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Managing Bone Stress Injuries in Athletes

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Clinical Vignette

N.R. is a 19-year-old freshman female collegiate cross-country runner without significant previous medical history or injury who presented for evaluation of right anterior shin pain that began three weeks ago. Initially, she noticed discomfort towards the end of her runs, but her symptoms progressed to the point where she could only run for 5-10 minutes before stopping. She was two months into her fall cross-country season and had increased her mileage to 80-90 miles/week. She reported losing about 10 pounds (7.5% previous total body weight) since starting college, which she attributed to an increase in running volume and denied intentional weight loss or dietary restriction. Menses began at age 13 and she reported 10 menses in the last 12 months, noting she missed her most recent period. On physical examination, the patient was a thin (body mass index 18.2 kg/m^2), well-appearing female. There was no visible bruising or swelling over the right lower extremity; however, there was a focal area of tenderness to percussion over the right medial aspect of the tibia at the midshaft. She had full strength and normal sensation throughout her lower extremities. Hopping on the right leg reproduced her symptoms. Standard radiographs of the right lower leg were normal. She was referred to the UPMC Physical Medicine and Rehabilitation Sports Medicine Clinic for help in treating this condition.

Defining the Problem

Bone stress injuries (BSIs) are overuse injuries that occur when repetitive submaximal mechanical loads exceed bone remodeling capacity.[1] BSIs are described on a continuum beginning with stress reactions where imaging may show periosteal or bone marrow edema and progressing to stress fractures, where a clear fracture line is seen. Stress fractures are characterized by their

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Disclosures

Drs. Bean and Fultz report no relationships with proprietary entities producing health care goods and services.

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occurrence in otherwise normal bone, differentiating them from pathologic fractures that occur at a site of disease (e.g., tumors, cysts, infection). Insufficiency fractures are stress fractures that result from overloading bone that is globally weakened due to an ongoing systemic process (e.g., osteoporosis, osteomalacia, osteogenesis imperfecta).

Risk Factors

BSIs most commonly occur in individuals participating in activities that require repetitive loading such as long-distance running or military training. The overall incidence of stress fractures has been estimated to be approximately 6.5% in male athletes, and 9.7% in female athletes.[2] Collegiate cross-country runners are at particularly high-risk of developing a BSI, with an annual incidence of up to 20% reported in one prospective study.[3] The vast majority of BSIs occur in the lower extremities, with the tibia being the most common location of injury[4], though sports requiring repetitive overhead or upper extremity movements may also place athletes at risk of upper extremity BSI. Common sport-specific locations of injury are shown in Table 1.

Table 1: Common BSI Location by Sport

Location	Sports
Tibia	Running, Basketball, Volleyball, Gymnastics, Ballet, Track & Field
Metatarsals	Running, Basketball, Volleyball, Gymnastics, Ballet
Lumbar Spine	Cricket, Tennis/Racquet Sports, Gymnastics, Ballet
Ribs	Rowing, Baseball, Tennis, Swimming
Olecranon	Baseball
Pelvis	Running/Endurance Sports, Soccer

Adapted from Beck and Drysdale, 2021[5]

Risk factors for BSI can be broadly divided into intrinsic or extrinsic factors (Table 2). The greatest predictor of developing a stress fracture is a history of prior stress fracture.[6] Up to 20% of stress fractures in high school and collegiate athletes were recurrent injuries.[7,8] Sudden increases and/or high training volumes are also commonly associated with developing BSI.[6] Both underweight (<19 kg/m^2 BMI) and overweight (>25 kg/m^2 BMI) individuals have also been shown to be at increased risk of BSI.[9,10] In addition to BMI, lower lean mass and higher fat mass also correlate with higher likelihood of BSI[11]. Medications such as glucocorticoids, antiepileptics and contraceptive agents have been shown to alter bone metabolism that may also increase an individual's risk of developing BSI.[11]

Table 2: Risk Factors for BSI

Intrinsic Factors	Extrinsic Factors
Prior stress fracture	Low Vitamin D intake
Female sex	Low energy availability or weight loss
Oligomenorrhea	Increased exercise patterns
Early/late onset of menarche	Biomechanical factors
Low or high BMI	Smoking
Low lean or high fat body mass	Alcohol consumption
Low bone mineral density	Medications

Adapted from Abbott et. al. 2020[12]

