CHAPTER **14**

CALCANEAL STRESS FRACTURES: A Pathomechanical Etiology

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Stress fractures of the foot are a rather common occurrence. The majority of these occur in the lesser metatarsals and may or may not have an identifiable enthesis. The second most common site is the calcaneus, but surprisingly there are few statistical accounts other than those from the military training populations. Most of these series also describe specific maneuvers, techniques and regimens that are known to precipitate the event.[1-5]

Another cause of stress fractures in the calcaneus involves the resumption of activity subsequent to major fractures of the lower leg. These reports include those patients that have had long periods of convalescence and non-physiologic loading to the heel. The mediating factor is thought to be disuse osteopenia with loss of secondary trabeculae.[6] A less common but contributing factor following lower extremity fracture is the possible angulatory deviations that may be accepted as a consequence of healing.[6] Furthermore there are some well-established metabolic states that also escalate the incidence of stress fracture in the calcaneus. Perhaps the best known one is the alteration of bone resiliency following sodium fluoride therapy for osteoporosis.[7]

Calcaneal stress fractures are infrequently reported in the orthopedic and podiatric literature for the non-military population. It is difficult to ascertain whether the paucity of reports reflects the true incidence of this entity, or whether calcaneal fractures are so common that the average foot and ankle practitioner fails to initiate a case report.

Although the calcaneus is often thought to be a resilient bone, the penetrance of stress fractures is not rare. Perhaps this misconception is fostered by the requirement of high energy impact trauma for overt fracture of the calcaneus, or rather the extremely high percentage of patients with heel pain that are diagnosed as having plantar fasciitis. Nevertheless, this clinical problem often evades the unsuspecting clinician until radiographic evidence is painfully obvious and often embarrassing.

When one examines the forces that act on the calcaneus during bipedal gait, it may become somewhat surprising that more of these fractures do not occur. The trabecular pattern is designed primarily to support the subtalar joint as it serves to transmit body weight in torque conversion role.[8] Specifically, the primary trabeculae are arranged in a tripod-like fashion to support the facets of the subtalar joint (Figs. 1, 2). Patients that transmit more force through the calcaneus by increased body weight or increased activity would be expected to fortify the trabecular pattern with

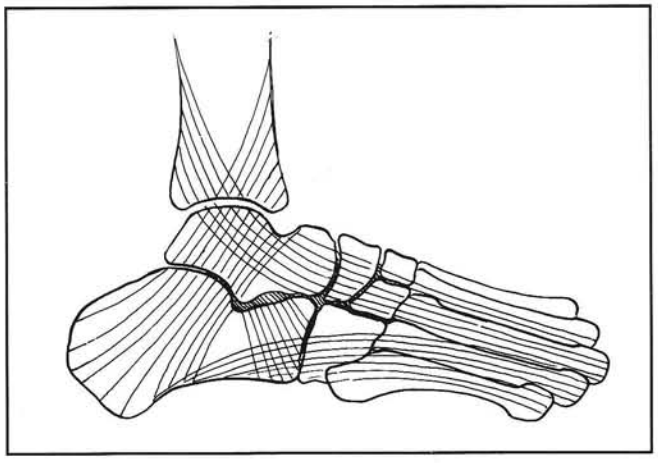

Figure 1. The arrangement of the trabecular pattern of the calcaneus. It is designed to support body weight primarily through the facets of the subtalar joint.

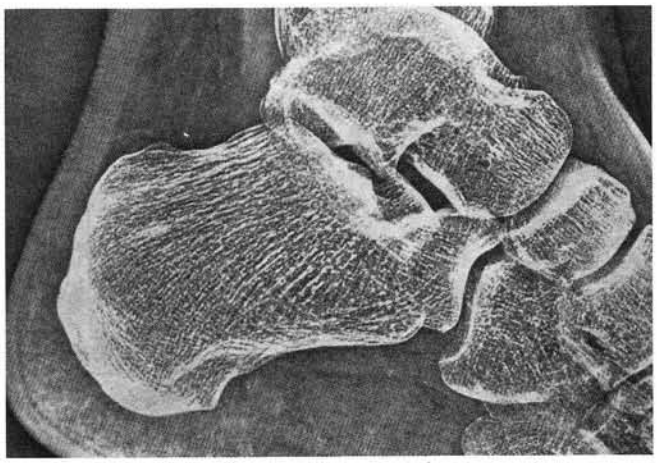

Figure 2. Xerogram of the heel showing the arrangement of the primary trabeculae. Note the primary trabeculae are most evident between the subtalar joint and the weight-bearing tuberosities.

