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Case Report

From a small heel lesion to calcaneal exposure in diabetic heel salvage complicated by ischemia and infection: a case report

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ABSTRACT

Heel ulcers are among the most threatening manifestations of diabetic foot disease due to their lack of soft-tissue coverage, their critical location, which causes constant loading, and their reliance on distal perfusion. We report an unusual case of a woman who experienced rapid progression of a right heel lesion to necrosis with calcaneal exposure despite an assuring ankle-brachial index, highlighting the importance of distal perfusion assessment and a multimodality approach in the diagnostic workup. We report a case of a 54-year-old female who experienced rapid progression of a lesion in her right heel, eventually manifesting as necrosis with calcaneal exposure. Her ankle-brachial index was within normal limits (0.92), but her toe-brachial index was critically low (0.14). CT angiography confirmed severe peripheral arterial disease. As such, her treatment involved staged revascularization, debridement, and partial calcanectomy. Additionally, she received culture-directed antibiotics and negative-pressure wound therapy. She eventually recovered with improvement of her heel, and thus avoided the need for amputation. Management of diabetic heel ischemia and successful limb salvage requires multiple interventions, including revascularization, debridement, wound stabilization, and infection control through antibiotics. A normal ABI may be misleading; as such, other modalities should be used as part of the diagnostic workup.

Keywords: Diabetic heel ulcer, Chronic limb-threatening ischemia, Toe-brachial index, Partial calcanectomy, Negative pressure wound therapy, Case report

INTRODUCTION

Vascular reconstruction has significantly transformed the treatment of diabetic foot disease, and limb salvage is now possible in most cases that would have been regarded as only for amputation. Advances in open bypass surgery, endovascular intervention, and perioperative care have improved limb salvage, especially when revascularization is incorporated into a limb-salvage pathway.[1,2] Despite this development, however, outcomes remain inconsistent, and certain areas of the body still exhibit disproportionately poor recovery.

One of the most difficult manifestations of diabetic foot disease is heel ulcers. Unlike forefoot lesions, the heel is confined by a small soft-tissue envelope, fixed biomechanical loading, and reliance on distal arterial perfusion via specific angiosomes, which are often impaired in severe peripheral arterial disease.[3,4] When deep tissue loss has occurred, physiological reserve is low, and the margin of error for ischemia and infection is small. Moreover, diabetic foot infection paired with ischemia enhances necrosis and significantly raises the chances of hospitalization and amputation.[5,6] We describe a case of a rapidly progressive ischemic diabetic heel ulcer with calcaneal exposure, which is unusual because the lesion

progressed despite a near-normal ABI, whereas the severely reduced TBI and angiographic findings revealed critical distal ischemia, highlighting the clinical gap between macroscopic inflow restoration and actual tissue viability and underscoring the importance of staged wound stabilization in cases where definitive reconstruction is biologically constrained.

CASE REPORT

A 54-year-old female patient with a long-term history of type 2 diabetes mellitus, uncontrolled hypertension, dyslipidemia, and advanced ischemic heart disease reported progressive worsening of a right heel lesion. She had a history of several coronary events with previous percutaneous coronary intervention (PCI), ischemic stroke with no residual deficit, and previous oncologic surgery with adjuvant radiotherapy. Two years ago, she had been managed for a diabetic ulcer of the right hallux. About six weeks before admission, she had observed a wound on the medial side of the right heel. No relevant family history or psychosocial factors were identified. Subsequent dark discoloration with progressive local degeneration despite conservative management ensued (Figure 1), and she had been previously reviewed elsewhere, where surgery was not recommended. She became unable to bear weight and eventually needed a wheelchair. She did not complain of classical rest pain or frank gangrene.

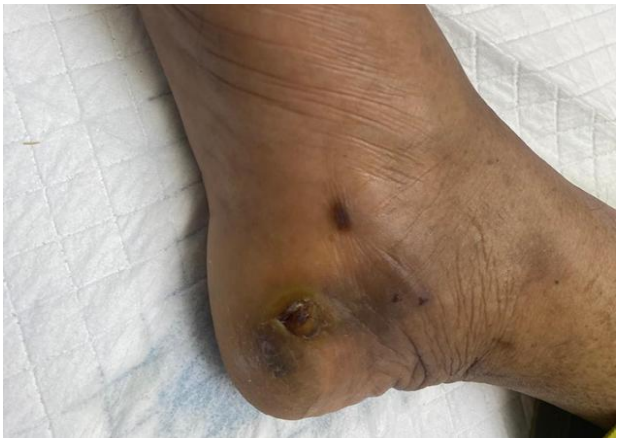

Figure 1: The right heel lesion is progressing early, with focal dark discoloration of the medial heel and progressive local tissue loss.

