnesium, phosphate
Albumin and pre-albumin
Alkaline phosphatase
Vitamin D
Estrogen/estradiol and progesterone
Testosterone
Thyroid and parathyroid hormones
GnRH, FSH, LH
ANA, HLA-B27
Specific tests for GI malabsorption disorders

and tests for gastrointestinal malabsorption disorders may be required to determine underlying causes and/or risk factors in athletes with stress fractures [15].

Nonoperative Treatment

Currently, there is no established best treatment for sacral stress fractures. Figure 15.6 shows an algorithm employed by the authors for evaluation and treatment of these injuries. Medications such as non-steroidal anti-inflammatory agents, acetaminophen, or in severe cases opioid analgesics may be used in the short term for pain control. In female athletes with a history suggestive of the female triad, oral contraceptive pills may be used to address amenorrhea.

Early mobilization for stable sacral stress fractures which do not require surgical intervention should be considered [16]. Non-weight-bearing may be discontinued where there is no more pain with ambulation. Cross-training with biking, elliptical, or swimming may begin after 1–2 weeks of no symptoms in order to avoid deconditioning [10]. Physical therapy can then be initiated, which includes a core and hip strengthening/stabilization program.

Operative Treatment

Operative treatment is reserved for selected cases with impending or present non-union of pelvic or sacral stress fractures. Successful outcome has been reported in a college football player who was diagnosed with an ischial stress fracture that went on to become a symptomatic non-union after extensive nonoperative management. The athlete was treated with open reduction internal fixation utilizing a tension band plate and screws [26]. Recently developed surgical techniques have been used for directly injecting calcium sulfate or bone graft substitutes into areas of low bone density in order to increase structural integrity of the bone and stimulate healing [27]. Additionally, procedures employing orthobiologic treatments for bone stress injuries such as concentrated bone marrow aspirate have been developed and shown

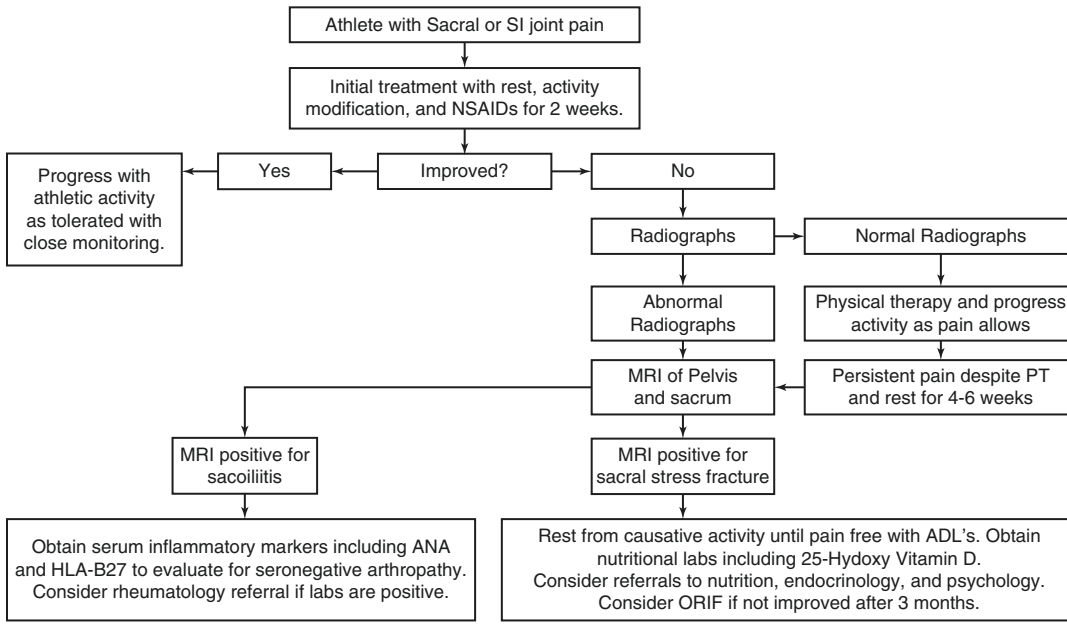

Fig. 15.6 Authors' recommended algorithm for evaluation and treatment of sacral pain concerning for sacral stress fracture. (Reproduced with permission from Vajapey et al. [3])

promise for enhancing bone healing including areas of the pelvis and sacrum [27].

Adjunctive Treatment

Adjunctive treatment includes addressing underlying nutritional and metabolic deficits, as well as altering the local fracture environment [28]. Vitamin D levels correlate highly with quality of local bone microenvironment and reparative ability after repetitive stress [27]. Numerous studies have evaluated the use of vitamin D supplementation in the military and in collegiate athletes. Patients with vitamin D insufficiency and deficiencies show delayed healing; however, vitamin D treatment alone without calcium does not appear to reduce stress fracture risk [27, 29–31]. A general nutrition consult along with evaluation by a sports psychologist should be performed in athletes with concerns of improper nutrition or disordered eating.

Conclusion

The diagnosis of pelvic and sacral stress fractures remains difficult, though increasing awareness of these injuries may expedite their diagnosis, treatment, healing, and return to sports. Distance runners, gymnasts, and military recruits are at highest risk for pelvic and sacral stress fractures due to the repetitive high-impact nature of the required training. They can occur in males and females, though females may be at greater risk due to the female athletic triad and RED-S. Sacral stress fractures may have atypical presentations including acute pain and radicular symptoms and may even coincide with lumbar disk herniation or stress fractures at multiple sites. Sports medicine clinicians must have a high index of suspicion for this injury. Diagnosis typically requires advanced imaging with MRI being the most sensitive and specific. Nonoperative treatment is the mainstay and should consist of rest from high-impact activities for at least a period of 6 weeks along with nutritional support including calcium and vitamin D supplementation. Surgery should be reserved for the rare cases of symptomatic nonunions. The physician must mitigate risk factors for fatigue fractures by addressing nutritional status, vitamin deficiencies, and psychological conditions including eating disorders and malabsorption conditions in order to prevent recurrence or worsening of these injuries.

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Stress Fractures of the Hip and Femur

16

Joshua D. Harris, Jessica Le, and Vijay Jotwani

Introduction

Stress fractures are common overuse injuries observed in athletes in a wide variety of sports. Endurance athletes require either high intensity and/or extended periods of repetitive hip motion and loading that may tip the balance of osseous homeostasis to bone stress injury. Over 80% of athletic hip injuries are due to overuse [1]. Although most athletic hip and femur injuries are intra-articular (e.g., femoroacetabular impingement [FAI] syndrome, labral tear, arthritis, dysplasia), bony stress injuries of the hip and femur are common extra-articular causes of hip and groin pain that may limit athletic performance. Stress fractures of the femoral neck and shaft are common, accounting for 5–11% and 3.5–7% of all stress fractures, respectively [2].

Stress fractures of the hip and femur can be characterized by location, type, and grade, which allow determination of a stress fracture's risk status – "low-risk" versus "high-risk." Low-risk stress fractures, regardless of location, generally have low risk for fracture displacement, short- or long-term complications (e.g., avascular necrosis, delayed union, and non-union), or need for surgical treatment [3]. High-risk stress fractures carry a significant risk of serious consequences if left untreated. Thus, in evaluation of patients possibly prone to high-risk stress fractures, an aggressive approach to diagnosis, including plain radiographs and advanced imaging (e.g., magnetic resonance imaging [MRI], computed tomography [CT], nuclear medicine bone scan), is required to commence early and appropriate treatment. Around the hip (Fig. 16.1), tension-sided (superior) stress fractures of the femoral neck are designated "high risk," while compression-sided (inferior) stress fractures of the femoral neck "low risk." Femoral shaft stress fractures are generally considered low risk, as they tend to have an excellent vascular supply available for healing. Subtrochanteric stress fractures due to overuse, in the presence of normal bone mineral density and metabolism, are considered low risk. Subtrochanteric stress fractures in the presence of poor bone mineral density (atypical, often secondary to medication use [e.g., bisphosphonate]) are considered high risk. Most, but not all, low-risk stress fractures can successfully be treated non-surgically, while most, but not all, high-risk stress fractures require surgery.

Stress fractures frequently occur secondary to one of the following three mechanisms: (1) abnormal stress on normal bone (e.g., fatigue fracture; endurance athletes, runners, excessive load); (2) normal stress on abnormal bone (e.g., insufficiency fracture; low bone mineral density,

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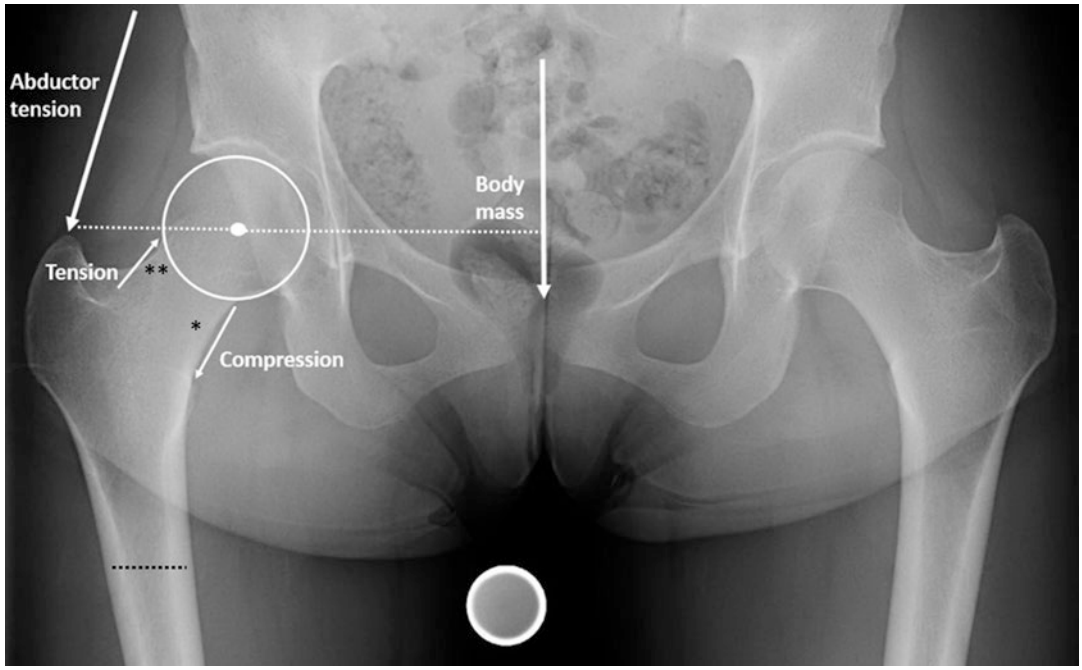


Fig. 16.1 Anteroposterior pelvis weight-bearing radiograph illustrating force across the hip and proximal femur. Around the center of rotation of the hip (white dot), body mass tilts the pelvis to the left, requiring abductor (gluteus medius, gluteus minimus) force to maintain coronal balance. Two-dimensionally, this tilt imparts a tensile stress to the superior femoral neck and a compressive stress to the inferior femoral neck, which accounts for high-risk tension-sided femoral neck stress fractures (two black asterisks) and low-risk compression-sided stress fractures (one black asterisk). Femoral shaft stress fractures tend to be transverse in the proximal shaft (single black dotted line) or subtrochanteric region

osteoporosis, medication use [e.g., bisphosphonate]); and (3) both abnormal load and abnormal bone [4]. Repetitive loads on the lower extremities (e.g., ultra-marathon runners, armed forces personnel during basic training, long-distance obstacle course athletes, triathletes, gymnasts, ballet dancers) increase stress fracture risk in the hip and femur [5]. Relative energy deficiency in sports (RED-S) is a clinical syndrome that entails both significant health risks and performance problems secondary to low energy availability [6]. Low energy availability is the underlying theme of both RED-S and the female athlete triad/tetrad and is the result of a simple imbalance between energy output (basal metabolism and sports/exercise/training) and input (caloric consumption). As opposed to the "female athlete triad" (menstrual dysfunction, low bone mineral density, and low energy availability with or without eating disorder) and "tetrad" (triad plus endothelial dysfunction) syndromes, RED-S is applicable to both males and females [7]. In women, low energy availability presents as menstrual irregularities, and in men, it presents as irregularities due to low testosterone levels [8]. These endocrinopathies are associated with decreased bone health and subsequent increased stress fracture risk.

Anatomy

Normal hip and femur anatomy is complex, but based on simple physics and force body diagrams. Thus, an understanding of normal osseous homeostasis requires only a basic understanding of axial loads, moment arms, and torque. Deviation from normal hip anatomy may impart abnormal stress on the bone, increasing bone injury risk. In the coronal plane,

normal femoral neck-shaft angle (NSA; also known as CCD [caput-collum-diaphyseal] angle) is approximately 125° (normal range 120–135°), and this value plays a significant role in stress fracture risk across the femoral neck [9]. Coxa valga is defined as NSA greater than 135°, with the tip of the greater trochanter below the center of the femoral head. This is typically accompanied by decreased offset from the abductor insertion to femoral head center of rotation, requiring greater abductor tension to maintain a level pelvis. This places more axial compressive force (and less tensile; decreased bending moment) on the superior side of the neck, thereby decreasing the risk of tension-sided stress fractures. Coxa vara is defined as NSA less than 120°, with the tip of the greater trochanter above the center of the femoral head. The increased offset from abductor insertion to hip center of rotation (and increased bending moment) places greater tensile stress across the superior neck, thereby increasing tension-sided stress fracture risk [10].

In the axial and sagittal planes, femoral version plays a role in both femoral neck and shaft loading. The shaft has a normal anatomic anterior bow (convex side apex anterior), with the tension side anterior and the compression side posterior. Depending on the technique of measurement, normal femoral version is approximately 10–20° (distal axis is posterior aspect of posterior femoral condyle line; proximal axis is variable, from as low as the lesser trochanter to as high as the mid-femoral neck) [11]. Increased femoral version (>20–25°; relative anteversion) effectively shortens the coronal plane lever arm, with more posterior positioning of the greater trochanter abductor insertion, decreasing the distance to the center of rotation and femoral offset, and subsequently increasing axial compression (less tensile stress) on the superior and posterior neck and decreasing tension-sided stress fracture risk. Decreased femoral version (<5–10°; relative retroversion) effectively lengthens the coronal plane lever arm, thereby increasing femoral offset and tensile stress (and stress fracture risk) across the superior neck.

Patient Evaluation

History

Patients with stress fractures of the hip or femur typically present with activity-related pain, usually with weight-bearing, excessive in distance, intensity, or both. Pain location is deep anterior, anteromedial, from the groin down the anterior/medial thigh. Patients with femoral neck stress fractures will commonly complain of pain in an intra-articular location, demonstrating a "C sign" or "between the fingers sign", but distinguished from FAI syndrome and labral tears due to provocation mechanisms – femoral neck stress fractures have increasing pain with increasing weight-bearing, versus FAI syndrome which is a motion- and position-dependent entity associated with increasing hip flexion and rotation [12]. Nonetheless, the former and latter may coexist and an astute diagnostician must be aware of and treat both, when present. Patients with femoral shaft stress fractures have increasing pain with increasing weight-bearing, but typically the pain is perceived in the anterior/medial thigh and knee.

A critical component of the history of present illness requires a thorough evaluation of the patient's training that led to the onset and/or progression of pain – has there been a rapid increase in weekly mileage, weekend long run distance, number of days run per week, increased speed work, increased plyometric cross-training, new shoes, new inserts/orthotics, form changes (hindfoot-, to midfoot-, to forefoot-strike, or vice versa), additions of weighted-vest running? All these questions are necessary to help properly evaluate the subjective history of athletes at risk for stress fracture. It is not uncommon to encounter these patients leading up to or just before a significant competitive event, as the increase in training load is common during this preparation [13]. In running, the pain will present with weight-bearing onset during the initial part of the run, will progressively worsen with time/miles, and does not abate until the run stops. Unfortunately, some athletes' pain tolerances and thresholds exceed the pain experienced, which

may lead to stress fracture formation, propagation, and potential completion [14]. In the case of the femoral neck, if a stress fracture is not appropriately managed and the athletes continue to load it, the consequences could be devastating – fracture completion and displacement, with resultant urgent surgical treatment necessary, and consequent non-union (5–25%) and avascular necrosis (10–40%) risk [15].

Physical Examination

A systematic examination should be performed in any patient suspected to have a hip or femur stress fracture. The physical examination begins with gait observation walking into the clinic or the examination room (with or without gait aids, such as crutches), then inspection, palpation, motion (hip, knee, spine), strength, and special testing. In the case of stress fracture, gait may be normal or antalgic (shortened stance phase). Inspection is frequently normal, without any cutaneous abnormalities, such as atrophy, skin lesions, or deformities. Palpation entails a detailed evaluation of all bony and soft tissue structures around the hip, thigh, pelvis, and lumbosacral spine. An obvious tenet to this evaluation mandates absolute consideration for modesty and genitourinary and gastrointestinal systems. Pre-examination discussion with the patient and their family (e.g., parents) about the latter ensures appropriateness and reduces risk of misperceived examination techniques, especially palpation. The hip joint, including the femoral neck, and femoral shaft are deep structures basically impossible to palpate. Specific areas to be palpated and documented for tenderness include the greater trochanteric facets, abductor tendons, iliac crest (from anterior superior to posterior superior iliac spines), inguinal canal (including inguinal hernia evaluation), pubic symphysis, pubic bone, rectus abdominis, sacroiliac (SI) joint, spinous processes (including asymmetry in coronal and sagittal plane alignment – scoliosis, kyphosis, and lordosis; rib humps with forward bend), deep gluteal space, ischiofemoral space (lateral to ischium), sciatic nerve (plus Tinel evaluation), proximal hamstring, ischial tuberosity, adductor longus, quadriceps muscle, iliotibial band (from hip to tibia Gerdy's tubercle), quadriceps tendon, patella, patellar tendon, peripatellar retinacula, and knee joint for effusion.

Hip (flexion, extension, abduction, adduction, internal and external rotation) and knee (flexion, extension) motion should be measured and compared with the uninjured side. Strength (MRC classification, x/5) of the paraspinal muscles and all lower extremity muscles is measured. Special testing for stress fracture evaluation should avoid "hop tests" (to avoid potential fracture displacement). Other tests include log roll, axial load (while supine), axial distraction, external rotation recoil, dial test, impingement testing (anterior via FADIR [flexion, adduction, internal rotation], subspine via straight sagittal plane maximal flexion, lateral via straight coronal plane maximal abduction, and posterior via external rotation [pain for impingement, apprehension for instability], and FABER [flexion, abduction, external rotation] distance to table asymmetry [>4 cm for FAI] versus SI joint pain), iliopsoas evaluation (Ludloff, Stinchfield, iliopsoas test, iliopsoas snap [audible "pop"]), iliotibial band snap (visible "pop"), Ober test, long- and short-lever adductor squeeze, resisted sit-up/crunch, and Valsalva examination (hernia, sports hernia/core muscle injury). It cannot be emphasized enough that if sufficient concern exists for stress fracture and/or displacement of a known stress fracture, then vigorous examination techniques should be avoided.

Imaging: Plain Radiographs

Plain radiographs need to be obtained at initial evaluation for any patient with concern for hip or femur stress fracture. Radiographic analysis should include high-quality imaging with detailed scrutiny of the femoral neck for direct (fracture line, with or without displacement) and indirect (callus, cortical thickening, asymmetric cortical sclerosis, cortical thickening, periosteal thickening) signs of fracture and/or healing (Fig. 16.2). Fracture non-union may be observed via smooth sclerotic edges around a fracture line. For the hip, an anteroposterior (AP) pelvis and at least one

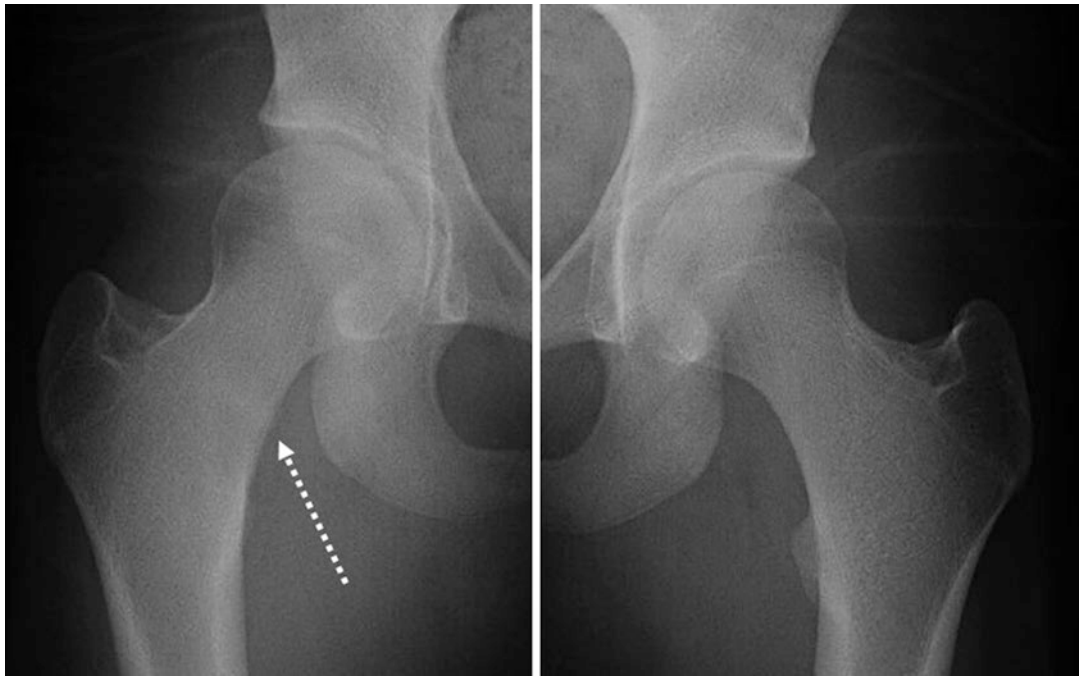


Fig. 16.2 Zoomed-in views extracted from a single anteroposterior (AP) pelvis weight-bearing radiograph showing a 16-year-old female cross-country runner with 6 weeks of significant right groin pain, worse with running, better with rest. A subtle sclerotic line along the inferior femoral neck (white arrow; compression-side) is observed. When taken in the context of the left hip, a difference can be seen. If only a single AP hip view of the right hip would have been obtained, then this subtle finding could have been overlooked and missed

lateral hip view (Dunn 45, Dunn 90, false profile, frog-leg lateral, cross-table lateral, Meyer lateral, Lauenstein lateral) are required. For the femur, an AP and lateral femur are required. In addition to fracture evaluation, plain radiographic analysis should include evaluation for arthritis, dysplasia, morphology associated with FAI syndrome (cam, pincer, prominent anterior inferior iliac spine [AIIS]), avascular necrosis, slipped capital femoral epiphysis, Legg–Calve–Perthes disease, synovial chondromatosis/osteochondromatosis, and any extra-skeletal calcification/ossification.

