Heel (Calcaneal) Gait and Plantar Heel Ulcerations as a Complication in DM

Conservative and surgical options are available to treat these DFUs.

BY ADAM R. JOHNSON, DPM

Diabetes Mellitus and Ankle Equinus

Throughout most of the podiatric profession, the terms "diabetes mellitus" and "ankle equinus" often go hand-in-hand. Of course, one or the other may be present without the other, but individuals with uncontrolled diabetes mellitus often present with some degree of ankle equinus deformity.

There are multiple factors that lead to the biomechanical equinus imbalance in the lower leg. Physiologically, as we age, our muscles become tighter. This may be compounded by a sedentary lifestyle. With diabetes mellitus, there is an elevated rate of damage to collagen segments[1] which make up tendons, further exacerbating this condition.

Over the years, the podiatric profession has developed a respect for the advancing ankle equinus in individuals diagnosed with diabetes mellitus and diabetic foot ulcerations. In the simplest terms, when ankle equinus is present, so are increased forefoot pressures. The increased plantar forefoot pressure leads to damage of the local skin and soft tissue, and a diabetic foot ulcer may soon follow.

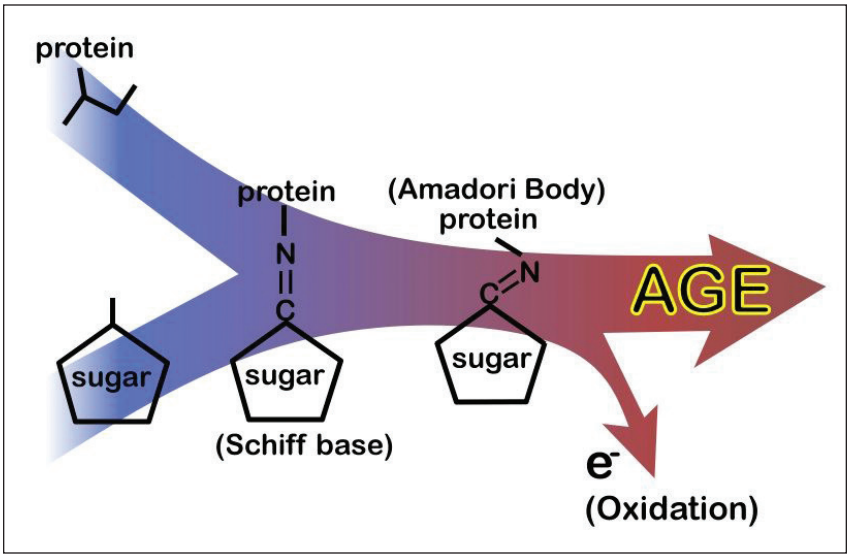

Diagram 1: Schematic diagram showing the pathway through the Maillard reaction. A simple sugar and protein form a Schiff base with a double carbon to nitrogen bond. The Schiff base rearranges to form a more stable Amadori body which can then be oxidized to create an irreversible Advanced Glycation End Product.

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Of course, there is more to the formation of, and delayed healing of, diabetic foot ulcerations than just ankle equinus. As mentioned, diabetes mellitus causes collagen damage. The damage is caused by glycation leading to the formation of advanced glycation end products (AGEs) (Diagram 1). But it does not end with collagen. The increases in blood glucose cause glycation of many proteins in our body. We measure the extent of glycation with a blood test evaluating a patient's hemoglobin A1c. The amount of hemoglobin that has become glycated may be used as a barometer for the amount of damage occurring in all other proteins in the body of individuals with diabetes mellitus.

Vasculopathy occurs as advanced glycation causes increased

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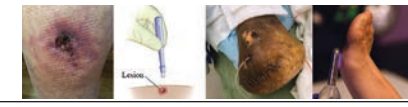

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stiffness and calcification of blood vessels.[2] The advanced glycation of low-density lipoproteins (LDL) can prevent their receptors within the walls of blood vessels from recognizing them, leading to a build-up in circulation and an accelerated formation of atherosclerosis-associated fatty streaks and plaques.[3] Damage to the vascular circulation may delay normal cellular turnover and slow the healing process.

