Fibular Stress Fractures in Runners

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The incidence of stress fractures of the fibula and tibia is increasing with the growing emphasis on and participation in jogging and aerobic exercise. The diagnosis requires a high level of suspicion on the part of the clinician. A thorough history and physical examination with appropriate x-ray examination and often technetium 99 methylene diphosphonate scan are required for the diagnosis. With the advent of the scan, earlier diagnosis is possible and earlier return to activity is realized. The treatment is complete rest from the precipitating activity and a gradual return only after there is no longer any pain on deep palpation at the fracture site. X-ray findings may persist 4 to 6 months after the initial injury.

A stress fracture is best described as a dynamic clinical syndrome characterized by typical symptoms, physical signs, and findings on plain x-ray film and bone scan.[1] It is a partial or incomplete fracture resulting from an inability to withstand nonviolent stress that is applied in a rhythmic, repeated, subthreshold manner.[2] The tibiofibular joint is the most frequent site.[3] Almost invariably the fracture is found in the distal third of the fibula, although isolated cases of proximal fibular fractures have also been reported.[4] The symptoms are exacerbated by stress and relieved by inactivity. The patient presents with bone tenderness and positive findings on technetium 99 methylene diphosphonate scan.

Many synonyms have been used to label the stress fracture syndrome: insufficiency fracture, Deutschlander's fracture, exhaustion fracture, fatigue fracture, spontaneous fracture, pseudofracture, and march fracture. The condition was first described in the early 1900s, mostly by military physicians.[5] The first report from the private setting was in 1940, by Weaver and Francisco,[6] who proposed the term *pseudofracture* to describe a lesion that always occurred in the upper third of one or both tibiae and was characterized on roentgenograms by a localized area of periosteal thickening and new bone formation over what appeared to be an incomplete V-shaped fracture in the cortex. They concluded that these cases were the result of a nonsuppurative osteomyelitis from which causative organisms were never isolated.

Devas and Sweetnam[7] reported on a series of 50 stress fractures of the fibula in trained athletes. They characterized the classical presentation, physical findings, and x-ray changes and localized the common fracture sites in both the fibula and the tibia.[7,8]

The incidence of fibular stress fractures in athletes accounts for 25 percent of all reported stress fractures.[9] Because of the nature of the injury, fe-

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male cadets at the United States Military Academy were found to be ten times more susceptible to stress fractures than their male counterparts.

Mechanism of Injury

The mechanism of injury was first described by Devas and Sweetnam.[7] They stated that stress fractures of the fibula in athletes most commonly result from running on hard surfaces, and proposed that recurrent rhythmical contraction of the plantar and long toe flexors transmitted stress through their origin on the fibula, approximating the fibula to the tibia. Running, therefore, causes a "to-and-fro" movement of the fibula with the point of maximal stress being near the tibiofibular joint. A stress fracture is therefore not an event but a continual process. Brahms et al[2] added that the muscular system provides shock absorption, and when the system becomes fatigued, it loses its efficiency and allows the biomechanical forces to be transmitted to the bone.

At the molecular level, the response of the bone to these repeated stresses is that of a normal remodeling process.[10] The initial stage is bone resorption followed by the formation of new bone in a pattern more appropriate for the new stress.[3] Because new bone formation is significantly slower than bone resorption, the bone weakens. With continued stress, the weakening can result in a fracture, usually of the hairline type. Bone does not gain strength as rapidly as muscle, which may explain why most of the runners who maintain a level of training are not prone to stress fractures.[4] This fact may also explain the 10 to 1 male-to-female ratio of incidence of stress fractures in cadets at West Point.

Diagnosis

The diagnosis of stress fracture, as is that of every other clinical entity, is based on a careful history, thorough physical examination, and appropriate x-ray studies.[11] The diagnosis requires a high index of suspicion on the part of the physician. The history is that of insidious onset of pain not associated with a particular event. Initially, the pain occurs at the end of the workout, but with time it occurs earlier in the activity and is of greater intensity.[7,8] The pain is typically relieved by rest. Usually the patient relates a change in training with added strain, a harder surface, longer distances, greater speed, or a new running shoe with a thinner sole.[3] Often, however, the only symptom is pain, and a history of increased activity is lacking.[10]

Physical examination does not constitute the major diagnostic clue. Full range of motion without any muscular atrophy is usually present.[1] Tender irregularities along the surface of the suspected bone may be appreciated.[12] Perifracture soft-tissue swelling may be present. Comparison of the normal and injured legs is best appreciated with the patient standing with legs rotated inward so that the fibulae may be seen in profile.[7] The pain is often reproduced by "springing" the lower extremity by straining the leg against a fulcrum.[8]