To appropriately characterize femoral neck stress fractures, multiple classification systems exist. The most reliable and valid system is the Kaeding–Miller system with five grades: grade 1 is a painless, asymptomatic stress response visible on imaging; grade 2 is a symptomatic stress response, without a fracture line; grade 3 is symptomatic, with a non-displaced fracture line; grade 4 is symptomatic, with a displaced fracture line; and grade 5 is a non-union [4]. This system applies to stress fractures of the femoral shaft as well. The Devas classification system dichotomizes femoral neck fractures into either tension or compression sides [16]. The Fullerton-Snowdy classification system has three types: nondisplaced tension side, nondisplaced compression side, and displaced side [17]. The Blickenstaff-Morris classification system has three types: type 1, callus without fracture; type 2, nondisplaced fracture line; and type 3, displaced fracture [18].

Imaging: MRI

If plain radiographs reveal no sign of osseous stress injury, but concern exists for stress fracture, then advanced imaging is indicated. MRI sensitivity and specificity for stress fracture approach 100% [19]. While triple-phase bone scintigraphy has excellent sensitivity, it does not have accuracy in anatomic localization like MRI, and also necessitates ionizing radiation,

which is why MRI is the gold-standard advanced imaging modality [20]. Non-contrast MRI of the hip and/or pelvis with a 1.5-Tesla or 3.0-Tesla magnet strength, without arthrogram, is appropriate for concern of proximal femoral stress fracture (Fig. 16.3). The advantage of pelvis imaging is the ability to evaluate for symmetry in any subtle amounts of edema in the proximal femur and pelvis (pubis, pubic rami, ischia, iliac wings, iliac crests, sacrum). If the fracture concern extends distal to the lesser trochanter, non-contrast MRI of the thigh with a 1.5-Tesla minimum magnet strength is indicated (Fig. 16.4).

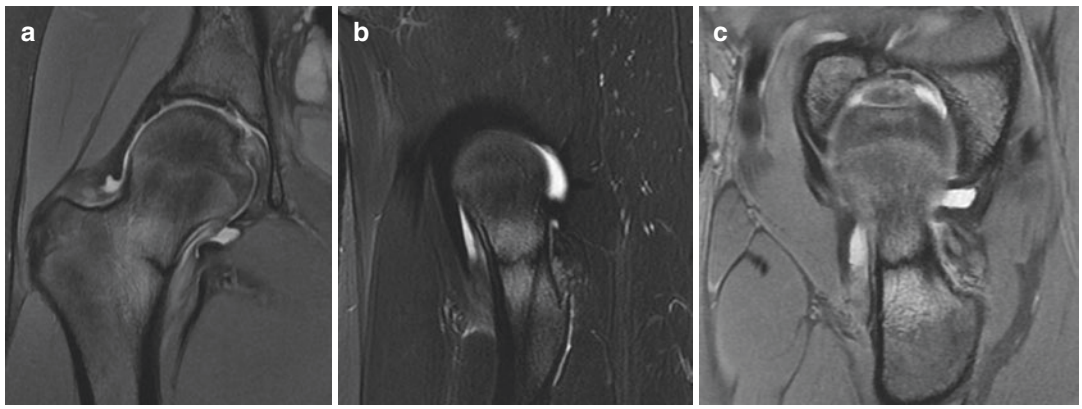


Fig. 16.3 Magnetic resonance imaging of the same patient as in Fig. 16.2; fluid-sensitive fat-suppressed T2-weighted coronal (**a**), sagittal (**b**), and axial oblique (**c**) series are shown, illustrating a fracture line, without displacement

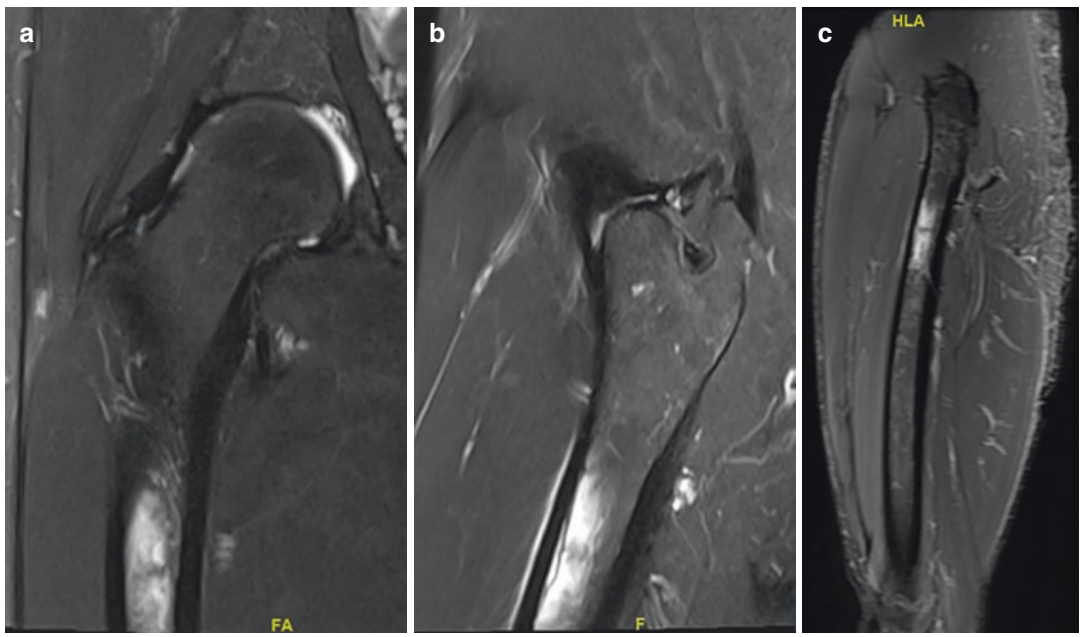


Fig. 16.4 Fluid-sensitive T2-weighted fat-suppressed coronal (**a**) and sagittal (**b**) right hip MRI without contrast of a 46-year-old multiple time sub-3 hour female Boston Marathon runner with no family or previous personal history of low bone mineral density or osteoporosis/osteopenia demonstrating a large amount of proximal femoral shaft edema with a small fracture line; (**c**) illustrates the importance of obtaining a thigh MRI if concern for stress fracture distal to the lesser trochanter exists, as the significant amount of edema extends distal to the most distal location for the hip series, but is fully visualized on the thigh imaging

In addition to osseous evaluation, MRI has the ability to evaluate intra- and extra-articular soft tissue structures around the hip – labrum, articular cartilage, hip capsule, ligamentum teres, joint synovial fluid (e.g., effusion, loose bodies), all musculotendinous units crossing the hip joint and thigh, and nerve (e.g., sciatic, femoral) and vascular structures. For both the hip and the thigh, axial, sagittal, and coronal at a minimum are necessary. Axial oblique, sagittal oblique, and radial series add additional information regarding the labrum and cam morphology. Fluid-sensitive techniques (e.g., T2-weighting, short-tau inversion recovery [STIR] with fat suppression) are the best to observe the spectrum of bone stress injuries.

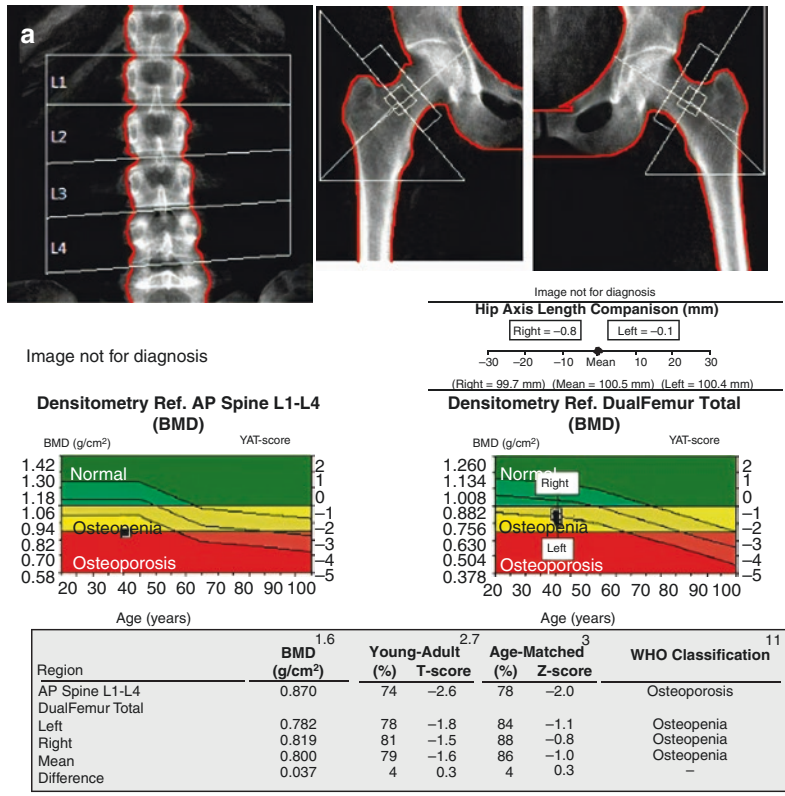
Management: Non-surgical

Non-surgical treatment of femoral neck fractures is indicated in incomplete compression-side stress fractures. This includes Kaeding–Miller grades 1, 2, and most 3 (depending on the length of the fracture line – if less than 50% of compression-side width of neck, non-surgical treatment trial first; if 50% or greater (moving across to tension-side), surgical treatment is indicated. If the stress fracture is on the tension side (grades 1–5), surgical treatment is indicated. Any displaced femoral neck fracture indicates urgent surgical reduction and fixation. Non-displaced incomplete (<50% width) femoral shaft stress fractures indicate non-surgical treatment. Displaced and incomplete (>50% width) femoral shaft stress fractures should indicate surgical treatment. For both femoral neck and femoral shaft stress fractures, non-surgical treatment is a minimum of 6 weeks, including protected weight-bearing (toe-touch, no more than 5 pounds) using crutches or walker. During this period of protection, a full stress fracture medical evaluation is needed. This includes serologic workup, including comprehensive metabolic panel (especially calcium, magnesium, phosphorus), albumin, alkaline phosphatase, vitamin D, and endocrine and sex hormones (estrogen, progesterone, GnRH, FSH, LH, thyroid, parathyroid). In addition, DEXA (dual-energy X-ray absorptiometry) is valuable in the management of bone stress injuries, regardless of non-surgical or surgical treatment, as its direct measurement of bone mineral density permits evaluation of osteoporosis and osteopenia (Fig. 16.5a). The trabecular bone score (TBS) is extracted from the DEXA imaging and helps evaluate bone microarchitecture and potential fracture risk (Fig. 16.5b). While protecting weight-bearing, core strengthening, non-weight-bearing hip motion, and non-weight-bearing hip and pelvis strengthening may continue.

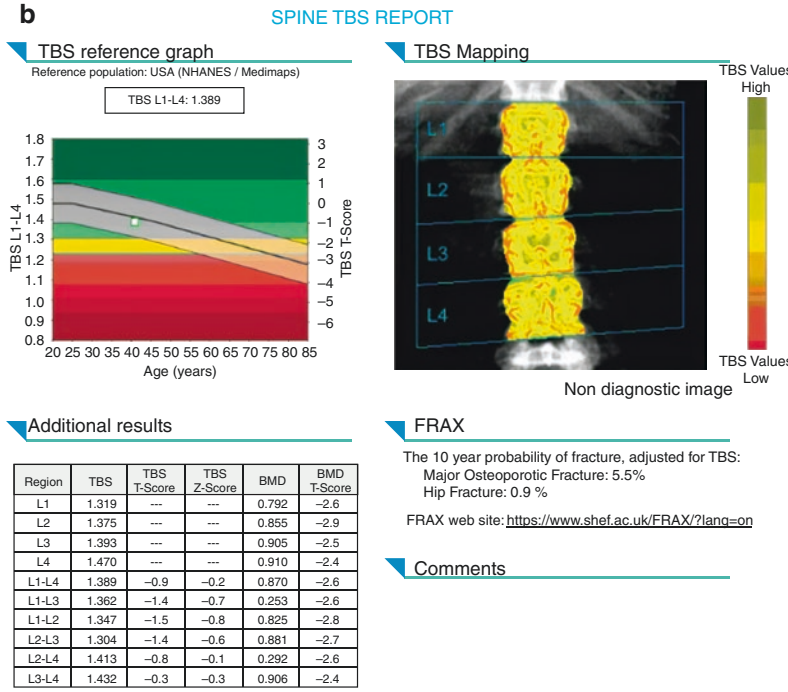
After a 6 week trial of non-surgical treatment with protected weight-bearing, repeat imaging is indicated. As the fracture heals, the fracture line will become more sclerotic, the periosteum will thicken, and the line may ultimately disappear. If the fracture does not heal, non-union is evident with a persistent fracture line with sclerotic fracture ends. If after 6 weeks there is no evidence of fracture healing, then MRI is indicated. If the fracture is persistent without significant change, or worse, then surgery is indicated. Fracture displacement at any time during non-surgical treatment is an indication for surgery – anatomic reduction and fixation.

Management: Surgical

Surgical treatment is indicated for femoral neck fractures in the situation of (1) any displacement, (2) fracture line >50% of the width of the neck, (3) tension-side fracture, and (4) compression-side fracture that failed a minimum 6 week trial of compliant non-surgical treatment. The gold-standard treatment of choice for non-displaced fractures is percutaneous screw fixation using 6.5-mm or 7.3-mm partially threaded cannulated screws. The gold-standard treatment of choice for displaced fractures is anatomic reduction and internal fixation. If anatomic reduction cannot be achieved with closed methods, then open reduction via anterior Smith-Petersen approach is indicated. In order to reduce the risk of avascular necrosis, reduction and fixation should be performed as soon as possible. For femoral shaft



Region	BMD (g/cm²) 1.6	Young-Adult (%) 2.7	Young-Adult T-score	Age-Matched (%) 3	Age-Matched Z-score	WHO Classification 11
AP Spine L1-L4	0.870	74	−2.6	78	−2.0	Osteoporosis
DualFemur Total						
Left	0.782	78	−1.8	84	−1.1	Osteopenia
Right	0.819	81	−1.5	88	−0.8	Osteopenia
Mean	0.800	79	−1.6	86	−1.0	Osteopenia
Difference	0.037	4	0.3	4	0.3	–



Additional results

Region	TBS	TBS T-Score	TBS Z-Score	BMD	BMD T-Score
L1	1.319	---	---	0.792	−2.6
L2	1.375	---	---	0.855	−2.9
L3	1.393	---	---	0.905	−2.5
L4	1.470	---	---	0.910	−2.4
L1-L4	1.389	−0.9	−0.2	0.870	−2.6
L1-L3	1.362	−1.4	−0.7	0.253	−2.6
L1-L2	1.347	−1.5	−0.8	0.825	−2.8
L2-L3	1.304	−1.4	−0.6	0.881	−2.7
L2-L4	1.413	−0.8	−0.1	0.292	−2.6
L3-L4	1.432	−0.3	−0.3	0.906	−2.4

FRAX

The 10 year probability of fracture, adjusted for TBS:
Major Osteoporotic Fracture: 5.5%
Hip Fracture: 0.9 %

FRAX web site: https://www.shef.ac.uk/FRAX/?lang=en

Comments

Fig. 16.5 (**a**) Dual-energy X-ray absorptiometry (DEXA) imaging and sample report from the lumbar spine (L1–L4; black dot illustrates osteoporosis, BMD 0.870 g/cm^2, T-score -2.6, Z-score -2.0) and bilateral proximal femurs (black dots illustrate osteopenia, BMD 0.800 g/cm^2, T-score -1.6, Z-score -1.0). BMD (bone mineral density). (**b**) Spine TBS (trabecular bone score) imaging and sample report from the same patient in Fig. 16.4. The TBS is related to bone micro-architecture and fracture risk. This information is complementary to the information obtained from DEXA

stress fractures, indications for surgery include (1) displacement, (2) incomplete, but greater than 50% of the width of the femur, and (3) incomplete, less than 50% width of femur, but failed non-surgical treatment trial. Femoral shaft surgical treatment of choice is intramedullary nail fixation.

Femoral neck fracture surgery requires general anesthesia, radiolucent bed, and gentle limb positioning and support. The latter is absolutely critical, as displacement can occur if unintentional excessive bending or rotational torque is applied to the fracture. Positioning should ensure complete visualization of the hip on AP and multiple different lateral views. A single large C-arm fluoroscopic unit (low-dose, pulse for low-dose protocol) is sufficient for closed or open reduction and internal fixation. However, two large C-arm units can be used with one positioned as an AP and the other as a lateral. Positioning for this technique requires more setup time. However, it may offset via reduced C-arm moving time during screw placement. An inverted triangle configuration is standard of care. The inferior (AP view) central (lateral views) screw is placed first, followed by superior (AP view) anterior (lateral views), and finally superior (AP view) posterior (lateral views) screws. The authors prefer to place all three guidepins first, then place the screws over the pins, rather than go pin-screw, pin-screw, pin-screw. Washers are not required, but can be advantageous if greater trochanter/lateral femur cortical integrity is a concern (if entry point repositioning is required, then the screw head may plunge through the cortex). During the entirety of pin and screw placement, the surgeon must ensure that no violation of the joint occurs. All screws should enter the femur above the lesser trochanter level (on AP view) to avoid a stress riser effect. Titanium and stainless steel screws are both viable options. However, titanium permits better postoperative MRI analysis than stainless steel if MRI is ever needed, with less artifact.

If open reduction is required, then a Smith-Petersen approach is optimal. Capsular exposure anteriorly permits full visualization of the iliofemoral ligament for "T" or "Z" capsulotomy, hemarthrosis evacuation, anatomic reduction, and then fixation. During the procedure, the medial femoral circumflex artery's terminal superior retinacular/lateral ascending vessels in the lateral synovial fold must be respected and not violated as they are the primary vascular supply to the femoral head.

Femoral shaft fracture surgery requires similar positioning as to the femoral neck. The primary difference is the necessity to fully fluoroscopically visualize the femur in AP and lateral views all the way to the knee. This is required to visualize appropriate distal placement of the nail and accurate distal interlocking screw placement. Antegrade trochanteric tip entry nails are preferred, with a vertical split in the gluteus medius/minimus insertion, gentle retractor placement for guidepin, reaming, and nail placement, followed by suture repair of the gluteal entry interval. Piriformis entry antegrade nails risk the femoral head blood supply and are not preferred. Far-lateral entry nails do not spare the abductor insertion. They tend to remove the actual tendinous insertion. The tip of trochanteric entry position enters at or above the myotendinous junction. Thus, there is less risk for actual tissue removal from sequential reaming as may occur with lateral entry nails. Retrograde nails violate the knee joint and may risk posterior cruciate ligament injury and are not preferred.

Following screw fixation for nondisplaced fractures, weight-bearing as tolerated is permitted, with crutches used for comfort only. Running may commence 6–8 weeks after surgery. Following open reduction and internal fixation, 6–8 weeks of protected weight-bearing is necessary, with up to 25% interval increases per week to full weight-bearing by 10–12 weeks.

Conclusions

Athletic hip and thigh injury evaluation requires an assessment for possible stress fracture. Stress fractures of the femoral neck and shaft typically occur via overuse in endurance athletes. Stress fractures at the hip are primarily located at the femoral neck. Compression-side femoral neck

stress fractures are usually of low risk, with non-surgical treatment typically successful. Tension-side femoral neck stress fractures are usually of high risk, with surgical treatment indicated. Femoral shaft stress fractures are usually of low risk, with non-surgical treatment typically successful. Screw fixation is the gold-standard surgical treatment for femoral neck fracture. In non-displaced fractures, no reduction is required. In displaced fractures, urgent anatomic reduction is necessary. For femoral shaft fractures indicated for surgery, antegrade greater trochanteric tip entry intramedullary nail placement is the preferred surgical technique. All patients with stress fractures warrant bone mineral density and RED-S (relative energy deficiency in sports) evaluation to optimize not only osseous health and the musculoskeletal system but also the health of all body systems in the entire individual.

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Stress Fractures of the Tibia

17

Joshua D. Harris, Kevin E. Varner, and Timothy L. Miller

Introduction

Stress fractures are common overuse injuries. Repetitive high intensity or extended duration of training places athletes at risk for stress fractures [1, 2]. Stress fractures are most commonly observed in the tibia (24%), tarsal navicular (18%), metatarsal (16%), fibula (16%), and femur (7%) [3]. Athletes in cross country, track and field, recreational and competitive running, triathlon, soccer, basketball, and dance are at risk for tibial stress fractures [3]. Military recruits are also at increased risk for overuse injuries, including stress fractures of the tibia [4]. History, physical examination, and imaging studies are essential to quickly and efficiently make the diagnosis of a tibial stress fracture. The most common differential diagnoses for chronic leg pain in a running athlete include medial tibial stress syndrome ("shin splints"), posterior tibial tendinopathy, tumors, exercise-induced compartment syndrome, and popliteal artery entrapment syndrome.

Once the diagnosis of a tibial stress fracture has been made, the appropriate treatment plan must be determined based on the athlete's risk factors and the likelihood of fracture progression. As for all high-risk stress fractures, a holistic approach to the injury must be employed to obtain the fastest healing and minimize the risk of recurrence. A discussion is warranted with the athlete and their family members along with his or her coach, athletic trainer, agent, and team. The conversation often must include a discussion of the timing of the injury and the willingness of the athlete to comply with activity restrictions in order to promote healing. For elite and professional athletes, extended complete rest is often impractical and unacceptable. It is paramount that Sports Medicine physician understand the probability of fracture progression and convey that to the athlete and the rest of the treatment team.

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Relevant Anatomy and Biomechanics

The tibia is the second longest long bone in the human body. It is the primary weight-bearing bone of the leg (up to 93% load transmission; 7% via the fibula) [5]. Its strong diaphysis is composed of thick cortical walls and is triangular in cross-sectional area, with proximal and distal metaphyseal and epiphyseal flared expansions. From the distal medial tibia projects the medial malleolus, a rigid bony stabilizer of the ankle mortise and the origin for the stout deltoid ligamentous complex. The soft tissue coverage of the tissue is asymmetric, with abundant muscular coverage laterally and posteriorly. However, the anteromedial aspects are superficial with only skin, minimal subcutaneous tissue, and periosteum coverage present.

The biomechanics of the tibia are primarily dependent upon the knee and ankle articulations and bridging musculotendinous units [6]. Distally, the ankle mortise is highly congruous with intimate articulation between the tibia, fibula, and talus in all positions of ankle dorsiflexion and plantarflexion. Ankle dorsiflexion is coupled with talar external rotation and fibular posterolateral translation and external rotation. Ankle plantarflexion is coupled with internal talar rotation. Even small amounts of ankle mortise disruption can lead to dramatic increases in articular contact pressures (1 millimeter of lateral talar displacement reduces the articular contact area by 42%, with subsequent increase in contact pressure and supraphysiologic articular cartilage load and wear) [7]. In addition to articular biomechanics, the powerful gastrocnemius-soleus complex, with explosive ankle plantarflexion seen in jumping sports, places a large tensile stress on the anterior tibia and a subsequent anterior bend and convexity. This, coupled with relative anterior tibial hypovascularity, increases the risk of poor healing if a stress fracture occurs at this site. Similarly, a pronated foot in endurance athletes, including runners, with weak subtalar inversion may permit excessive proximal rotational torque at the tibia, with a consequent increase in risk of tibia stress injury and medial tibial stress syndrome.