Females are at increased risk of developing BSI compared to males.[13] Recognition that several factors contribute to this higher risk led to the development of the term Female Athlete Triad. Female Athlete Triad is a metabolic injury that occurs when there is an imbalance between energy intake and energy expenditure. In 1993, Yeager et. al. defined Female Athlete Triad as the presence of three conditions: disordered eating, amenorrhea, and osteoporosis.[14] However, it has been increasingly recognized that increased risk of developing BSI does not require the presence of all three of the conditions. Female Athlete Triad is now instead recognized as a spectrum of three risk factors: low energy availability, low bone mineral density (BMD) and menstrual dysfunction.[15]

While females overall have greater risk of developing BSI compared to males, it has been increasingly recognized that some male athletes may also be at higher risk.[16] In 2014, the International Olympic Committee proposed a new term, "Relative Energy Deficiency in Sport," in order to acknowledge the effects of low energy availability on reproductive and bone health in both sexes in high-level athletes.[17] In 2018, the Female Athlete Triad Coalition was renamed to the Female and Male Athlete Triad Coalition. A recently published consensus statement was produced by this group describing Male Athlete Triad, a syndrome of three inter-related conditions: low energy availability, impaired bone health, and suppression of the hypothalamic-pituitary-gonadal (HPG) axis.[18]

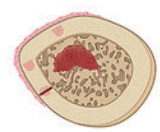
Initial Clinical Evaluation

The first step in diagnosis of a BSI is a complete history and physical examination. A thorough history should include questions regarding prior and current medical issues including history of BSI, age of onset of menses and frequency, training load, dietary intake, and medications. A family history of osteoporosis or other bone injury can be useful if present. Patients with BSI typically report progressive pain that worsens with activity and improves with rest. Physical examination often reveals tenderness to direct or indirect percussion, while bruising or edema are less common but may be present at more advanced stages.[19] A positive hop test, where the patient's pain is reproduced with jumping up and down on the injured leg, can be helpful in diagnosing lower extremity BSI.[20] The tuning fork test, where the examiner places a vibrating tuning fork at the presumed area of injury, has also been shown to elicit localized pain in the setting of stress fracture.[21] The fulcrum test can be utilized to localize a femoral shaft BSI. In this maneuver, the examiner places one arm under the symptomatic thigh, moving it proximal to distal, creating a fulcrum as the examiner's other hand presses down at the knee.

Imaging

Plain film radiographs are typically the first diagnostic test of choice for BSI due to high accessibility and low cost. However, radiographs have low sensitivity (~10%) in the early stages of injury leading to a high false negative rate.[22] Sensitivity increases to 30-70% after 3 weeks, with subtle cortical radiolucency often being the earliest radiographic finding.[23] If there is high suspicion for a BSI with negative radiographs, further imaging with magnetic resonance imaging (MRI) is recommended. MRI is considered the gold standard imaging modality for the assessment of bone stress injury, given its high sensitivity and specificity of up to 99% and 97%, respectively.[24] MRI can detect early signs of stress injury, such as periosteal and soft tissue edema prior to the development of a fracture line.[25] Nuclear scintigraphy was previously considered the gold standard for diagnosis of BSI, but is less commonly used now due to its lower sensitivity compared to MRI and high levels of radiation exposure. Computed tomography has lower sensitivity than MRI or nuclear scintigraphy, but its high specificity (98%) can help with confirming diagnoses if other imaging is equivocal.[24] Ultrasound has been increasingly utilized for musculoskeletal injuries and has the advantage of point-of-care imaging, but has a high false positive rate making it better as a screening test to rule out a stress fracture given a negative result.[24]

Table 3: MRI Grading Scales for BSI

Grade	Fredericson, et. al.[19]	Nattiv, et. al.[26]	Illustration
1	Mild/moderate periosteal edema on T2; Normal marrow on T1/T2	Mild marrow or periosteal edema on T2; Normal T1	
2	Moderate/severe periosteal edema on T2; Marrow edema on T2	Moderate marrow or periosteal edema on T2; Normal T1	
3	Moderate/severe periosteal edema on T2; Marrow edema on T1/T2	Severe marrow or periosteal edema on T2 and T1	
4	Moderate/severe periosteal edema on T2; Marrow edema on T1/T2; visible fracture line	Severe marrow or periosteal edema on T2 and T1; visible fracture line	

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A number of MRI classification systems for BSI have been proposed. The Fredericson Stress Injury Grading System was published in 1995 to grade severity of tibial bone stress injuries (Table 3).[19] More recently, Nattiv, et. al. developed a grading scale that could be applied to BSI in any anatomic region.[26] In both scales, a higher grade is indicative of a more advanced injury and predicts a longer period before full return to activity.

