

CALCANEAL FRACTURE LEADING TO OSTEOMYELITIS AND NEUROPATHIC HEEL ULCER: Case Report

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INTRODUCTION

A patient presented to my practice with a conundrum. It appears that there was reluctance to treat the initial tongue type displaced calcaneal fracture in a diabetic patient who would have been challenged to be totally non-weightbearing. The nonunion of the calcaneal fracture led to calcaneal gait. In a neuropathic foot what ensued were two heel ulcers and ultimately osteomyelitis.

INITIAL PRESENTATION

MP, a 49-year-old diabetic woman with a body mass index of 52.9, presented to my office late January of 2011 with two plantar heel ulcers of the right foot following a calcaneal fracture suffered in 2007. MP had a very flat affect, making it difficult to read her motivation. However, it appeared that MP was resolved to having these ulcers indefinitely. MP related that an odor was coming from the wounds. She denied fever, chills, nausea, or vomiting. There was some ambiguity about the fracture treatment but surgery was not performed in lieu of a boot of some type. Since that time MP had been going to the hospital-based wound care center for treatment of the ulcers. They were debriding the wound on a regular basis. Antibiotics were prescribed on

multiple occasions over the last four years. MP was told that based on magnetic resonance imaging (MRI) there was a bone infection. A bone biopsy was never performed. The recommendation from the wound care physician, the orthopedic surgeon, and several other doctors was to amputate the leg. At present MP was treating the wound at home by cleaning with alcohol-based hand sanitizer that was named and applying a dressing on a daily basis. MP had been referred to our office by another patient of my practice that had undergone successful limb salvage.

On physical examination, the patient had two ulcers on the plantar heel of the right lower extremity. The most distal ulcer measured 4 cm x 3 cm in diameter and the proximal measured 2 cm x 2 cm in diameter. There was a granulomatous appearing mass at the base of the larger ulcer (Figures 1, 2). The smaller lesion was granular. Both lesions showed macerated borders, distal greater than proximal. There was minimal drainage with no purulence. No erythema, edema or increased calor was appreciated. Bone was not visible within the wound but it was apparent that the distal ulcer was deep and the bone was in close proximity. When asked to plantarflex, she had very little power and was graded at 4/5. Otherwise muscle testing was graded at 5/5 bilaterally and clearly there was profound neuropathy.

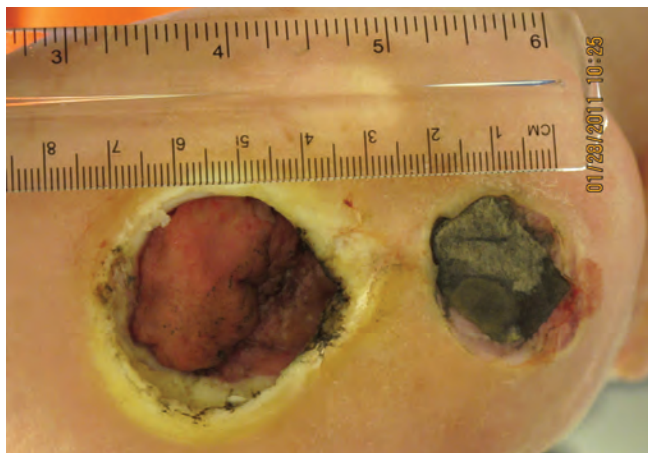


Figure 1. Plantar ulcers on initial presentation, January 28, 2011.



Figure 2. MP presenting with two plantar heel ulcers January 28, 2011.



Figure 3. MP lateral radiograph demonstrates a sclerotic body of the calcaneus and nonunion of the posterior tuber with cystic changes. The soft tissue window shows both plantar heel ulcers.

