The Diabetic Foot

Chronic Wounds: Combating an "Axis of Evil" with the "Forces of Healing"

This is a war which must be won.

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Aiding the healing of chronic wounds and preventing consequences such as amputation can, in a sense, be likened to the "War on Terror." The "terror" that is present in these patients is the fear of amputation resulting in loss of independence. We already know that 84% of amputations are preceded by a foot wound.[1] We know that an infected wound is a significant risk factor for limb loss.[2] Directing resources toward keeping wounds from getting infected, and assisting them to close in a timely fashion, can maximize the potential for limb preservation. The "Axis of Evil" consists of ischemia, infection, and pressure.

A patient with a lower extremity wound and contributing peripheral ischemia should have a consult for a vascular intervention.

Ischemia
Ischemia in extremities with open wounds prolongs or prevents healing and is a risk factor for amputation. Ischemia must be recognized and treated. The history, clinical exam, and imaging are all used to make the diagnosis. Patients should be questioned about intermittent claudication and rest pain. Frequently, a patient with diabetic neuropathy may lack the true symptoms of intermittent claudication, but rather have fatigue after walking, which is relieved by rest. The American Diabetes Association has published guidelines for screening of peripheral arterial disease in persons with diabetes recommending intervals of 5 years.[3]

A patient with a lower extremity wound and contributing peripheral ischemia should have a consult for

Figure 1: The VersaJet (Smith & Nephew) hydroscalpel debriding a full-thickness ulcer.

Figure 2: Maggot Debridement Therapy used to convert a fibrotic or necrotic wound, to a granular one.

a vascular intervention. Although distal bypass is considered the "gold standard" for limb revascularization, an endovascular procedure may restore enough flow to the limb to allow the wound to heal—achieving the desired endpoint.

Infection

Infection is a clinical diagnosis. A culture should only be taken if the wound is clinically infected and is used to direct therapy, not to make the diagnosis of infection. The Infectious Diseases Society of America (IDSA) classifies diabetic foot infections as uninfected, mild, moderate, and severe.

Uninfected wounds have no purulence or manifestations of inflammation.

Mild infections have >2 manifestations of inflammation (purulence, erythema, pain, tenderness, warmth, induration);

Figure 3: The Wound V. A. C. (KCI, San Antonio) produces topical negative pressure and promotes granulation tissue.

AND any erythema extends less than 2 cm from the wound, AND infection is limited to skin/superficial subcutaneous tissue without systemic illness.

Moderate infections have one or more of the following descriptions: cellulitis extending greater than 2 cm from the wound, lymphangitis, deep tissue abscess, gangrene, involvement

Figure 4: Puracol (Medline) is 100% collagen and is moistened with a few drops of saline and placed on the wound.

Figure 5: A split thickness skin graft (STSG) placed on the dorsum of the foot, after 1 week the interstices are healing.

Figure 6A & 6B: The use of GraftJacket (Wright Medical) to replace "like with like" tissue on the plantar foot.

of muscle/tendon/joint/bone AND the patient is systemically well and metabolically stable.

Severe infections have the characteristics of a moderate infection, but the patient has additional signs/symptoms of systemic toxicity such as fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, hyperglycemia or azotemia.[2]

Our group has since validated this classification system, noting a substantial increase in hospitalization and amputation with increased infection severity.[4]

Pressure

Pressure is probably the most commonly overlooked component of wound chronicity. High foot pressures are a major etiologic factor in wound development[5] and can keep a foot ulcer from healing. Foot deformities are predictive of elevated pressures, such as hallux valgus, hammered digits, and Charcot foot.

Some deformities are not visible to inspection. Limited joint mobility (LJM) is as important for prediction of foot ulcerations as deformity.[6] The joints in the foot that should be checked for restricted range of motion are the first metatarsophalangeal joint and the ankle joint. The hallux should have at least 50 degrees of passive dorsiflexion.[7] Less than 10 degrees of dorsiflexion at the ankle is an equinus deformity. Ankle equinus increases plantar forefoot pressures and the risk for ulceration.[8] Likewise, a tendo- Achilles lengthening has been shown to reduce plantar forefoot pressures and is beneficial to heal or prevent forefoot ulcerations. Lin, et al. reported rapid healing of previously recalcitrant plantar wounds with an Achilles tendon lengthening procedure.[9] Mueller, et al. subsequently reported a similar trend toward lower ulcer recurrence when performing an Achilles tendon lengthening.[10]

The "Forces of Healing" are debridement, promotion of granulation

tissue, and wound closure. These steps must be completed in order. It is assumed that before one begins the healing treatments below, the "axis of evil" components (vascular, infection, and pressure) all have been addressed.

