CHAPTER 7

AVASCULAR NECROSIS OF THE TALUS

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Avascular necrosis of the talus is seldom considered as a diagnosis outside of the realm of trauma. However, there are certain patient groups that may be at increased risk for this condition. The purpose of this article is to raise the index of suspicion in these particular patient groups.

Avascular necrosis is a descriptive term that actually characterizes the end stage of the disease. Other terms used to describe this condition include ischemic necrosis of bone (INB) and locally as osteochondritis dessicans. Atraumatic avascular necrosis can also occur at the femoral head, humeral head, distal femur, and proximal tibia.

Risk factors for developing this condition include systemic lupus erythematosis, corticosteroid use, gout, alcohol and tobacco abuse, rheumatoid arthritis, hemophilia, and Gaucher's disease. Other possible factors are Raynaud's phenomena and physical stress associated with exercise or on-the-job physical stresses.

The single most common factor associated with avascular necrosis of bone (ANB) is systemic lupus erythematosus (SLE). Patients with this disease have been noted in several studies to have AVN of multiple sites such as bilateral femoral head AVN. This frequent diagnosis is even more of a challenge due to the relatively symmetrical appearance of the bone scans used to evaluate the suspected condition. The increased risk of developing AVN in the patient with SLE seems to be associated with earlier age of onset of the disease, higher and/or more prolonged steroid dose, and perhaps increased risks when the patient suffers from both SLE and Raynaud's phenomena.

Steroids, which are frequently used to treat SLE, present a risk for development of ANB. Higher doses over an extended period of time equal increased risk. Conversley, very high doses for short periods of time do not seem to generate the same risks. Similarly endogenously produced high levels of corticosteroids also do not seem to present the same level of risk. Higher dose risks have been confirmed by studying different groups of renal transplant patients treated with varying protocols of steroid for immunosuppression. The groups treated with higher doses for an extended period of time were also subject to the ANB problem just as seen in the SLE patients. This raises the interesting question of whether the bigger risk of developing ANB is SLE or other disease process itself or the corticosteroid treatment.

Diagnosis of this condition may be quite challenging because the patient may initially present with pain and unremarkable radiographs. The key to timely diagnosis and treatment lies in an increased index of suspicision, and a thorough history. With a higher index of suspicion a TC-99 bone scan and MRI can be ordered earlier for assistance in diagnosis. Other tests which may also help with diagnosis but are most likely of more academic and historic significance include bone marrow pressure testing and intraosseous venography.

The following case illustrates the potentially difficult process of diagnosis. However, a careful review of the medical history and social factors, as well as a review of the available literature raises the index of suspicision for avascular necrosis without underlying trauma.

CASE PRESENTATION

The patient is a 51-year-old female. She was seen after referral from a local podiatrist for concern of possible tibialis posterior tendon dysfunction that was unresponsive to approximately 6 months of conservative care including antiinflammatory medications and protected weight bearing using a fracture walker brace.

Review of the patient's past medical history showed a history of cirrhosis. The patient reportedly had discontinued alcohol abuse for the last eight years. However, the odor of alcohol was noted on several occasions by the staff. The patient's social history was positive for tobacco use with a reported >33 pack-a-year history.

Laboratory evaluation prior to referral of the patient showed elevated alkaline phosphatase, elevated SGOT, and elevated SGPT. Rheumatoid arthritis test results and the erythrocyte sedimentation rate were unremarkable. Plain film radiographs showed normal maintainence of the joint space (Figure 1).

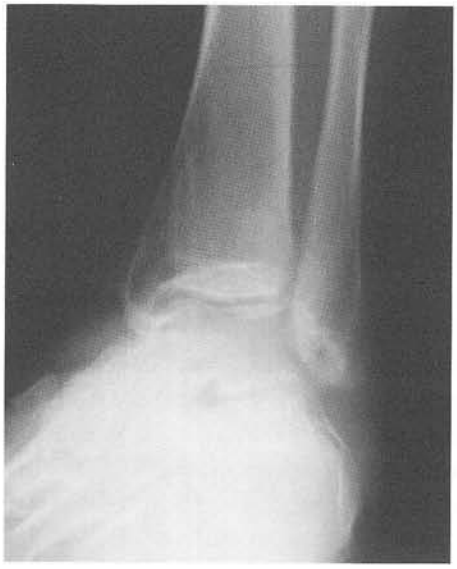

Figure 1A. Pretreatment mortise view of the ankle.