The lateral radiograph (Figure 3) demonstrated a posterior tongue type calcaneal fracture with the posterior tuber rotated and displaced cephalad. The posterior tuber was cystic and radiolucent. The entire remaining body of the calcaneus was significantly sclerotic with irregular margins plantarly and along the fracture line. The fracture line through the posterior facet was clearly visible with lateral process of the talus sheared off. The ulcers were visible in the soft tissue window. We were able to procure a copy of radiographs performed in November of 2008, one year after the trauma (Figure 4). These films did show the ulcers present at that time as well as the tongue type fracture of the calcaneus.

TREATMENT TIMELINE

Treatment, 2011

From the initial presentation we verified that MP had adequate nutritional and vascular status for wound healing. We had off weighted the heel ulcer in a cam walker and ordered a crow boot. By March the proximal, smaller ulcer had resolved. At that time there was a 20% reduction in the size of the distal ulcer (now 4 cm x 4 cm). We had purchased a rolling knee walker. Infectious disease consult was ordered. Another physician told MP that the bone was infected and that her life expectancy was measured in months. We elected to perform a bone biopsy on April 6, 2011. From the initial presentation in late January to the surgery, there were nine appointments with debridement of the ulcer at each step. The distal ulcer was now 3.5 cm x 3.5 cm, a reduction in area by 40%. At no time prior to the surgery were there overt signs of clinical infection. However there was on occasion odor and a fair amount of serous appearing drainage from the wound.



Figure 4. MP lateral radiograph from over two years earlier dated November 26, 2008 one year after the initial trauma. There is a significantly displaced posterior tuber of the calcaneus. Soft tissue window shows signs that she had developed the plantar heel ulceration at this time.

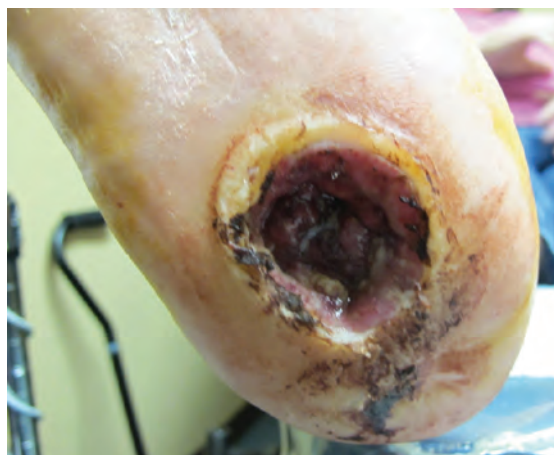


Figure 5. Plantar heel ulcer after surgical debridement, bone biopsy and culture on April 4, 2011.

Surgery, April 6, 2011

The bone was biopsied and cultured and the granulomatous tissue debrided (Figure 5). The bone biopsy was done under fluoroscopy and directed in the center of the body of the calcaneus and came back negative. Bone culture came back positive for *Staphylococcus epidermidis*.

At this time MP was having difficulty with the knee walker. Computed tomography (CT) verified that there was a full nonunion of the posterior tuber. By May we had home health start a wound vac protocol. Her Vitamin D level was 18.5 and we recommended 5,000 IU daily of Vitamin D3.

Hospital admission, August 15, 2011

The patient presented to the office with malaise, vomiting, and a temperature of 101.1. There were no clinical signs of foot infection. The ulcer at this time was 4 cm x 4 cm. Infectious disease work up for fever was performed and intravenous (IV) antibiotics were initiated. During this four-day admission radiographs and MRI were ordered.

They were read as positive for osteomyelitis. Orthopedic consult was ordered but refused since MP was aware that the recommendation was a below knee amputation. Vascular consult was ordered and below knee amputation was recommended. MP was ultimately discharged to home with the recommendation of the below knee amputation by the hospitalist.