Additionally, neuropathy occurs both indirectly—as the microcirculation to the nervous system is damaged—as well as directly—as the advanced glycation of myelin leads to its dysfunction. The result of this damage is often the inability to sense something is wrong in the foot. It is this neurological manifestation that often separates those who develop foot ulcers and those who do not.[4]

Other factors play a role as well. While one may not instinctively think that kidney disease would compound the effects, it should be stated that it does. Often termed as the "renal foot," patients with end-stage renal disease (ESRD), even without diabetes, develop secondary hyperparathyroidism, which leads to increased blood vessel stiffness, atherosclerotic plaques, and medial calcific sclerosis.[5] When the cause of kidney disease is diabetes mellitus, the effects are amplified.

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Achilles Tendon Injuries

While little to no research exists in terms of the rate of Achilles tendon injury and rupture in patients with diabetes mellitus, any specialist who treats the foot and ankle understands that it may be a costly injury to this population. A traumatic rupture may be neglected. The injury that leads to the rupture may be subtle in nature, the already compromised tendon ripe for injury after increased stress. Neuropathy may prevent the injury from being painful. Chronic leg swelling from renal disease and cardiovascular complications may hide what might otherwise be an obvious injury. In addition, the often sedentary lifestyle that accompanies those with complicated diabetes mellitus prevents the loss of function from being fully appreciated (Figure 1).

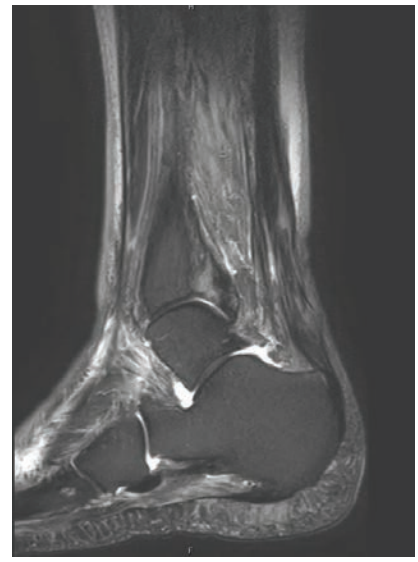

Figure 1: MRI demonstrating rupture of Achilles tendon.

Iatrogenic injury may occur after the best intentions. Lengthening the Achilles tendon is a common surgical procedure. It is performed to decrease forefoot pressures and assist in the healing of chronic forefoot ulcerations.[6] It is also performed alongside partial foot amputations as a preventative measure to decrease the risk of future amputation stump breakdown.[7] The percutaneous triple hemi-section of the Achilles tendon (TAL) is a simple procedure which may be performed under a local block without sedation in a clinic or procedure room setting. However, it may also impart damage to the Achilles tendon already unhealthy from advanced glycation. Even if the tendon does not completely rupture, potential for excessive lengthening and loss of posterior muscle group function due to a longer level arm may still lead to complications.

It has been suggested that an alternative to the TAL is the use of the gastrocnemius muscle recession. Performed via multiple described incisional methods[8-10], a common approach is a medial calf incision at the distal margin of the medial head of the gastrocnemius muscle performed with the patient in the supine position. Through this approach, the aponeurosis of the gastrocnemius muscle is severed to provide length without imparting release on the soleus muscle, preventing risk for significant loss of push-off strength. In addition, with the surgical site being distant from the vascular "watershed" region of the Achilles tendon, risk of compounding the potential for rupture at this location is avoided.

An alternative to the TAL is the use of the gastrocnemius muscle recession.

While the potential for excessive weakening of the posterior muscle group is limited with a gastrocnemius muscle recession, it may still exist. Excessive cutting of the gastrocnemius aponeurosis, or sectioning of the soleus aponeurosis, may increase this risk.

The Heel (Calcaneal) Gait

If an Achilles tendon injury occurs and fails to heal, or heals with excessive elongation of the Achilles tendon, potential weakness in the leg may occur as a long-term

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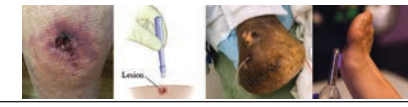

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complication. As previously mentioned, disproportionate tightness in the posterior calf muscle may lead to equinus and increased plantar forefoot pressures. After an injury to the Achilles tendon, the pendulum may swing in the opposite direction.