Assessment in a multidisciplinary diabetic limb salvage clinic showed a necrotic medio-plantar heel ulcer with exposed calcaneal bone, necrotic wound edges, and moderate hemorrhagic-serous exudate (Figure 2). Anterior heel soft-tissue defect involving the calcaneus was subsequently observed in a lateral radiograph of the right foot, which confirmed the degree of local tissue loss (Figure 3). Popliteal, pedal, and femoral pulses could not be palpated on the affected side on vascular examination. Bedside examination revealed a near-normal ankle-brachial index (ABI) of 0.92. Still, the toe-brachial index (TBI) was significantly lower at 0.14, indicating severe distal ischemia despite the seemingly encouraging ABI.[7–9] Extensive peripheral arterial disease was then confirmed by CT angiography. The abdominal aorta and iliac arteries were of sufficient calibre, and there was diffuse distal atherosclerotic disease with total non-opacification of the distal popliteal artery and its posterior tibial and peroneal branches, which were heavily calcified. The anterior tibial artery showed slight opacification along its path. These results were suggestive of critical ischemia of the heel angiosome and supported the ineffectiveness of conservative wound care, given the development of calcaneal exposure.

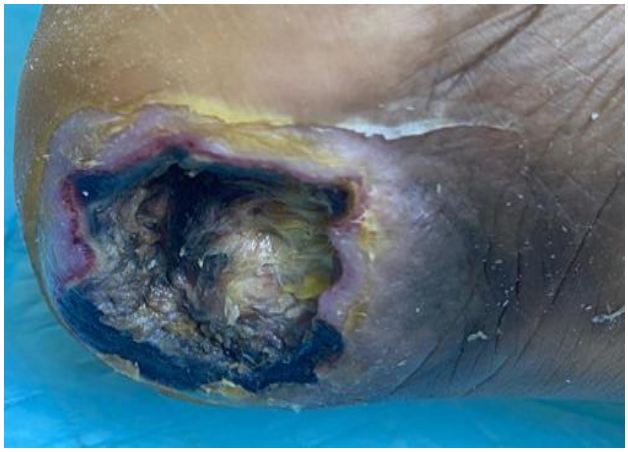

Figure 2: Advanced necrotic medio-plantar heel ulcer with extensive tissue loss, exposed calcaneal bone, and devitalized wound edges at presentation to the diabetic limb clinic.

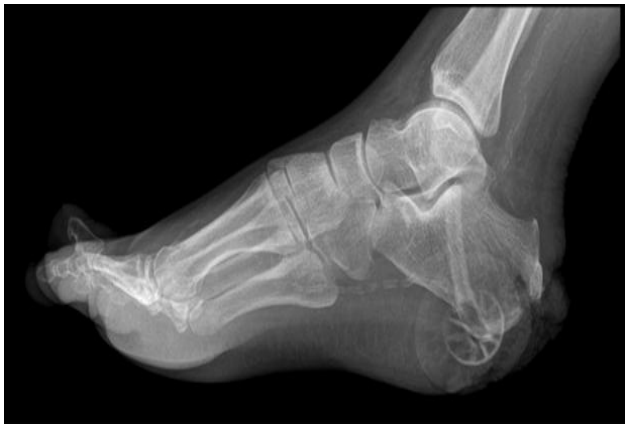

Figure 3: Lateral foot radiograph that shows a posterior heel soft-tissue defect over the calcaneus, which is in favor of deep local tissue destruction with calcaneal involvement.

A staged limb-salvage approach was adopted, with restoration of perfusion taking precedence over attempts at definitive wound reconstruction. The patient underwent a common femoral endarterectomy with profundaplasty and patch repair, and a superficial femoral artery-to-distal anterior tibial artery bypass using an ipsilateral reversed great saphenous vein graft. Aggressive debridement of the

right heel was done in the same operative setting, with partial calcanectomy. Intraoperative observations revealed extensive calcaneal involvement and detachment of the Achilles tendon insertion.

Polymicrobial infection complicated the postoperative course and prolonged it. Serial tissue and bone cultures showed *Staphylococcus haemolyticus, Enterococcus faecalis,* multidrug-resistant *Klebsiella pneumoniae*, and subsequently multidrug-resistant *Pseudomonas aeruginosa*. Antimicrobial treatment was intensified in liaison with microbiology, and recurrent operative debridement was necessary. Negative pressure wound therapy (NPWT) was used to aid wound stabilization.

A donor-site wound developed into a raw and clinically infected wound during treatment. After debridement, the wound edges were slowly brought together with sterilized non-medical self-locking plastic ties at intervals along the skin margins, which served as a temporary external tensioning system (Figure 4). Sequential tightening allowed progressive edge advancement without high-tension closure. When the wound condition improved, and the edges could be approximated safely, the ties were removed, the wound was re-debrided, and definitive primary closure was performed with sutures.

