



ACSM Sports Medicine Basics

STRESS FRACTURES



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Incidence

Stress fractures and other bone stress injuries comprise up to one-sixth of all athletic injuries and have been reported in virtually all bones of the body. Athletes most at risk of stress fracture are runners, jumpers, gymnasts and dancers, for whom the lower extremity is most commonly affected. Vertebral stress fractures occur in cricket, fast bowlers and gymnasts, while upper extremity stress fractures are typically reported in swimmers, rowers and racquet sport players. Lower extremity stress fracture incidence among military recruits is slightly higher than in athletes, with particularly high rates reported for females.

Etiology

Exercise-related loading causes a degree of deformation (strain) in bone. Bone exhibits an intrinsic ability to adapt to changes in patterns of strain (magnitude or type) in order to minimize excessive deformation that may cause microdamage. Bone also has the capacity to repair itself. Ironically, for some the processes of adaptation and repair may transiently increase the risk of stress fracture, as described below.

Bone adaptation occurs via increased modeling and/or remodeling. Modeling is a response to chronically increased or decreased loading when bone tissue is either deposited or removed in order to modify the shape and/or size of a bone. The addition of bone tissue around the perimeter of a long bone shaft will increase its resistance to bending and is therefore an adaptive response to exercise that is likely to reduce the risk of stress fractures. Remodeling involves bone resorption followed, after a delay, by deposition of replacement bone. The remodeling process occurs continuously throughout life in order to mobilize calcium for numerous essential physiological processes. Remodeling is **upregulated** under a variety of metabolic conditions, following exposure to certain drugs, and in response to load-induced microdamage. Resorption (by osteoclasts) acts to remove damaged bone, after which osteoblasts deposit replacement bone. This “self-healing” characteristic of bone is a remarkable phenomenon that maintains the integrity of the skeleton, but its success can be compromised by excessive loading during the resorption phase. That is, the initial increase in porosity from increased resorption

will render a bone temporarily weaker until replacement bone can be deposited and mineralized. Untimely loading during this window of reduced bone strength may lead to more microdamage and the coalescence of damage into one or more stress fractures.

Risk Factors

Many factors have been purported to contribute to the risk of stress fracture, but quality evidence to support those claims is often lacking. The best available evidence suggests that a change in training, low fitness and low muscle strength will increase the risk of stress fracture. There is conflicting evidence that female sex, low bone mass, low serum vitamin D, and narrow bones predispose to stress fracture. While anecdotal reports or findings from lower quality studies have suggested age, race, pes planus (flat feet), pes cavus (high arch), genu valgus (knock knees), leg length inequality, tall thin body type, reduced flexibility, inadequate calories, inadequate calcium, previous injury, alcohol, smoking and corticosteroids are all associated with increased risk of stress fracture, no robust human data exists to support those claims.



Diagnosis

Positive symptoms of stress fracture include local tenderness, pain with loading, pain with direct or indirect percussion, and the presence of night pain. Confirmation of clinical diagnoses with imaging can be fraught due to false positives and negatives, however triple phase technetium⁹⁹ bone scans are generally highly sensitive to stress fracture. Magnetic resonance imaging (MRI) is now considered to provide the best impression of injury severity due to the ability to differentiate periosteal and marrow edema; the latter being indicative of greater severity. Plain radiographs and CTs are inadequately sensitive for the purposes of diagnosis. Ultrasound and tuning forks are similarly inadequate diagnostic modalities for stress fracture. Generally, historical symptoms and physical signs provide adequate bases for diagnosis. Imaging signs can also be misleading in terms of recovery; therefore the clinical resolution of symptoms provides the best guidance for return to training.

Management Recommendations

As stress fractures are typically a consequence of the normal adaptive or restorative response of bone to modified loading, unloading (rest) and graduated return to training is the most effective method of treatment in most circumstances. Recovery times vary according to site with small bones healing more quickly than large, and some locations being particularly problematic (e.g. anterior tibia, superior femoral neck). As there is some evidence that non-steroidal anti-inflammatory drugs (NSAIDs) and even acetaminophen are disruptive to bone healing, pain medications should be avoided when possible. Ice,

stretching, taping and massage will not enhance the rate of healing. Prevention is undoubtedly the best management approach.

Recommendations to minimize the risk of stress fracture and promote recovery include the following:

Before beginning or substantially modifying training:

1. Ensure old injuries have been fully rehabilitated (particularly if the individual has been immobilized for any length of time)
2. Enhance muscle strength around chronically loaded bones
3. Ensure adequate nutrition, calcium and vitamin D

During training:

1. Increase training intensity or alterations gradually over a period of four to six weeks, depending on the nature of the training
2. Ensure correct technique to minimize bone impact
3. Run initially on very uniform surfaces, such as level, short grass or asphalt. Progress to longer grass, soft soil, sand and uneven terrain, thereafter varying the training surface.
4. Maintain a minimum of 1000 mg/day dietary calcium and 800 IU/day vitamin D

When injured, do:

1. Rest from pain-provoking activities at the first sign of bone discomfort, and resume offending activity only when pain-free with loading
2. Maintain aerobic fitness with exercise that does not overload the injured bone

3. Resume training gradually, restarting at lower intensity than when injured
4. Use pneumatic braces for long bones as they may be helpful during return to training

When injured, do not:

1. Completely immobilize the injured site
2. Engage in activities that generate pain at the injured site
3. Engage in heavy resistance training of the injured site
4. Take pain medications if avoidable

New Directions

Preliminary evidence suggests that low intensity pulsed ultrasound may enhance stress fracture healing. Electrical or electromagnetic stimulation may also be helpful, but more data is needed. The benefit of training in bare feet or minimal shoes requires further investigation.

Summary

Stress fractures are a recognized complication of chronic, intensive athletic and military training. Bones are most susceptible to stress fracture when weakened by remodeling-related porosity – a primary stage in the adaptive or restorative response of bone to changes in patterns of loading or microdamage. Prevention is the most appropriate management approach, best achieved through graduated training increments to maximize muscle strength and general fitness. Adequate nutrition, including calcium and vitamin D is also important. While bone scans and MRI can be used to detect stress fracture, clinical diagnosis is normally adequate. The goal of stress fracture treatment is to temporarily reduce loads on the injured site to prevent further microdamage and allow the bone tissue to remodel. Thus, rest from pain-provoking activities remains the most effective, if often prolonged, intervention approach. Clinical resolution of symptoms is the most reliable guide for timing of return to training. Return to full training intensity must be highly graduated.

About the Author

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