Medial tibial stress syndrome (MTSS), most often referred to a "shin splints," is an overuse injury whose symptoms commonly overlap with a stress fracture over the posterior medial distal metaphysis. On physical exam and on MRI or bone scan, this injury is more diffusely distributed along the medial tibial shaft than the point tenderness or "hot spot" of a stress fracture. Although it has been suggested that medial tibial stress syndrome may occur within a continuum of tibial stress injuries, this is debatable [8]. Simply, MTSS is periostitis of the tibia due to the tensile pull of the posterior calf musculature, with co-existent tendinopathy, periosteal remodeling, and stress reaction of the tibia [9].

Definition and Classification

Stress fractures represent a wide spectrum of bone injury and no two stress fractures behave exactly the same way. Even stress fractures at different sites within the same bone behave biomechanically individually. They are characterized by an imbalance between repetitive microtrauma and repair. The continuum of injury begins with the inability of the bone to repair microcracks that occur with overuse. As the microcracks increase in both absolute number and size, a distinct "macrocrack", or fracture, may occur and be visualized with imaging studies. Though crack initiation is a normal component of bone homeostasis, crack propagation is the beginning of structural failure within the bone. Left untreated, incomplete or nondisplaced fractures may eventually displace and progress to nonunion. This variable spectrum of stress responses in bone is best illustrated in the Kaeding–Miller classification of stress fractures (Table 17.1) [10]. This grading system and its implication on treatment strategy are described in more detail in Chap. 5 of this textbook, though it has shown increased time to return to sports by approximately 1 month for each grade of severity [2, 11].

Stress fractures may additionally be dichotomized into "low-risk" and "high-risk" fractures [12]. High-risk stress fractures are those prone to displacement, nonunion, delayed union, and

Table 17.1 The Kaeding–Miller classification system for stress fractures [10]

Grade	Pain	Radiographic findings (X-ray, MRI, CT, or bone scan)
I	–	Imaging evidence of stress injury *No* fracture line
II	+	Imaging evidence of stress injury *No* fracture line
III	+	Non-displaced fracture line
IV	+	Displaced fracture (≥2 mm)
V	+	Nonunion

refracture. High-risk fractures present a challenging situation, often requiring either surgery or a lengthy duration of non-surgical management, which, for some competitive athletes, may be potentially career-ending. Not all stress fractures of the tibia, however, are of high risk. Posteromedial tibial stress fractures, frequently seen in runners and other endurance athletes, as opposed to the tension-sided anterior cortical fracture, are on the compression side of the tibial shaft and respond more favorably to non-surgical treatment. They are therefore considered to be low-risk injuries [9].

Risk Factors

Stress fractures may occur in two distinct scenarios: abnormal stresses applied to normal bone (fatigue) and normal stresses applied to abnormal bone (insufficiency), though they are not mutually exclusive [13]. Fatigue fractures are overuse injuries. Thus, training errors, competition, nutrition, equipment, extremity biomechanics, hormonal imbalance, and bony alignment all play significant roles in the development of fatigue stress fractures at the tibia and elsewhere (Table 17.2). Female athletes are at particular risk for stress fracture if they present with symptoms of the "female athlete triad" [14] or have body mass index of 19 kg/m^2 or less [2, 11]. More recently the term relative energy deficiency in sport (REDS) has been applied to both male and female athletes to indicate inadequate caloric intake to replace the energy being used during endurance training [2, 15]. Athlete with risk factors for these conditions often exhibit

Table 17.2 Risk factors for tibial stress fractures in runners

Rapid increase in training intensity
Rapid increase in training mileage
Leg-length discrepancy
Increased hip adduction
Subtalar eversion and foot pronation
Pes cavus
Female athlete triad
Hard running surfaces
Old, worn running shoes
Vitamin D insufficiency
BMI ≤ 19 kg/m^2

Table 17.3 Clinical history pearls for evaluation of tibial stress fracture

History of prior stress fracture
Recent increase in training intensity, duration, or equipment
Focal pain localization with activity, including weight-bearing, running, jumping
Pain progression ranging from after activity, to with activity, to activities of daily living, to occurring at rest
Female athlete triad/relative energy deficiency syndrome

characteristics of both fatigue and insufficiency fracture, with amenorrhea or oligomenorrhea, overall energy deficiency imbalance, and low bone mineral density [14, 16]. Further nutritional risk factors include inadequate or insufficient serum vitamin D levels. This has been demonstrated in multiple studies internationally to increase risk of stress fractures, particularly in military personnel and athletes training at high northern latitudes [17, 18].

History and Presentation

The patient's history is a key component of establishing a correct diagnosis in the patient assessment for a suspected bony stress injury (Table 17.3). It requires not only asking specific questions pertinent to a chief complaint but also actively pursuing questions related to nutritional, psychological, biomechanical, and hormonal risk factors that may predispose the athlete to a stress fracture. Characterization of the principal symptoms attrib-

utable to the chief complaint (usually worsening leg pain) should describe seven entities [19]: location, quality, severity, timing (onset, duration, frequency), setting, exacerbating and relieving factors, and associated manifestations. History should also elicit the time of year and time of training program during which time symptoms began to occur and if the symptoms have progressed to the point of affecting activities of daily living. Pertinent positive and negative findings from the past medical history, family history, social history, review of systems, tobacco use, and medication list are all also very relevant. Patient demographics and epidemiology must also not be overlooked, especially age and gender, as these factors do play a significant role in evaluating overuse injuries.

Pain Localization

Pain location is an important component in clinical diagnosis. In the diagnosis of tibial stress fracture, it is vital to localize exactly where the patient feels the most severe pain, either at rest or with activity. If necessary, it may be beneficial to have the patient run or jump for 5–10 minutes to reproduce the symptoms in order to accurately delineate the location of pain. This is mostly useful for defining a stress fracture versus MTSS. In diaphyseal tibial stress fractures, the pain is frequently insidious in onset, over a prodromal 2–4 week course, and often coincides with a change in training volume and/or intensity. Initially, the pain is after activity, then progresses to occur during activity, especially weight-bearing during running or jumping on the affected. Eventually symptoms progress to affect activities of daily living and/or at rest. Anterior or anterolateral, tension-sided, tibial stress fractures often present in jumpers, ballet performer, or dancers at the central one-third of the diaphysis and the athlete typically points with one finger stating "this is where it hurts." Compression-sided fractures often present in runners at the posteromedial tibia (similar to medial tibial stress syndrome). However, these fractures may present proximally, distally, or in the mid-shaft. Patients may point to a focal area of pain or may more broadly or vaguely state that "it hurts around this area." Patients with medial tibial stress syndrome similarly complain of vague diffuse exertional pain along the posteromedial border of the tibia at the mid-distal portion of the shaft typically 6–12 cm proximal to the medial malleolus.

Patients with stress fractures in other locations of the tibia often complain of focal tenderness to touch and weight-bearing pain. In patients with proximal tibia stress fractures, activity-related weight-bearing pain is common at the location of the fracture which is most often the medial proximal diaphysis and commonly associated with an insufficiency fracture.

Exacerbating and Relieving Factors

Circumstances that aggravate a painful sensation often clue the clinician in to the diagnosis. Factors that relieve pain include rest, medications, immobilization, and procedures. In patients with a tibial stress fracture, exacerbating factors include running (e.g., compression-side posteromedial tibia), jumping or dancing (e.g., tension-side anterior tibia due to force of posterior calf musculature), and weight-bearing (e.g., ambulation). Relieving factors usually include rest, reduction in weight-bearing and loading of the leg, anti-inflammatory medications, and ice cryotherapy.

A helpful history pearl that may distinguish medial tibial stress syndrome and tibial stress fracture is the pain response to a training session. Patients with stress fractures tend to have worsening of pain in the location of the fracture with a single training session as the session progresses to the point that the athlete sometimes has to stop due to pain. Athletes with medial tibial stress syndrome, in the early stages, may actually have pain at the beginning of a training session that gradually subsides during that training session.

Other Findings

The clinician must be cognizant of other possible contributing coexistent pathology that may predispose the athlete to a stress fracture. A

thorough foot exam may reveal rigid pes cavus, subtalar pronation, tarsal coalition, muscle imbalance, and weakness or stiffness. Pes planus has also been shown to predispose to medial tibial stress syndrome due to tensile forces created at the medial tibia with excessive pronation [9]. However, other studies have shown, rather than pes planus, it is the ratio of subtalar eversion to inversion strength (in favor of eversion) that is predictive of increased risk of medial tibial stress syndrome [20]. Evaluation of leg length is necessary as a significant leg-length discrepancy is associated with tibial stress fracture [21].

Physical Examination

The physical examination for the lower limb with an overuse injury should be comprehensive and systematic. This allows for consistency and reproducibility during examination of patients with not only tibial stress fractures (Table 17.4) but also all potential causes. It should, just as questioning during a proper history, be adaptive as well. Physical assessment of any limb or joint requires visual inspection, palpation, motion, strength, and special testing (e.g., vibration tuning fork [22], tuning fork with stethoscope [23], and single leg hop and fulcrum [24]). Further, in order to understand whether pathology is present in the involved tibia, the clinician must also thoroughly examine the contralateral tibia as well, with the knowledge that bilateral stress fractures may coexist without the athlete realizing it. Extensions of the physical examination of tibial stress fracture require evaluation from as far proximal as the lumbar spine and down the entire lower extremity as needed. This requires an assessment of coronal plane alignment, femoral version, tibial torsion, and pedal arch. Further, assessment of core strength, hip impingement, knee and ankle stability, and musculotendinous unit tightness (e.g., hamstring, hip adductors, iliotibial band, gastrocnemius-soleus-Achilles, plantar fascia) is warranted. Although the physical examination of the possible tibial stress fracture should focus on the presenting chief complaint, a comprehensive physical examination should also identify other abnormalities that may predispose the patient to other overuse injuries (i.e., injury prevention). These can be further assessed dynamically with the use of video running gait analysis.

Table 17.4 Physical examination pearls in the evaluation of tibial stress fractures

Focal point tenderness at the site of the fracture
Edema, palpable periosteal thickening
Positive single-leg “hop” test, although non-specific
Positive fulcrum test
Absent compartment swelling, nerve symptoms
Evaluate for possible contributing coexistent pathology (e.g., muscular tightness, contracture, or deformity)
Running gait observation and analysis

Tibia-Specific Physical Examination

The key physical examination finding to distinguish a tibial stress fracture from other causes of leg pain is primarily focal point tenderness at the location of the fracture, usually the anterior or medial tibia. As opposed to the latter, patients with medial tibial stress syndrome frequently have more diffuse, nonfocal tenderness along the posteromedial middle to distal one-third of the tibia and not the anterior tibia or the surrounding soft tissues. If the patient reports that the pain only occurs after an activity, such as running, then the clinician should consider have the athlete provoke the symptoms by running to the point of symptoms and then re-evaluating in the same clinical setting. Pain and tenderness may be localized by a tuning fork test. The tuning fork test has a sensitivity, specificity, positive predictive value, and negative predictive value of 75%, 67%, 77%, and 63%, respectively. The single-leg hop test is also frequently utilized in the evaluation of all lower extremity stress fractures, not just the tibia [25]. However, this test is non-specific, as it is also positive in up to 46% of patients with medial tibial stress syndrome [26]. Edema and palpable periosteal thickening are also observed in patients with tibial stress fractures. In patients with medial tibial stress syndrome, edema is usually absent. Fulcrum testing of the long bones including the femur and tibia may be performed by applying a

3-point bending force across the site of pain with the end of the examination table of the examiner's forearm (Fig. 17.1).

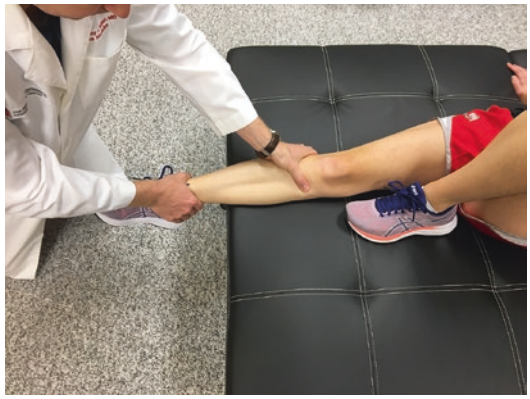

Fig. 17.1 A fulcrum test is performed on an athlete's tibia by applying a 3-point bending force across the end of the examination table

Inspection

Thorough inspection of the tibial stress fracture requires observation of the core and entire lower extremity. In addition to the patient himself/herself, shoe wear patterns should also be evaluated. One should also note prior surgical incisions and observe any deformity or asymmetry in alignment, swelling, calluses, and blisters. The clinician should inspect for any swelling, edema, ecchymosis, or erythema. Gait evaluation by observation either in clinic or outside of clinic – on a treadmill or via digital video analysis – gives a real-time evaluation of biomechanical factors that may predispose to stress injuries of the tibia (Fig. 17.2a–c).

Palpation

This is a key component in the tibial stress fracture evaluation. All osseous and soft tissue struc-

Fig. 17.2 (**a**) Posterior still-frame photograph from a treadmill running gait analysis of an elite-level distance runner demonstrating 7 degrees of pelvic tilt. (**b**) Lateral still-frame photograph from the same runner demonstrating stride length with neutral foot-strike. (**c**) Still-frame photograph of gait analysis performed on an elite-level long jumper via aquatic treadmill as the athlete recovered from a tibial stress fracture

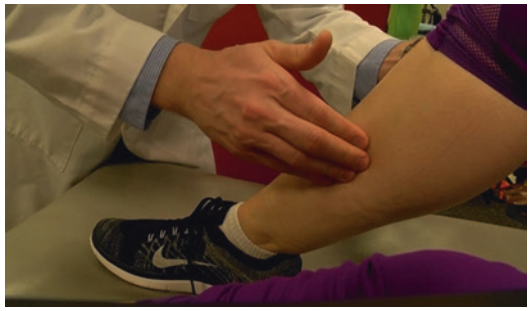

Fig. 17.3 The posteromedial distal tibial diaphysis is palpated for tenderness indicative of a stress fracture or medial tibial stress syndrome

tures warrant palpation (Fig. 17.3). In the leg, this includes, among others, the subcutaneous tibia, the knee joint, tibial tubercle, fibular head, medial and lateral malleoli, the popliteal fossa, calf musculature and Achilles tendon, and plantar fascia. The patient with a tibial stress fracture may exhibit focal tenderness with percussion testing, a positive tuning fork and single-leg hop test, and edema. Patients with medial malleolar stress fractures have tenderness over the medial malleolus and pain with forced passive ankle dorsiflexion and rotation or inversion.

Motion, Strength, and Special Testing

Assessment of motion and strength in the evaluation of tibial stress fractures mandates analysis of both limbs for comparison. It is pertinent to examine the "normal" uninvolved limb before examination of the involved limb. It is important to assess for tightness or contracture in certain muscle groups especially the iliopsoas, iliotibial band, common adductors, hamstring complex, gastrocnemius-soleus-Achilles complex, and plantar fascia. The Thomas test may be utilized to assess for hip flexor tightness [27]. The Ober test may elicit iliotibial band tightness [28]. The Silfverskiöld test may be used to determine gastrocnemius tightness (improved ankle dorsiflexion while the knee is flexed) versus Achilles tightness (no difference in ankle dorsiflexion with knee flexion or extension) [29]. The clinician must also rule out chronic exertional compartment syndrome in patients with exertional leg pain and overuse injury. Progressive leg pain, swelling, clumsy foot, and numbness or tingling with activity warrants an evaluation with post-exercise compartment pressure testing with a slit catheter. Nerve entrapment is infrequent in the athlete's lower extremity, but must be ruled out in patients with neurological symptoms such as numbness, tingling, "pins and needles", or burning pain. Possible affected nerves include the saphenous, common peroneal, deep peroneal, superficial peroneal, and tibial. Reproduction of the nerve symptoms with compression and a positive Tinel's sign are suggestive of nerve entrapment syndrome.

Imaging

Imaging evaluation for tibial stress fractures includes plain orthogonal radiographs, non-contrast magnetic resonance images in three planes (axial, sagittal, coronal), computed tomography, and Technetium-99m-labeled methylene diphosphonate bone scan (triple phase bone scintigraphy). In the early course of a tibial stress fractures, plain X-rays are usually negative (10% sensitivity) [3]. After 3 weeks, radiographs may illustrate direct or indirect signs of fracture (periosteal or cortical thickening or sclerosis, endosteal thickening or sclerosis, a discrete fracture, or callus [30–70% sensitivity]) [30]. When the "dreaded black line" (Fig. 17.4) is present, the fracture takes on more characteristics of a nonunion and rarely responds to conservative treatment, often requiring intramedullary nail placement (Fig. 17.5) [31, 32] or stabilization with a plate and screws. Magnetic resonance imaging provides the best anatomic detail. It has demonstrated higher specificity than bone scan and either equal or superior sensitivity versus bone scintigraphy [30, 33] along with better accuracy for predicting length of time away from sport. If the diagnosis is needed earlier than the appearance of plain radiographic findings, magnetic resonance imaging may demonstrate the pres-

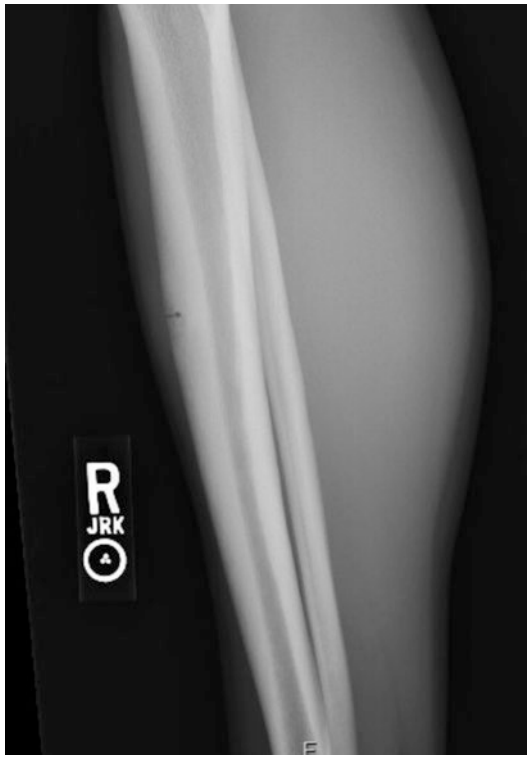

Fig. 17.4 Lateral radiograph of the tibia and fibula in a patient with an anterior cortex tibial stress fracture, demonstrating the "dreaded black line"

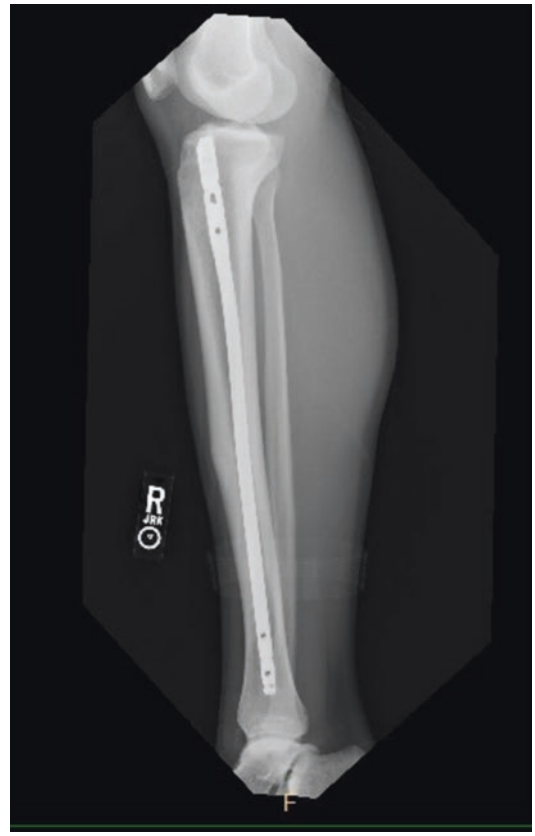

Fig. 17.5 Lateral radiograph of tibia and fibula showing complete healing of an anterior cortex tibial stress fracture following intramedullary rod fixation

ence of a stress fracture (Fig. 17.6). Sensitivity, specificity, accuracy, and positive and negative predictive values for magnetic resonance imaging in tibial stress fractures were 88%, 100%, 90%, 100%, and 62%, respectively [33]. These values for computed tomography scan were 42%, 100%, 52%, 100%, and 26%, respectively [33]. The sensitivity of bone scan was 74%. Although magnetic resonance imaging is advantageous based on its diagnostic performance and lack of ionizing radiation, it is the most expensive of the imaging modalities available to diagnose tibial stress fracture.

Treatment

Initial management of tibial stress fractures should include a period of rest, activity modification, immobilization, and reduced weight-bearing. For both low- and high-risk stress fractures, a metabolic bone disease evaluation should be performed and further laboratory workup instituted based on risk factors including a history multiple stress fractures (Table 17.5). Metabolic deficiencies, such as calcium and/or vitamin D, may be easily identified and corrected, as long as the underlying cause is identified and addressed. Currently, athletes with calcium and vitamin D deficiencies should take 1000–1500 milligrams and 1000–3000 International Units of calcium and vitamin D daily, respectively. Although parathyroid hormone derivatives have been shown to improve stress fracture repair in animal models, their clinical use has no high-level evidence in either the prevention or treatment of tibial stress fractures [34]. Electrical osseous stimulation with low-intensity pulsed ultrasound may be beneficial in the treatment of tibial stress fractures,

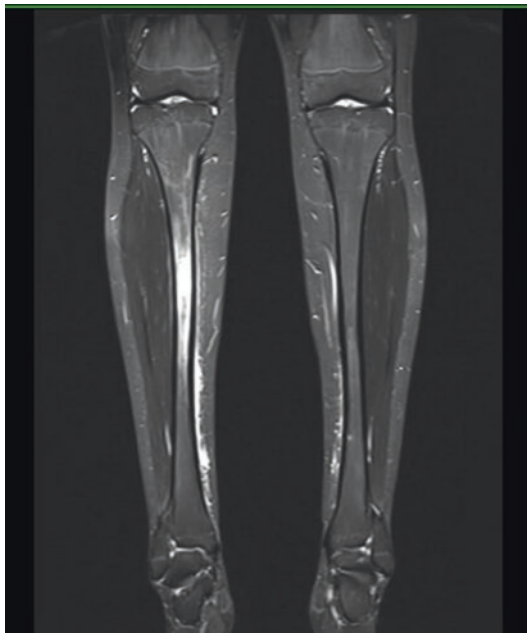

Fig. 17.6 Coronal T2-weighted bilateral leg magnetic resonance image of a 16-year-old female lacrosse play with leg pain demonstrating a tibial stress reaction (Kaeding–Miller classification, grade II) in the right middle tibial diaphysis without a discrete fracture line visible

Table 17.5 Metabolic bone disease serum laboratory evaluation

Comprehensive metabolic panel (especially calcium, phosphorus, magnesium)
Albumin
Alkaline phosphatase
Vitamin D
Endocrine and sex hormones (e.g., thyroid, parathyroid, estrogen, progesterone, GnRH, FSH, LH)

with possible reduction in time to osseous union [35]. The mechanism of action of ultrasound is via a cascade of effects with initial integrin activation, leading to upregulation of COX-2 (cyclooxygenase), VEGF (vascular endothelial growth factor), and BMP-2, 4, 6, and 7 (bone morphogenic protein) [36–38]. This may translate to increased extracellular matrix formation in soft callus, increased enchondral ossification, osteoblast differentiation, and mineralization in hard callus, and remodeling of mineralized callus [37, 39–41]. Despite the latter basic science evidence, clinical evidence for treatment of tibial stress fractures with ultrasound-based or electromagnetic field-based stimulation is limited and should be utilized on a case-by-case basis [42]. In the setting of delayed union, nonunion, or stress fractures of the tibia, low-intensity pulsed ultrasound bone stimulators may be of use. Thus, in patients with or without delayed union of a tibial stress fracture, bone stimulation should be considered. Pneumatic leg braces have demonstrated efficacy in rehabilitation of tibial stress fractures, with faster healing and return to sport than control [43–45]. However, other studies have failed to show any difference between pneumatic braces and controls [24]. There is limited evidence illustrating that extracorporeal shock-wave therapy has benefit in the treatment of recalcitrant tibial stress fractures [46].