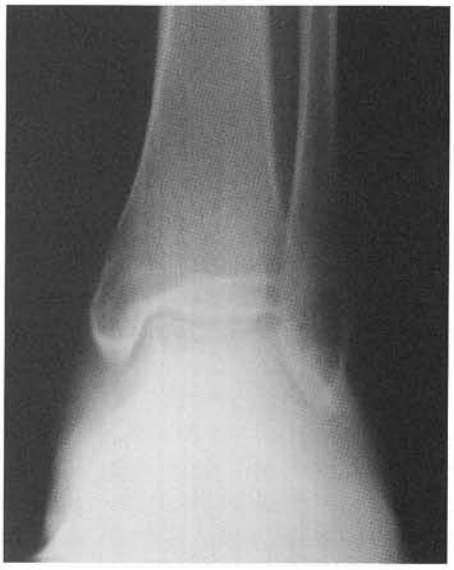

Figure 1B. Pretreatment AP view of the ankle with normal joint space.

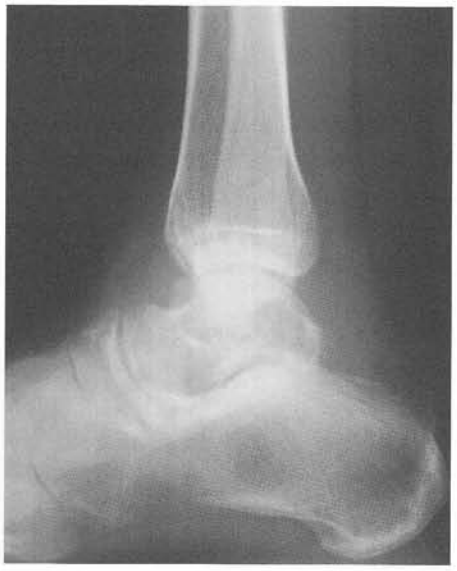

Figure 1C. Pretreatment lateral view of the ankle.

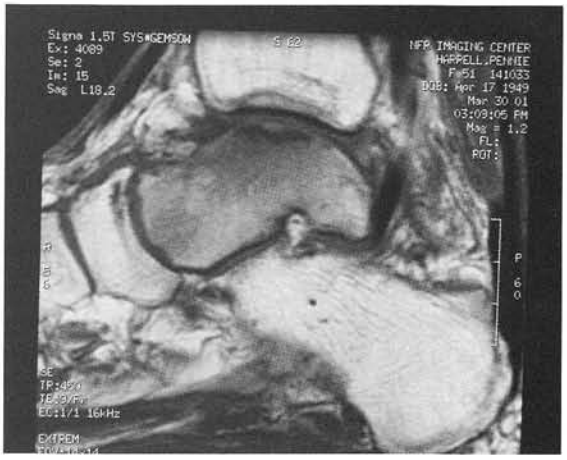

Figure 2A. Pretreatment sagittal T1-weighted image showing decreased signal intensity immediatly inferior to the articular surface dorsally with altered signal intensity surrounding the area.

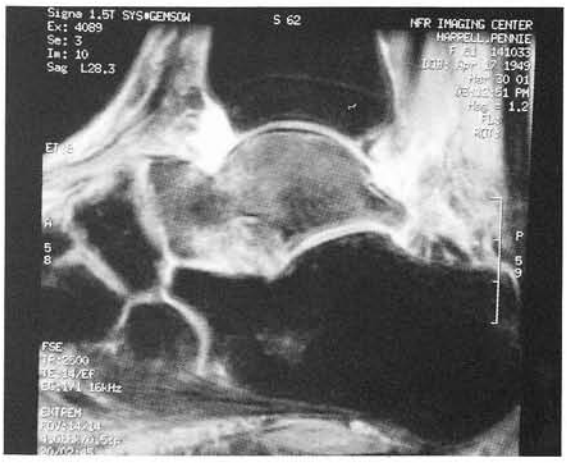

Figure 2B. Sagittal proton dense image pretreatmant

On initial evaluation the patient showed acute findings on the left foot consistent with tibialis posterior tendon dysfunction. Due to the patient's significant history and extensive previous conservative care, she was initially immobilized in a nonweightbearing short-leg cast.

