Avascular Necrosis of the Calcaneus 17 Years After Kidney Transplant: A Case Report

Avascular necrosis (AVN) of the calcaneus is a known complication of high dose, long term steroid use, however, it rarely occurs in the calcaneus. AVN can be caused by numerous etiologies such as steroid use, infection, trauma and sickle cell anemia to name a few. A helpful mnemonic is ASEPCTIC: anemia, pancreatitis, steroid, trauma, infection, and vasculitis disease (1). 3-9% of post-transplant patients develop AVN and most often it is in the femur or other long bones (2, 3). This is a case report of a 48 year old male who developed calcaneal AVN with a pathologic stress fracture 17 years after kidney transplant.

Case Report

A 48 year old male presented to the office complaining of left heel pain. The pain was described as an ache that worsened with weight bearing. Stretching and rest alleviated the pain, but were less effective as time went on. The patient had the prior to seeking medical attention. There was no history of trauma or changes in activities. His past medical history was significant for renal failure in 1997 requiring a kidney transplant. Tacrolimus one milligram daily and prednisone five milligrams daily was in immunosuppressant regimen. He reported no history of graft rejection requiring methylprednisolone.

Physical exam revealed loss of protective sensation and vibratory sensation to both feet, tenderness to palpation at the plantar aspect of the left heel and bilateral gastro-soleus equinus with ankle dorsiflexion loss than five degrees. Radiographic examination at the first visit demonstrated an intra-calcaneal exostosis and a geographic sclerotic lesion in the posterior calcaneus with satellite sclerosis descending to the plantar aspect of the body (Fig. 1).

The patient underwent eight weeks of physical therapy with instructions for at home Achilles tendon stretches and received a pair of custom orthotics. He reported 100% relief of symptoms. Radiographs were obtained at the eight week visit which demonstrated increased sclerosis of the calcaneus and a possible fracture through the bone island (Fig. 2). An MRI was obtained that confirmed a stress reaction of the calcaneus involving the site of avascular necrosis (Fig. 3). The patient was placed in an air cast for eight weeks after which time the pain and swelling had resolved.

At twelve month follow-up the patient continued to be seen for verruca plantaris, however, reported no recurrence of heel pain and had resumed all physical activities.

Literature Review

There are nine case reports describing calcaneal AVN. The most common etiology was prolonged, daily steroid use for disease control. Abravins-zadek and colleagues reported on the largest case series which included MRI investigation of six patients with calcaneal AVN. All patients were taking daily prednisolone for systemic disease control. These patients had either bilateral or unilateral calcaneal AVN in the posterior half of the bone (4, 5).

Literature Review Continued

Similar to our patient, Howze and colleagues reported a case of calcaneal AVN approximately three years after a heart transplant. The patient was a 58 year old female, who underwent two courses of methylprednisolone 1 gram daily for three days because of transplant rejection. The patient was also taking cyclosporine A, azathioprine, and prednisolone as part of her immunosuppression regiment. The authors hypothesized that the methylprednisolone increased her risk for developing AVN. The patient’s heel pain resolved with conservative treatment (2).

It is reported that 3% of transplant patients will develop AVN; most often it occurs in the femoral head and condyles (2). Guchelaar and colleagues analyzed the incidence of AVN in post-transplant patients based on bone mineral density (BMD) and medication regimen. The incidence was 8.9% of AVN, one of which occurred in the foot in the metatarsal bones. AVN occurred at a mean of 2.4 years after transplant. They found that high 4-month steroid doses, cyclosporine therapy, low BMD, and elevated cholesterol are all risk factors for developing AVN. Steroid treatment increases the risk of AVN because it decrease bone formation, causes apoptosis of osteocytes, and increases fat emboli in the microvasculature of bone (3).

Andermahr and colleagues performed a cadaver study that evaluated the lateral calcaneal artery. The study revealed the lateral calcaneal artery (anterior tibial artery) is joined by the lateral tarsal artery (dorsalis pedis artery) and supplies the lateral aspect of the calcaneus. The medial side is supplied by the plantar lateral artery. Given the location of the lateral calcaneal artery it is susceptible to injury during fractures and standard surgical approach on the lateral side. It is possible that damage to this artery could increase the risk for calcaneal AVN (6). A second theory, is a watershed zone in the posterior calcaneus where the recurrent vessels anastomose with the trans sphyphal arteries. The manner in which the vessels converge with in the calcaneus creates a pseudo-single dominant vessel pattern as seen at the femoral neck (4).

Conclusions

This is the second case report of calcaneal AVN after a transplant. The location of calcaneal AVN is in the posterior half of the bone, which likely represents a watershed zone, despite the absence of significant injury. Conservative treatment has thus far been a reliable treatment choice for symptomatic calcaneal AVN. As demonstrated above prolonged steroid use can cause AVN of any bone, including the calcaneus. This is yet another diagnosis to keep in mind when dealing with heel pain in immunosuppressed patients.