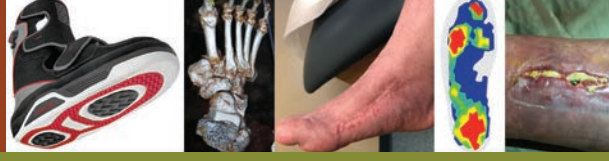

Case Report: Successful Treatment of a DFU Using a Human Acellular Dermal Matrix Allograft

This skin substitute was shown to support wound healing.

BY JEAN ARCHER, DPM

Abstract

This case report details the management of a heel pressure ulcer in a 40-year-old diabetic male with neuropathy. A human acellular dermal matrix allograft was utilized in this case to support wound healing. The literature supports the use of local wound care, antibiotics, and offloading as part of the treatment strategy. All too often, the successful management of this condition necessitates surgical intervention via debridement, partial or total calcanectomy, revascularization in the presence of peripheral vascular disease and/or use of free tissue flaps. Oftentimes, amputation is considered, especially in cases where surgical intervention and/or revascularization has failed, there is poor prognosis for successful healing using traditional methods, and high risk for surgical intervention exists. Even with these strategies, prognosis may remain poor. This article revisits this condition and details a successful outcome in the management of this frequently encountered condition. The wound detailed went on to close in about 14 weeks and no additional surgical intervention in the way of a calcanectomy and/or major amputation was required. The patient was followed at six months, there was no re-ulceration of the wound, and the revascularization efforts continued to be successful. More long-term studies are needed to predict re-ulceration rates in this high-risk patient population as well as stent patency.

Introduction

A pressure ulcer (PU), otherwise known as a pressure injury, is the result of localized injury/trauma to the skin, commonly occurring over bony prominences, directly due to friction and/or shear forces.[1] Factors contributing to this condition include maceration of the skin, dryness of the tissue, systemic disease, malnutrition, limb ischemia, and physiologic stress.[2,3]

The anatomy of the heel is well designed for function. Its primary blood supply comes from the posterior tibial artery (PTA) and peroneal artery. The lateral aspect and skin of the heel is supplied by the perforating branches of the peroneal artery, and the heel pad gets its blood supply from the medial calcaneal branch of the PTA. The plantar surface of the heel is comprised of the superficial fascia, interspaced adipose tissue, and deep fascia which aid in shock absorption. There are also attachments of the abductor digiti minimi and flexor digitorum brevis muscles. Fibrous septa are also present connecting the underlying periosteum of the calcaneus to the overlying dermis.[4]

Where there is immobility or pressure forces occurring that may result in injury to the heel, the posterior aspect of the heel becomes prone to ulceration and early necrosis of the superficial adipose tissue.

However, where there is immobility or pressure forces occurring that may result in injury to the heel, the posterior aspect of the heel becomes prone to ulceration and early necrosis of the superficial adipose tissue. This occurrence is partly due to the thin skin and lack of muscle coverage in this area, marginal blood supply, and decrease in strength of tissue with advancing age.[5-7]

The classification system for pressure ulcers (PUs) list several stages, depending on the degree of

Continued on page 134

TABLE 1

Classification of Pressure Ulcers (PUs)

Stage 1	Intact skin with non-blanching erythema in a localized area, typically over a bony prominence
Stage 2	Partial thickness loss of dermis, with a shallow open ulcer
Stage 3	Full-thickness tissue loss. Subcutaneous fat may show but not muscle, tendon or bone
Stage 4	Full-thickness tissue loss with exposed muscle, tendon or bone

Source: Fowler E, Scott-Williams S, McGuire JB. Practice recommendations for preventing heel pressure ulcers. Ostomy Wound Manage 2008; 54:42–57.

DFU Treatment *(from page 133)*

tissue loss (Table 1). Most recent updates to the literature list five different stages, from 1-1V and a final or fifth stage referred to as unclassifiable. Stage 1 is non-blanching erythema of the skin over a bony prominence well after removal of the pressure and persistent erythema of the skin without tissue loss. Stage 2 involves partial thickness loss of the dermis, with a shallow open ulcer. Stage 3 involves full thickness tissue loss including subcutaneous tissue loss but not muscle tendon or bone. Stage 4 involves full thickness tissue loss with exposed muscle, tendon, and bone. Most recently, the term "deep tissue injury" has been added to describe a final and fifth stage. In this stage, the skin is intact covering the underlying damaged tissue, which may be mushy or boggy on palpation. Once skin is breached, the wound bed can be examined and the classification scheme employed.[1]

Pressure Ulcers

Pressure ulcers can occur on any part of the body where there is prolonged pressure. The heel and the sacrum are the most frequently affected locations.[9] Observational studies show that a large preponderance of heel ulcers were grade 4[10] requiring surgical intervention. The development of a PU is associated with a high mortality rate owing to the co-existing disease.[11-13] Of all the PUs, heel wounds are associated with the poorest outcomes and increased amputation rates.[14]

The Braden scale is a PU risk assessment tool examining several criteria: sensory perception, moisture, activity, mobility, nutrition, and friction and shear.