deposition of additional bone mass in the area of load transmission

However, the actual orientation of calcaneal stress fractures usually occur at right angles to the primary trabecular pattern and often are quite removed from the direct transmission of forces. In the metatarsals, the pattern and location of most stress fractures is in the midshaft, and can easily be seen to be a result of excessive cantilever bending. Although such cantilever bending is a sub-optimal load to failure, the rate and repetition of that load are responsible for the propagation of the fatigue fracture. The protective nature of the muscles surrounding any long bone, against fracture, is well established.[9]

There seems to be an almost paradoxical mechanism in the calcaneus. Because the calcaneus is not technically a "long bone" it is often not thought of as subject to bending forces. As described, the calcaneus serves as the interface for weight transmission and torque conversion. However careful analysis of the anatomical arrangement of the soft tissue envelope encasing the calcaneus may explain the high incidence of stress fractures. The Achilles tendon complex inserts on the middle third of the posterior surface of the calcaneus, but actually extends distally along the distal third of the tuber and becomes contiguous with the plantar fascia. Similarly, the plantar fascia originates from the tuberosity of the calcaneus and courses distally from there. However, close exam shows that the continuity of the insertion of the Achilles and the origin of plantar fascia serves as an anchor point or control point for the position of the tuber (Figs. 3A, 3B). As such, the spatial position of the tuber in the sagittal plane is determined by the synergy of the forces generated by the plantar fascia and the Achilles tendon.

During normal gait, maximum tension may be generated across the plantar fascia surrounding the resupination process. This event is quite temporally associated with full knee extension which is followed by heel-off. During normal biomechanical function of the foot and the gait process, the simultaneous or near simultaneous generation of maximal tensile forces of the plantar fascia and the Achilles tendon would normally be realized by a compressive load, that is directed approximately 45° from the supporting surface toward the subtalar joint. Because bone is normally quite resistant to compressive loads, the entire anatomical construct behaves like an internal tension band. This mechanical phenomenon is applied surgically during the execution of the closing Dwyer calcaneal wedge osteotomy. In order to generate a closing, compressive force, the surgeon simply dorsiflexes the foot and the intact plantar fascia and Achilles insertion mediate closure of the osteotomy by shifting the posterior fragment in an anterior direction.

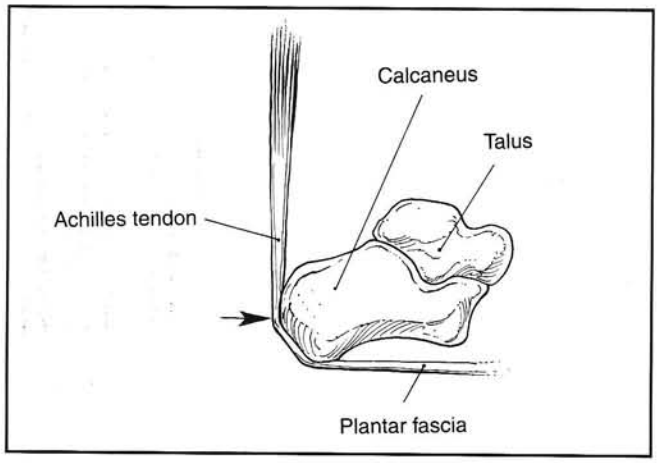

Figure 3A. The contiguous nature of the achilles tendon and the plantar fascia.