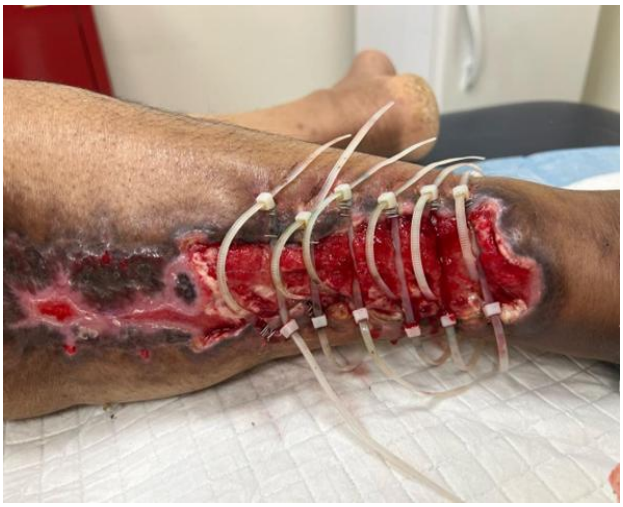

Figure 4: Donor-site wound managed with sterilized non-medical self-locking plastic ties applied as a temporary external tensioning system to approximate the wound edges following debridement gradually.

Follow-up showed that the heel had improved biologically with healthy granulation tissue over the calcaneal remnant, less exudate, and no clinical signs of uncontrolled infection. The donor-site wound also improved significantly, and limb preservation was achieved without major amputation progression at the last reported follow-up (Figure 5). The patient also reported overall improvement both clinically and aesthetically, and was able to walk again. Good adherence was assessed clinically through continued attendance for inpatient treatment and follow-up.

Figure 5: Follow-up image with limb preservation and biological healing of the heel wound and significant healing of the donor-site wound without major amputation.

Timeline

At the 2 years before presentation with right hallux diabetic ulcer.

Six weeks before admission with right heel wound appeared.

Thereafter the progressive discoloration and tissue loss despite conservative management.

At presentation the exposed calcaneus with severe distal ischemia despite near-normal ABI.

Treatment done were staged revascularization, debridement, partial calcanectomy, antibiotics, and NPWT.

Follow-up for wound improvement, limb preservation, and walking regained.

DISCUSSION

This case demonstrates the difference between restoring arterial inflow and restoring tissue viability. Although vascular reconstruction may restore macroscopic blood flow, it does not always reverse downstream microvascular and cellular dysfunction in diabetic patients.[3,10] This discrepancy is especially noticeable in the heel, where physiological reserve is low and local tissue demand is high.

The interplay between ischemia and infection further increases tissue susceptibility. Ischemia impairs the delivery of immune cells and penetration of antibiotics, and infection elevates metabolic load and necrosis.[5,11] When deep infection or osteomyelitis has developed, it becomes difficult to eradicate and may require repeated debridement and long-term culture-directed antimicrobial therapy, as in this case.

The limited utility of ABI in diabetes was also highlighted in this case. Calcification of the medial arteries may yield misleading, reassuring ABI values, especially when distal perfusion is severely impaired.[7] Conversely, medial sclerosis has less influence on toe pressures and TBI, which could be more indicative of distal ischemia in wound healing and amputation risk.[8,9] In this case, the near-normal ABI was in stark contrast to the severely impaired TBI, which is in line with angiographic findings of severely impaired distal runoff.

In this context, management should thus be perceived as a gradual biological process rather than a technical intervention. Revascularization is essential, but on its own it may not be enough to overcome the combined effects of severe ischemia, infection, tissue loss, and impaired healing. Repeated debridement, infection control, and temporizing wound care measures may be required before any definitive reconstruction or closure can be considered. NPWT is essential in this step-by-step process, as it aids granulation, exudate management, and stabilization of complex wounds when immediate closure is not biologically possible.[12]

In general, this case supports the idea that diabetic heel ulcers remain among the most threatening lesions of the limbs.[3,5,6] The key to maximizing limb-salvage outcomes is early identification of heel ischemia, avoiding false reassurance from ABI, realistic expectations about healing potential, and acceptance of staged biological stabilization.

This report is limited by its single-case design, limited generalizability, and the absence of longer-term follow-up. Its strength lies in showing the mismatch between apparently adequate ABI findings and true distal ischemia in a limb-threatening heel lesion, as well as the value of staged limb salvage in a biologically constrained setting.

CONCLUSION

Technical success in revascularization of ischemic diabetic heel ulcers may not be sufficient to achieve tissue viability to enable definitive reconstruction, especially when a seemingly reassuring ABI does not reflect the severity of distal ischemia demonstrated by TBI and angiography. In cases of delayed convergence of ischemia and infection, limb salvage can be achieved through staged infection control, repeated debridement, and wound-stabilization interventions such as NPWT.

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