On August 8, 2011 the ulcer was 2.0 cm x 2.5 cm. with a reduction in size of 75%. We discussed the recent admission with the infectious disease physician, who was convinced MP had osteomyelitis despite the negative biopsy and culture. We elected to perform four bone biopsies in all quadrants of the ulcer, bone debridement, and obtain additional cultures

Surgeries and Long-Term Acute Care: August to October

On August 31, 2011 the aforementioned surgery was performed and MP was immediately admitted to the long-term acute care facility (Figure 6). One biopsy was positive for osteomyelitis. Methicillin resistant *Staphylococcus aureus* (MRSA) was identified from the cultures. Hyperbaric oxygen therapy and IV antibiotics were initiated.

On September 2, 2011 we elected to remove the Dermaclose device and perform a partial calcaneotomy with application of Vancomycin beads once again. Two additional bone biopsies were performed. A bone biopsy sample was positive for osteomyelitis.

On September 9, 2011 further bone debridement was performed to attempt clean margins with three additional bone biopsies. Delayed primary closure over plain packing was performed. Of the three final biopsies performed one was negative, one “mild” and one “very mild” osteomyelitis.



Figure 6. Intraoperative photograph following Dermaclose application on November 3, 2011.

Based on this data and the radiographs showing a significant reduction in the size of the calcaneal body, we elected not to debride any further bone.

On September 28, 2011 we debrided the ulcer and closed once again. The deep layers of the ulcer were not healing well and we were concerned that due to the longevity of the ulcer and underlying osteomyelitis, we may not be able to heal the wound.

After six weeks of IV antibiotics, hyperbaric oxygen, four surgeries, and local wound care including a wound vac for part of the admission MP was discharged from long-term acute care on October 21, 2011.

Treatment To Healed Ulcer

From the discharge to resolution of the ulcer on August 21, 2012, MP had an additional 24 appointments with my office. Although reduced in size to 1 cm x 1.5 cm and a 92% reduction size, the ulcer tracked deeply close to the level of the calcaneal body (Figure 7). During that time there were no overt clinical signs of infection. Included in the care were six Dermagraft applications. MP had been dispensed a motorized scooter. We viewed MP's home on Google maps and had the landlord build a ramp to the apartment for the scooter and wheelchair. Once we had optimized the non-weightbearing status albeit with moderate success, we performed delayed primary closure of the wound in the office. Serial radiographs were performed along the course of the care with no acute changes. CT scan continued to show nonunion, but again no acute changes. On August 21, 2012 we finally had resolved the ulcer (Figures 8,9). We ordered custom shoes and double upright brace.



Figure 7. Plantar heel ulcer resolved after hyperbaric oxygen, four surgical debridements, six Dermagraft applications, wound vac therapy and delayed primary closure, August 21, 2012.



Figure 8. Ulcer resolved on 8/21/2012.



Figure 9. Ulcer still resolved but lesions present on the hallux and fifth digit. January 23, 2013.



Figure 10. Hallux ulcer presents on January 23, 2013.



Figure 11. Fifth digit ulcer on the same date.

Hallux Malleus and Hammertoe Fifth Ulcer

On October 4, 2012 MP developed irritation of the hallux and 5th digit secondary to hallux malleus and a 5th toe hammertoe. My estimation was this was compensation for the calcaneal gait. MP had hallux varus from prior surgery that included fibular sesamoidectomy, but no complications from the varus. We stretched the shoe and referred back to the orthotist for shoe modification. On December 17, 2012, MP had developed a hallux and fifth digit ulcer (Figures 10,11). We start treating the ulcers and fit MP for a modified cam walker pending new shoes. We scheduled surgery for hallux interphalangeal joint fusion, tibial sesamoidectomy and proximal interphalangeal joint

arthroplasty of the fifth digit for March 6, 2013. Prior to the scheduled surgery, all ulcers were resolved. Unfortunately the diabetes destabilized with a blood sugar of 350 and HbA1C of 11 and we elected to delay surgery to get this under control.