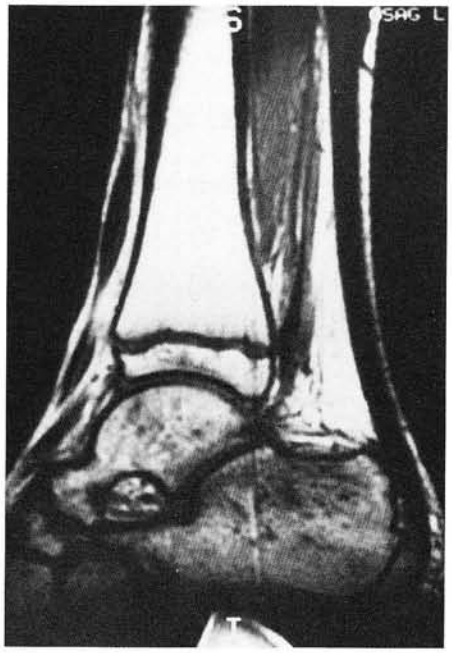

Figure 3B. MRI confirming the anatomic continuity of the two structures.

Therefore, it stands to reason that the pathogenesis of calcaneal stress fractures may be mediated by an over-activity, increased mechanical advantage, or weakness of one of the tethering structures. Rather than a resultant compressive force, asymmetric pull or action by one of the tethering structures can place an undampened shearing force across the tuber of the calcaneus. This shearing force would occur perpendicular to the primary trabeculae of the calcaneus, but parallel to the secondary trabeculae. It is well established that the loss of secondary trabeculae occurs prior to the alteration of the primary trabeculae in most bone sparing processes. However this is not to suggest that there is necessary depletion of secondary bone structure as a prerequisite to calcaneal stress fracture. Rather, the morphology and orientation of the vast majority of calcaneal stress fractures perpendicular to the primary bone structure, only speaks to shearing forces that are generated.

Although it is beyond the scope of this paper to explore all of the possible ramifications of the mismatch of forces, it is not difficult to postulate on some of the more obvious scenarios. Patients with marked gastrocnemius or gastro-soleal equinus may compensate with early heel off, which in turn will generate a moment of force in a superior direction before the plantar fascia may have become taut. With even a slight increase in walking activity over normal, there may be sufficient imbalance to initiate the fatigue failure. On the other side of the spectrum, a patient with accentuated calcaneal inclination and bone-block equinus, may generate the same mismatch in forces (but only in an inferior direction), as the posterior muscle group cannot generate enough tension to counteract that generated by the plantar fascia.

Whatever the aberrant biomechanical process, the forces acting on the tuber of the calcaneus are controlled by the Achilles tendon and plantar fascia, causing the tuber to behave much like a "log in the sea." If one end of the restraining mechanism attains mechanical advantage, the resultant shearing force disrupts the trabecular pattern, and stress fracture ensues. Inspection of the trabecular pattern shows the sparsest concentration, aside from the neutral triangle, that lies between the subtalar joint and the medial and lateral tubercles. This corresponds to the calcaneal thalamic system and is the most common site for calcaneal stress fractures.

CLINICAL FEATURES

Historical Findings

Superficially, the historical features of the calcaneal stress fracture may exhibit little difference than those in patients presenting with any type of heel pain. However, careful scrutiny and elicitation of certain patterns may help sort out patients with an evolving stress fracture from these other forms of heel pain.

Antecedent increases in activity may be elicited, but one should not be fooled if this historical piece of information is not forthcoming. Patients may not realize that a simple change of surfaces at work (due to a temporary assignment) or other modifications may be sufficient to incite the injury. Other obvious precipitating events may be elicited, such as recovering from a fracture of the ipsilateral leg, followed by the resumption of weight bearing.

The onset of pain may be just as insidious as that from plantar fasciitis, but pain from a stress fracture is usually present with activity and dissipates with rest. Although patients with impending stress fractures may have pain upon the first few steps after arising or recumbence, the distinguishing feature is that pain from a stress fracture does not wane with activity.

The location of symptoms from a stress fracture also are fairly unique in most cases. As described previously, the pain is usually along the medial and/or lateral wall, inferior to the subtalar joint line. It may masquerade as peroneal tendon tenderness, or flexor tendonitis, but careful examination by palpation can usually differentiate them. Clearly, the pain is usually not plantar and not posterior, which would suggest other diagnoses more common to those anatomic areas. Lastly radiating pain or neurologic symptoms are not common, but a wide, poorly localized and diffuse area of pain may be described by the patient.