Low-risk, posteromedial tibia stress fractures are initially managed non-operatively. Relative rest from running, jumping, and repetitive weight-bearing load may be employed during the competitive season. In the non-competitive season, cessation of the causative activity is instituted until the patient is asymptomatic with activities of daily living. If this is unsuccessful after approximately 3–4 weeks, then a trial of complete non-weight-bearing and immobilization may be used prior to return to activities. High-risk, anterior tibial cortex stress fractures are at significantly greater risk of nonunion, delayed union, and fracture completion and displacement than low-risk posteromedial tibia stress fractures [47, 48]. Thus, they are more frequently treated surgically with reamed intramedullary nailing [32]. An alternative to intramedullary nailing is open reduction and internal fixation with anterior tension band plating with or without bone grafting (Fig. 17.7) [49]. Both of these options have demonstrated high rates of healing but have recently shown a high rate of symptomatic hardware. Chronic anterior knee pain after intramedullary nailing is the greatest concern for jumping athletes [50, 51]. Medial malleolar stress fractures are prone to nonunion due to the high shear forces at the fracture site [52]. Thus, in patients with a discrete fracture line (≥Kaeding–Miller grade III) or nonunion, surgical treatment is often recommended with two 4.0-millimeter partially-

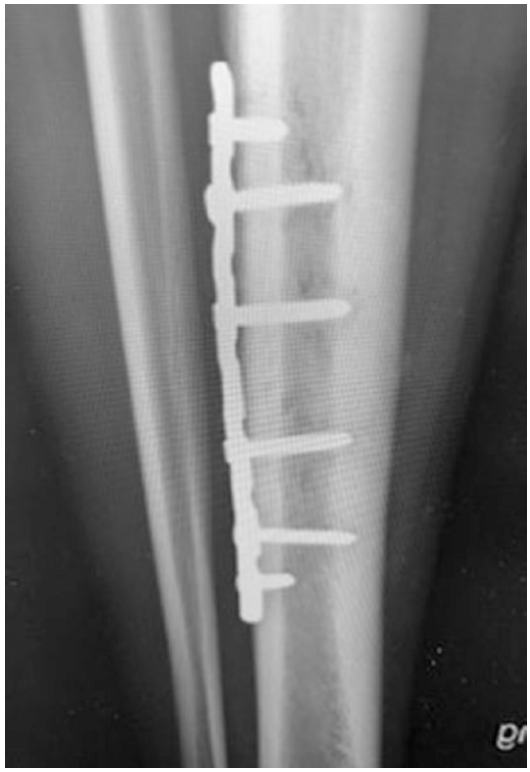

Fig. 17.7 Anteroposterior radiograph of 24-year-old female ballet performer following fixation of an anterior tibial stress fracture with an anterolateral compression plate

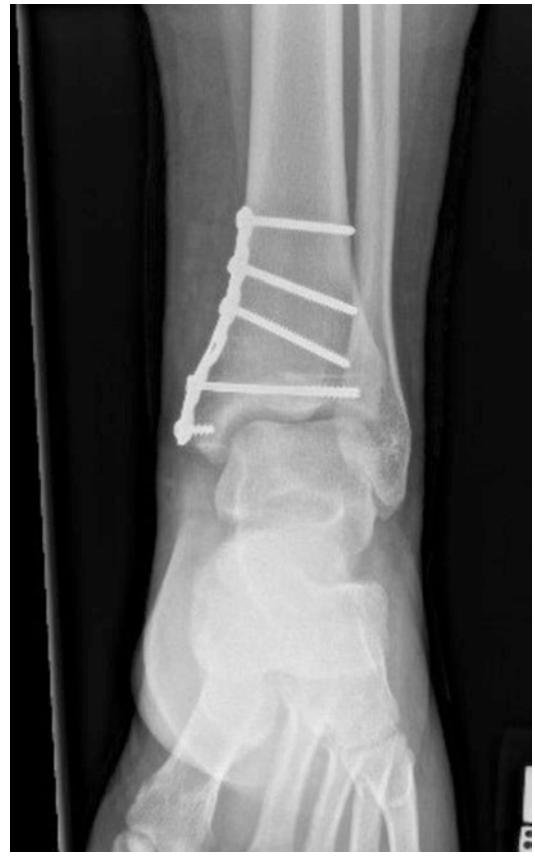

Fig. 17.8 Anteroposterior radiograph of an 18-year-old male lacrosse player following open reduction internal fixation of a vertical medial malleolar stress fracture with an antiglide plate

threaded cancellous screws or a low-profile antiglide plate and screw construct (Fig. 17.8).

Biologic Healing Augmentation

Recently, efforts have been made to use biologic treatment strategies to expedite healing and recovery from stress fractures [53]. *Osteobiologics* is a term used to describe materials that have been identified and developed to promote bone healing. These options can be divided into two groups: direct injectable modalities and indirect systemic stimulating treatments. It should be borne in mind that not every option is ideal for every bone or even each area of the affected bone. Some options may be used in combination or as an adjunct to internal fixation with hardware. Each option carries its own risks and benefits. Direct injectable modalities include concentrated bone marrow aspirate, autologous platelet-rich solutions, and injectable bone graft substitutes. Indirect systemic-stimulating treatments include vitamin D supplementation, pulsed parathyroid hormone, extracorporeal shockwave therapy, and electrical osseous stimulation. These biologic healing enhancement options may be considered for an athlete requiring quick return to sport but scientific evidence proving their efficacy is lacking [53].

Summary

Tibial stress fractures are common injuries particularly in distance runners and military recruits. The diagnosis can be made if a high index of suspicion is maintained and proper imaging studies are obtained. The Kaeding–Miller classification system for stress fractures characterizes these injuries based on the patient's symptoms as well as their position on a radiologic continuum of severity. A holistic approach to treatment that takes into account the importance of nutritional, hormonal, psychologic, and biomechanical factors is necessary for treatment success. Stress fracture management should be individualized to the patient or athlete by taking into consideration injury site (low versus high risk), grade (extent of microdamage accumulation), the individual's activity level, competitive situation, and risk tolerance. High-risk stress fractures such as the anterior tibial cortex are primarily loaded in tension, have a poor natural history, and commonly require surgical intervention. Low-risk fractures at the posteromedial tibial diaphysis are more common and loaded in compression. They have a better prognosis and are unlikely to progress to complete fracture. The recommended treatment is based upon injury severity and the biomechanical environment in which the stress fracture is located. In addition to the traditional treatment strategies of rest, immobilization, and surgical stabilization, recent modalities for enhancing healing potential have been utilized with some success from further research being required to fully confirm their efficacy.

Critical Points

- The anatomy and biomechanics of the tibia play a critical role in the development and management of tibial stress fractures.
- The differential diagnosis of patients with overuse injuries of the leg includes stress fracture, medial tibial stress syndrome, exertional compartment syndrome, popliteal artery entrapment syndrome, and multiple nerve entrapment syndromes, among others.
- Risk factors for tibial stress fracture include prior stress fracture, recent increases in training intensity and/or duration, improper training technique or equipment, and the female athlete triad, among others.
- The history and physical examination of a patient with a tibial stress fracture generally indicates focal point tenderness at the site of the fracture, unremitting pain with weight-bearing activity, and pain with single-leg hop and tuning fork testing.
- Magnetic resonance imaging is the imaging modality with the highest diagnostic utility.
- Non-operative treatment is generally successful in low-risk posteromedial tibial stress fractures.
- Surgical treatment, including reamed intramedullary nailing and anterior tension-band plating, is a successful treatment for chronic anterior mid-diaphyseal tibial fractures that have failed non-surgical treatment with high union rates and high rates of return to sport.

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Stress Fractures of the Ankle and Hindfoot

18

Christopher E. Hubbard and Martin J. O'Malley

Introduction

Stress fractures of the ankle and hindfoot are injuries that require a high index of suspicion. Bone stress injuries account for 15% of all musculoskeletal foot and ankle injuries in elite collegiate athletes [1] and 10% of injuries in recreational and competitive athletes [2].

Stress fractures of the ankle and hindfoot can be a result of intrinsic and extrinsic factors. Intrinsic factors relate to the patient's anatomy and biology. Females have been shown to have a higher incidence of stress fracture with a reported incidence of 3% versus 9.2% in males in military populations and 6.5% versus 9.7% in athletes [3]. Other intrinsic factors can include poor bone density, vascular supply, foot structure such as cavus feet or forefoot varus, tarsal coalition, hormonal imbalances, or heel cord contractures. Extrinsic factors can include training regimen, specific sport, improper footwear, and exercise terrain. Intrinsic factors are more difficult to modify than extrinsic factors.

The clinical evaluation of patients with a potential stress fracture should start with a thorough history and physical examination. Most fractures can be attributed to a recent change in training regimen or shoe wear. Athletes will typically complain of an insidious onset of pain or swelling over the past few weeks and can sometimes be difficult for the athlete to localize. The pain is usually activity related and relieved by rest. A thorough history should include recent training, diet, and any risk factors for low bone density. Physical examination starts with a weight-bearing assessment of both lower extremities for alignment and comparison of any differences in swelling. A single-limbed heel rise can help localize the anatomic area of pain. Inspection of gait, range of motion, and strength testing is performed. Tenderness to palpation is not always indicative of location of stress injury. Standing radiographs of the foot or ankle are often negative if symptoms are less than a few weeks old, with a sensitivity of 10% reported for the detection of stress injury at initial presentation [4], which increases up to 30–70% after 3 weeks [5]. Radiographic findings are dependent on the chronicity, specific bone involved, and even location within each bone. If a stress fracture occurs in cancellous bone, such as the calcaneus, initial radiographic finding is a faint trabecular sclerosis due to microcallus formation [6]. In contrast, if the cortex of a long bone is involved, the initial radiographic finding is a subtle cortical lucency followed later by a periosteal reaction and endosteal callous formation [6]. In more high-grade

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injuries, a frank cortical break will be evident. In chronic presentation, evidence of sclerosis can be seen at fracture line on radiographs. However, often these fractures are difficult to visualize radiographically and may result in a delay in diagnosis.

The decision to proceed with further imaging if radiographs are negative is dependent on the specific suspected fracture and the potential for altering the treatment plan. For instance, a suspected calcaneal stress fracture can likely be managed with a walking boot and follow-up with repeat radiographs in 2 weeks which by then will likely be positive. However, for a suspected navicular stress fracture in an athlete, further imaging would be recommended to further clarify the extent of injury to determine whether surgical intervention is indicated.

MRI is the preferred imaging technique when initial radiographs are negative. MRI findings of stress fracture include periosteal and bone marrow edema, with intracortical signal changes or intramedullary, low-signal intensity fracture line only able to be visualized relatively late in the pathogenesis of stress fractures [6]. A stress reaction represents a clinical syndrome thought to be due to early accumulation of microdamage and likely represents an early stress injury [6]. The MRI findings of stress reaction include bone marrow edema like signal without a distinct fracture line. A stress reaction becomes a stress fracture once a cortical break develops. Not all bone marrow edema, however, predisposes athletes to later stress fracture. One study of 21 asymptomatic college distance runners demonstrated a 43% incidence of bone marrow edema on MRI [7].

CT scans are useful in distinguishing a fracture line better than MRI, and in fractures of the medial malleolus and navicular, they can help determine the need for surgical intervention. Ultrasound imaging is becoming more assessable in the office setting and can identify a cortical break, but evaluation of marrow space is not possible.

Medial Malleolus

Stress fractures of the medial malleolus are relatively uncommon injuries, accounting for only 0.6–4.1% of all lower extremity fractures [8]. Shelbourne first described stress fractures of the medial malleolus with the presentation of chronic or subacute pain over the medial malleolus, tenderness to palpation along the medial ankle, and a history of running activity at the time of injury or running activities aggravating the pain [9]. Because athletes with this injury often present with nonspecific ankle pain and normal radiographs, clinicians should include this fracture in their differential diagnosis of medial ankle pain in the running or jumping athlete. Failure to diagnose in the athlete can result in fracture progression, nonunion, chronic pain, and extended delay in return to athletic activity. Radiographs may appear normal for up to 2 months after symptoms appear [10]. When there is clinical suspicion of a medial malleolar stress fracture with normal-appearing radiographs, MRI is recommended for further imaging evaluation which typically shows bone marrow edema localized to the medial malleolus. CT scan is then helpful to determine whether a fracture line is present in the setting of extensive medial malleolar edema and can help in surgical planning (Fig. 18.1). The majority of medial malleolar stress fractures are vertically oriented, and the fracture line typically extends proximately from the junction of the tibial plafond and medial malleolus [11]. Jowett and colleagues indicated that a major intrinsic risk factor in professional athletes is the presence of anteromedial distal tibial osteophytes [12]. The location of these osteophytes was shown in an anatomic study to involve the non-weight-bearing anteromedial cartilage of the distal tibia, which extends up to 3 mm proximal to the tibiotalar joint line [13]. The initiation of these osteophytes is thought to be caused by repetitive trauma to the cartilage, which then responds by the formation of scar tissue and subsequent calcification [14]. Damage to this cartilage can also be caused by supination

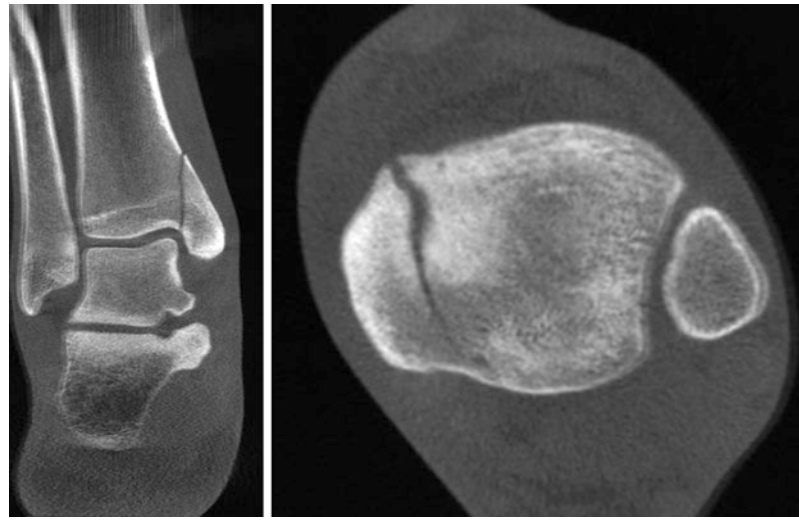

Fig. 18.1 Coronal and axial CT scans demonstrate vertical fracture line of medial malleolar stress fracture

trauma, particularly on the medial side in the case of ankle instability, and can lead to osteophyte formation [15]. These bone growths are theorized to impart rotational forces to the medial malleolus during end dorsiflexion of the ankle. Foot alignment, specifically cavus foot, has been a proposed intrinsic risk factor for medial malleolar stress fractures by transferring similar forces to the medial tibia. Medial malleolar stress fractures can be treated either conservatively with immobilization and reduction in weight-bearing activities or with surgery. However, conservative treatment has been associated with prolonged healing times and tendency toward nonunion [16]. With conservative treatment, fracture union and return to full activity can take as long as 6 months. With surgical treatment, return to play can be expected as early as 3 months following surgery [18]. Historically, the operative treatment of medial malleolar fractures has been placing two cancellous screws perpendicular to the fracture line to prevent superior displacement of the fracture. A recent study has found that an antiglide plate construct provides the stiffest initial fixation while withstanding higher load to failure for vertical medial malleolar fractures when compared to unicortical and bicortical screw fixation alone [17]. The senior author O'Malley has recently reported on the results of six professional basketball players treated with surgical fixation and iliac crest bone marrow aspirate graft, with five of the six players able to return to play by the 12th postoperative week [18] (Fig. 18.2).

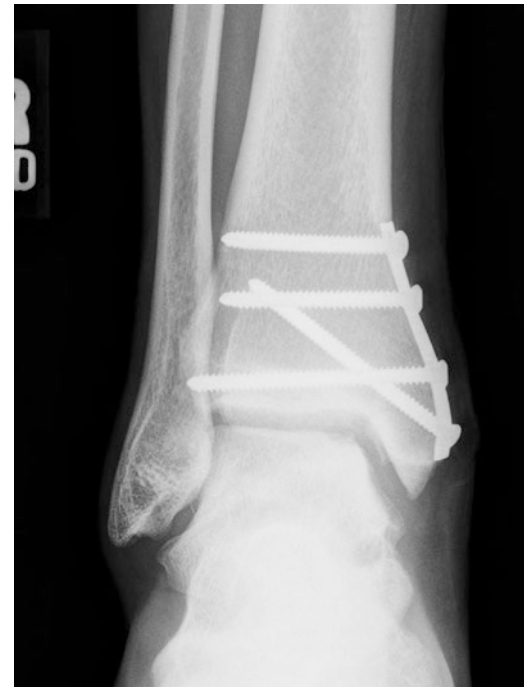

Fig. 18.2 Surgical fixation of medial malleolar fracture with antiglide plate and screws perpendicular to fracture line

Additionally, it is important to address any tibial or talar osteophytes arthroscopically or by open treatment in addition to fixation of the fracture. Calder reported on 16 professional soccer players treated with surgical fixation and arthroscopic osteophyte debridement [19]. All the 16 patients had bone osteophytes on the tibia and/or talus. Ten patients had spurs on both the tibia and the talus, while six patients had isolated tibial spurs.

Distal Tibia

The posterior medial tibial shaft is the most common location for stress fractures, most typically reported in military recruits and running athletes. Hard surfaces pose a higher risk for stress fractures [20]. Tibial strain rates in runners were 48–285% higher when running over ground compared with running on treadmills [21]. Worn running shoes may increase the risk for stress fracture because of decreased shock absorption. A distal tibial stress injury can initiate as a stress reaction where no fracture line has developed and progress to a frank cortical fracture [10]. The location along the posterior medial tibia is a result of repetitive impaction and muscular forces. Compressive repetitive forces from the gastrocnemius-soleus complex and pull of the deep plantar flexors have been thought to be mechanical factors [22]. Athletes will report a pain along the medial distal tibia that is worse with impact. Typically, patients will have tenderness along the posterior medial distal tibia to palpation. Radiographs initially are normal or can show a subtle cortical lucency followed by periosteal reaction and cortical thickening [10] (Fig. 18.3). MRI is recommended with clinical suspicion of stress fracture and is often diagnostic (Fig. 18.4). Treatment is almost always conservative as these injuries have a high likelihood of healing with rest and immobilization. One study has looked at gait retraining to reduce lower extremity loading in runners, which resulted in 20% decrease in vertical force impact peak and 30% decrease in vertical force loading rates which were maintained at 1 month follow-up [23]. This decrease in forces may reduce their risk of tibial stress fractures. MRI grading is described according to the Fredricson classification (and Kijowski modifications) and can be helpful in estimating time to return to athletic activities [24]. The shortest time to return is in a

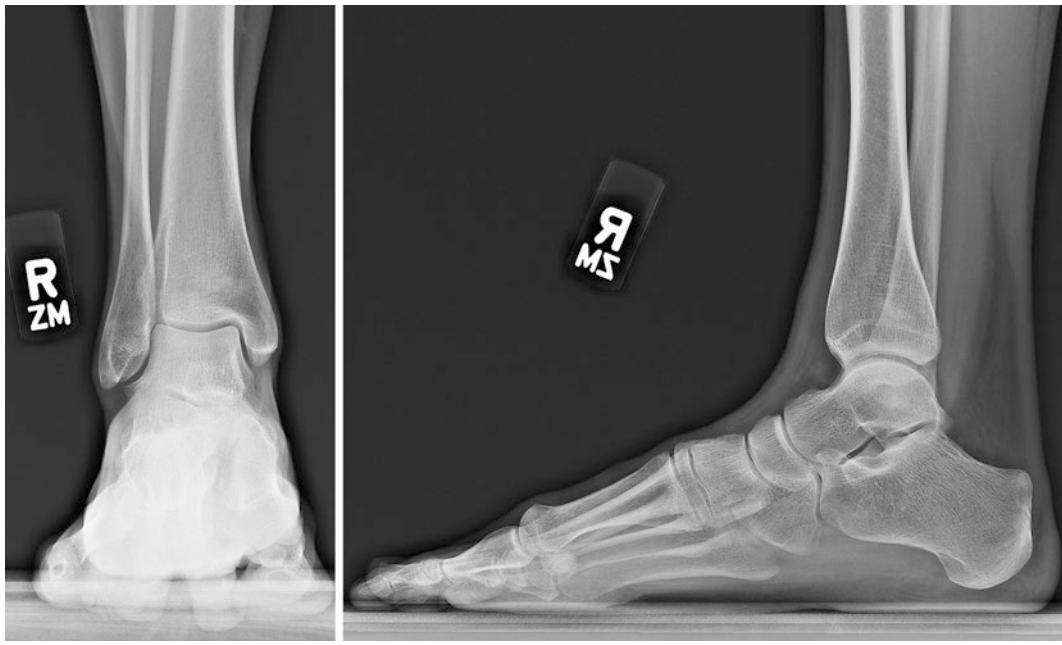

Fig. 18.3 AP and lateral ankle X-rays reveal faint intramedullary sclerosis of the distal tibial metaphysis

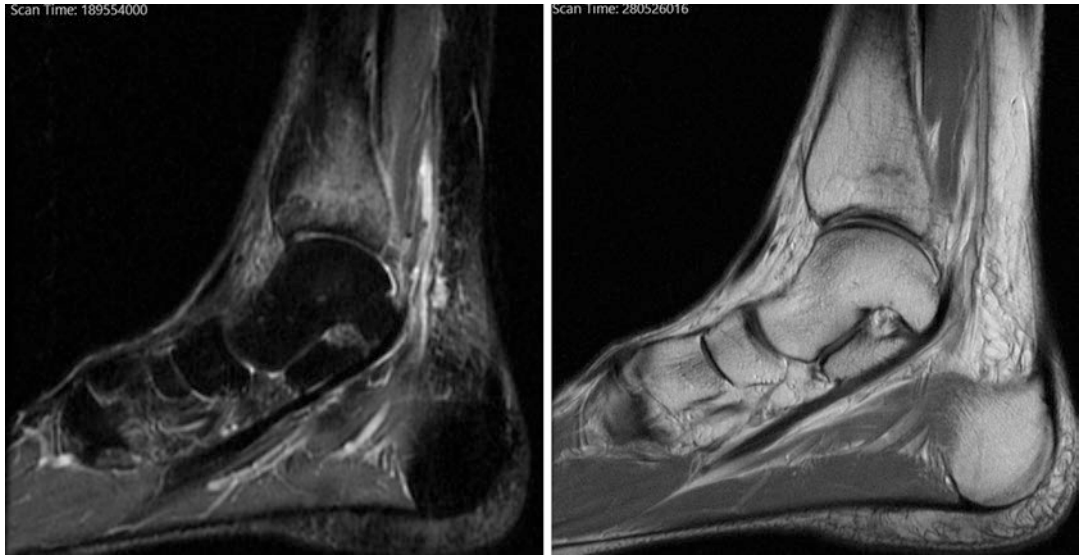

Fig. 18.4 MR demonstrating intense bone marrow edema distal tibia on T2-weighted image and trabecular fracture line on T1-weighted image

grade 1 injury, which presents on MRI as a periosteal tibial edema with normal marrow signal, and results as a mean time of return of 16 days, compared to a grade 4B injury, which demonstrates a linear cortical fracture line, and the longest time of 71 days to return to play. Distal tibial stress fractures have also been reported in adolescent athletes as a stress fracture of the distal tibial physis, for example, in a 9-year old female gymnast and dancer [25]. She was made non-weightbearing for 6 weeks and then allowed to ambulate in a walking boot. She was not able to return to activities until 6 months after presentation. The distal tibial stress fracture is different from the anterior cortical tibial stress fracture which is described elsewhere in this book.