Diagnosing Female/Male Athlete Triad

Athletes are diagnosed with Female or Male Athlete Triad if they are identified as having one or more components along the Triad spectrum. Cumulative risk assessments have been developed to stratify female and male athletes into low, moderate, or high-risk categories based on their dietary, menstrual, bone mineral density and stress fracture history[15]. Individuals in the moderate- and high-risk categories have been found to be more likely to develop BSI.[27]

Diagnosing Low Energy Availability

Indications of low energy availability include a BMI <17.5 kg/m^2, <85% expected body weight for height in adolescents, or recent weight loss >10% total body weight.[15] Findings on examination such as tooth decay, parotid gland hypertrophy, or callus on the proximal phalangeal joints may be signs of an eating disorder.

Diagnosing Low BMD

Evaluation of BMD using dual energy x-ray absorptiometry (DXA) is recommended for individuals who are determined to be at high risk based on weight, presence or history of disordered eating or clinical eating disorder, menstrual history and stress fracture history.[28] Low BMD is defined as a Z-score < -1.0 in female athletes participating in weight bearing sports by the American College of Sports Medicine,[29] and the same cutoff is recommended for males by the Female and Male Athlete Triad Coalition. For females and males in non-weight bearing sports, a Z-score < -2.0 is recommended as a threshold for diagnosing low BMD.

Diagnosing Amenorrhea/Suppression of the HPG-axis

Female athletes reporting abnormal menses should undergo further evaluation to rule out pregnancy or endocrinopathies. Testing may include a complete blood count, thyroid function testing and sex hormone levels (e.g., luteinizing hormone, follicle stimulating hormone, prolactin, estradiol and/or progesterone challenge test, testosterone, and dehydroepiandrosterone). Pelvic ultrasound may be used to assess for potential causes of primary amenorrhea or diagnosis of hyperandrogenic conditions such as polycystic ovary syndrome or virilizing tumors.[15] Males should be screened for symptoms of hypogonadism such as decreased libido, loss of morning erections, or reduction in growth of facial hair. Small testicular size and sparse pubic hair can be suggestive of long-standing hypogonadism. If there is concern for hypogonadism, additional testing including thyroid stimulating hormone, as well as total and free testosterone can be considered.[28] Referral to endocrinology should be considered if the diagnosing practitioner is not experienced with diagnosis or management of amenorrhea or hypogonadism.

Management

Initial Management

Management of BSIs vary depending on grade and location. Low grade BSIs are managed conservatively with relative rest and reduced weight bearing as needed to minimize pain during activities of daily living. Patients with leg or rearfoot BSI can wear cushioned shoes/insoles to decrease forces through the injured bones, while those with mid- or forefoot BSI may consider wearing stiff-soled shoes. For individuals who cannot maintain a normal walking pattern without pain, crutches or a walking boot can be utilized to reduce weight bearing forces until increased weight bearing is tolerated. To maintain cardiovascular fitness, participation in reduced weight-bearing activities such as deep water running, swimming, or cycling are recommended, as long as the patient remains pain-free during and after activity. Tibial stress injuries should focus on strengthening of the plantar flexors, as these muscles produce the highest strain on the tibia during activity.[30] A graduated return to running program is typically recommended once the patient is able to perform ADLs with full weight bearing without increased pain for 5 days. Emphasis is maintained on a progression that does not provoke onset of symptoms.