Novel Emed Pedobaraph

During the course of my care we had discussed possible fracture repair to restore Achilles function. The assumption was that the calcaneal gait caused increased plantar pressures leading to the ulcer. Once MP understood that this might be an option, she requested this surgery at every visit. At this time the plantar heel ulcer was resolved and the plan was to

monitor that closely and keep the double upright brace and custom shoes. MP was fairly functional at this time. However, pain and tightness deep in the foot were related. There was also a request to shed the double upright brace. Although MP wished to return to work, it was difficult to recommend that based on the history. Fortunately at this time we were able to measure the plantar pressure with a Novel emed pedobaragraph. We elected to walk MP on the pedobaraph to see if the hypothesis would be supported and document any delta in heel pressures.

The data from the pedobaraph demonstrated a more than three-fold increase in plantar pressures with a much larger area of increased pressure when compared to the contralateral limb (Figure 12). The peak heel pressure on the unaffected limb was 34 Newton/cm² (N/cm²) and on the affected side 117 N/cm². The three-dimensional representation of the peak pressures and the center of pressure visually quantifies the difference between left and the right affected side (Figure 13). The pedobaraphic data did support the hypothesis that the lack of a functional Achilles was significantly increasing the plantar heel pressure. Also, noted was elevated pressure in the forefoot of the unaffected limb, explained by the inability to off weight the affected side's heel. Since the ulcer was not present at the time of the fracture and developed some time later, the conclusion was that this was the etiology of the ulcer. Although we had resolved the ulcer, we understood that osteomyelitis could still be a contributing factor should there be re-ulceration.

Also noted on the pedobaraph was the spike in plantar tibial sesamoid pressure on the affected right side. This peak pressure was still lower than the submetatarsal head one pressures on the contralateral limb. MP had previously undergone fibular sesamoid excision. These data and the varus hallux led us to remove the tibial sesamoid when correcting the hallux malleus.

Recurrent Heel Ulcer

While waiting to stabilize the diabetes so that we could resolve the forefoot deformity, MP developed a recurrence of the heel ulcer. We elected to correct the forefoot deformity in spite of the heel ulcer so we could get MP back into the double upright brace as soon as possible (Figure 14).

On June 12, 2013 a proximal interphalangeal joint arthroplasty of the fifth digit, tibial sesamoidectomy with lateral capsular reefing and hallux interphalangeal joint fusion were performed (Figures 15, 16). MP developed cellulitis of the hallux and wound dehiscence treated with clindamycin and delayed closure. The heel ulcer was now 2 cm x 2 cm in diameter. By July 11, 2013, the cellulitis

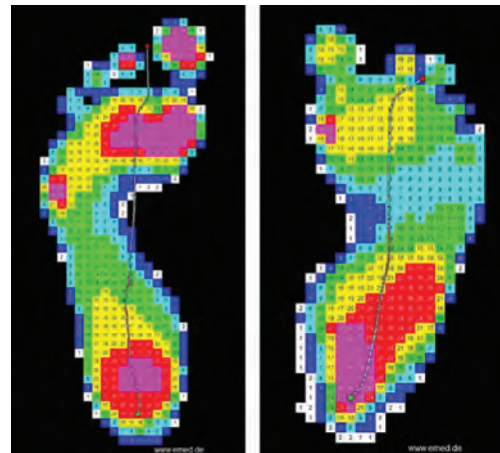


Figure 12. Two-dimensional pedobaraphic data with numeric pressure values in Newton/meters². The black line represents center of pressure in gait. Right foot is on the right and vice versa.

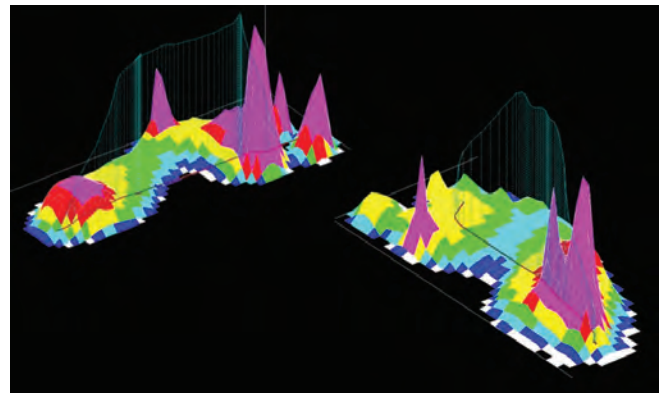


Figure 13. Three dimensional pedobaraphic data right foot on the right and vice versa.