Debridement
Debridement is a key component to the management of foot ulcerations. Debridement removes senescent cells and reduces bioburden. Armstrong, et al. found that diabetic foot ulcers that were debrided on each visit had a 5.3-times greater chance of healing in 12 weeks than ulcers that were debrided less often.[11] There are different forms of debridement: sharp/surgical, enzymatic, mechanical, autolytic, and biosurgical. When debriding a wound, it is important to remove all necrotic/non-viable tissue, and any undermining. The surgeon must not be concerned with the defect caused by the initial debridement, since removing this tissue is important to attain closure. Most wounds require serial debridements.

New tools are available to make debridement easier and can help discriminate between healthy and nonviable tissue. The VersaJet (Smith & Nephew) is a "hydroscapel" which uses a thin spray of saline at pressures greater than 10,000 P.S.I. to debride tissue, layer by layer (Figure 1). The SonicOne (Misonix) is a device that uses ultrasonic energy to create cavitation which separates non-viable tissue.

Maggot debridement therapy (MonarchLabs com) is the only FDA-approved device that is a living organism. Maggots arrive in a sterile vial containing 250-500 larvae. They are placed inside a fibrotic wound and covered with a mesh dressing for three days (Figure 2). The species Phaenicia sericata (green blowfly) is unique in that it only consumes fibrotic/necrotic tissue and will not digest living granulation. Occasionally, multiple applications are required.

Promotion of Granulation
After a wound has been debrided,

Figure 3: The Wound V. A. C. (KCI, San Antonio) produces topical negative pressure and promotes granulation tissue.

via any modality granulation tissue must be promoted. Multiple methods exist, but the Wound VAC (KCI, San Antonio) is by far the most superior method (Figure 3). With an acute surgical debridement, we wait 24 hours to allow hemorrhage to stop; then, we apply the Wound VAC. Most often the wound bed can be prepared for closure within four weeks.

Granulation tissue can also be promoted by using collagen dressings (Puracol or Promogran), which act as a substrate for MMPs and elastase, preventing breakdown of native collagen in the wound (Figure 4). Growth factors, such as PDGF (Regranex), can be useful in a minority of cases. The living skin equivalent, Apligraf, which contains live cells, acts like a "growth factor factory". We have found it more useful at promoting granulation tissue than providing wound epithelialization.

We have shown that marrow-derived stem cells are potent, rapid stimulators of granulation tissue, providing wound bed preparation in an average of 16 days.[12] The marrow aspirate can be harvested from the distal tibial metaphysis and reinjected into the periwound area. We have placed the Wound VAC on these wounds after 24-48 hours.

Wound Closure
The best method of wound closure depends on the location of the ulcer and the stresses that will be present during and after wound closure. Dorsal foot wounds are quite amenable to split thickness skin

grafting (Figure 5). We use the Wound VAC as a bolster dressing at 125 mmHg, continuous for three to five days post-grafting. It is important to place a non-adherent dressing, such as the silicone Mepitel (Molnlycke), between the foam and the graft, as not to avulse it during the VAC removal. The VAC helps to reduce the two main factors responsible for graft failure, shearing force and hematoma/seroma. Dorsal wounds can also be closed with skin stretching devices. Medial, lateral, and plantar wounds require more resilient treatments, given the pressures that these surfaces are subjected to during ambulation. Plantar wounds have been responsive to GraftJacket (Wright Medical) and this seems to replace "like tissue with like tissue" (Figure 6). The subsequent healed plantar skin is of good quality. GraftJacket undergoes a phase during which it almost liquefies, but we have found that leaving it alone and keeping it covered with a non-adherent dressing allows it to respond as desired. Plantar wounds can also be closed with a fasciocutaneous flap. A medial plantar artery rotational flap allows the plantar arch skin to be rotated in the midfoot area. A split thickness skin graft can then be placed in the donor arch area, which is not subjected to much weight-bearing force (Figure 7). Medial and lateral wounds can respond to a variety of treatments.

Unite (Pegasus) is an equine pericardium graft used for tendon repair (Figure 8). It is extremely tough and resistant to breakdown since the processing does not destroy the cross-linking of collagen.

Figure 8: The xenograft Unite (Pegasus) is made from equine pericardium and can be used as a skin substitute.

"Mission Accomplished— Victory Over Wounds"

Once treatment has begun, clinicians should not be satisfied with delayed healing in wounds. We recommend using a wound healing prediction model to track progress, such as described by Sheehan, et al., which states that a wound which does not reduce in area (simple length x width) by at least 50% in four weeks, has a 91% chance of not healing in 12 weeks.[13] This means that the wound should be re-evaluated and an alternate treatment should be considered. The best offense is a good defense. The post-ulcerative patient should be closely monitored, as re-ulceration rates are very high. The etiology of the wound should be managed, with close attention given to pressure. Prescriptive footwear and prophylactic surgery should be considered. Neuropathic patients should be encouraged to purchase a dermal thermometer (TempTouch, Xilas) and monitor their foot temperatures daily. A spike in foot temperature can predict ulcerations days in advance, when an intervention might prevent the wound.[14] ∎

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