STRESS FRACTURES IN ATHLETES

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INTRODUCTION

Stress fractures represent 20% of all injuries treated in sports medicine. Stress-related injuries can lead to discomfort, reduced performance, lost training time, and medical expenses. Early diagnosis and adequate management are the keys to avoid complications such as progression to complete fracture, chronic pain, prolonged recovery, or disability (1). The incidence of stress-related injuries has increase over the past few years in both, amateur and competitive athletes. Furthermore, women are at increased risk for stress fractures. Wentz et al showed that the incidence of stress fractures was approximately 9.2% in female military recruits compared to 3% in male military recruits, and 9.7% in female athletes as opposed to 6.5% in male athletes (2). The combination of energy deficiency, amenorrheic state, and osteoporosis constitute the female athlete triad, which is a significant risk factor for stress fractures. The most commonly affected bone is the tibia (49.1%), followed by tarsal bones (25.3%), metatarsals (8.8%), fibula (6.6%) and sesamoid bone (0.9%) (3).

PHYSIOPATHOLOGY

According to Wolff's law, bone grows and remodels in response to the mechanical stress. A rapid increase in the intensity, duration, or frequency of physical activity without sufficient rest may lead to pathologic alterations in the bone. In fact, when the balance between formation and resorption is disturbed, bone weakens and becomes more susceptible to micro fracture (3). The histological analysis of bone stress injury done by Johnson et al showed that early discontinuation of repetitive stress prevented the progression to a fracture (4, 5).

ETIOLOGY

Stress fractures are generally separated in two different categories based on their etiology, fatigue fracture and insufficiency fracture. Fatigue fractures are a result of increase stress applied to a normal bone. Although often seen in athletes and military recruits, the general population is susceptible when undergoing an increase in activity (6). The stress applied is greater than the body's reparative process, which results in the breakdown of osseous structures. Athletes should progress activity levels gradually, which means no more than a 10% increase in load per week (7). Insufficiency fractures happen when the bone is weakened by a systemic or direct process that leads to bone loss and subsequent fracture with normal weightbearing. The best example is lack of weightbearing after surgery, which can result in atrophy and high risk for fracture at rehabilitation. Systemic processes include osteomalacia, hyperparathyroidism, osteoporosis, steroid use, and rheumatoid arthritis.

DIAGNOSIS

The patient with a stress fracture will often complain of immediate onset of limb pain that occurs at the end of the physical activity. It is also often associated with a significant change in training regimen in the preceding weeks. As the microfractures progress to stress fracture, the symptoms may persist even during ambulation. On physical examination, local tenderness and swelling are often noticed over the involved bone. Clinical tests useful in diagnosis include the hop test, hyperextension test, and fulcrum test.

The most useful diagnostic modality for initial assessment of stress fracture is plain radiography. In stress fractures that involve cortical bone, early findings may involve poor definition of the cortex or fine radiolucency. Later findings include periosteal new bone formation or sclerosis. If the stress fracture involves cancellous bone, it generally appears as a band of sclerosis oriented perpendicular to the trabeculae (8). It is important to keep in mind that their sensitivity is only 10% in the early stages of the injury. In the case of negative radiographs and the presence of concerning clinical findings, repeat the radiographs two weeks after initial imaging. If urgent diagnosis is needed, other diagnostic studies should be performed like bone scan and magnetic resonance imaging (MRI).

Radionuclide bone scans can detect stress fracture as early as 2 to 8 days after the first clinical symptoms. However, use is limited by a low specificity and the lack of visualization of the fracture line (9). In fact, focal tracer uptake can be the result of any processes that remodel bone such as tumor, infection, or stress reaction without fracture. MRI has become indispensable for evaluation of stress fracture that cannot be diagnosed with standard radiographs. Fluid-sensitive sequences are highly sensitive for endosteal marrow edema and periosteal edema, which are generally one of the

earliest features (Figure 1), A hypointense line through the cortex or medullary cavity can also be visible on MRI. MRI is at least as sensitive and more specific than bone scan in detection of stress fracture (8). Computed tomography (CT) is not frequently indicated for initial evaluation of suspected stress fractures because it has a greater radiation dose than radiography and lower sensitivity than MRI.

CLASSIFICATION

Stress fractures are described as high or low risk. This classification is important in the management of such injuries. High risk fractures include those likely to progress to complete fracture, delayed union, or nonunion; those are the ones that most likely will require surgical repair and to be non- or partial-weightbearing. The most detrimental fractures involving the lower extremity include the femoral neck, patella, and anterior tibia. Stress fractures in the talar neck, navicular, sesamoids, and fifth metatarsal are most debilitating in the foot. Those fractures occur on the tension side of the biomechanical axis. Low risk fractures respond well to conservative treatment, and include the femoral shaft, the medial tibia, fibula, the calcaneus, and the first through the fourth metatarsals.