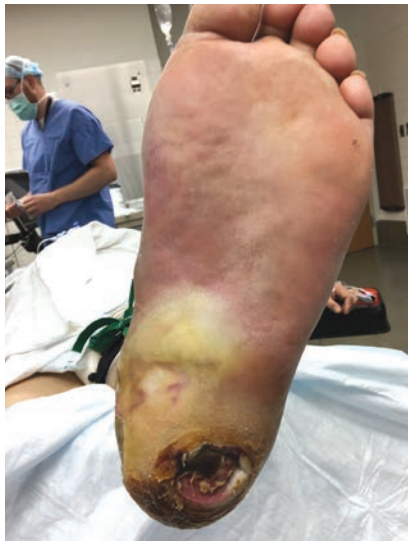

Figure 2: Infected plantar heel ulceration.

Rather than increased forefoot pressures, there may now be increased plantar rearfoot pressures. Biomechanically, this results in a "heel gait," otherwise known as a "calcaneal gait." When a calcaneal gait is present, the resultant gait is one in which forefoot push-off is diminished and the progression to heel-off is delayed. This increases terminal stance and causes prolonged time and pressure on the plantar heel. The increased load causes inflammation in and around the heel, leading to pre-ulcerative hyperkeratosis. While in the sensate this may just lead to discomfort, in patients with neuropathy, the pain is absent and the potential for skin ulceration occurs (Figure 2).

How Is The Ulcerated Heel (Calcaneal) Gait Addressed?

Plantar heel ulcerations are difficult to heal and conservative measures to offload are limited. Total contact casting (TCC) is considered the gold-standard for pressure offloading of the forefoot and it has been shown to be effective in offloading the heel, as well.[11] However, it should be noted that with the use of casting heel ulcerations close slower than their forefoot counterparts.[11] The ability for a TCC to heal ulcerations on the plantar heel may be multifold; it may be hypothesized that a well-crafted total contact cast applied to the lower leg offers calf muscle and/or patellar tendon bearing properties. It may also be hypothesized that the redistribution of plantar pressures relieves areas of excessive pressure.

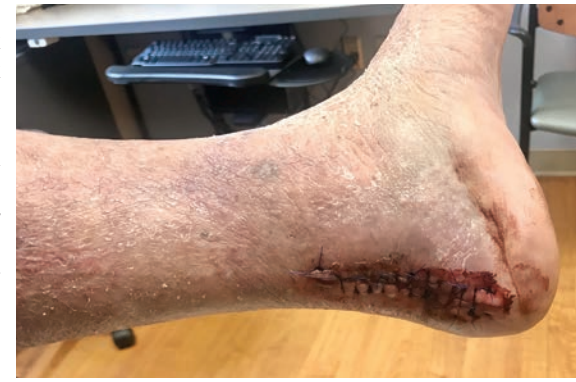

Figure 3: Post-operative image of incision used to access the FHL tendon for transfer to the calcaneus.

One surgical option to aid in the push-off strength of the foot is to transfer the flexor hallucis longus (FHL) tendon to the calcaneus.

While the TCC is considered the gold standard, the application of a total contact cast requires careful attention to detail with application to avoid complication. Furthermore, multiple applications are required to allow for periodic removal to evaluate the underlying ulceration. A pre-fabricated pneumatic walking brace, again designed to maintain a total contact fit through the lower leg to provide calf muscle and/or patellar tendon bearing properties, has also been shown to offer reduction through the entire plantar foot.[12] Alternatively, a custom molded patellar-tendon-bearing foot and ankle orthosis (PTBO) or a calf-muscle-bearing foot and ankle brace could also be fabricated to provide proper heel offloading as well as allow for periodic removal.

If conservative measures fail, either to heal the wound or prevent chronic reoccurrence of skin integrity breakdown, surgery may be indicated.