Distal Fibula

Stress fractures of the distal fibula most commonly affect the lateral cortex of the fibula and are commonly reported in the military and athletic populations [26]. Distal fibular stress fractures have been reported in 6.6% of all stress fractures in athletes [27]. Stress fractures of the distal one-third of the fibular are more common than stress fractures of the proximal two-thirds with the majority of these fractures occurring 4–7 cm proximal to the lateral malleolus. Devas and Sweetham proposed that the mechanism of injury in these fractures was related to running on hard ground and that recurrent contraction of the plantar and long toe flexors transmitted stress through their origin on the fibula, approximating the fibula to the tibia and creating a bending moment that results in the stress injury [28]. Alternatively, it is thought that the area of the fibula just proximal to the syndesmotic ligaments is susceptible to increased forces of running and impact activities. Athletes will complain of lateral ankle pain, and the pain is most common after increasing or changing exercise regimen. The differential diagnosis includes peroneal tendon pathology and lateral ankle ligament injury. Initially radiographs are normal within the first 3–4 weeks of symptoms but then will show a periosteal reaction [10]. Intramedullary sclerosis, callous formation, or discrete fracture in a transverse pattern may be seen later (Fig. 18.5). Treatment is generally conservative with walking boot immobilization, and return to activity is usually in 6–8 weeks. MRI and further imaging often are often unnecessary unless one is concerned

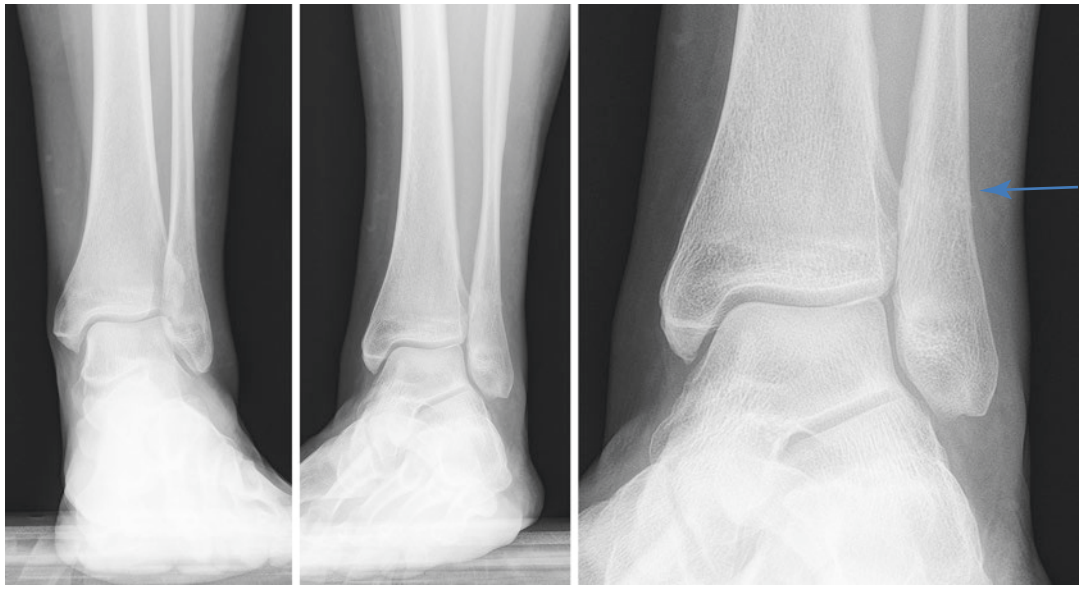

Fig. 18.5 X-rays of ankle demonstrate faint sclerosis distal fibula

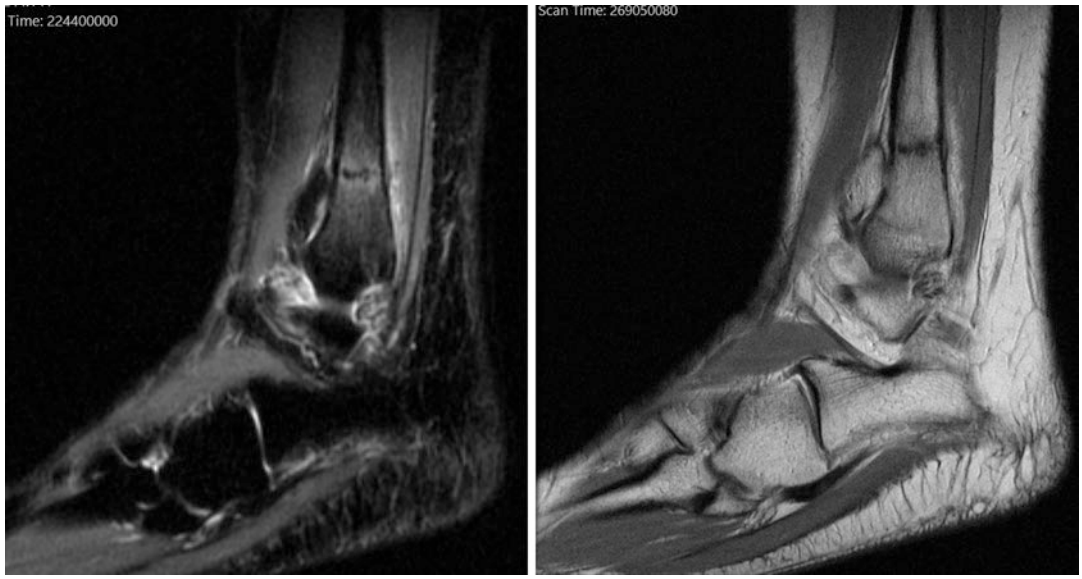

Fig. 18.6 MR T1 and T2 sagittal images demonstrate distal fibula stress fracture

about tendon or ligament pathology but can confirm diagnosis (Fig. 18.6).

A separate mechanism exists for distal fibula stress fractures in the patient or athlete with a flat foot. Patients or athletes with a posterior tibial tendon dysfunction can result in a degeneration and elongation of the posterior tibial tendon, which then results in a flatfoot deformity. The lateralization of the load axis of the lower leg then contributes to weight-bearing across the fibula [29]. The fibula typically plays a secondary role in weight-bearing with approximately 6.4–17.2% of total body weight applied to the fibula [30]. One study demonstrated a lateral shift of contact

area and peak pressure in a flatfoot model and suggested this causes a transfer of load off the talar dome [31]. The increased load in a flatfoot deformity concentrates stresses on the fibula and can lead to a stress injury. Initial treatment of these fractures is similar to the non-flatfoot fibular stress fracture, but longer-term treatment involves orthotic and shoe wear modifications and possible surgical intervention to address the posterior tibial tendon and foot deformity.

Talus

Stress injuries of the talus are relatively uncommon with mostly case reports in the literature. McGlone was the first to report on a stress fracture of the talus in 1965 [32]. The precise mechanism for stress injury to the talus is unclear. Proposed theories include the increased compression of the talar body against the navicular during pushoff [33] and excessive subtalar pronation and plantar flexion causing the lateral process of the calcaneus to impinge on the posterolateral corner of the talus [34]. One retrospective study reviewed MRI findings in military recruits with foot or ankle pain and reported that 51 recruits exhibited bone stress injuries in the talus during the study period of 96 months [34]. This yielded a person-based incidence of 4.4 per 10,000 person-years. Bilateral injuries were seen in five cases, and in 86% of the cases with talar bone edema, there was also bone marrow edema in other tarsal bones. The diagnosis can be difficult to make as the athlete typically will complain of a vague and nonspecific pain, and it is often difficult to elicit any focal tenderness on physical examination. Radiographs are usually unremarkable, and MRI typically demonstrates bone marrow edema (Fig. 18.7). Of the 56 bone stress injuries reported in military recruits, 40 occurred in the head, 15 in the body, and 5 in the posterior part of the talus [34]. The median time from the reported onset of pain to the date of diagnosis of talar stress injury on MRI was 62 days. Treatment of talar stress injuries is generally a

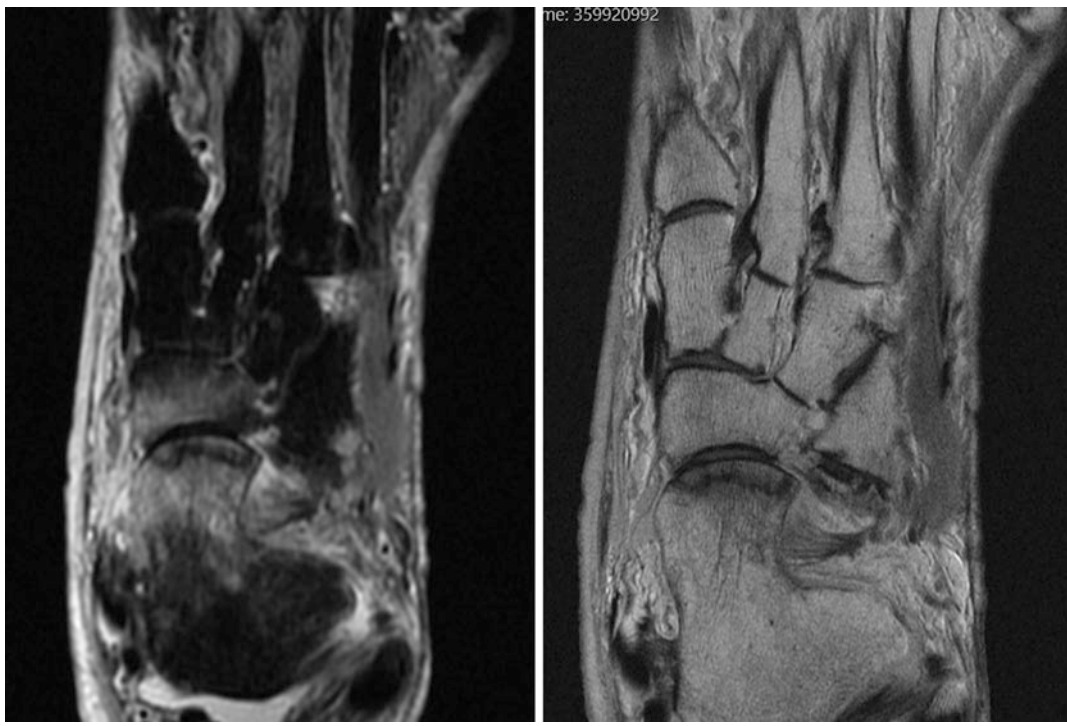

Fig. 18.7 Axial T2 and T1 MR images demonstrate stress fracture in the head of talus

walking boot and some period of non-weight-bearing if a fracture line is visible on imaging. Bone marrow edema, however, can be a non-specific finding that can be present in infections, osteonecrosis, malignancies, and bone contusion. In an MRI study of 12 random professional ballet dancers, 75% demonstrated bone marrow edema of the talus [35]. Studies describing the incidence and outcome of actual stress fractures of the talus with demonstrable fracture line on MRI are even less common. In a study following eight military recruits with a talar fracture line visible on MRI, five had mild to moderate symptoms after a mean follow-up time of 45 months [36]. All recruits were treated with reduced activity or weight-bearing restrictions based on initial symptoms and were symptom free at an average of 64 days. Five patients displayed subchondral degeneration and edema near the original area in the follow-up MRI, and in two of these patients, the degeneration was also visible on the plain radiographs.

Stress fractures have also been described of the lateral process of the talus in a runner [37] and in a competitive tennis player [38]. Both athletes had a history of greater than 1 year of vague lateral foot pain and had multiple prior diagnoses. The runner had a supinated foot which has been shown to increase pressures along the lateral talus. Stress fracture of the talus has also been reported after resection of a talocalcaneal coalition with a new onset of medial ankle pain 3 months post-surgery [39].

Calcaneus

Stress fractures of the calcaneus are quite common and reportedly comprise up to 20% of all stress fractures of the foot [40]. They are often associated with running and jumping sports and are correlated with heel strike and non-cushioned shoe wear and hard training surfaces. The pull of the Achilles tendon insertion in resisting plantar flexion of the foot is also thought to contribute. The athlete will present with posterior heel pain, most often after an increase in training activity. The examination is usually positive for tenderness with simultaneous compression of both medial and lateral aspects of the calcaneus. The differential diagnosis can include insertional Achilles tendinopathy, plantar fasciitis, and distal tarsal tunnel syndrome. Calcaneal stress fracture has been reported in injuries observed in the minimalist runners [41]. Calcaneal stress fractures can be visualized on radiographs as soon as 10 days after the onset of symptoms and appear as a sclerotic line perpendicular to the trabeculae which run in arcs perpendicular to the posterior cortex of the calcaneus [10] (Fig. 18.8). MRI will demonstrate low signal intensity line with surrounding edema (Fig. 18.9). In an MRI study of military recruits, 26% of calcaneal stress fractures occurred in the anterior region of the calcaneus, 18% in the middle, and 56% in the posterior calcaneus [42]. A total of 79% occurred in the upper region of the bone and 21% in the lower region of the calcaneus. Fifty-nine percent of the injuries were of a higher grade with a fracture line that was visible on MRI. A total of 22 of the 30 cases were associated with stress injuries of the talus, navicular, or cuboid. Treatment is conservative and involves protected weight-bearing in walking boot until symptoms diminish which generally takes 6–8 weeks. With the high association of other associated stress injuries of the foot, treatment plans can be altered.

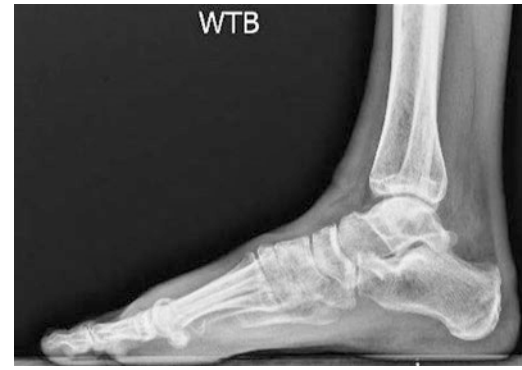

Fig. 18.8 Sagittal X-ray demonstrating sclerotic line in posterior calcaneus signifying stress fracture

Stress fractures of the anterior process of the calcaneus are rare. There have been two reports associated with a calcaneonavicular coalition, with the lack of normal motion from a coalition leading to increased pressure along the anterior process. In one case, the bar was resected and a screw placed across the calcaneal stress fracture

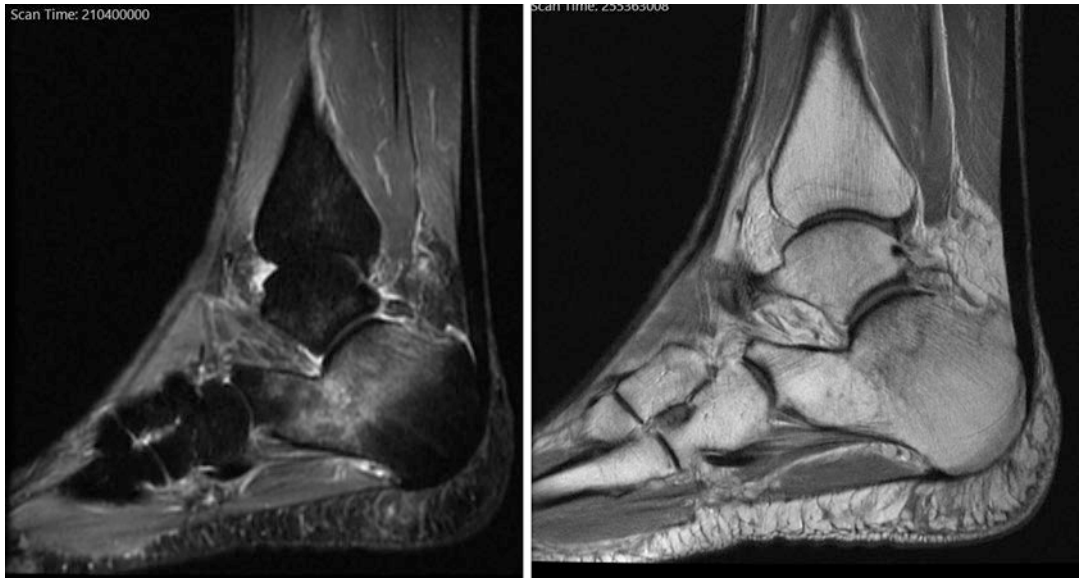

Fig. 18.9 MR demonstrating edema on T2 sagittal image and trabecular line on T1

[43]. A case report of a 14-year-old female basketball player described a stress fracture of an elongated anterior process and was subsequently treated with drilling of the fracture after failure of conservative care [44].

Navicular

Stress fractures of the navicular are high-risk fractures commonly seen in track and field [45], tennis [46], and basketball athletes. First reported in the orthopedic literature by Towne in 1970 [47], navicular stress fractures have been described to account for almost 35% of all bone stress fractures [48]. These fractures can have significant effect on the athlete's career. Anderson reported on players at the NFL combine with a history of navicular stress fracture, and overall only 28.6% of players with fracture played over 2 years in the NFL compared to 69.6% that did not have a navicular injury [49]. Talonavicular arthritis was present in 75% of athletes with injury.

The navicular is a saddle-shaped bone that articulates with the talus proximally and with the medial, middle, and lateral cuneiforms distally [48]. That poster tibial tendon inserts on the medial tuberosity, and the calcaneonavicular spring ligament inserts along the plantar beak. The foot can be divided into two parallel columns consisting of a more rigid medial column and a more flexible lateral column. The navicular is the keystone of the medial column and provides stability to the longitudinal and transverse charges of the foot [48].

The vascular supply to the navicular comprises medial tarsal branches of the dorsal pedis artery as well has branches from the superficial branch of the medial plantar artery. A recent cadaver study reported that 12% of specimens had an avascular region in the dorsal central third of the bone corresponding to the usual location of navicular stress fracture [50]. The navicular's decreased vascularity in this region has implications for healing and can result in delayed healing, high risk of nonunion, and prolonged time out of sport. In addition to the vascular properties of the navicular, specific biomechanical properties are thought to contribute to stress fracture at the central one-third. It has been theorized that during the foot strike phase of running, compression forces are generated from distal to proximal across the medial and lateral aspects of the navicular through the first and second metatarsal cuneiforms joints [51]. The forces across the first metatarsal and medial cuneiform are shared by the talar head, where those forces across the second metatarsal and middle cuneiforms are not, and result in a sheer force at the central one-third

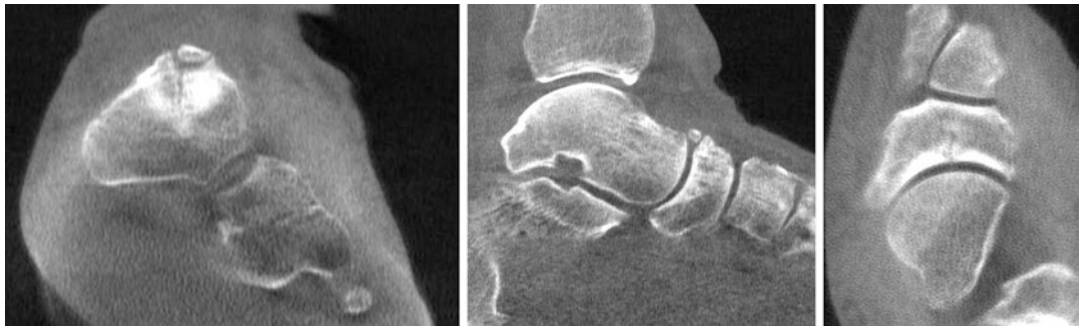

Fig. 18.10 CT images showing navicular stress fracture with os supranaviculare or previous dorsal avulsion fracture

of the navicular bone. Runners who demonstrate increased rearfoot eversion and reduced forefoot abduction during stance may be at risk of developing navicular stress fractures [52]. The presence of an os supranaviculare, an accessory ossicle at the proximal dorsal cortex of the navicular reported in 1% of individuals, has been implicated in the development of a navicular stress fracture [53]. The typical dorsal navicular depression under the os supranaviculare is localized at the area of maximal stress on the navicular and contributes to the propagation of stress fractures (Fig. 18.10). An osteochondral lesion of the tarsal navicular has also been reported with a stress fracture of navicular in high-level athletes [54].

Delay in diagnosis is common and has been reported up to 6 months on average and in a study by Saxena of up to 8.8 months [55]. Typically the athlete complains of a slow onset of vague medial and dorsal foot pain that radiates along the medial arch of the foot. The pain is worse with activity and generally relieved at rest. Running, jumping, and cutting activities exacerbate the symptoms. Runners often alter their gait to compensate for their pain, minimize their symptoms, and typically have a high threshold for pain.

On physical examination, there is no swelling of the foot, and athletes generally have a normal range of motion and strength. Tenderness to palpation of the central third of the navicular is called the "N" spot, and Torg described the tenderness to palpation in 81% of patients with navicular stress fractures [56]. A single leg heel rise or hop test often elicits pain along the midfoot.