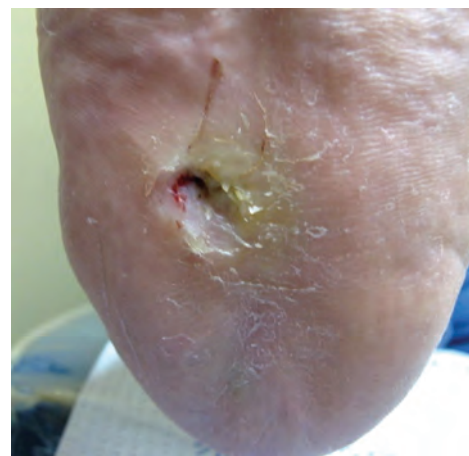


Figure 14. Heel ulcer returns on presentation it is nine months to the day from having it resolve on May 21, 2013.



Figure 15. Postoperative anterior-posterior radiograph following surgical correction of hallux malleus, tibial sesamoidectomy and proximal interphalangeal arthroplasty.

and all three incisions were healed from the latest surgery without any further forefoot complications.

From the resolution of the forefoot surgery on July 11, 2013 to present, MP has had an additional twelve visits. During this time we have provided debridement, local wound care, and tried to re-establish full non-weightbearing status unsuccessfully. A CT scan showed continued fracture with no acute changes. Vitamin D level is still well below normal at 11.8. We are again recommending 5,000 IU of Vitamin D3. Nutritional markers are within normal limits. We have consulted the orthotist for modified patella weightbearing brace and modified shoe gear to optimize heel protection.

DISCUSSION

A Pubmed literature search was performed on keyword combinations in an attempt to locate other cases and the treatment of plantar heel ulcers secondary to loss of Achilles function. We searched on the following keywords: Achilles rupture and ulcer; calcaneal fracture and ulcer; calcaneal osteomyelitis and ulcer; calcaneal gait and ulcer. Reviewing the abstracts of all resultant searches left one paper that discussed one case of calcaneal gait secondary to a spinal cord injury. The patient had developed the heel ulcer from



Figure 16. Postoperative lateral radiograph following the surgery showing the latest presentation of the calcaneus.

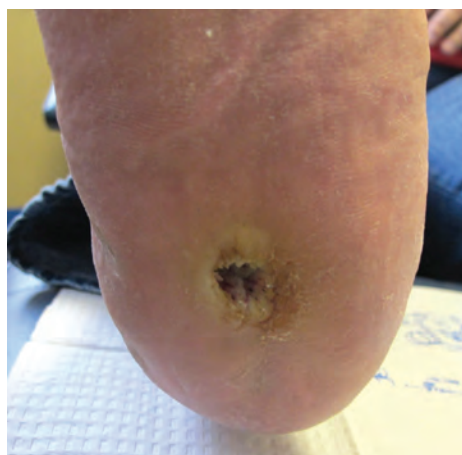


Figure 17. Most recent clinical photograph showing the current ulcer at 1.25 cm x 2 cm following debridement on November 25, 2013.

pregnancy weight gain that resolved post partum due to significant weight loss and conservative treatment(1).