Identification of Risk Factors

Identification of risk factors for developing PUs is paramount to the prevention of PUs and involves a careful skin assessment.[15] The Braden scale is a PU risk assessment tool examining several criteria: sensory perception, moisture, activity, mobility, nutrition, and friction and shear.[16,17] Patient populations that seem to have increased risk of developing PUs include the elderly, patients with stroke or spinal injury, patients with peripheral sensory loss or neuropathy, patients with renal disease, patients with hypoalbuminemia, patients with peripheral vascular disease, and patients undergoing surgery and immobilization for long periods of time.[18-23] The literature also points to an ankle equinus as one of the most common causes of foot ulceration leading to increased pressures on the plantar aspect of the foot during ambulation.[24] Among diabetic patients, contracture of the triceps surae is thought to occur, and this contributes to ulcer formation. It is not clear in the current literature if the pathophysiology leading to equinus is the same between diabetic and non-diabetic patients.[24]

Identifying the Pathogenesis

Identifying the pathogenesis can lead to correct and timely treatment. Assessments and interventions are essential in breaking the cycle that interferes with wound healing and leads to re-occurrence.[25]

In the successful management of heel PUs, consider addressing several key critical areas which include the vascular state, presence of infection, and pressure relief,[26] involving a multi-disciplinary approach, in the efforts to reduce rates of major amputation and recurrence rates.[27,28]

In cases where there is extensive soft tissue infection, osteomyelitis, or vascular insufficiency, surgical intervention is warranted.

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Continued on page 135

DFU Treatment *(from page 134)*

sue, debridement is key to promoting successful wound healing. The presence of moist infected necrotic tissue stalls wound healing by prolonging the inflammatory phase of wound healing.[29] Debridement types include: autolytic debridement using the body's own enzymes or endogenous enzymes under an occlusive dressing to remove the non-viable tissue; chemical/enzymatic debridement uses topical agents for example collagenase ointment; mechanical debridement uses moistened dressings which when allowed to dry, adhere to the necrotic tissue and debride the wound, and also uses pulsatile lavage to achieve similar results. Biologic debridement uses maggots or the green fly larvae. Surgical debridement can offer the fastest and most efficient method of debridement, especially in cases where there is extensive tissue loss and infection. This type of debridement can be performed at bedside or in the operating room.[30,31]

Calcaneal Osteomyelitis

Calcaneal osteomyelitis can occur through contiguous transmission between the soft tissue ulceration and bone. The most common infecting organism is Staphylococcus and Pseudomonas.[32,33] The diagnosis is made using a series of tests, including a complete blood count, erythrocyte sedimentation rate, c reactive protein, and x-rays. Magnetic resonance imaging with and without contrast aids in the diagnosis, lending to its increased sensitivity and specificity.[34] The diagnosis of osteomyelitis is further aided when the wound probes to bone.[35] Antibiotic therapy, intravenous and/or oral remains the first-line treatment after the identification of the relevant pathogen.[36] This may be performed in conjunction with traditional methods of wound healing. Partial calcanectomy[37,38] or total calcanectomy[39,40] is the surgical procedure of choice for calcaneal osteomyelitis. These procedures are considered limb salvage procedures as an alternative to below-knee and above-knee amputations.[41]

In cases where there is extensive tissue loss and infection in the absence of calcaneal osteomyelitis, where debridement is performed leaving exposed bone, consideration should be given to the use of heel flaps to achieve coverage. This can be performed during the initial debridement followed by daily wound cleansing and debridement, followed by secondary closure of the heel flap over the calcaneus.[42]

In a majority of cases, revascularization plays a critical role in the healing of these wounds in the presence of PVD.