Radiographs are often negative but can evaluate other causes of foot and ankle pain

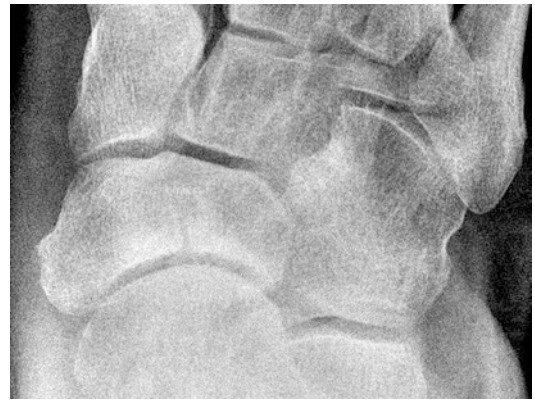

Fig. 18.11 AP X-ray image demonstrating sclerotic line in navicular indicating stress fracture

(Fig. 18.11). In a study by Saxena [57], only 2 out of the 22 patients had their fracture visible on plain X-ray. If initial radiographs are negative and there is clinical suspicion of a navicular stress fracture, then MRI is recommended. With a positive MRI for navicular stress fracture, a CT scan is indicated for further clarification of fracture line. Saxena proposed a CT classification and treatment scheme [57]. A type I fracture involves a dorsal cortical fracture of the navicular (Fig. 18.12). A type II fracture extends from the dorsal cortex into the navicular body. A type III fracture penetrates a second cortex (plantar, medial, or lateral.) They later added a type 0.5 to indicate stress reaction.

Treatment for navicular stress fractures in the athlete remains a topic of debate. Nonoperative treatment relies on immobilization and protected weight-bearing in a cast. Torg et al. treated 10 patients with non-weight-bearing cast for

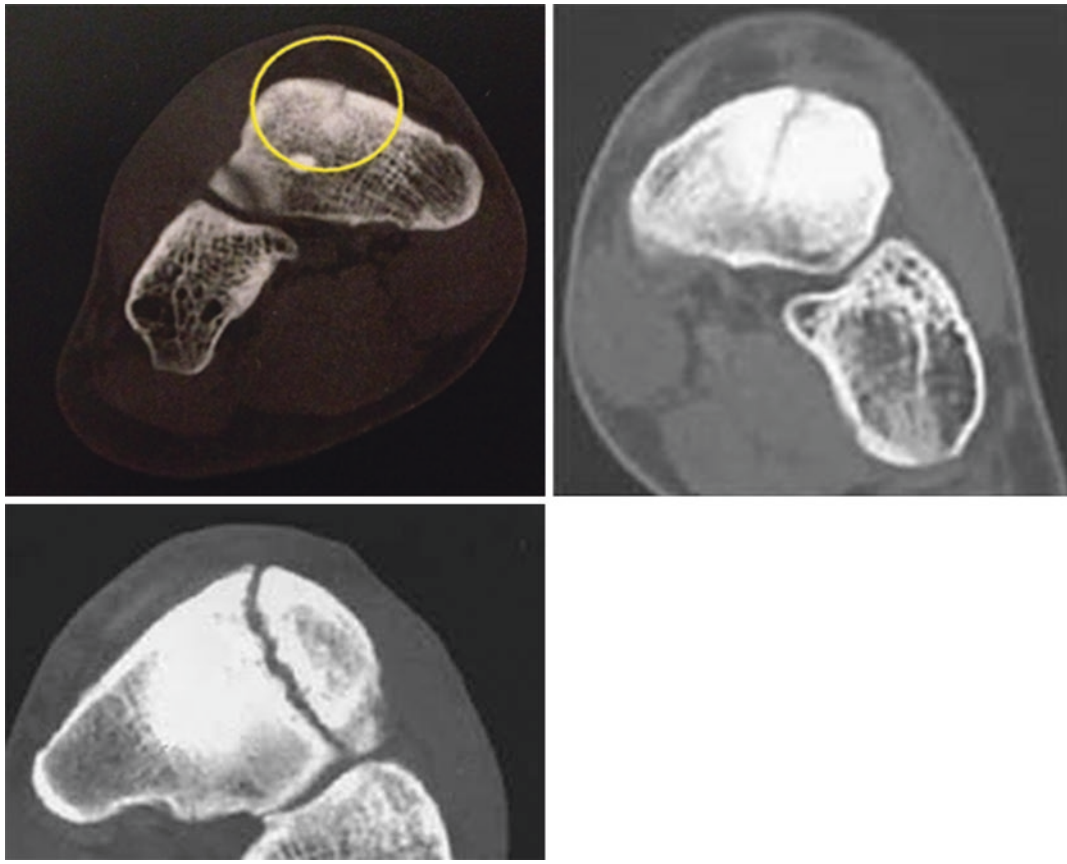

Fig. 18.12 CT axial images demonstrating Saxena classification. Type I is a fracture through the dorsal cortex of the navicular; type II is a fracture that extends into the navicular body; and type III is a fracture that penetrates through a second cortex

6–8 weeks and had a 100% healing rate without complications, but with return to activity an average of 3–6 months [56]. In a cohort treated with a walking cast, 78% could not resume sports because of pain. Khan also demonstrated a significantly worse return to full activity in athletes who used a weight-bearing cast compared to non-weight-bearing cast [57]. Surgery has been proposed for nondisplaced fractures involving two cortices, displaced fractures, fractures with sclerotic changes, and athletes who failed conservative treatment or cannot tolerate a long recovery course. Saxena and Fulham [58] found that there were no clinical differences in those patients who were treated nonoperatively versus those who underwent surgery for fixation. Surgery was recommended for type II and III fractures, and return to activity was similar for both populations at 3.9 months. A meta-analysis that evaluated outcomes of navicular stress fractures treated with surgery versus non-surgical non-weight-bearing management concluded that there was no statistical significant difference [59]. Weight-bearing as a conservative treatment was shown to be significantly less effective than either non-weight-bearing or surgical treatment. Mallee reviewed 200 stress fractures of the navicular in athletes but did not perform a statistical analysis comparing success of immobilization. However, the researchers did note that the weighted mean time to return to sports was 16.4 weeks in those treated with surgery versus 21.7 weeks in patients treated conservatively with non-weight-bearing cast for greater than 6 weeks [60].

Surgery should be strongly considered with athletes with type II and III navicular stress fractures, especially those with cystic changes, sclerosis, or osteonecrosis. These

fractures have a frequency of delayed union and refracture which can result in unpredictable healing times. A shortest time to return to play is important for athletes and is often the determining factor when deciding on treatment recommendations. Saxena described outcomes of navicular fractures in athletes using their protocol of non-weight-bearing for type I fractures, and surgery for type II and III fractures resulted in greater than 90% of athletes being able to return to activity at their preinjury level [54]. All 21 elite or professional athletes were able to return to activity. Patients who underwent open reduction and internal fixation had a return to activity of 4.56 months compared to those who had undergone a nonoperative treatment who had an average return to activity of 3.97 months.

For type I navicular stress fractures treated surgically, percutaneous fixation with solid screw (but cannulated technique) placed lateral to medial is recommended (Fig. 18.13). For type II and III fractures, an open dorsal approach with autograft bone, iliac bone marrow aspirate, and two screws placed lateral to medial through a separate lateral incision can be utilized (Fig. 18.14).

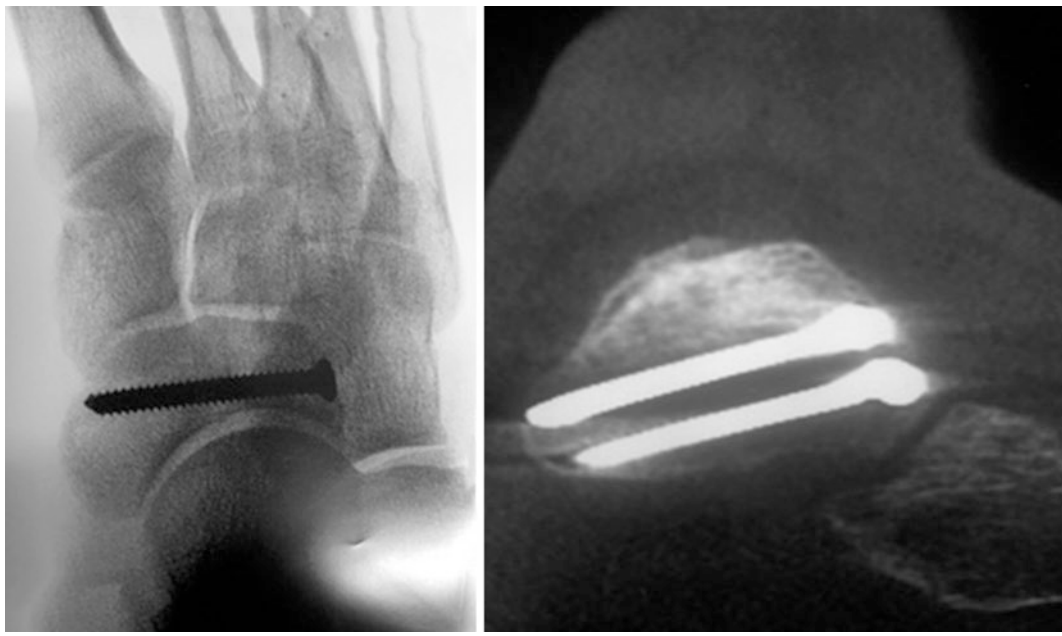

Fig. 18.13 Surgical fixation of navicular stress fracture treated with solid screw placed lateral to medial

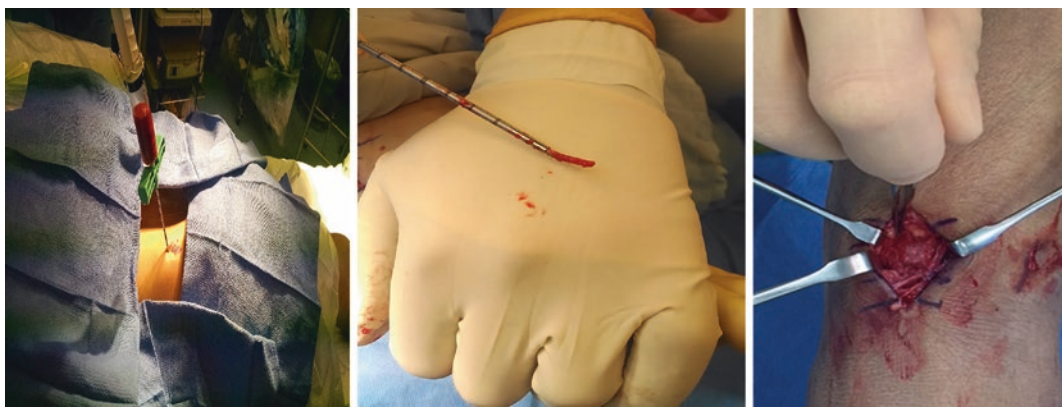

Fig. 18.14 Iliac crest aspirate plus cancellous autograft utilized for type II and type III navicular stress fractures

The screws should be placed perpendicular to the fracture line, and intraoperative CT scan can aid in technique if available. For refractures or nonunions, a localized bone graft technique that was described by Nunley should be performed [61] (Figs. 18.15 and 18.16).

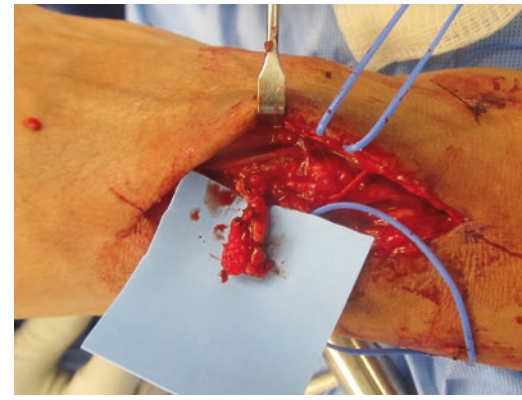

Fig. 18.15 In refractures or nonunions of navicular stress fracture, a pedicled cuneiform graft can be utilized

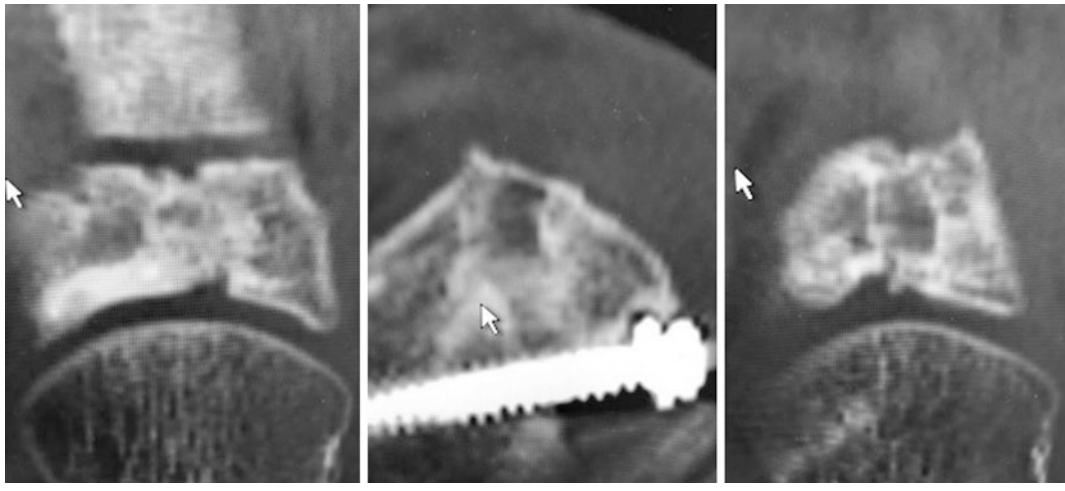

Fig. 18.16 CT images post-vascularized graft showing healed fracture with excellent incorporation of graft

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Stress Fractures of the Midfoot and Forefoot

19

Justin J. Hicks, Parth Vyas, Jonathon Backus, and Ljiljana Bogunovic

First Through Fourth Metatarsal Stress Fracture

Introduction

Metatarsal stress fractures are the most common stress fractures of the foot. In one study of 250 stress fractures, lesser metatarsal stress fractures accounted for 38% of the cases described [3]. Coined "march fractures" in the late 1800s because of their high prevalence among members of the military, these fractures are often activity related, caused by the summation of stresses that ultimately lead to fatigue and failure of bone [4]. They are often seen in athletes participating in running and jumping sports including ballet and track and field. These stress fractures should be considered in any athlete or military recruit when a patient complains of dorsal forefoot pain. The second and third metatarsals are most commonly involved with an incidence of 52% and 35%, respectively [5]. First and fourth metatarsal stress fractures are less common accounting for 8% and 5% of metatarsal stress fractures, respectively [5]. One study of 827 military recruits found that 11% of metatarsal stress fractures were located at the first metatarsal [6]. First metatarsal stress fractures are typically located about the proximal metadiaphyseal junction, while second, third, and fourth metatarsal fractures are often in a diaphyseal in location [7].

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Pathophysiology

Repetitive or chronic stress of the foot predisposes patients to metatarsal stress factors especially in patients with altered biomechanics. This alteration can be caused by acquired or congenital cavovarus or hallux valgus, neuropathy, osteoporosis, rheumatoid arthritis, plantar ganglion cysts, osteochondromas, and iatrogenic causes such as metatarsal osteotomies or resections and corrective bunion procedures [8–15]. Significantly higher strains about the metatarsals are also associated with pes planus. Moreover, strains encountered by the metatarsals increase 100% with resection of the plantar fascia [16].

Second metatarsal stress fractures in athletes tend to occur either at the metatarsal base or the distal diaphysis. Distal fractures are related to high-impact and high-stress activities such as running. Metatarsal base fractures are usually associated with lower bone mass, low-volume training, and multiple metatarsal fractures [17]. These fractures are associated with prolonged recovery time and higher rates of nonunion [18, 19]. Changes in shoe wear have also been associated with metatarsal stress fractures in runners. In a case study of two experienced runners, switching to a barefoot-simulating shoe was associated

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with second metatarsal stress fractures [20]. Ballet dancers are particularly at risk for the second and third metatarsal base fracture secondary to the unique foot positions such as standing "en pointe" which involves supporting their body weight on the toes with the foot plantarflexed [21–28]. These stress fractures have also been observed in classical Irish dancers [29].

Although the exact pathogenesis of metatarsal stress fractures has yet to be described, it is believed that the material properties of the lesser metatarsals are more important than the overall geometry. In an in vitro study of second metatarsals, volumetric cortical bone mineral density correlated with load to failure in cantilever bending, whereas geometric variables did not [30]. Other literature regarding animal bone properties suggests that repeated stress leads to the formation of microcracks where a threshold exists that could either allow a bone to remodel or form a stress fracture [31, 32]. Nevertheless, when microcracks were studied in human metatarsals, no link could be established to stress fracture formation [33].

Diagnosis

Clinical Presentation

Athletes usually present with generalized dorsal forefoot pain and swelling [21, 34–36]. The average time to presentation typically ranges from 2 to 6 weeks. Pain is activity related and worsened by weight-bearing [37]. Many athletes will be unable to run or complete training activities [37]. Although rare, stress fractures can also occur in the metatarsal heads and mimic metatarsalgia or plantar plate injuries as well.

Imaging

Radiographic evaluation should begin with anteroposterior, lateral, and oblique plain radiographs. Initial findings are typically subtle periosteal reactions that may show progression overtime (Fig. 19.1). First metatarsal stress fractures are usually associated with linear sclerosis that is located perpendicular to the direction of stress. Periosteal reactions are less common on plain radiographs of the first metatarsal [5]. In athletes, MRIs are the preferred advanced imaging for these injuries and are particularly useful in identifying stress fractures as symptoms first start and plain films are often negative. Radionuclide bone scanning can also be used to help diagnose bony stress injuries. Technetium TC 99 diphosphonate triple-phase scanning has been shown to diagnose stress injuries 2–8 days following the onset of symptoms [38]. This test has largely fallen from favor as it is more invasive, takes significantly longer to perform than MRI, and exposes the patient to ionizing radiation. Recently, ultrasound has also been reported to aid in the early detection of metatarsal stress fractures with the potential benefit of costs savings [35, 39, 40]. In one study, high-resolution ultrasound was found to be 83% sensitive and has specificity of 76% for the diagnosis of metatarsal stress fractures [39].

Treatment

Nonoperative Management

Initial treatment of lesser metatarsal stress fractures is conservative. Many of these injuries heal with cessation of the offending activity and stiff-soled shoe wear. Protected weight-bearing in either a postop shoe, CAM walking boot, or weight-bearing cast is allowed unless there is concern for decreased healing potential in athletes with malnutrition or excessive pain. Choice of foot orthoses is determined primarily by provider preference. It is the authors' preference to avoid use of a CAM walking boot or cast immobilization in order to minimize stiffness and deconditioning in the athlete which may prolong recovery. An exception to this treatment plan is a stress fracture at the base of the second metatarsal in ballerinas. These injuries can potentially involve the Lisfranc ligament complex and should be protected with a CAM walking boot and a 4-week period of non-weight-bearing [26].

Early mobilization and physical therapy can help decrease the duration of recovery. Nonimpact activities are allowed after symptoms have ceased with ambulation for 1–2 weeks. Many stress

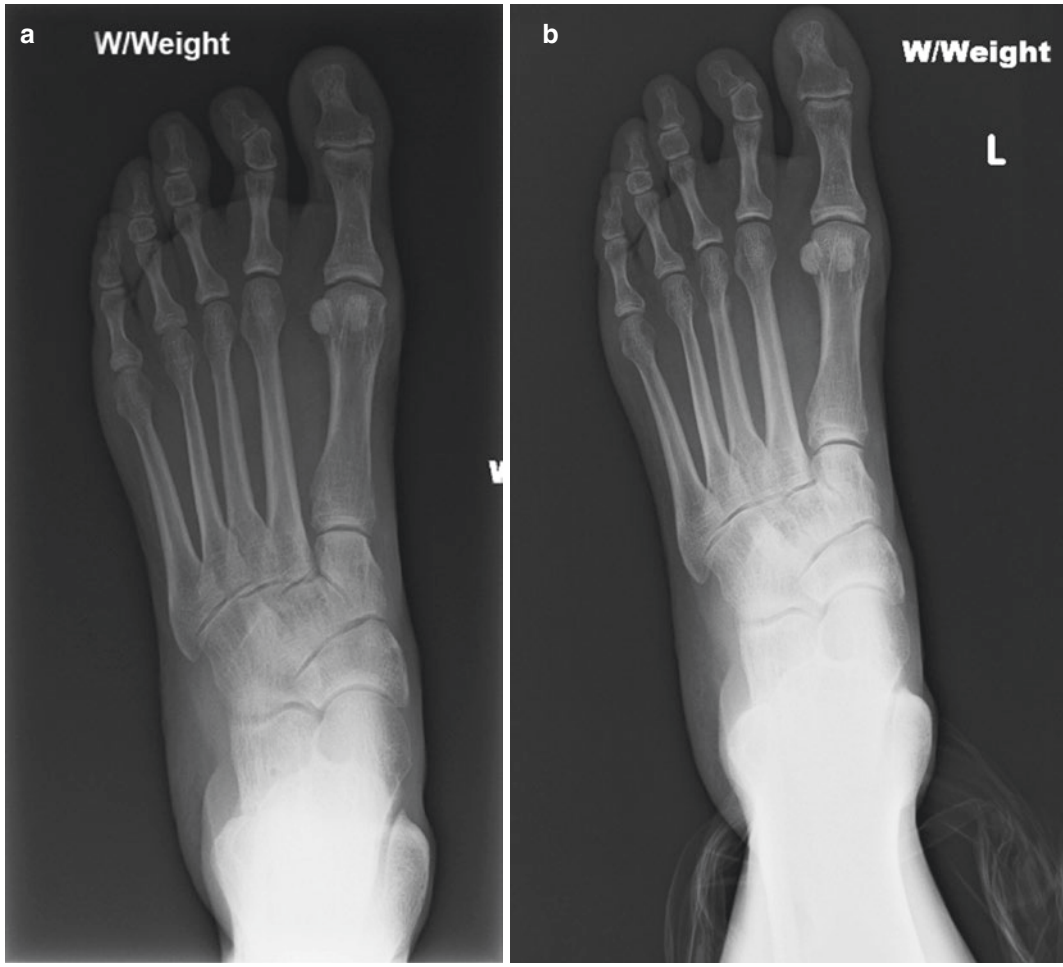

Fig. 19.1 Left: Anteroposterior radiograph at the time of initial presentation demonstrating no acute fractures of the third metatarsal. Right: Anteroposterior radiograph of the same patient after 3 weeks demonstrating healing stress fracture of the third metatarsal at the metatarsal neck. Note the presence of callus

fractures will show radiographic healing and symptom resolution within a 6–8-week period [1]. Running and impact activities are allowed once this point of recovery has been achieved. Pathologic foot shapes and gait patterns should be assessed, and custom orthotics may be warranted to help prevent future injuries. It is also appropriate to advise the athlete on appropriate nutrition and gradual increase in training volumes and intensity. In the absence of metabolic or endocrine abnormalities, recurrent stress fractures are rare and are very unlikely to recur at the same site [41].

Bone stimulator use has also been described in treating metatarsal stress fractures. In a study of 19 ballet dancers with second and third metatarsal base stress fractures, both external shock wave therapy and pulse electromagnetic field stimulators were used in conjunction with 3–5 weeks of non-weight-bearing. All patients returned to dance at a mean of 4.6 weeks and returned to full en pointe dancing at a mean of 18 days following this. No patients had persistent pain or nonunion [42].

