Tibial Stress Injuries in Athletes

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Tibial stress injuries account for up to 75% of cases of lower extremity pain in athletes.[1,2] The spectrum of tibial stress injuries includes stress-induced periosteal and marrow reaction provoked by increased muscle and fascial tension (also known as medial tibial stress syndrome), stress fractures, and, rarely, displaced fractures. Stress or fatigue injuries occur with repetitive, strenuous activities, often in athletes who suddenly increase the intensity or duration of their training.[1,2] Activity-related pain aggravated by resisted plantar flexion that resolves with rest, localized tenderness, and occasional soft-tissue swelling are the typical presenting symptoms, commonly termed shin splints. Distinguishing tibial stress fractures from medial tibial stress syndrome is clinically challenging, but necessary, as the entities have different treatments and clinical courses.

Where stress fractures occur within the tibia depends on the inciting activity. Recreational and competitive runners have a higher incidence of fractures within the proximal posteromedial cortex, whereas ballet dancers and athletes whose sports involve jumping (eg, basketball) more often sustain fatigue fractures within the anterior cortex. Anterior tibial cortex stress fractures are considered higher risk injuries, as they are prone to delayed union or nonunion, particularly when diagnosis is delayed. More than one stress fracture can occur in a single bone, and both lower extremities may be affected simultaneously.[2]

The gray cortex sign—a focal cortical area of decreased bone density—is the first radiographic finding of stress fracture. This sign is followed by a localized periosteal reaction, a cortical break, and callus formation[3] (Figure 1A). However, radiographs fail to detect up to 94% of cortical stress fractures, and a periosteal reaction may also be seen in medial tibial stress syn-

drome. Computed tomography is excellent in detecting subtle cortical breaks (Figure 1B) but poor in assessing the surrounding soft tissues and associated marrow changes. Triple-phase bone scintigraphy is a highly sensitive imaging modality but is time consuming, involves radioisotope injection, and does not provide an evaluation of the surrounding soft tissues (Figure 2).

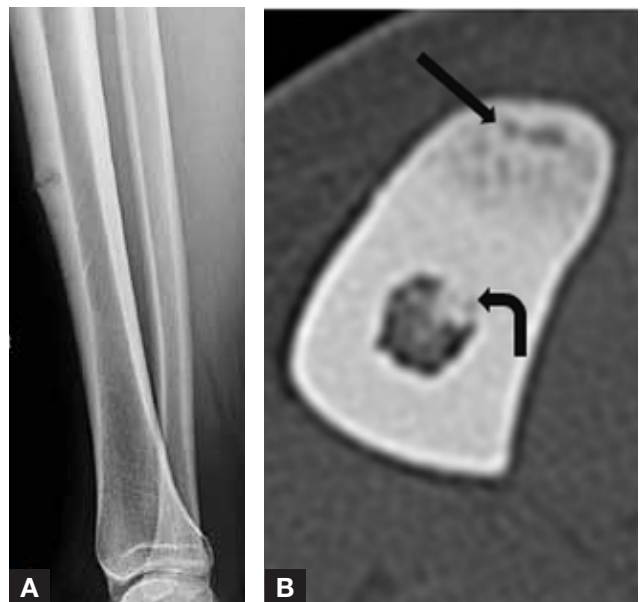Magnetic resonance imaging (MRI) is the most sensitive and specific tool for distinguishing the vari-

Figure 1. (A) Lateral radiograph shows anterior periosteal thickening and linear lucent line consistent with tibial stress fracture. (B) Axial computed tomography shows focal cortical break (arrow) and periosteal and endosteal reaction (curved arrow).
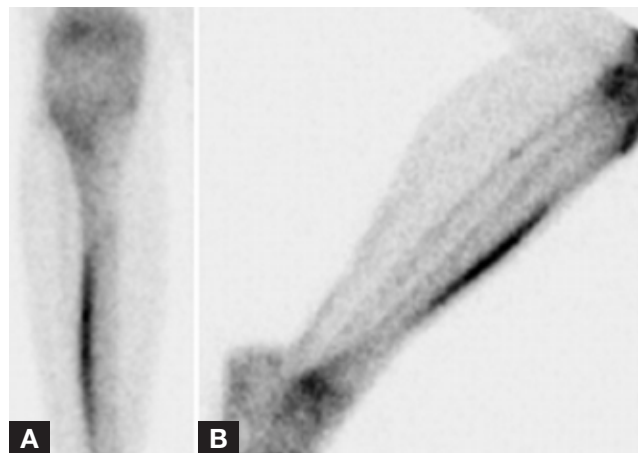

Figure 2. Frontal (A) and lateral (B) bone scan images of right lower extremity show linearly oriented area of increased radionuclide accumulation along cortex of tibia, consistent with tibial stress reaction (medial tibial stress syndrome).