Revascularization

In a majority of cases, revascularization plays a critical role in the healing of these wounds in the presence of PVD. Despite an underlying etiology of pressure, adequate blood flow to the foot is seen as mandatory for the healing of pressure ulcers with co-existing limb ischemia. Revascularization of the wound may be achieved by surgical bypass (often to pedal vessels) or by endovascular techniques. Successful revascularization must be performed in conjunction with appropriate debridement, wound care, offloading, antibiotics, nutrition intake, as well as risk stratification. Even so, utilizing this algorithm does not guarantee the successful healing of the PU.[43]

Patients who are being considered for revascularization should undergo diagnostic testing including duplex ultrasound and angiographic studies (magnetic resonance angiography (MRA), computed tomography. In the presence of arterial calcification, MRA is preferable,[44] however, in cases where one must avoid nephrotoxicity particularly in patients with renal disease, CTA is a safer alternative.[45] A system developed by Gentile, et al. grades the radiologic degree of pedal ischemia in necrotic heel PUs and highlights the importance of a patent PTA:[46]

Grade 1: Patent PTA

Grade 2: occluded PTA with revascularization via the peroneal artery

Grade 3: occluded PTA with revascularization via the dorsalis pedis

Grade 4: no reconstitution but patent heel tributaries

Grade 5: an avascular heel

Although open surgical bypass is considered the gold standard in revascularization, endovascular revascularization has become increasingly popular in suitable patients as a first-line intervention. This is further supported by its lower mortality and morbidity rates. Despite there are low long-term patency rates with the endovascular approach, a temporary improvement in blood supply may permit wound healing.[47,48] In deciding on target vessel revascularization, especially for those with crural disease, there is a preponderance of evidence highlighting the importance of the theory of angiosomes of the ischemic wound. First described by Taylor and Palmer,[49] an angiosome is considered tissue supplied by a specific artery and vein. The foot and ankle have six angiosomes. The posterior tibial artery (PTA) angiosome supplies three areas of the foot, the peroneal artery supplies two areas, and the anterior tibial artery supplies one area.[50] Heel ulcer perfusion is achieved by re-perfusing the PTA and peroneals. It stands to reason that direct revascularization vs indirect revascularization improves wound healing and lowers amputation rates.[51]

Certain clinical scenarios mandate amputation, such as extensive non-salvageable tissue loss in the context of acute sepsis. Some authors advocate primary amputation in those who are bed-bound with severe flexion contractures. Renal failure has been consistently shown to predict poorer outcomes after successful revascularization of heel ulcers complicated by ischemia. Certain authors caution against revascularization

Continued on page 136

DFU Treatment *(from page 135)*

in such patients because of the low rates of limb salvage and suggest that primary amputation is considered as the first-line treatment.[52,53] Nehler, et al.[54] examined the issue of revascularization for critical limb ischemia and questioned whether it was always appropriate. They proposed that three factors should be considered: technical issues with bypass surgery; associated comorbidity; and the potential length of time for wound healing before revascularization is considered. Their conclusion was that this subgroup of patients are best served by primary amputation.

Certain clinical scenarios mandate amputation, such as extensive non-salvageable tissue loss in the context of acute sepsis.

Case Report

A 40-year old male was admitted to Noyes Health/University of Rochester Medical Center with an infected heel ulcer of the right foot and involving cellulitis. The patient reports that the ulcer had been present for over two months duration before seeking care. The patient presented to the emergency room with a chief complaint of a non-healing ulcer and worsening of his condition. He did not recall any injury or trauma but admits his diabetes has been poorly controlled for many years. The patient reports he self-treated his condition for months prior to his admission.

The patient's medical history was significant for uncontrolled diabetes, hypertension, hyperlipidemia, and peripheral neuropathy. He reports tobacco use for over 20 years. Upon initial presentation, it was noted that the patient's vital signs were within normal limits; however, his vascular neurologic and biomechanical exams were not. The patient had reduced pedal pulses, diminished sensation bilaterally, and on biomechanical examination, exhibited an equinus gait most likely secondary to a gastric-soleus contracture on the right side. The etiology was unclear.

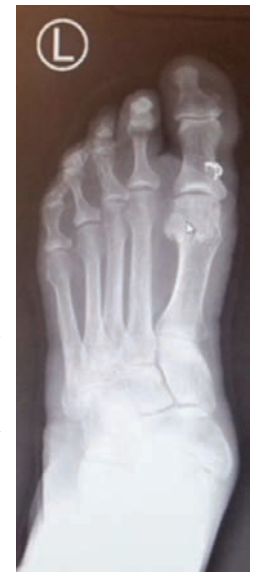


Figure 2: Operative management of a diabetic patient with a large infected heel ulcer.

