Stress Fractures in Runners

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KEYWORDS

- Stress fracture • Running injuries
- Overuse injuries • Lower limb

Running has many beneficial effects, including cardiovascular and skeletal health. Poor training technique and a variety of risk factors may predispose runners to lower-limb overuse injuries affecting muscle, tendon, and bone. Injuries to the bone include stress reactions to full-fledged stress fractures. This article is designed to provide an understanding of the general concepts involving bone strain, bone injury risk factor assessment, and evaluation and treatment strategies for the runner with a stress fracture. The second half of the article presents more detail regarding each specific fracture location, grouped as high risk and low risk, common to runners.

PATHOPHYSIOLOGY OF STRESS FRACTURES

An understanding of stress fractures requires an understanding of basic bone biology and the general response of bone remodeling to applied stress. There are two subtypes of bone. Cortical (compact) bone is located in the diaphysis of long bones and the shell of square bones (vertebral bodies and tarsals). Cortical bony turnover is much slower and most stress fractures occur in cortical bone. Cancellous (trabecular) bone is located in the metaphysis and epiphysis of long and square bones. More active remodeling occurs in cancellous bone. Bone mineral density (BMD) measurements are taken of cancellous bone (vertebral body and femoral neck) because of the increased rate of turnover; thus changes in BMD are more readily observed. Stress fractures of cancellous bone correlate with low BMD more so than fractures of cortical bone; therefore, any runner with a cancellous bone stress fracture should have a BMD evaluation.[1]

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The underlying principle of bone's response to stress is Wolff's law, which states that every change in the form and function of bone leads to changes in its internal architecture and its external form. Simplistically stated, bone is an active substance that adapts to the loads it is placed under. The response of bone to repetitive stress is increased osteoclastic activity over osteoblastic new bone formation, which results in a temporary weakening of bone. The eventual adaptive response is periosteal new bone formation to provide reinforcement. However, if physical stress continues, the osteoclastic activity may predominate resulting initially in microfractures (commonly seen as bone marrow edema on MRI, consistent with a stress reaction) and eventually a true cortical break (stress fracture) may result. Thus, there is a continuum of normal bone strain leading to appropriate remodeling, but if strain becomes excessive or adequate rest is not allowed, stress reaction and eventually stress fracture can result.

It is important to delineate the differences between fatigue and insufficiency type stress fractures. Fatigue fractures are the typical overuse stress fractures observed in athletes and military recruits with normal bone density. Fatigue fractures result from an imbalance in the bone's ability to keep up with skeletal repair from excessive bone strain with progressive accumulation of microdamage. An insufficiency fracture is seen in those with low BMD, such as runners with the female athlete triad; metabolic bone disease; or osteoporosis. Insufficiency fractures result from poor bone remodeling (increased resorption and depressed formation) in response to normal strain. Simply stated, fatigue fractures occur in normal bone under excessive or abnormal strain and insufficiency fractures occur in abnormal bone under normal strain.

RISK FACTORS

Defining the causative risk factors for stress fracture is difficult because there are many interrelated variables that make a risk assessment problematic to independently study. Most studies are case series and many pertain to general overuse injuries in runners, which are not necessarily specifically focused on bone injury. However, these risk factors can be subdivided into extrinsic and intrinsic factors.

Extrinsic Factors

Training variables in runners commonly predispose to stress fractures. An increase in frequency, duration, or intensity of runs is often cited as a primary risk factor. Hard or cambered training surfaces are also factors associated with lower-limb overuse injuries.[2] In a small study, Milgrom demonstrated that treadmill running produced significantly less in vivo tibial strain than over-ground running suggesting treadmill runners are at lower risk for tibial stress fractures.[3] Failure to schedule rest days after higher intensity runs can also contribute to overuse injury risk. Periodization of training is an important coaching technique that maximizes performance gains but also decreases injury risk. Periodization in run training generally follows a 3- to 4-week cycle, with 3 weeks of a progressive buildup of intensity, duration, or distance, followed by an off week of less intense training to allow for rest and the subsequent metabolic training adaptations to occur before entering the next buildup period.

Training in shoes older than 6 months is a risk factor for stress fracture,[4] which is likely related to the decrement in shock absorption as a shoe ages. There is data demonstrating that shock-absorbing foot orthoses decrease the risk for stress fracture in military recruits, though this doesn't necessarily translate to the running athlete.[5,6] A general rule of thumb that most coaches and trainers suggest is changing running shoes every 300 to 500 miles logged to limit excessive risk for lower-limb overuse injuries.