Physical Findings

Physical findings in the patient with stress fracture are relative sparse but distinct. Focal edema, mild warmth and discrete tenderness to palpation along the lateral or medial wall are the hallmarks. One should remember that the edema is often best visualized along the lateral wall where there is little soft tissue that may confound the diagnosis. This is in contradistinction to the plantar heel pain

where visible edema is either non-existent or subtle at best. Detailed palpation must be done to differentiate and segregate the specific anatomic areas. One should be suspicious for other diagnoses if the patient has pain to palpation in other areas of the calcaneus that are not the usual presenting locations.

Radiographic Findings

Radiographic findings typically are often quite subtle and non-diagnostic, particularly in the early stages of the entity. As with any stress fracture, positive radiographic findings are confirmatory and are present because of the reparative or healing process. Patients with symptoms may have totally normal radiographs, yet have established stress fractures that have not healed enough to exhibit a visible change in the bone density. This so called lag period must be considered in those patients in which the history and physical findings indicate stress fracture, yet the radiographs do not. This phenomenon would indicate serial radiographic examination to confirm the diagnosis.

Positive radiographic findings may be described as an area of sclerosis over the affected area. Since the calcaneus is mostly cancellous bone, there is often little or no periosteal callus. Furthermore, even if the fracture has propagated through the cortex, the lateral projection of the x-ray will obscure the periosteal response, unless one is lucky enough to have an axial projection where the x-ray beam is tangential to the callus tissue.

Other diagnostic imaging techniques are usually confined to radionuclide bone scans, specifically technetium. The delayed phase will often show an intense band of uptake which may resemble the morphology of the fracture itself, if the scanner is set for high resolution. Or a more non-specific pattern of intense uptake may also be present in a diffuse distribution. In either case, increased uptake in the delayed phase is often confirmatory for stress fracture. Needless to say, none of the tests are diagnostic in isolation and the clinical information must be analyzed, interpreted and assimilated into a sensible diagnosis.

TREATMENT

Once the diagnosis is confirmed, treatment is relatively simple and usually supportive. It is quite unlikely for a stress fracture to propagate into a frankly displaced or intra-articular injury, in the otherwise healthy patient. In fact most stress fractures have inherent stability that would mitigate any sort of migration. However, patients often have significant pain with weight bearing and/or ambulation and seek relief.

Although various forms of supportive therapy are available, treatment should be based upon protection of the part. If this can be accomplished with simple maneuvers such as strapping or soft forms of immobilization, then it can be utilized to achieve the goal of pain relief. However, in the author's experience, more rigid forms of immobilization and protection are necessary to alleviate pain. Short-leg casts provide excellent protection of the part, and may even allow the patient to bear full weight while in the cast. In more severe or symptomatic cases, non-weight bearing may need to accompany the cast immobilization

The period for immobilization is variable, but in most cases symptoms are quiescent enough to discontinue the cast after 4 weeks. Essentially the practitioner and patient must titrate the amount of immobilization with the level of symptoms and desired level of function.

Careful radiographic monitoring during the healing process will ensure healing without displacement. One should base the length of time of protection on the symptoms, and not the radiographic appearance, as the fracture callous or fracture line may persist well past the point of functional recovery. The change in radiographic bone density always lags behind the actual physiologic and biologic process.

Other measures of supportive treatment such as analgesics and physical therapy may be appropriate throughout the course. If there is a clear-cut etiologic event or pathomechanical process that caused the fracture, then it should be eliminated or modified to minimize the chance of recurrence. However the assignment of precise etiologies to this injury is difficult and speculative at best. Recurrence is so rare that this discussion of prevention may be unnecessary.

Special consideration and care should be afforded the neuropathic patient. Here the actual pathogenic mechanism of the fracture may be distinctly different, and thus the behavior of the fracture is different. Specifically, Charcot arthropathy influences fractures of any bone to respond differently to the same set of exogenous stress placed upon them. Although the initial stages of

fracture propagation may fit the model described above, clearly the response does not. Conversely, the propagation and mechanism may be diametrically different, but the same fulminant response to the neglected fracture ensues.