Physical examination revealed a full thickness that was unstageable ulceration of the posterior lateral aspect of the right heel (Figure 1). Tissue composition was boggy with the presence of some fibrotic tissue as well as moderate purulent drainage and malodor. Increased swelling and redness accompanied the wound which extended up the right lower leg. The wound did not probe to bone.

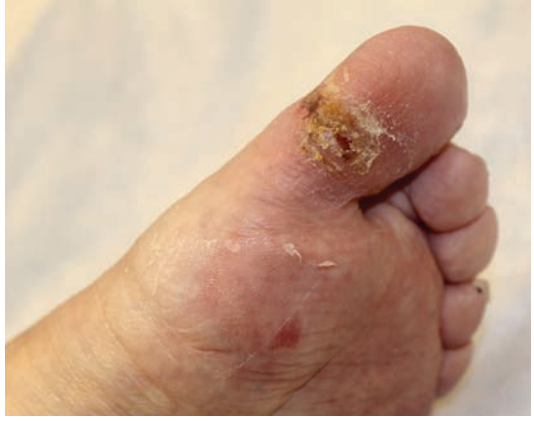


Figure 1: Infected ulcer at the posterior aspect of the heel

On admission, laboratory and diagnostic testing were performed. The abnormal findings were the following: (1) an abnormally elevated white blood cell count, (2) an abnormally elevated erythrocyte sedimentation rate and c-reactive protein (3) elevated hemoglobin A1C (4) an abnormal duplex ultrasound demonstrating monophasic waveforms.

The decision was made to bring the patient to the operating room and perform an incision and drainage/debridement of the infected ulcer. Deep wound cultures were performed, and specimens of tissue were submitted to pathology for analysis.

A CT angiogram was performed by our interventional radiologist. We did not have a vascular surgery service. Angiography of the right lower extremity revealed a short segment (approximately 3 cm) of critical stenosis of the right posterior tibial artery which was treated with a 2.5 and 3 cm angioplasty performed by interventional radiology. On post-procedure, arterial spasm was seen in the posterior tibial artery rendering accurate angiography post-procedure not possible. However, triphasic waveforms were audible on Doppler in the right posterior tibial artery and biphasic in the right dorsalis pedis artery.

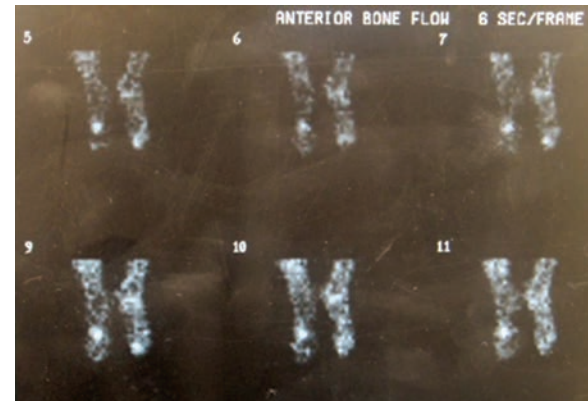


Figure 3 : Debridement of the heel wound and application of the human acellular dermal graft.

A second debridement of the ulcer was performed (Figure 2) as well as an application of the human acellular matrix graft to support healing (Figure 3). Once the graft was applied, absorbable sutures and 3.0 Vicryl were used centrally to anchor the graft to the wound base and non-ab-

Continued on page 137

DFU Treatment (from page 136)

sorbable, 3.0 nylon sutures were used to secure the graft at its periphery to the wound edges. Xeroform was used to prevent desiccation. The patient was brought back to the office weekly for similar dressing changes consisting of moistening with saline, application of xeroform, as well as gauze dressings. Pressure relief was achieved using a pressure relief boot.

The ulcer went on to heal uneventfully within 14 weeks (Figure 4). The patient was followed at six months, there was no re-occurrence of the ulcer, and stent placements remained patent. Once the ulcer healed, the patient was fitted for custom orthoses and added-depth footwear.

Discussion

Despite the various surgical and non-surgical options available to treat patients with heel pressure ulcers, a significant number will require amputation. There will be a small subgroup of patients who will be candidates for revascularization, and this will be necessary to promote wound healing. In this case, the patient's chances for successful healing were increased with the treatment implemented, and he did not require an amputation.

It is important to identify at-risk patients by performing risk assessments, and then take steps to prevent ulcerations.