Table 4: Risk Classification of Stress Fractures Based on Location

Low-Risk	High-Risk
Pars articularis of the spine	Femoral neck
Pelvis	Patella
Posteromedial tibia	Anterior tibial cortex
Fibula/lateral malleolus	Medial malleolus
Calcaneus	Talus
2nd-4th metatarsal shaft	Navicular
Ulnar shaft	Fifth metatarsal head
Calcaneus	Sesamoids
Ribs	
Upper extremity	

BSI can be classified as high- or low-risk based on anatomical location (Table 4).[31] High-risk regions are more likely to suffer from delayed or non-union, particularly if diagnosis is delayed, and typically require a longer time to full recovery.[32] The most common high risk fractures that may require surgical management include femoral neck and the anterior tibial cortex stress fractures.[4] Prior systematic reviews suggest higher return-to-sports rates with surgical management of anterior tibial stress fractures (96%) following failed initial treatment when compared to continued conservative management.[33] Follow-up radiographs are recommended for high-grade BSI, with full return to activities delayed until there is radiographic evidence of union.[34]

Several modalities have been utilized for treatment of BSI in addition to activity modification and strengthening. Pulsed ultrasound has been trialed, but a systematic review showed no evidence of improved outcomes or accelerated healing.[35] Extracorporeal shockwave therapy may aid in healing nonunion injuries; however, no randomized control trials have been published to date.[36] Anti-gravity treadmills, which can allow reducing forces up to 80% of body weight using a specialized air pressure control system, have been increasingly utilized in the rehabilitation process,[37] though no randomized control trials have been performed in individuals with bone stress injury.

Use of pharmacological therapies in the initial treatment of BSI is limited. Non-steroidal anti-inflammatory drugs are not recommended except in the acute phase (<7 days) for resting pain, as some studies have shown that prolonged use can impair bone healing.[38] Bisphosphonates, which are commonly used for treatment of osteoporosis in post-menopausal women, are not FDA approved for treatment of BSI. Any consideration of the use of bisphosphonates should involve consultation with an endocrinologist or bone metabolic disease specialist.[15] Vitamin D supplementation is recommended if 25-hydroxyvitamin D levels are less than 32 ng/mL.[28]

Management of Female/Male Athlete Triad

A multidisciplinary approach is important for management of athletes with BSI who are identified to have risk factors for Triad. The first step should be to address any energy imbalance in the athlete. Energy deficiencies can be normalized through modification of dietary intake and/or alterations in physical activity.[15] Increasing body weight to a BMI >18.5 kg/m^2 may correct underlying physiological imbalances including menstrual irregularities. Referral to a sports dietitian is recommended for individuals suspected to have low energy availability, and those identified to have either disordered eating patterns or a clinical eating disorder should be referred to mental health providers. Once energy availability is addressed, recent randomized clinical trials have found that transdermal combined oral contraceptives may be helpful for treating oligomenorrhea and low BMD.[39] There is currently insufficient evidence to recommend testosterone or other hormonal replacement in males with HPG-axis dysfunction.[28] However, endocrinology referral is recommended for individuals with low BMD and/or menstrual or HPG-axis dysfunction if pharmacological treatment is being considered.

Prevention

Screening for risk factors of Triad during pre-participation physicals is recommended to allow for early intervention and prevention of future BSI. Questionnaires have been developed for female[15] and male[28] athletes focusing on eating habits, medication, and bone health history. Positive screening questions should prompt a more thorough investigation into risk factors and any abnormalities should be addressed. While Vitamin D and/or calcium supplementation has been shown to reduce the incidence of stress fractures in athletes and military personnel,[40,41] there is no current consensus on whether athletes should be screened for Vitamin D deficiency in the absence of other risk factors. Additionally, there is no evidence that imaging is indicated in asymptomatic individuals, as early MRI findings do not appear to predict who will go on to develop symptoms in the future.[42]

Clinical Vignette Outcome

To further evaluate N.R.'s condition, MRI was ordered and showed increased intracortical signal and thickening of the posteromedial tibial diaphysis with periosteal and endosteal edema on T2 weighted images and normal appearing T1 weighted images, consistent with a Fredericson grade 2 bone stress injury. Additional laboratory workup revealed a negative pregnancy test as well as a 25-hydroxyvitamin D level of 42 ng/mL. N.R. was instructed to wear cushioned shoes and utilize axillary crutches to reduce weight bearing in order to ambulate without pain. She was encouraged to begin cross-training with deep water running to maintain cardiovascular fitness, which she was able to do without symptom provocation during and after activity. On re-evaluation two weeks after her initial presentation, she could ambulate and perform all ADLs without pain and began a supervised rehabilitation program. She started a return-to-running program that included training on an anti-gravity treadmill with gradual increase in body weight. She also met with her team's nutritionist who adjusted her diet to increase overall calorie intake. Six weeks after initial presentation, she was fully cleared to return to running and continued to train with her teammates throughout the winter track season without recurrence of pain.

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