In terms of preventative treatment, several studies in military populations have suggested

that the use of custom orthoses to modify foot biomechanics can result in decreased metatarsal stress fractures and lower-extremity injuries [36, 43, 44]. In a study of military recruits, 400 individuals that were determined to have medium- to high-risk anatomic features of the foot or plantar pressures (as determined by a proprietary algorithm after walking on a force plate) were randomized to receive 3D-printed custom orthotics or standardized military footwear. The group that received the custom orthotics had significantly fewer injuries than the control group (61 vs 21, $p < 0001$) [44]. Metatarsal stress fractures were decreased in the data presented; however, no subanalysis was performed.

Operative Management

In rare cases of chronic pain and nonunion, open reduction and internal fixation with bone grafting can be considered [45]. Operative intervention for a symptomatic nonunion includes takedown of the nonunion, internal fixation with a plate, and possible use of adjuvant bone grafting. There is one case series utilizing axial screws for fourth metatarsal stress fractures without the complication of tarsometatarsal joint degeneration with up to 10-year follow-up [46].

Fifth Metatarsal Stress Fractures

Introduction

Fifth metatarsal fractures were originally described by Sir Robert Jones in 1902 in a case series including his own fracture sustained while dancing [47]. Since that time, greater attention has been paid to fractures about the proximal aspect of the fifth metatarsal leading to many subclassifications and intensive analysis of their presentation, treatment, and outcomes. Stress fractures of the fifth metatarsal usually occur in athletes involved in running or jumping sports [48]. These injuries are deemed high risk given their tendency toward delayed union, nonunion, and refracture [49–52].

In a recent case series of lower-extremity stress fractures in professional basketball players in the National Basketball Association (NBA), 55% involved the foot with fifth metatarsal stress fractures being the most common stress fracture overall [2]. DeLee et al. defined a proximal fifth metatarsal stress fracture by three criteria: (1) a prodrome of symptoms over the lateral aspect of the foot, (2) radiographic evidence (intramedullary sclerosis, periosteal reaction, and cortical hypertrophy) of a proximal fifth metatarsal stress reaction in the bone, and (3) no previous treatment of an acute fifth metatarsal fracture [49, 53].

Patients typically present without an acute injury to the foot with a history of prodromal symptoms about the dorsolateral surface of the forefoot and radiographic evidence of stress reaction. It is important to have a high clinical suspicion in these athletes because a delay in diagnosis or inadequate treatment can result in significant delays in return to play and can even be career-ending. In a study of NBA players, only 43% who sustained fifth metatarsal stress fractures were unable to return to sport [2].

Anatomy

The anatomy of the proximal fifth metatarsal predisposes this bone to stress injury. To appropriately diagnose and manage injuries about the fifth metatarsal, a firm understanding of the anatomy of the fifth metatarsal is imperative. The proximal fifth metatarsal has traditionally been divided into three zones (Fig. 19.1). Zone 1 is the most proximal and encompasses the proximal highly vascular cancellous tuberosity. Zone 2 is defined by the metaphyseal–diaphyseal junction as well as the fourth and fifth metatarsal articulation. Zone 2 is the location of a true Jones fracture. Zone 3 starts distal to the intermetatarsal ligaments extending 1.5 cm distal into the tubular portion of the diaphysis. The lateral band of the plantar fascia inserts on the plantar aspect of the metatarsal styloid, while the peroneus brevis inserts on the highly vascular tuberosity in Zone 1, and the peroneus tertius inserts on the dorsal surface of the metaphyseal diaphysis (Zone 3) (Fig. 19.1).

The diaphysis of the fifth metatarsal has a curve located in about the distal 1/3. The dorsal–

plantar cortex is thinner than the medial–lateral cortex. The fifth metatarsal has an apophysis located at the proximal fifth metatarsal that can persist into adolescence. It is often present bilaterally and can be distinguished from stress reactions, fractures, and nonunion given its smooth rounded edges and presence on contralateral radiographs.

Classification

There are several classifications for proximal fifth metatarsal fractures. The three-zone concept described by Dameron is the most common classification utilized [54]. Zone 1 is the most proximal and encompasses the highly vascular cancellous tuberosity. Zone 2 is defined by the metaphyseal–diaphyseal junction between the fourth and fifth metatarsal articulation. Zone 2 is the location of a true Jones fracture. Zone 3 starts distal to the intermetatarsal ligaments extending 1.5 cm distal into the tubular portion of the diaphysis (Fig. 19.2) [54].

Torg et al. described a commonly utilized system for proximal fifth metatarsal fractures based on the presence and amount of periosteal bone reaction (acute on chronic) vs intramedullary sclerosis (late) (Fig. 19.3) [50]. Torg type I represents an acute fracture where radiographs show prior periosteal reaction, a plantar-based fracture line, and no medullary sclerosis. Torg type II

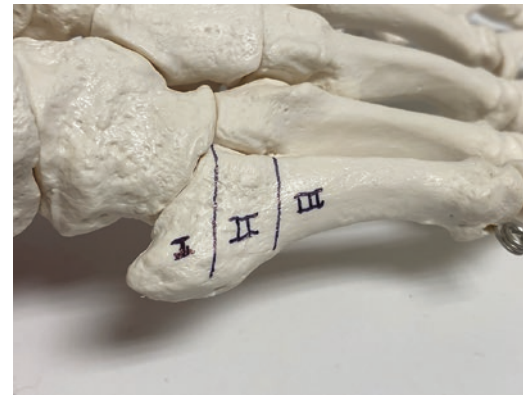

Fig. 19.2 A saw bones model with the three different zones of the proximal fifth metatarsal as described by Dameron et al. [48]

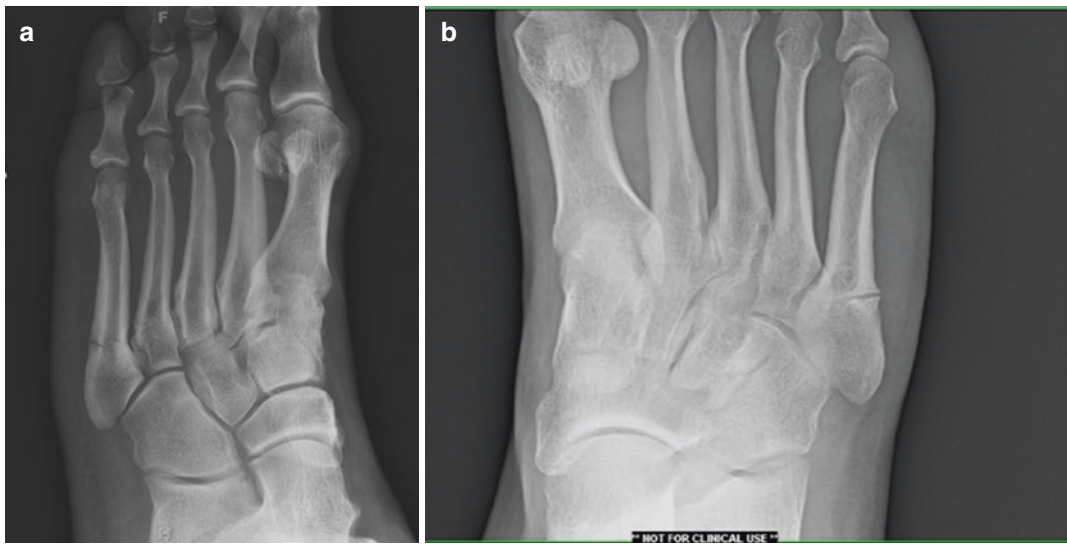

Fig. 19.3 Left: Oblique radiograph of the fifth metatarsal, demonstrating an acute fracture distal to the tuberosity. There is some cortical hypertrophy, an indicator of chronic stress, but the fracture line is narrow. It involves both cortices, and it is not associated with intramedullary sclerosis. Right: Oblique radiograph demonstrating nonunion. Note the widening of the fracture line with cortical hypertrophy and dense intramedullary sclerosis completely obliterating the medullary cavity. (Images courtesy of Sandra Klein, MD, Washington University in St. Louis, MO)

fractures occur in the setting of medullary sclerosis and narrowing and are associated with delayed union. Torg type III fractures have complete obliteration of the medullary canal and represent a nonunion.

DeLee et al. classified fractures based on the chronicity as well as the presence of a plantar fracture gap measured on the standard oblique radiograph [49]. Type A1 represents an acute, complete fracture of the proximal fifth metatarsal at the metaphyseal–diaphyseal junction. Type A2 represents an acute-on-chronic complete fracture at this location. A type B1 fracture is an incomplete fracture with a plantar gap measuring less than 1 mm. Finally, a type B2 fracture is an incomplete fracture with a plantar gap measuring 1 mm or greater.

Pathophysiology

The fifth metatarsal is particularly at high risk due to the stresses it experiences and its blood supply. Smith et al. state that there are three sources of blood supply to the fifth metatarsal: the nutrient artery, periosteal arteries, and metaphyseal perforators [55] (Fig. 19.4). The single nutrient artery that feeds the fifth metatarsal enters the medial cortex at the junction of the proximal and middle third diaphysis; the base and tuberosity are supplied by epiphyseal and metaphyseal arteries. This distribution of the blood flow results in a watershed area near the proximal metadiaphyseal junction leading to impaired reparative biologic response to stresses at the junction [51]. In addition to the poor blood supply, this area also experiences a high level of stress in athletes. During gait, the fifth metatarsal experiences stress when the heel is off the ground and body weight is transferred to the lateral column. This causes adduction of the fifth metatarsal, which is resisted proximally by ligamentous attachments, resulting in a repetitive varus stress at the metaphyseal–diaphyseal junction [47, 56]. Stress typically starts laterally and progresses medially leading to plantar lateral gap due to the tensile forces plantarlaterally and compressive forces dorsomedially. This stress is worsened because the metatarsal head is more mobile than the proximal base (capsular and ligamentous attachments) causing a fulcrum effect at the meta-diaphyseal junction [57]. Some studies in athletes have suggested that the greatest pressure differential between the base of the fifth metatarsal and the head occurs during the acceleration phase of running [58]. Based on the results of this study, injury prevention programs have started to emphasize longer recovery time between accelerations to prevent buildup of stress in the metadiaphyseal junction.

Fig. 19.4 Cadaveric specimen demonstrating the vascular anatomy of fifth metatarsal. This specimen illustrates the main blood supply to the fifth metatarsal. The watershed area lies at the transition between the proximal branch of the intramedullary nutrient artery and the proximal metaphyseal vessel and is marked with an asterisk. (Image courtesy of Sandra Klein, MD, Washington University in St. Louis, MO)

Diagnosis

Clinical Presentation

Patients with fifth metatarsal stress fractures may present with an acute fracture or may have an insidious onset of activity-related pain about the proximal fifth metatarsal. Patients typically present with lateral foot pain, tenderness to palpation over the fifth metatarsal, and pain with bone percussion test. Swelling may be present; however, there is often very little swelling.

The goal of the history and physical exam would be to identify both extrinsic and intrinsic risk factors responsible for fifth metatarsal stress fracture. Intrinsic factors would include a high longitudinal arch, leg length discrepancy, cavovarus, or hindfoot varus position which may

predispose to fifth metatarsal stress fractures. Extrinsic factors include an intense training regimen, prolonged running, poor training, or incorrect footwear [59]. Special attention should be paid in obtaining a thorough understanding of level of intensity and training schedule in an athlete suspected of having a fifth metatarsal stress fracture.

Imaging

Radiographic evaluation should always include an AP, lateral, and oblique radiograph (preferably weight-bearing). Initial radiographic findings generally include periosteal reaction as well as varying degrees of progressive intramedullary sclerosis [4]. A computed tomography (CT) scan can be helpful for diagnosis as well as preoperative planning. CT scan can further elucidate the degree of sclerosis and intramedullary canal obliteration.

Magnetic resonance imaging (MRI) and Technetium-99 m-labeled diphosphonate bone scans are both sensitive and specific for stress fractures and are useful in the setting of negative plain films [60]. Findings on an MRI that suggest a stress fracture of the fifth metatarsal include endosteal marrow edema and periosteal edema of the proximal fifth metatarsal on T2. A nondisplaced fracture line may also be seen on T1 sequence. An increased uptake in the region of stress reaction may be seen on bone scan. Both of these imaging modalities can be used to make an early diagnosis of a stress reaction in the proximal fifth metatarsal [48, 51, 61]. MRI is often the modality of choice.

Treatment

Nonoperative Management

Nonoperative management for fifth metatarsal stress fractures can be successful [62]. This typically includes 4 weeks in a non-weight-bearing cast followed by 4 weeks in a weight-bearing cast or boot or weight-bearing orthosis for older less demanding patient [63]. It should be noted that nonoperative management is associated with longer time to union as well as high risk of delayed union, nonunion, and refracture [64]. This should be considered when treating athletes, especially at the elite level. This cohort of patients may benefit from earlier surgical intervention of nondisplaced fractures.

Other nonoperative treatment modalities for fifth metatarsal stress fractures such as extracorporeal shockwave therapy (ECSWT) and electromagnetic bone stimulation have been studied [65]. The high-energy acoustic waves of ECSWT have been shown to induce neovascularization and bone healing in animal models. Strong clinical evidence for ECSWT is lacking; however, it is an option especially in lower-demand patients.

Electromagnetic bone stimulation has also been used in treating proximal fifth metatarsal fractures and has shown some promising results in the setting of stress fractures, delayed union, and nonunion; however, these are largely level IV studies. This modality may be useful as an adjunct to surgery, but high-level evidence is still lacking [57].

Operative Treatment

Multiple studies have demonstrated that surgical management leads to earlier union, lower incidence of nonunion, and reduced rates of refracture. Due to the high incidence of delayed union or nonunion, aggressive management has been recommended especially in elite athletes, those with persistent pain, and those patients who develop a pseudoarthrosis [61].

Several operative treatment options have been described. These include intramedullary screw, inlay cortical cancellous graft, tension band wiring, and combinations of screw placement and grafting [49, 50, 53]. Intramedullary screw is preferred as initial fixation to reduce the incidence of prominent hardware in this area of the foot. Huh et al. found that in a cadaveric Jones fracture model, intramedullary screw fixation demonstrated bending stiffness and resistance to early torsional loading that was superior to that offered by plate fixation [66]. Relative contraindications include an obliterated or sclerotic intramedullary canal and an acute fracture in a nonathlete [63].

There has been much debate regarding the biomechanics of screw selection such as solid

versus cannulated, length of screw, diameter of the screw, and headless versus headed. Shah et al. demonstrated that a 5.5 mm screw diameter did not improve 3-point bending failure load strength when compared to a 4.5 mm screw diameter [67]. However, Kelly et al. noted that a 6.5 mm screw has a greater pullout strength than the smaller-diameter screws; hence, a larger-diameter screw can be used to fill larger intramedullary canals [68]. Regarding screw length, typically a longer screw is not necessary and in certain cases can even be detrimental given the curvature of the bone distally and the risk of cortical perforation medially which may even lead to lateral gapping and distraction at the fracture site. Recently a method utilizing a pointed reduction clamp to prevent iatrogenic displacement and gapping during placement of a solid intramedullary screw was described [69]. Porter et al. studied 23 athletes treated surgically with a 4.5 mm cannulated screw for fifth metatarsal Jones and found that fixation with a stainless steel 4.5 mm cannulated screw had 100% clinical healing and near 100% healing as shown on radiographs [70]. All athletes returned to sport at the meantime of 7.5 weeks. Despite numerous studies showing reliable outcomes with the use of a cannulated screw, a majority of the surgeons today continue to use a solid screw partly due to the superior fatigue strength of a solid screw when compared to a cannulated screw [71]. Sides et al. showed that headless compression screws are effective in resisting bending strength but do not offer equivalent resistance to thread pullout. In nonunion cases plate fixation is often considered due to increased bony sclerosis at the medullary canal, and the fracture is often opened anyhow for bone grafting [72].

Fractures with a large plantar lateral gap (greater than 1 mm) have been shown to demonstrate delays in time to union [73]. In fractures with a large plantar lateral gap and sclerosis about the fracture, autogenous bone graft is recommended [49, 57, 74]. Miller et al. have utilized bone marrow aspirate concentrate in this scenario; however, strong prospective long-term studies of its efficacy are lacking [75]. There is currently a double-blinded randomized controlled trial underway to study the effects of BMAC on fifth metatarsal stress fractures [76].

Patients generally do well following internal fixation of proximal fifth metatarsal stress fractures; however, some patients do require hardware removal after fracture healing secondary to persistent pain, while others endorse shoe-wear irritation about the surgical incision [53, 77, 78]. Screw fracture and malpositioning of intramedullary screws have also been reported [79]. Begly et al. retrospectively reviewed 26 basketball players with joint fractures over 19 NBA seasons and found that 85% of athletes returned to their preinjury level of competition and there was no decrease in performance on their return to play [80]. Additionally, Lareau et al. studied 25 NFL players who underwent Jones fracture fixation with an intramedullary screw and found that with an aggressive rehabilitation protocol, early return to play (average of 8.7 weeks) was achievable with a low refracture rate [81].

Navicular Stress Fractures

Introduction

Approximately one-third of stress fractures of the foot and ankle involve the tarsal navicular [82, 83]. These injuries are most often seen in athletes participating in track and field, basketball, and gymnastics [82–84]. Several characteristics unique to the tarsal navicular predispose this bone to stress injury including its anatomic, biomechanical, and vascular properties. Positioned between the talar head and the cuneiforms, the navicular experiences significant shear stress at the central third of the bone, resulting in a common site of stress injury [85]. These forces are accentuated during the foot strike phase of running [63]. The blood supply to the tarsal navicular arises from branches of the dorsalis pedis and tibialis posterior arteries [85] (Fig. 19.5). A relatively avascular zone in the central third of the navicular has been described and felt to contribute to the development of stress fractures in this area [86, 87]. The classic navicular stress fracture line typically occurs within this avascular zone, extending in an oblique fashion from dorsomedial to plantar–lateral.

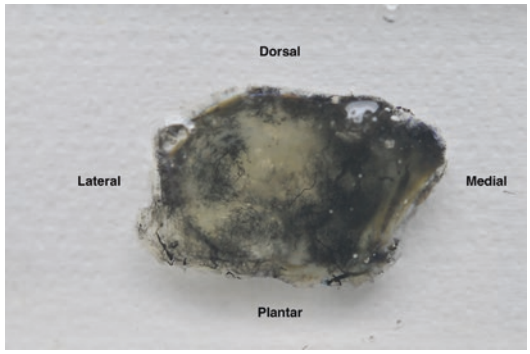

Fig. 19.5 Cadaveric specimen demonstrating the vascular anatomy of the navicular. Navicular stress fractures often occur in the hypovascular area located in the central third of the navicular as seen here. (Image courtesy of Sandra Klein, MD, Washington University in St. Louis, MO)

Diagnosis

Clinical Presentation

The pain associated with stress fractures of the navicular is often insidious and vague. Many initially describe a soreness or cramping on the dorsomedial aspect of the foot. Others may find the pain difficult to localize and report pain in the distal tibia, ankle, and/or arch. Given these subtle clinical signs, diagnosis of this injury is frequently delayed, with the average patient receiving treatment 6–8 months after the onset of symptoms [84, 85]. Initially, pain is only present with activity. Explosive movements such as jumping, sprinting, and cutting typically exacerbate symptoms [85]. As the injury progresses, the athlete may develop pain with activities of daily living or even at rest.

A high index of suspicion is required to make the diagnosis when evaluating an athlete with foot and/or ankle pain. On physical examination, patients will often have tenderness with palpation over the dorsal navicular, referred to as the "N spot" [88, 89]. They may also report pain with dorsiflexion of the ankle and range of motion of the subtalar joint [18]. Dynamic tests such as single-limb hop may also reproduce pain to the injured navicular [85].

Imaging

Weight-bearing X-rays of the foot and ankle should be obtained in all patients with a suspected navicular fracture. In chronic cases a fracture line may be visible over the dorsal cortex on the lateral view, but in the majority of cases, plain radiographs are normal [63, 88, 89]. In a patient with a suspected stress fracture and negative plain films, advanced imaging such as a bone scan, MRI, or CT scan should be obtained. Despite 100% sensitivity for detecting stress fractures of the navicular, bone scans are not very specific and are unable to differentiate stress fractures from other painful conditions involving the navicular such as a symptomatic accessory navicular and posterior tibial tendonitis [63]. CT scan and MRI provide both sensitivity and specificity in addition to detailing the fracture pattern (Fig. 19.6). Despite exposure to radiation, CT scan may be slightly advantageous to MRI in that it allows for classification of the fracture type which can guide treatment and prognosis [4, 84]. Data also exists that suggests a slight advantage of CT scan over MRI in more accurate diagnosis of navicular stress fractures [84, 90]. If the CT scan imagining is negative and a high clinical suspicion for navicular stress injury exists, MRI is recommended as it can identify as stress reaction prior to the development of a stress fracture.

Classification

Saxena et al. established a navicular stress fracture classification system based on the fracture characteristics as seen on CT imaging [84]. This classification system is often utilized to guide fracture treatment and does appear to correlate with patient outcome. Type 0.5 fractures are stress reaction injuries were edema is seen within the navicular on MRI, but no fracture line is visible on CT. Type I fractures are incomplete fractures in which the fracture line only involves the dorsal cortex of the navicular. Type II fractures are incomplete fractures in which the fracture line extends from the dorsal cortex to the body of the navicular. Type III fractures are complete fractures that extend through both cortices of the navicular. Fractures are further subclassified if additional findings indicative of chronicity, including cystic and sclerotic changes, are evident on CT imaging. Saxena et al. reports pro-

longed return to sport with more severe fractures (Type II and III) compared to more mild injuries (Type 0.5 and Type I) [84].

Treatment

Nonoperative Treatment

Navicular stress fractures are considered "high-risk" stress injuries due to the elevated risk of delayed healing, nonunion, fracture progression, refracture, or talonavicular arthritis development without adequate treatment [84, 88–91]. The treatment for navicular stress fractures, however, remains controversial. While some promote a trial of conservative management of all navicular stress fractures regardless of fracture type, others suggest limiting conservative management as first-line management to less severe injuries such as Type 0.5 and Type 1 fractures [84, 89].

Despite the controversy, successful management of both partial and complete navicular stress fractures with conservative treatment has been described [63, 89, 90]. The treatment protocol involves non-weight-bearing cast immobilization for 6–8 weeks followed by gradual progression to weight-bearing in a boot until pain-free. Impact activity such as running and jumping is allowed to resume at approximate 12–16 weeks provided the patient is asymptomatic [63]. Full return to activity is typically allowed after 4 months. Repeat CT scan can be performed prior to return to sport to confirm healing.