After approximately 3 weeks the patient called complaining of pain in the cast. The cast was removed and the patient examined, but no positive signs of DVT or local skin irritation were noted. Because the patient could not tolerate the cast she was placed in a CAM walker, but instructed to remain nonweightbearing. A course of oral prednisone (60 mg/day, tapered to 10 mg/ day for 13

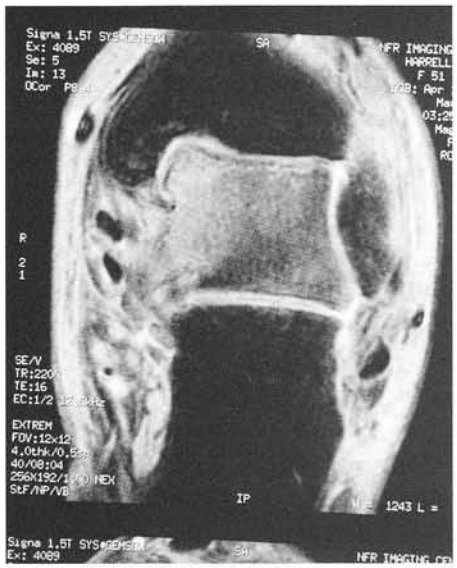

Figure 2C. Coronal proton dense image showing significant bone marrow edema of entire talar body.

days). After completing the oral prednisone therapy and maintaining a period of partial nonweightbearing (the patient was noncompliant with the full nonweightbearing instructions) no significant improvement was noted. An MRI was obtained to confirm the suspected significant tibialis posterior tendon pathology. However, MRI showed extensive bone marrow edema of the entire talar body and a small subchondral insufficiency fracture beneath the talar dome (Figure 2).

After consulting with the radiologist, a presumptive diagnosis of atraumatic avascular necrosis of the talus was established. Treatment involved application of a nonweightbearing short leg cast and placement of ultrasound bone stimulator after an approximately four-week delay due to obtaining the bone stimulator and patient noncompliance with appointments. In addition to application of the cast and bone stimulator the patient was counseled extensively regarding tobacco use. She was placed on Wellbutrin, but despite all attempts she continued to smoke at least a half pack per day.

After approximately 3 months of immobilization and 2 months of ultrasound bone stimulation a follow-up MRI was obtained. Fortunately at this point normalization of the signal intensity within the talar body was noted. No significant collapse of the talar dome was noted on MRI ,consistent with the serial plain film radiographic findings obtained in the office (Figure 3).

Progressive return to weightbearing was then

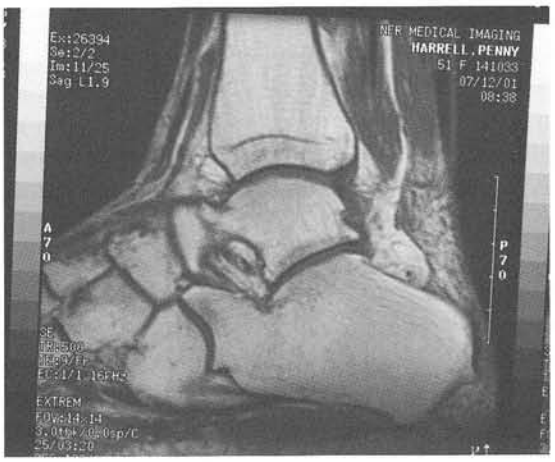

Figure 3A. Sagittal T1-weighted MRI after 3 months of immobilization and use of ultrasound bone stimulator.

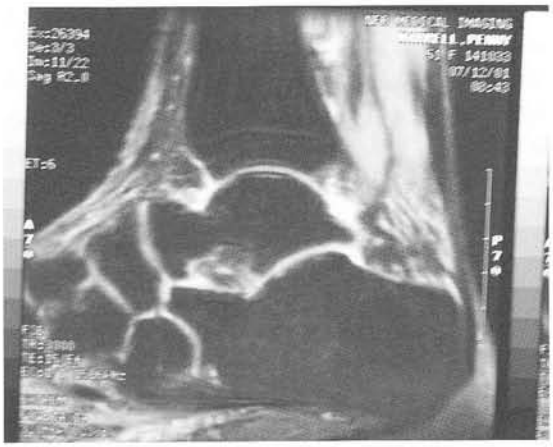

Figure 3B. Sagittal proton dense image showing complete resolution of previous talar body marrow edema.