Propagation of the fracture and subsequent collapse can occur rapidly and with vigor if the patient continues to load the injured heel. In patients with any neuropathic process and a diagnosis of stress fracture, the affected extremity must be unweighted and immobilized. The period of these conditions is protracted compared to the non-neuropathic patient, often by a 2- to 3-fold increase.

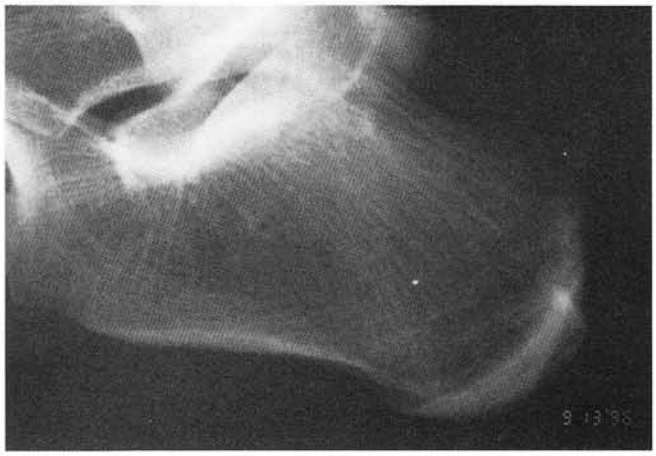

Figure 4. Lateral radiograph at the time of presentation of patient I. It was interpreted as negative for fracture.

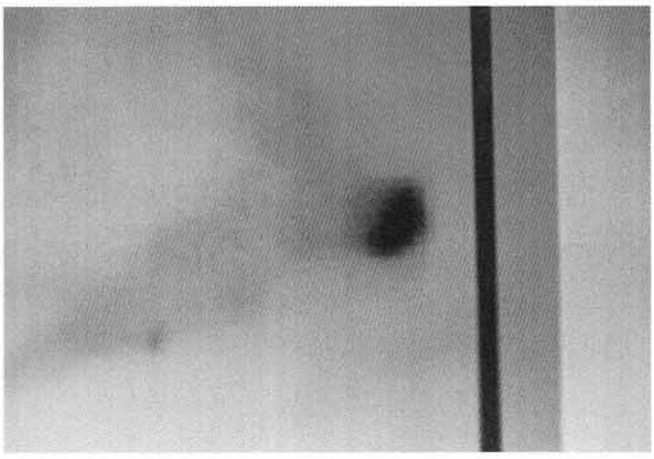

Figure 5. Bone scan of patient I, showing intense uptake in the posterior portion of the calcaneus and the site of fracture.

CASE I

A 45-year-old department store salesman had increasing pain in the heel. He described the pain only when weight bearing and it was totally relieved by rest. He had no post-static dyskinesia. He was treated initially with nonsteroidal anti-inflammatory medications and over-the-counter arch supports over a three-week period without significant relief. Initial radiographs were negative (Fig. 4). Following referral to the author's office one week later, physical examination findings were consistent with pain to palpation over the tuber on both the medial and lateral aspects. There was mild warmth and mild edema in a focal distribution. A bone scan showed intense uptake in the region of the calcaneal body (Fig. 5) and a follow-up radiograph confirmed the presence of stress fracture (Fig. 6).

The patient was treated with cast immobilization for a period of three weeks with complete resolution of the pain. Physical findings showed some focal edema but no tenderness to palpation over the fracture site. However, residual edema persisted for another 2 months.