PREVENTION

Education and prevention are the best management approachs. Medications that have been used to manage osteoporosis are also used to prevent stress fractures. Diphosphonates, intravenous pamidronate, and risedronate are most commonly prescribed (10). Daily intake of milk, calcium, vitamin D, and dairy products are related to a decreased rate of stress fractures. Nieves et al found that the strongest protection was from skim milk. Every cup of skim milk was associated with a 62% reduced fracture risk. An evaluation of diet patterns showed that individuals with high dairy and low fat intake had reduced risk for stress fractures (8). More research is being done on the use of anabolic agents for fracture prevention and healing. Also more studies need to be done to determine the role of mesenchymal stem cells, platelet-rich plasma, and bone morphogenetic protein with stress fracture healing (10).

GENERAL TREATMENT PRINCIPLES

A complete metabolic panel, including serum calcium, albumin, alkaline phosphatase, and serum vitamin D levels, as well as a calculated glomerular filtration rate, thyroid function, and complete blood count should be ordered for all patients with stress fractures. Hormonal levels should be assessed if the patient has a history of amenorrhea, oligomenorrhea, sexual dysfunction, or certain endocrinopathies. In addition,

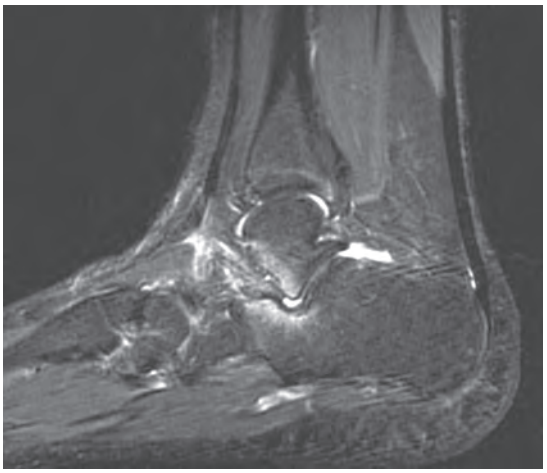

Figure 1. Magnetic resonance image showing a stress reaction of the calcaneus and the talus.

bone densitometry can be ordered for patients with a history of multiple stress fractures.

If laboratory workup reveals low levels of calcium, vitamin D, or phosphorus, underlying causes should be investigated and, at a minimum, a strict regimen of supplementation should be instituted. The recommended daily allowances of calcium and vitamin D are 1,000 mg and 800-1,000 IU, respectively. However, debate exists regarding whether this amount is too low for athletes. Shindle et al recommend 1,200 to 1,500 mg/day of calcium and 800 to 3,000 IU/day of vitamin D (10, 11).

Andrish described a 2-phase protocol for the treatment of low risk stress fracture. Phase 1 is initiated with pain control with different modalities including ice, physical therapy, and oral analgesic medications (12). The use of nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided at least during the first weeks after a stress fracture due to the potential adverse effect on bone healing (13). Ziltener et al showed that the harmful effects of NSAIDs vary depending on the substance and treatment duration. A delay in bone consolidation has been described multiple times in the literature with the use of NSAIDs (14-16). However, use of NSAIDS is not really justified in the case of a stress fracture, analgesics should be sufficient to manage the pain (13).

Sports should be discontinued and the patient should be weightbearing as tolerated. Cam walkers are recommended for patients who are unable to ambulate without pain. Minimal-impact aerobic activities such as cycling and swimming can help maintain cardiovascular fitness. When the athlete is pain free for 10-14 days, phase 2 begins. At first, it is recommended to run only every other day for the first 2 weeks and gradually increase to the preinjury level over 3 to 6 weeks. The progression of the sport should be dictated by the patient's pain level. The use of pneumatic braces may speed the healing of tibial stress fractures (16).

High risk stress fractures require different management. Conservative treatment including weightbearing restriction and immobilization is usually sufficient for Grade 1 and 2 (17). It is recommended to wait until complete healing before the athlete can return to sports to prevent progression to a full fracture. The site of the fracture, a high grade of fracture, and competitive participation will help determine whether surgery is the treatment of choice.

ADJUNCTIVE TREATMENTS

Orthotics were thought to prevent stress fractures by absorbing shock and by changing the biomechanics of the lower extremity. The use of this device remains controversial (1). The antigravity treadmill is a new technology for the management of stress fracture. It provides adjustable body weight support and allows the patient to perform exercise in a hypogravity environment. It actually allows safe training for the athlete during the healing phase. However, further investigations are still needed to determine the physiologic effect of hypogravity environment in healing bone injury and its effect on clinical outcomes (10). Bone stimulation may speed the recovery from a stress fracture. In a study done by Saxena, athletes using bone stimulators returned to activity in 8.8 weeks versus 17.6 weeks (18). Extracorporeal shock wave therapy (ESWT) has proved to be effective for fracture healing in animals by inducing microfractures to the trabeculae. Additional studies are necessary to determine its effectiveness and setting for optimal results (10).

SUMMARY

Stress fractures are common injuries among athletes but also in the general population. Etiologies and treatment plans are multifactorial. Conservative management is the gold standard for low risk stress fractures. High risk fractures may require more aggressive treatments. Early diagnosis and adequate management are the keys to avoid complications such as progression to complete fracture, chronic pain, prolonged recovery, or disability.

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