In the process of writing this article approximately twenty hours were spent reviewing the patient chart from the office, radiographs, CT scans, MRIs and the hospital chart. One of my mentors in surgical residency would keep a hand written spreadsheet with one-line entries of each patient encounter to keep a bird's eye view of his patients. When I first entered private practice I did the same. When switching over to the electronic medical record it did not have a provision for this and we figured it would be not be necessary since digital information could be searched and reviewed quickly. We were wrong. While reviewing MP's chart for this report, the first step was to create this

spreadsheet. It is not clear had that timeline been available all along whether the course of treatment may have been expedited. Certainly, now that the spreadsheet is available the course of treatment is much clearer. We have now created an electronic spreadsheet inside all patients' electronic chart and will be returning to the one-line entries to track treatments and results. This will provide a birds eye view and better guide the decision-making process.

What is the value of a person's leg? We suspect that depends on the lens that we look through. Every physician involved in this case including wound care specialist, vascular surgeon, orthopedic surgeons hospitalist, and plastic surgeon (refused to see the patient based on the history) recommended amputation of the limb. The care of this limb, since January of 2011, has included six surgeries, a four-day hospital admission, six weeks of IV antibiotics, a course of hyperbaric oxygen therapy with six weeks in a long-term acute care facility, several CT scans and MRIs, endocrine, vascular, infectious disease consults and at least 90 outpatient encounters in my office that included six Dermagraft applications. Most, if not all would agree that attempting limb salvage is not worth a person's life. But, is it our place to decide the value of a limb or is it the patient's? It appears the patient is the driving force but there must be someone willing to treat.

What role does osteomyelitis play in the care of this limb? What does bone look like under a microscope after being fractured open then exposed to the environment for over 5 years? What does "mild osteomyelitis" mean? These questions were posed to the pathologists. They were kind enough to consider the questions, and search textbooks and their literature. There were no data or clear answers, but the diagnosis was still osteomyelitis. Certainly placing hardware in the presence of osteomyelitis increases the risk of recurrent osteomyelitis, infection and ultimately sepsis. If the ulcer could be resolved with bracing and modified shoe gear with reduced activity would this be an acceptable result? Is the patient's wish to not wear a brace and return to work worth risking further surgery?

At this time we have a recurrent ulcer that measures 1.25 cm x 2 cm (Figure 17), continued posterior tuber fracture and calcaneal gait with significantly elevated plantar pressures as quantified by the pedographic data. We are

considering open heel surgery and external fixation to try and establish a bony union of the posterior tuber and more functional gait. The external fixator would allow distraction of the posterior tuber of the calcaneus in position for fusion, for absolute protection of the ulcer and incision and eliminate the need for retained hardware.

Another consideration would be open reduction with internal fixation and subsequent removal of the hardware. We have reservations with this approach. One concern would be the ability to reduce this fracture at the time of surgery after six years. We estimate that we might have to remove the Achilles and subsequently repair it. If this interface becomes infected it may eliminate the ability to reach the objective of near normal plantarflexion power. We also would need strict non-weightbearing. The patient has yet to demonstrate the ability to provide this even in the long-term care facility setting. Without it, fusion and resolution of the ulcer are not likely.

Another consideration would be to create an internal double upright brace in the form of an ankle fusion. Mechanically, this would be a promising approach since with the external double upright brace, the ulcer stayed healed for nine months. Reservations with this approach are that this is not the source of the deformity. The preference is to attack the problem at the level of the deformity whenever possible. Secondly, if we should develop osteomyelitis or a nonunion of the tibia the prognosis is worsened. Unless an external fixator was used we still have the issue of weight-bearing status. An external fixator would require wires in the calcaneus. I am concerned with cross contamination of the tibia and talus with this approach.

I realize that any of these approaches may lead to below knee amputation. There are not a lot of data in the literature to support any of these options. An amputation might still be the best option for this patient. This will ultimately be resolved between the patient and a willing surgeon in the near future.

REFERENCE

1. Ward K, Sobel E, Kosinski MA. Cauda equina syndrome resulting in late sequela of calcaneal gait and neuropathic heel ulcer. *J Am Podiatric Med Assoc* 1997;87:60-5.