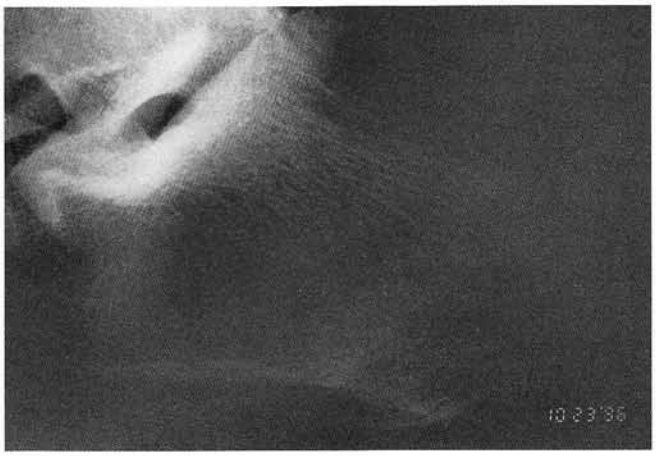

Figure 6.Follow-up radiograph 6 weeks after initial x-ray confirming presence of stress fracture.

CASE II

A 56-year-old female presented to the outpatient general clinic with a complaint of right heel pain lasting for approximately 2 weeks. Although she was later discovered to be a poor historian, she stated that the pain was present only with weight bearing. She was an insulin-requiring diabetic. A retrospective review of the chart showed sparse recorded physical examination findings. The patient was sent for an x-ray which was read as negative (Fig. 7) and given anti-inflammatory agents.

The patient returned almost 3 weeks later with continuing, and worsening pain, and according to the chart there was more swelling. New x-rays revealed a complete fracture through the calcaneus just posterior to the posterior facet of the subtalar joint, with collapse. The patient was referred to the author and was managed symptomatically, given a short-leg cast and told to be non-weight bearing. The patient did not return until 16 months later, with complaints of continued pain, limb shortening, and persistent swelling. She also complained of pain in the opposite (left) heel with weight bearing. The right heel was x-rayed and showed severe collapse of the calcaneus with compression of the posterior aspect of the body with depression of the talus (Fig. 8). Follow-up x-ray 8 months later showed consolidation of the fracture site with no significant increase in collapse.

The opposite side was also x-rayed at the time of symptoms and a diagnosis of stress fracture was also made. Radiographs confirmed its presence (Fig. 9). Similar instructions were given with a short-leg, non-weight-bearing cast. Although compliance with instructions was better than before (in spite of strict admonitions), it still was less than optimal. A follow-up radiograph 2 months later showed some minimal collapse of the heel (Fig. 10).

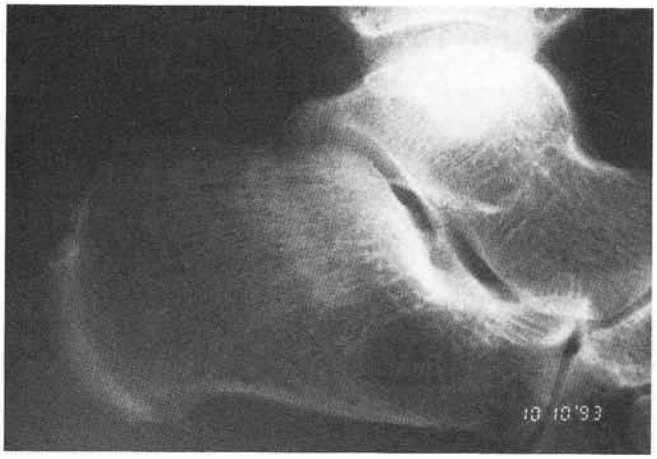

Figure 7. The initial radiograph of insulin diabetic female with heel pain. Note the stress fracture just behind the posterior facet

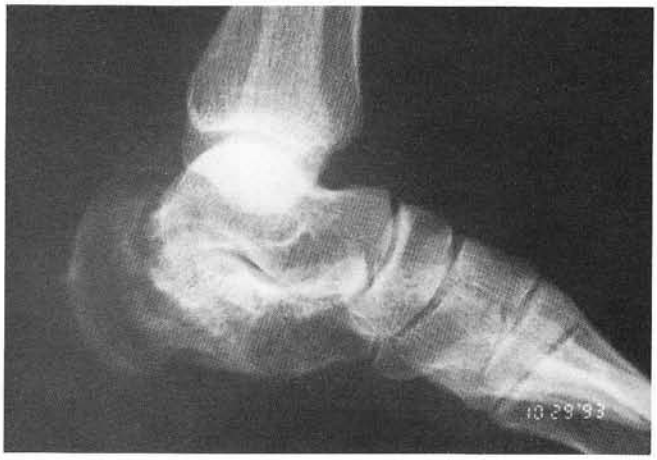

Figure 8. Follow-up radiograph of same patient showing collapse of calcaneus. This x-ray was taken 19 days after the initial film.