Conservative treatment employing weight-bearing immobilization has been showed to result in poor outcome including delayed union, nonunion, and fracture recurrence [88, 92, 93]. In a series of 86 navicular stress fractures, Khan et al. reported return to full activity in 86% of patients who were treated with a minimum of 6 weeks of non-weight-bearing immobilization. Average return to activity in these patients was 5.6 months. In comparison, only 26% who were treated in a weight-bearing boot for a minimum of 6 weeks were able to ultimately return to sport [88]. In a recent meta-analysis of 251 navicular stress fractures, non-weight-bearing and immobilization were successful in 96% of patients. Successful or unsuccessful was based on radiographic and/or clinical healing of the fracture and time from onset of treatment to return to activity. There was a trend toward successful outcome (96%) among patients who were treated with non-weight-bearing immobilization versus surgical fixation (82%); however, this was not statistically significant. In this same series, only 47% of patients allowed to weight-bear during conservative treatment experienced a successful outcome [92]. A recent retrospective review found that 11% of patients treated nonoperative experienced a refracture on average 5 years after the initial injury [84]. College-age or younger athletes should be counseled on these outcomes during discussions of nonoperative versus operative management. Further long-term studies are needed to assess the outcomes of patients managed nonoperatively.

Operative Treatment

Operative treatment of navicular stress fractures is indicated in those patients who fail a trial of non-weight-bearing immobilization [63, 84, 85]. Surgical fixation has also been advocated for patients with nondisplaced complete fractures, displaced fractures, fractures with evidence of chronicity including cystic and/or sclerotic changes, and fractures in high-level athletes who desire an expedient return to full activity [63, 84, 85]. An increased risk of refracture has been reported in athletes treated with conservative management compared to operative fixation [63, 84, 90, 94]. In a recent prospective study of 62 navicular stress fractures, Saxena et al. reported refracture in 11.2% of patients at an average of more than 5 years post-initial injury. Unlike patients who had been treated with previous operative fixation, all of those who experienced refracture had been treated with nonoperative management [84]. While many studies demonstrate no difference in return-to-sport time between conservative and operative treatment, Saxena et al. did report a faster return to sport for type II and III fractures when patients were treated with internal fixation compared to nonoperative management [95].

Operative fixation, when utilized, often involves compression screws placed from dorsal lateral to plantar medial across the fracture line (Fig. 19.6). This can be done percutaneously in the setting of nondisplaced fracture or with open reduction with or without debridement and grafting in displaced fractures and/or those with signs of chronicity such as sclerosis or cystic change. An open approach is often recommend given the natural variability of the neurovascular structures overlying the dorsal navicular [63]. The incision is typically centered over the fracture line over the middle third of the navicular. Referencing the CT scan can be helpful in localizing the fracture line which can often be difficult to identify intraoperatively. If sclerosis is present at the fracture site, debridement with a curette and/or rongeur is recommended in addition to the application of bone graft in the fracture site. When placing screws, care must be taken to ensure that the screw length is not too long so as to prevent injury to the posterior tibialis tendon. One or two screws are typically placed parallel to each other in a perpendicular fashion across the fracture line. A partially threaded solid screw (4.0) is preferred over a cannulated screw given its increased strength. Postoperatively, patients are kept in a non-weight-bearing cast for 6 weeks. Patients are subsequently

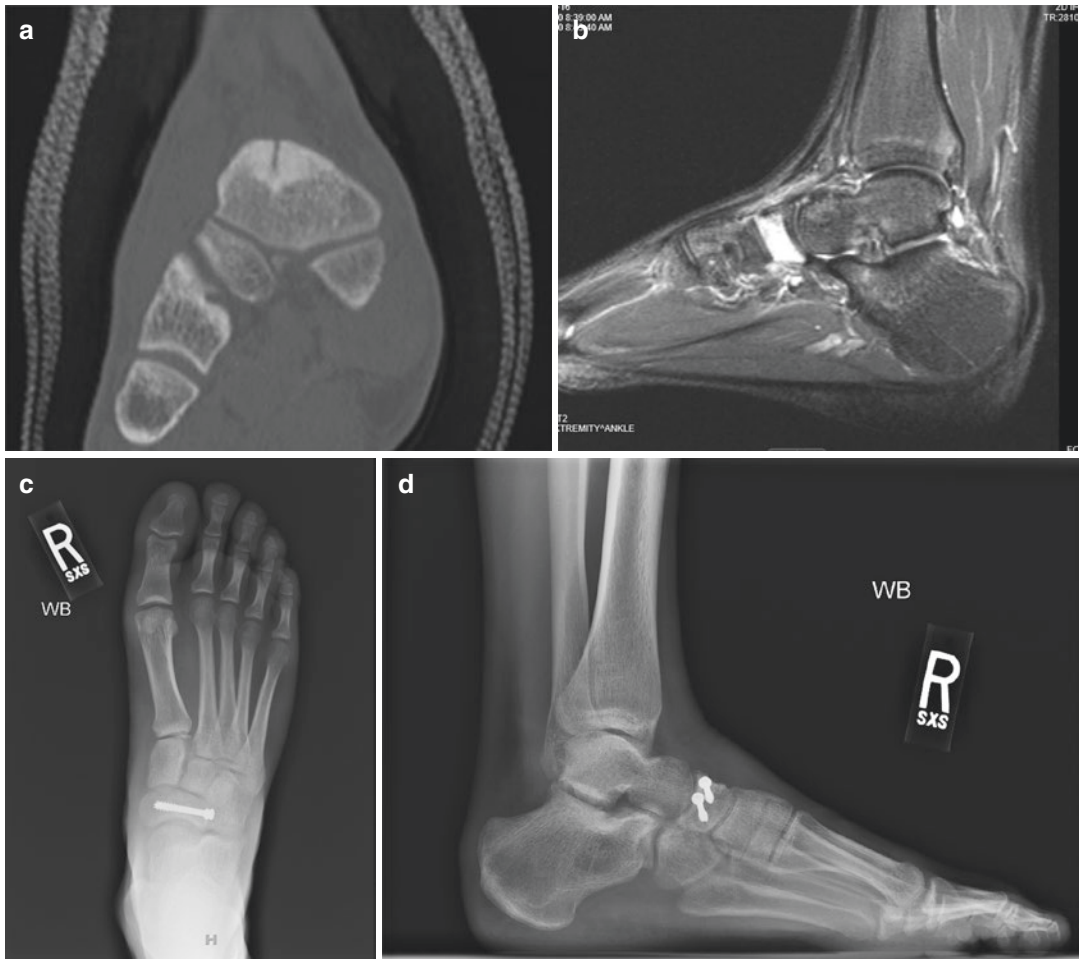

Fig. 19.6 Clinical example of a navicular stress fracture fixed with open-reduction internal fixation and bone grafting. Top left image is a coronal CT demonstrating an incomplete dorsal fracture line and sclerosis. Top right image is a sagittal T2 MRI with significant edema noted within the navicular body. Lower left and right images are an AP and lateral postoperative radiograph following fixation and bone grafting

started in physical therapy and transitioned to weightbearing in a boot. The boot is maintained for approximately 2–6 weeks depending on clinical and radiographic evidence of healing. Return to impact activity including running and jumping is typically initiated 4 months postoperatively.

Outcome following navicular stress fracture open reduction and fixation has been reported to be successful; however, nonunion rates have been reported as high as 20% [96]. In a case series of ten navicular stress fractures treated surgically with average follow-up of 42.4 months, eight out of ten went on to union. AOFAS and SF-36 scores in those patients were on average 92 compared to 74 in the 2 patients with nonunions. The nonunion cases were noted to be complete, displaced fractures, whereas partial navicular stress fractures achieved union. 50% Of displaced fractures reached union; of those that healed, autologous bone grafting was utilized [96].

Cuboid Stress Fractures

Introduction

Cuboid stress fractures are extremely rare. The cuboid is located in the lateral column of the midfoot and is not typically a weight-bearing bone. The entire midfoot bears approximately 8% of the body weight [97]. In a study involving 250 military recruits, there were 11 cases of cuboid stress fractures or ~4% of reported stress fractures [98]. However, stress fractures of the cuboid are usually even more rarely encountered with only 1 stress fracture of the cuboid reported in a series of 113 stress fractures in soldiers [99] and a single cuboid stress fracture among 1338 stress fractures in military recruits at Fort Dix [100]. Among athletes, there have only been a few case reports of cuboid stress fractures in the literature [101–105].

Pathophysiology

The etiology of cuboid stress fractures is not clearly elucidated. It has been postulated that a *nut-in-the-Nutcracker* phenomenon leads to cuboid stress fractures. This phenomenon involves repeated plantarflexion which leads to compression of the cuboid between the calcaneus and fourth/fifth metatarsals [106]. The peroneal longus tendon is also thought to play a role in cuboid stress fractures as it passes through the peroneal groove of the cuboid [107]. As the tendon contracts in the groove on the plantar aspect of the bone, it may concentrate excessive forces in this location during high-stress activities such as running [107, 108]. Cuboid stress fractures have also been associated with abnormalities and/or ruptures of the plantar fascia which may place excessive forces on the peroneus longus tendon [108]. Malalignment of the foot especially pronated or a cavus foot could also predispose to cuboid stress fractures. A pronated foot compresses the cuboid in between the calcaneus and the cuboid, while a cavus foot leads to increased weight-bearing on the lateral column leading to stress fractures [109].

Diagnosis

Imaging

Imaging of stress fractures begins with plain films which are usually negative early on with a sensitivity of 15–35% on initial examination that increases to 30–70% over 2–3 weeks [103]. Stress fractures of the cuboid are usually radiographically occult making MRI the mainstay for evaluation which would demonstrate bone marrow edema and occasionally fracture lines [4] (Fig. 19.7). CT and Technetium-99 m-labeled diphosphonate bone scans can be useful to diagnose occult cuboid stress fractures.

Treatment

Given the low incidence of cuboid stress fractures, there is no strong clinical evidence regarding their management in athletes. There are seven case reports of cuboid stress fractures in athletes reported in the literature (Table 19.1). Each case was treated conservatively with activity modification and/or immobilization with recovery and return to play in each case. Nonoperative man-

agement should include partial weightbearing +/− immobilization for 2–6 weeks followed by progressive return to low-impact activities. Return to play is possible once the athlete can participate in activities without pain. There is one report in the podiatric literature of two cuboid stress fractures that involved percutaneous injection of a calcium phosphate material in the cuboid for treatment in a patient with refractory pain (Fig. 19.6 – add on) [110].

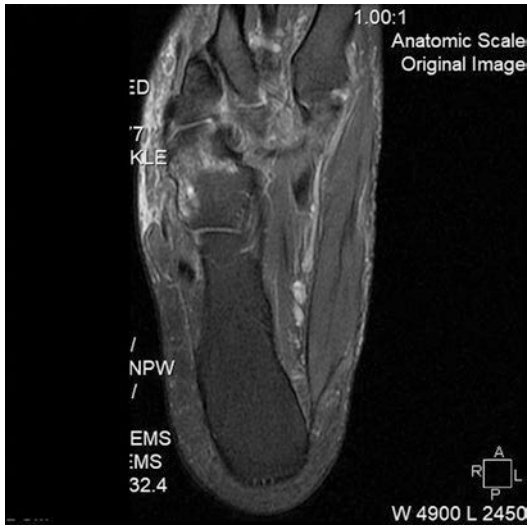

Fig. 19.7 T-2 weighted MRI demonstrating a cuboid stress fracture or reaction. This patient had normal, unremarkable radiographs. Increased T2 signal within the cuboid. Fracture line not well visualized. (Image courtesy of Daniel Ocel, Cornerstone Orthopaedics, Superior, CO)

Cuneiform Stress Fractures

Introduction

Cuneiform stress fractures are extremely rare. It was first described in 1936 as a "march fracture" of the first cuneiform associated with a second and fifth metatarsal fracture [117]. Since that time, there have only been a few case reports of cuneiform stress fractures in the literature [118–122].

Pathophysiology

Due to their location in the midfoot, the cuneiforms are subjected to bending and compressive forces that are responsible for the development of stress fractures [90, 118]. A significant proportion of body weight passes through the cuneiforms especially during the propulsion state of running, placing sprinters and runners at risk for stress fractures [123]. Similar to cuboid stress fractures, the plantar fascia is also theorized to play an important role in cuneiform stress fractures, likely secondary to increased lateral column stress [117]. It is felt that abnormalities of the plantar fascia can destabilize the lateral column resulting in these injuries; yet, cases of cuneiform and cuboid stress fractures have not been reported following plantar fascia release.

Table 19.1 Reported cases of cuboid bone stress fractures in athletes

Study	Age/gender	Sport	Duration of prodromal symptoms	Treatment
Mahler et al. [111]	20 years old/female	Gymnast	1 week	4 weeks Non-weight-bearing
Beaman et al. [112]	22 years old/male	Track and field	2 weeks	Plaster cast
Chen [107]	20 years old/female	Soldier (distance running)	1 week	Rest
Matsumoto et al. [113]	16 years old/male	Football	1 week	Brace
Battaglia et al. [114]	17 years old/male	Handball	4 weeks	Plaster cast
Kawahara et al. [115]	17 years old/male	High jump	3 weeks	Splint
Hagino et al. [116]	22 years old/female	Rugby	1 month	Brace

Diagnosis

Imaging

Evaluation of cuneiform stress fractures begins with AP, lateral, and oblique plain films. Similar to the cuboid, the cuneiform does not have a diaphysis and, therefore, is not typically characterized by periosteal callus formation [117].

Advanced imaging in the form of MRI, CT, or Technetium-99 m-labeled diphosphonate bone scans are often utilized if a suspected stress fracture is not visible on plain films. MRI is the imaging modality of choice. MRI would demonstrate bone marrow edema and low-signal intensity fracture lines [4].

Treatment

Nonoperative management is typically effective for cuneiform stress fractures. This includes initial partial weight-bearing on the affected extremity for 2–6 weeks with or without immobilization in a splint or cast followed by progression to low-impact activities [123]. Return to full activities can occur once the patient can perform low-impact activities without pain [123].

Sesamoid Stress Fractures

Introduction

Stress fractures of the hallux sesamoids are uncommon but challenging injuries that occur most often in running and jumping athletes [4, 123]. There are two hallux sesamoids: the medial or tibial and the lateral or fibular hallux sesamoid. The two hallux sesamoids are positioned deep to the head of the first metatarsal within the tendon of the flexor hallucis brevis. The tibial sesamoid is larger, longer, and lies within the medial head of the flexor hallucis brevis, while the smaller and rounder lateral sesamoid can be found within the lateral head and is positioned more proximal in relation to the medial sesamoid [124]. Portions of the adductor hallucis (medially) and the adductor hallucis (laterally) insert onto the sesamoids which are held together securely by the intersesamoid ligament [125]. Ossification of the sesamoids typically occurs between the ages of 7 and 10 years of age and are often in multiple areas of the sesamoid [125]. These ossification centers frequently fail to fuse leading to partitions also referred to as bipartite or multipartite sesamoids and can be misinterpreted as fractures. This occurs about ten times more common in the medial sesamoid with ranges reported between 10% and 33% and is often bilateral [126].

The sesamoids function to provide several important properties essential to the proper biomechanical function of the first metatarsal phalangeal joint (MTP). By elevating the first metatarsal head, the sesamoids increase the mechanical advantage of the flexor hallucis brevis tendons and provide protection to the flexor hallucis longus. Excision of the tibial sesamoid has been shown to correlate with a 10% loss of hallux push-off power [127]. The sesamoids also function to off-load forces across the metatarsal head as occurs with dorsiflexion. The tibial sesamoid, the larger and more central position of the two, receives a greater degree of load transmission in the more commonly injured [4, 61]. Anatomic variants of the foot and/or ankle that can be risk factors for the development of a sesamoid stress fracture include a plantar-flexed first ray, cavus heal alignment, or tight heel cord.

Diagnosis

Clinical Presentation

Patients often present with pain that is well localized to the plantar aspect of the great toe and worsens with weight-bearing. The pain is usually insidious in onset, worsened by athletic activity and climbing stairs. There usually is no inciting or traumatic event. On exam, tenderness about the plantar–medial hallux MTP joint is related to a symptomatic tibial sesamoid, while fibular sesamoids have tenderness directly plantar. Pain is exacerbated by dorsiflexion of the first MTP. Swelling and erythema may be present.

Imaging

Baseline imaging includes a standing anteroposterior (AP), lateral, and oblique radiograph of the foot and should be obtained in all patients with a suspected sesamoid injury. Additional radiographic views of the sesamoids including axial, tangential, and oblique images can also be helpful in identifying and categorizing potential injuries. Anatomic variants including a congenital bipartite sesamoid do exist and need to be differentiated from acute injury. These are ten times more common in the tibial sesamoid and occur in 10–33% of the general population [4]. Whereas acute fractures tend to have more sharp linear fracture margins, those of congenital bipartite sesamoids tend to be smooth and rounded on plain radiographs. Advance imaging with MRI or Technetium-99 m-labeled diphosphonate bone scans is typically warranted in the setting of a suspected sesamoid stress fracture given its high degrees of sensitivity and specificity in making the diagnosis. Imaging findings may vary from normal X-rays and edema on MRI to displaced fractures with signs of sclerosis and cystic change visible on plain films.

Treatment

Nonoperative Treatment

Nonoperative treatment management is recommended for initial treatment of sesamoid stress fractures. Similar to other overuse injuries, treatment starts with activity cessation and modification for 2–6 months, nonsteroidal anti-inflammatory medications, physical therapy, and orthotics [128]. Weight-bearing is typically not restricted. There has been success utilizing immobilization in a boot or short leg cast for 6–8 weeks [129]. Short leg cast can be modified with a toe spica component which will further relieve stress on the sesamoids [63]. This is followed by a molded orthotic and footwear modification and gradual resumption of sports as tolerated [129]. Sesamoid stress fractures should be followed by serial radiographs especially in runners as there is a risk of gradual diastasis between sesamoid fragments that can lead to a cock-up deformity of the hallux [63].

Operative Treatment

Surgical management is indicated after 6 or more months of failed conservative management or functional loss. There are essentially three surgical treatment strategies that have been described for management of persistently symptomatic nonunion of sesamoid stress fractures: (1) sesamoidectomy, (2) bone grafting, and (3) internal fixation. For longitudinal fractures as well as comminuted fractures, sesamoidectomy is recommended. Internal fixation is recommended for displaced transverse fractures, while internal fixation or bone grafting can be utilized for nondisplaced transverse fractures [128].

Sesamoidectomy

Total or partial sesamoidectomy has been used for the management of sesamoid stress fractures. Sesamoidectomy was first used successfully for the treatment of sesamoid stress fractures in 1982. Their report describes four patients (two medial and two lateral) who presented with persistently symptomatic stress fractures. All four patients were able to resume athletic activities without residual symptoms. Tibia sesamoidectomy can potentially lead to the loss of push-off strength of up to 10% which to the casual athlete my not be appreciated; however, it could be significant in runners. Bone grafting procedures have been advocated to avoid tibial hallux sesamoidectomies; however, in the setting of comminution, articular disease, or diastasis, this may not be feasible. Abductor hallucis tendon transfers have been utilized in an effort to minimize this loss of push-off strength [63]. Other concerns are the potential development of first MTP arthritis, hallux valgus, or hallux varus depending on which sesamoid was removed [130]. In athletes with hallux valgus deformity preoperatively, concomitant correction should be considered.

Bone Grafting

Anderson and McByrde described a bone grafting procedure primarily for athletes with nonunions of tibial sesamoid stress fractures in an effort to avoid loss of push-off strength [131]. Twenty-four patients underwent autogenous cancellous bone grafting after failure of conservative

management of minimally displaced (3 mm or less) tibial sesamoid nonunions. Twenty-one of 24 patients were evaluated at follow-up. In 19 cases, the stress fracture healed radiographically, and 17 patients had returned to their previous level of athletic activity. Subsequent studies have shown good results in tibial stress fracture nonunions with less than 2 mm of diastasis and no gross motion between the two fragments. Other contraindications include cystic fragmentation of the sesamoid, avascular necrosis, disproportionately sized fragments, and chondromalacia or frank arthritis of the sesamoid–metatarsal articulation.

Internal Fixation

Internal fixation is utilized for displaced or nondisplaced sesamoid stress fractures. Internal fixation can either be performed percutaneously through a stab incision or via open reduction through a medial incision followed by cannulated compression screw or suture fixation. Blundell et al. described percutaneous screw fixation in nine patients who had failed conservative management [132]. At 6 months after surgery, all nine patients had returned to their previous level of athletic activity with no reported complications. Subsequent studies have demonstrated similar good results of compression screw fixation [133, 134].

Return to Play

A recent systematic review of the literature analyzed the success of both conservative and surgical treatment of sesamoid stress fractures in athletes [135]. Fourteen publications were found, 7 prospective cohort studies, 4 retrospective cohort, and 3 case series. There were no randomized controlled trials. There were 195 fractures. Follow-up data were available for 168 fractures. Twenty-two stress fractures were managed conservatively vs 146 surgically after failed conservative management (mean 22 weeks, range 4 weeks to 1 year). Sesamoidectomy (*n*–99) was the most commonly used followed by bone graft ($n = 20$), internal fixation ($n = 21$), and partial sesamoidectomy. The return rates for each treatment modality can be found in Table 19.2. Sesamoidectomy was associated with faster return to sport, 10.5 weeks compared to 11.8 weeks for internal fixation ($p < 0.001$) and 13.9 weeks for athletes managed conservatively ($p < 0.017$). Internal fixation was associated with improved rates of return to sport, 100% compared to 96% for sesamoidectomy and 86% for athletes managed conservatively. Athletes who underwent internal fixation were also found to have higher rates of return to preinjury-level sport, 100% compared to 86% for sesamoidectomy and 64% for conservative management.

Summary

Stress fractures of the midfoot and forefoot are common injuries in athletes. Delays in diagnosis are common and often lead to prolonged pain, disability, and loss of time in sport. Treatment of these injuries is dependent on the location of the fracture and demands of the patient. First through fourth metatarsal stress fractures are successfully managed with protected weight-bearing, while surgical management is often recommended for stress fractures of the fifth metatarsal especially for the elite-level athlete. Navicular stress fractures can be managed conservatively initially;

Table 19.2 Return rates and return times to sport by treatment modality [135]

Mode of treatment	*n* (total)	Return rates to sport	Mean return times to sport	Return rate to pre-injury level
All	168	158/168 (94%)	11.4 weeks	114/135 (84%)
Conservative	22	19/22 (86%)	13.9 weeks	14/22 (64%)
Surgical	146	139/146 (95%)	11.0 weeks	100/113 (88%)
Sesamoidectomy	99	95/99 (96%)	10.5 weeks	57/66 (86%)
Partial sesamoidectomy	6	6/6 (100%)	14.7 weeks	5/6 (83%)
Internal fixation	21	21/21 (100%)	11.8 weeks	21/21 (100%)

however, patients must be counseled on the ~11% refracture rate. Surgical management is advocated for elite athletes with navicular stress fractures. Cuboid and cuneiform stress fractures are difficult to diagnose and rarely encountered; however, they commonly heal after a period of immobilization. Sesamoid stress fractures are initially managed conservatively with surgery indicated after of failed conservative management. Overall, early diagnosis and appropriate treatment of stress fractures of the mid- and forefoot are key to produce acceptable outcomes.

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Correction to: Stress Fractures of the Pelvis and Sacrum

Burak Altintas, Timothy L. Miller, and Mary Lloyd Ireland

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The spelling of the author name was inadvertently published as Burak Alintas in the Table of Contents and Chapter 15.

This has now been amended throughout the book as Burak Altintas.

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