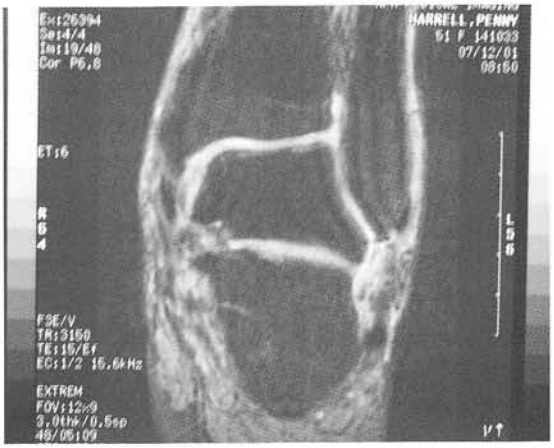

Figure 3C. Coronal proton dense image showing normalization of signal intensity after treatment.

allowed in the fracture walker brace. Serial radiographs were obtained as the patient progressed to return to regular weight-bearing. No further collapse of the talar dome was noted.

Subsequent to the patient's left ankle symptoms resolving, she began to have pain in the right ankle which showed noticeably different symptoms that were relieved with intraarticular injections. Because of the quick pain resolution, and the fact that this pain was significantly different from the left (AVN) ankle an MRI was not initially obtained.

In follow-up a second set of laboratory studies was obtained after having reviewed the literature to screen for any possible underlying seronegative arthritides which could be associated with atraumatic avascular necrosis of the talus. Results showed a positive anti-nuclear antibody with a "speckled pattern." The patient was referred for a rheumatology evaluation, but failed to comply with this recommendation. The patient was lost to follow-up after resolution of her symptoms on the left ankle. However, in phone calls to the patient she relates that she continues to be asymptomatic.

SUMMARY

This case illustrates several important points to remember in the case of ankle pain with no prior trauma history. A comprehensive patient history may help guide a thorough list of differential diagnoses. This becomes especially important when dealing with common symptoms as presented with patients with tibialis posterior tendon dysfunction or plantar fasciitis. It is very easy to exhibit a "knee-jerk" response of labeling these patients with the presumptive diagnosis only and effectively placing blinders on our other diagnostic skills. In this particular case the underlying history of alcoholism and cirrhosis should have led the practitioner to a higher index of suspicion and a more rapid diagnosis.

The prudent use of specialized imaging studies such as the MRI was particularly useful in this case. The MRI was ordered because of a lack of improvement after immobilization and a regimen of steroids. In this particular case the MRI was invaluable in relatively early diagnosis and treatment of this condition therefore preventing collapse of the talar dome.

A thorough review of the available literature on avascular necrosis without underlying history of trauma showed a fairly complete description of this pathology in the hip and several case reports involving the ankle. Most of these articles recognized SLE as the underlying etiology however one article specifically identified alcohol and tobacco abuse as significant factors. Several other articles also recognized these as ancillary factors.

Treatment for avascular necrosis has typically been supportive and protective in nature. One article obtained in the comprehensive review studied invasive bone stimulation for AVN of the femoral head. Surprisingly results were relatively poor. Mention however was made anecdotally to other studies looking at pulse electromagnetic field stimulation with more positive results noted early on. The findings in this case seemed to be consistent with this, showing fairly remarkable resolution of extensive bone marrow edema noted on the MRI within 3 months.

It should be noted that ischemic necrosis of the bone should be considered in any patient presenting with ankle pain without an underlying history of trauma and not responding quickly to conservative care. This consideration should be heightened in any patient with multiple risk factors such as chronic lupus steroid therapy, alcohol or tobacco abuse, or other underlying factors mentioned previously in this section. A comprehensive history with complete differential diagnosis may also accelerate early diagnosis and treatment therefore preventing significant morbidity associated with delayed diagnosis and treatment.

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