Outcomes vary based on a number of underlying factors, including co-existing morbidities as well as structural and biomechanical factors. It is thought that the patient's equinus deformity of unknown etiology increased pressure on the plantar aspect of the heel with ambulation. His neuropathy secondary to the hyperglycemia contributed to the loss of sensation of pressure caused by his tight-fitting shoes.

It is important to identify at-risk patients by performing risk assessments, and then take steps to prevent ulcerations. Although the majority of heel ulcerations are superficial, a significant number of these will require limb salvage measures which include debridement, partial or total calcanectomy, surgical or endovascular revascularization, and the use of tissue flaps. In this article, an alternative management of the pres-

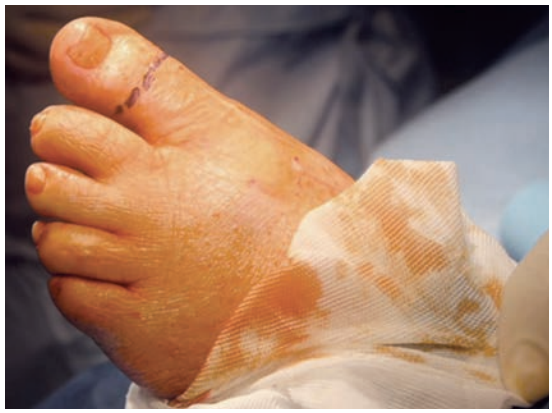
Continued on page 138

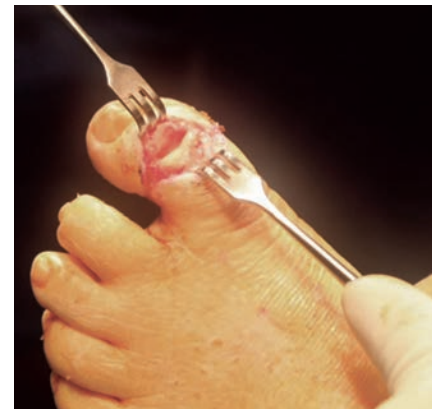
DFU Treatment (from page 137)

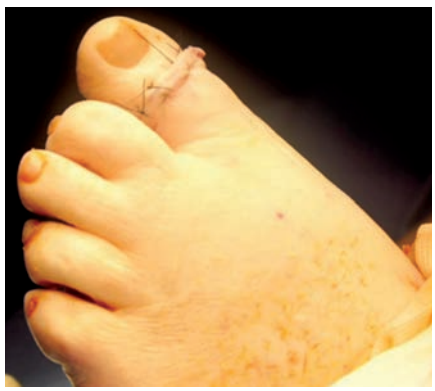
sure heel ulcer is presented with the use of a matrix scaffold for tissue regeneration, an acellular human dermal matrix allograft. Decellularized human skin has been used extensively to support wound healing and soft tissue reconstruction, thus saving limbs. The ability to utilize this skin substitute as an adjunctive measure proved to support wound healing.

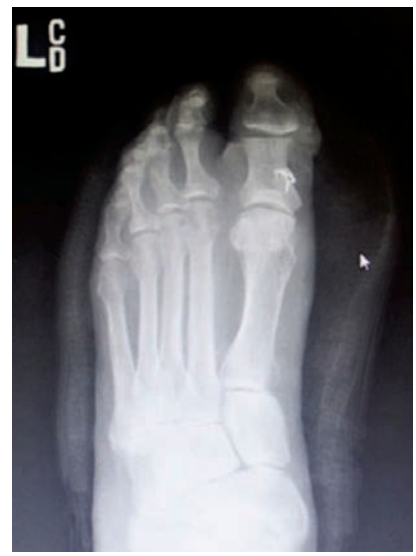
The patient in this case did not require a tissue flap. It was also important to address the underlying factors in order to prevent a re-ulceration. The patient was educated as to the importance of glycemic control and was prescribed added-depth footwear and custom orthoses in hopes of improving the equinus gait. Management of the ankle equinus has included stretching and physical therapy. In some severe cases, Achilles tendon lengthening may be needed to provide an increase in the range of motion. The patient reported here did not require an Achilles tendon lengthening procedure.

Poor glycemic control in patients with diabetes may lead to neuropathy; this may lead to ulceration as well as amputation. In the case reported, peripheral neuropathy and hyperglycemia were also underlying factors. Adherence to strict glycemic control should be implemented prior to surgical intervention to ensure good outcomes. **PM**









Figures 4A : Incorporation of the graft. B & C Weekly irrigation of the wound with saline. D Wound closure at 14 weeks

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DFU Treatment *(from page 138)*

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