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Figure 3. Coronal short TI inversion recovery image (left) and T₁-weighted image (right) show horizontal low signal intensity stress fractures (arrow) with adjacent marrow edema consistent with proximal tibial stress fracture.
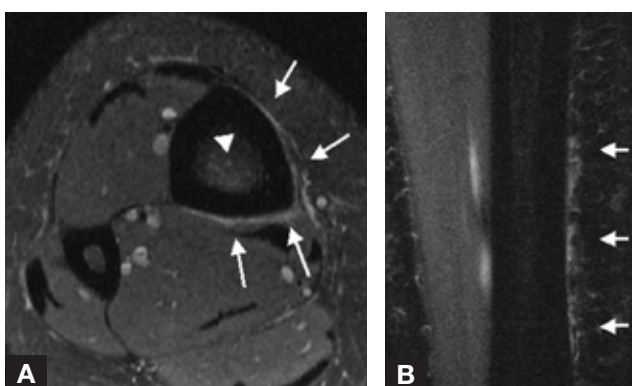

Figure 4. Axial (A) and sagittal (B) proton density images with fat suppression show subtle high signal bone marrow edema (arrowhead) and periosteal reaction (arrows) without cortical break, consistent with tibial stress reaction (medial tibial stress syndrome).

Figure 5. Sagittal (A) and coronal (B) proton density images show focal linear high signal cortical defect (arrows) with periosteal edema (arrowheads), consistent with tibial stress fracture.

Fredericson and colleagues[6] created a 4-grade system for characterizing tibial stress injuries on the basis of MRI findings. Grade 1 is mild to moderate periosteal edema; grade 2, severe periosteal edema with concomitant bone marrow edema on T_2-weighted images; grade 3, moderate to severe periosteal and marrow edema on both T_1- and T_2-weighted images (Figure 4); and grade 4, findings of grade 3 and a definite fracture line (Figure 5).

Among the conditions associated with tibial overuse injuries, stress fractures are often the most difficult to treat because of their prolonged healing times.[7] Medial tibial stress syndrome and most tibial stress injuries can be managed with conservative treatment, such as activity modification and use of nonsteroidal anti-inflammatory drugs. Other treatment options include electrical bone stimulators, extracorporeal shock wave therapy, low intensity ultrasound, and pneumatic lower leg braces. Despite these treatments, a certain subset of cases progresses to stress fracture nonunion, and surgical intervention is required.[7]

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

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ous tibial stress injuries. Given its superb soft-tissue contrast resolution, MRI can help differentiate stress fractures from periostitis and associated marrow reactive changes without using ionizing radiation.[4] For optimal detection of stress fractures, MRI (high resolution, small field of view) should be centered on the site of maximum perceived pain. Multichannel surface coils, small slice thickness with no gap, high matrix, and optimized fast spin echo techniques, with and without fat saturation, should be used. Stress fractures are seen as intramedullary linear low signal intensity lines on T_1-weighted images and T_2-weighted fat-suppressed or short TI inversion recovery images (Figure 3). Cortical involvement may manifest as linear high signal intensity on fluid-sensitive sequences. Associated reactive bone marrow edema appears as high medullary signal on fluid-sensitive images and as intermediate to low marrow signal on T_1-weighted images. Routine enhancement of marrow and adjacent soft tissues after intravenous administration of gadolinium contrast may mimic osteomyelitis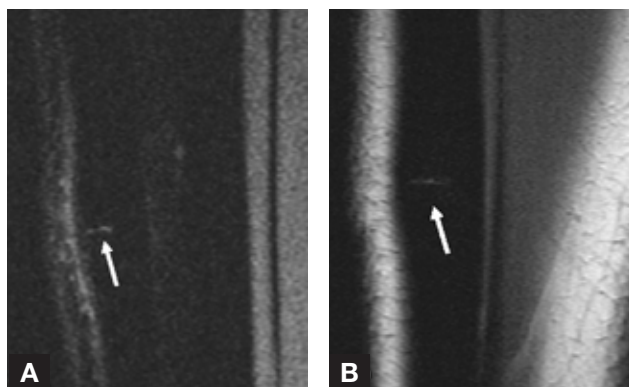 or even neoplasm. Gadolinium is not required for diagnosis.[5]