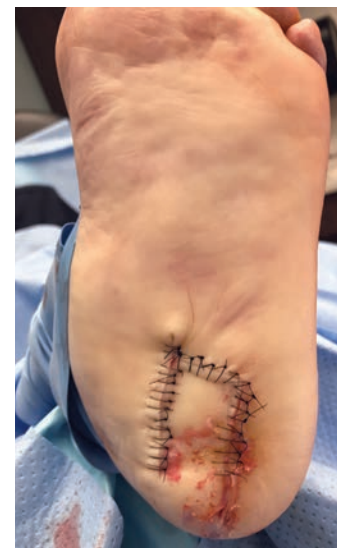

Figure 4: Excision and primary closure of plantar heel ulceration with use of rotational flap.

Flexor Hallucis Longus Muscle Transfer

One surgical option to aid in the push-off strength of the foot is to transfer the flexor hallucis longus (FHL) tendon to the calcaneus. This reliable and effective surgical procedure is able to be performed through a single incision at the posterior ankle with minimal operative time compared to other described surgical procedures.[13,14]

The technique involves positioning the patient prone on the operative table and making an incision medial to the Achilles tendon along the length of the posterior heel (Figure 3). Skin

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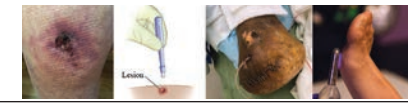

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incision is followed by dissection down to the FHL tendon which is readily accessible. The tendon is easy to identify as manual motion of the hallux will provide visualization of the tendon motion. The FHL tendon is cut as distal as possible along its visible course. It is then transferred to the calcaneus via placement of a drill hole and secured with a tenodesis screw while ensuring tension on the tendon with the foot in slight plantarflexion. If there are remnant fibers of the Achilles tendon present, the FHL tendon may also be sutured to these fibers to provide additional security.

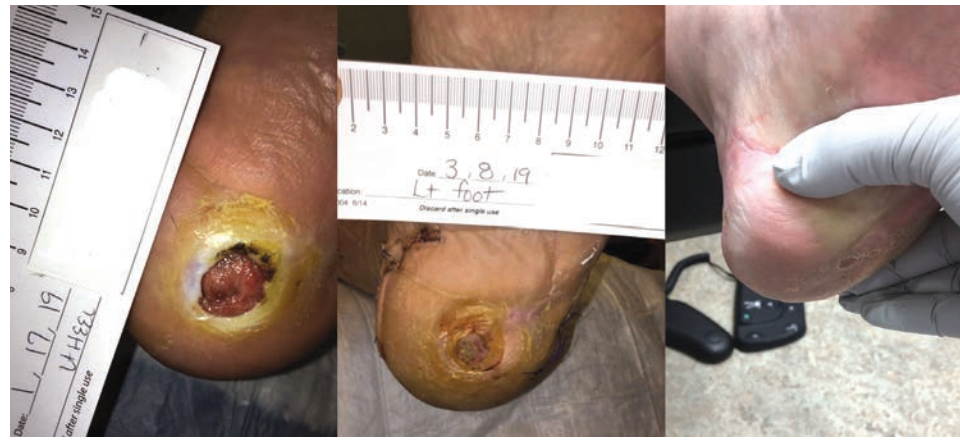

Figure 5: (a) Chronic ulceration at the plantar heel, (b) healing of ulceration after FHL transfer and augmentation with autogenous adipose transfer, (c) demonstration of same heel at 3-month follow-up after surgical intervention.

If there is significant loss of the plantar heel fat pad, an autogenous adipose transfer or allograft injection into the heel may also be considered.

Post-operative restriction recommendations include the continued use of off-loading with either use of a TCC, ankle immobilizing CAM boot, or previously constructed custom brace. The aforementioned off-loading options will also provide the necessary immobilization to allow for the tendon transfer to the heel. Non-weight-bearing should be continued for four to six weeks after surgery with the use of the off-loading device until ulceration is healed.

Adjunctive procedures may be considered at the same time, or at a later date. Aggressive debridement, if not complete excision of the ulceration, is advisable and should be performed before the surgical tendon transfer to reduce bacterial load. If complete excision is performed, this may be coupled with primary closure if local tissues allow and the rules for random flap placement are followed (Figure 4). If there is significant loss of the plantar heel fat pad, an autogenous adipose transfer or allograft injection into the heel may also be considered (Figure 5). **PM**

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