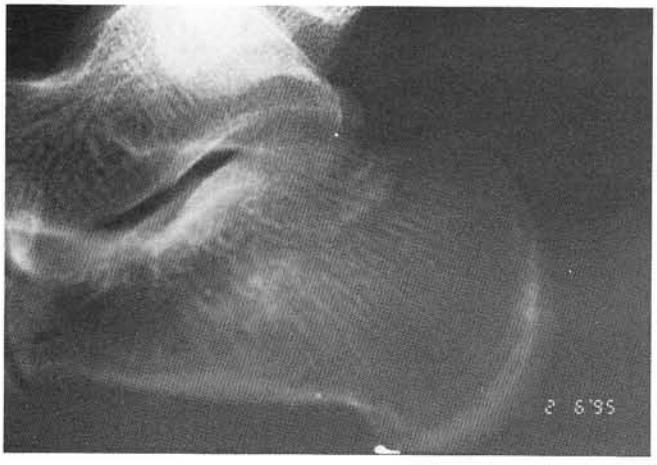

Figure 9. The opposite foot of the same patient with similar symptoms over two years later. The same evidence of stress fracture is seen.

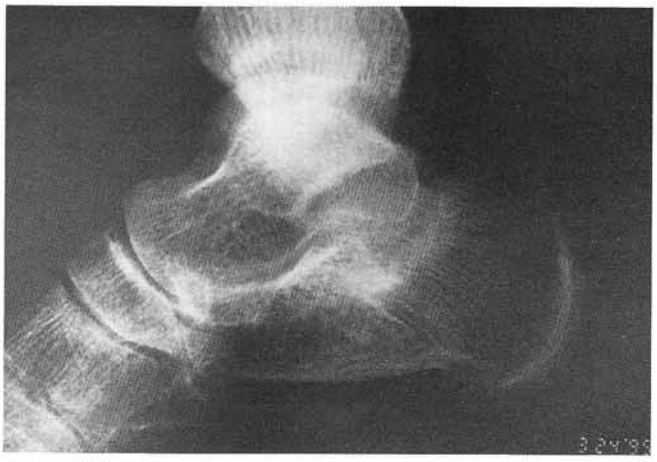

Figure 10. Lateral radiograph showing mild collapse despite immobilization and partial unweighting of foot.

DISCUSSION

The first case represents the typical scenario for patients presenting with calcaneal stress fractures. Although slightly inferior to the usual location, the proposed mechanism was probably still responsible for the propagation of this injury. Patients will usually respond to treatment with cast immobilization.

The second case perhaps represents an unusual presentation of bilateral cases separated by a two-year time period. However, the symmetry of the fracture pattern suggests a common mechanism which can either be explained by serendipitous, unrelated events in a diabetic caused by mechanical intra-osseous shear; Charcot arthropathy potentially mediated by the point of subtalar joint capsular attachment; or a combined mechanism probably caused by local hyperemia from the Charcot process and potentiated by the mechanical model. It is the author's belief that the third postulate is probably the most plausible one. However it is well known that fractures of this nature are quite common throughout the foot and ankle in neuropathic patients.

Although the outcome was quite different in each of the latter patient's feet, it was in part expected due to the delay in diagnosis and the poor compliance in the first fracture. The second fracture was diagnosed promptly, but an optimal result was confounded by patient non-compliance.

SUMMARY

Stress fractures of the calcaneus are possibly more common than reported. The diagnosis is made on a high index of suspicion. A pathomechanical mechanism is postulated based on the trabecular pattern of the bone and the attachment of the achilles tendon and the plantar fascia. Although the outcome is usually benign, special care and caution should be exercised with neuropathic patients.

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