Stress fracture is definitively diagnosed radiographically. These findings depend mainly on the time elapsed between the onset of the symptoms and the time of examination. The radiologic changes occur relatively late; they may not be visualized for up to four weeks.[7] The first visible change is subperiosteal new bone,[1] which appears as a hazy area of thin callus that may not be visualized unless "hot-lighted." The area of the fracture, sometimes seen as an oblique line, is characterized by a sclerotic band or consolidation of the callus along the fracture line. There is also a diffuse thickening of the cortex and subsequent narrowing of the medullary canal.[13] The overall picture is that of a localized mass of spindle-shaped reactive new bone, the center of which is the cortical fracture.[14]

A subacute stress fracture is often difficult to diagnose. The delay in changes visible on x-ray film may be three to four weeks, and occasionally, high-quality tomograms or xeroradiograms may be necessary to show the fracture line.[10] Scintillation bone scan with ^{99m}Tc is an effective means of evaluating obscure bone pain.[10,14,15] It is positive and accurate in 90 percent of cases.[1] Spencer et al[15] describes four variants of the appearance of stress fractures of the lower extremity on scintograms: focal lesions, diffuse lesions, combined tibial-fibular lesions, and fibular lesions with demonstrable changes in the bones of the foot.

To complicate matters, in the early stages of a stress fracture the scintogram and plain x-ray views resemble osteomyelitis, eosinophilic granu-

loma, or a malignant neoplasm.[14] A stress fracture can often be differentiated from these other disorders, but care must be taken. Osteomyelitis usually involves the entire thickness of the bone. The periosteal reaction is larger and often circumferential. The patient also usually has a febrile response, increased sedimentation rate, and leukocytosis. Eosinophilic granuloma produces a thick expanded shell of new bone. Tomograms show a spherical lesion. Ewing's sarcoma is indicated by a periosteal reaction that is delicate, interrupted, or regularly laminated with adjacent permeative bone destruction. Osteogenic sarcoma appears as a periosteal reaction that is dense, fluffy, and often spiculated. The involvement often extends away from the bone and into the soft tissue. In the late stages of repair, cortical osteoid osteoma must also be excluded. The lesion center appears round and lucent with a central opacity. The history is that of pain that is worse at night and relieved by aspirin.

The diagnosis of stress fracture is particularly difficult in children. The clinical findings are often suggestive of a primary malignancy, and biopsy has been required for a definitive diagnosis to be made.[10]

Recently, Nitz and Scoville[5] proposed the use of ultrasound in the diagnosis of stress fractures of the medial tibial plateau. Ultrasound has not proved to be beneficial to the treatment of fractures because it causes pain. They postulated that an increase in pain at the fracture site in response to 2 to 3 watts/cm^2 for less than 30 seconds would provide an easy, reliable test. In 90 percent of their patients who experienced pain with ultrasound, radiographic evidence of a medial plateau stress fracture subsequently developed. All those who did not have pain were found to be free of fractures radiographically. Thus, ultrasound may be a useful modality in the differential diagnosis of stress fractures because it is simple, inexpensive, and accurate, and it reduces the amount of radiation exposure required to routinely follow this injury.

Typical Case

At the Louisiana State University Runner's Clinic, a 28-year-old woman complained of a dull pain that had persisted over the lateral malleolus of her left ankle for one month. The pain, which was brought on by running and participation in an aerobic exercise class, could not be relieved by ice or aspirin but subsided when the patient was not exercising. The patient had been involved in the sport of running for three years, averaging 10 to 15 miles per week until three months earlier, when she had increased her mileage to 25 miles per week in preparation for the Crescent City Classic 10,000 meter road race. Her training mileage was done on concrete and asphalt.

Figure 1. Roentgenogram showing periosteal elevation with medullary sclerosis of the fibula

On physical examination, the patient had an apparent leg-length discrepancy of 1.0 cm (left leg longer than right). Flexibility of the hamstrings and gastrosoleus muscles was slightly diminished, but the strength was preserved. There was point tenderness with mild edema over the distal third of the left fibula, accentuated by pressure. No masses were palpable in that region.

Radiographic studies showed periosteal elevation with medullary sclerosis of the fibula, 6 to 8 cm from the distal end. No fracture line was appreciated (Figure 1).

Treatment

Once the diagnosis of stress fracture is made, the treatment is straightforward. Because the cause of the fracture is believed to be the result of bone remodeling in response to repeated stress, rest is the all-important and probably the only essential part of the treatment.[8] Rest interrupts the process to produce osteoblastic activity greater than osteoclastic activity, resulting in periosteal and endosteal healing. Wrapping and immobilizing the leg are unnecessary. Substitute activities that do not reproduce the stress are recommended, such as bicycling, swimming, or running in water with the aid of a flotation device.[11] Normal walking is allowed and encouraged.

The healing process is monitored by a follow-up x-ray examination at three to four months because residual x-ray findings may be present for that period. A gradual return to the preinjury activity may be started when there is no longer any localized tenderness on firm pressure (usually after about three weeks[7]). Full training may then be resumed over the next four to six weeks.[8]

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