

CURATIVE HEALING OF THE PLANTAR FIRST METATARSAL ULCERATION

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INTRODUCTION

The plantar first metatarsal head is a common location for diabetic ulcer formation. Nearly one-quarter of diabetic ulcerations occur at this site (1). A variety of both isolated and coexisting biomechanical overloading forces contribute to neurotrophic ulceration of this area. Determining the causative overloading forces can be elusive, and is essential to effective surgical offloading and curative healing of plantar first metatarsal ulcers.

Based upon the specific anatomic position of a plantar first metatarsal head ulcer, the purpose of this paper is to present an examination approach for delineating etiology. The particular location of an ulcer varies according to identifiable patient biomechanics and pathology. Functionally, specific areas of the first metatarsal head become wounded and the wound's location is the link to discovery of the biomechanical fault. Simply stated, ulcer location reveals its causation (Table 1). The goal of this examination approach is to identify indicated surgical procedures, which provide wound healing without recurrence (i.e., curative healing).

NEUROPATHIC BASIS OF FIRST METATARSAL ULCER FORMATION

Etiologically, the first ray becomes overloaded in the neuropathic foot due to motor neuropathy, muscular atrophy, and resultant muscle imbalance. Coexisting sensory neuropathy eliminates protective sensation feedback, which allows the overloading effect to inconspicuously create skin injury and ulceration.

Sequentially, motor neuropathy creates intrinsic muscle weakness, which is followed by extrinsic weakness of the anterior and lateral lower leg musculature. Intrinsic weakness contributes to digital instability including hallux valgus and hallux malleus deformity. Lower leg muscle imbalance creates a relative progression of strength and inflexibility of the posterior muscle group. The most profound effect of muscle imbalance at this level is expressed within the gastrosoleal complex. Both digital instability and

Table 1

FOCAL ETIOLOGY OF PLANTAR FIRST METATARSAL HEAD ULCERS

Plantar central	Rigidly plantarflexed 1st metatarsal
Plantar central-medial	Hallux valgus*
Plantar medial- pinch lesions	Hallux valgus

*Hallux limitus may be concomitantly present. The primary cause of medial shifted lesions is an unstable first metatarsophalangeal joint (i.e., medial column overload and hallux valgus).

gastrosoleal equinus are implicated in the formation of plantar first metatarsal ulcers.

Motor neuropathy will be compensated for at some anatomical level. Biomechanical compensation may occur at both intra-pedal and supra-pedal levels based upon an individual's anatomy, flexibility, and biomechanics. A critical wounding point is reached in the insensate foot when compensation is not adequate to match the progression of neuropathic muscle imbalance (although out of the scope of this paper, Charcot neuroarthropathy appears to develop by this same progression).

WOUNDING BIOMECHANICS OF PLANTAR FIRST METATARSAL ULCERS

The approach described in this section addresses peri-ulcerative pathomechanics. The recommended approach is to examine each patient while nonweight bearing, weight bearing, and during gait. Aside from ankle equinus, which will be discussed below it is assumed that the clinician will further evaluate each patient for additional contributory causes including: previous trauma, Charcot deformity, congenital and developmental

conditions, arthritic conditions of the foot and ankle as well as abnormal pelvis, hip, and knee motion. Although an in depth review of these conditions is beyond this paper's scope, a thorough consideration is required to optimize curative healing.

ANKLE EQUINUS

Ankle equinus is a nearly universal contributing clinical finding when neuropathic ulcers are present. Addressing the gastrosoleal complex is a key step towards curative surgical offloading for all neuropathic ulcers described. This abnormality needs to be repaired either through gastrocnemius recession or Achilles tendon lengthening to prevent ulcer recurrence. (To avoid redundancy the inclusion of this procedure is assumed and will not be discussed within each section below.)

PLANTAR FIRST METATARSAL ULCERS: POSITION AND ETIOLOGY

Of the 3 ulcer locations addressed herein, the plantar central-medial first metatarsal head ulcer provides the most subtle of causative hints. Specifically, a medial shift of the ulcer's center point by just a few millimeters predictably implicates a different etiology. Seeming insignificant, this slight positional shift is a critical finding that significantly affects procedure selection.

The biomechanical marker that differentiates medially positioned lesions and the central lesion is first metatarsophalangeal joint (MTPJ) stability. Medial and central-medial lesions occur with an unstable first MTPJ, whereas, central lesions occur with a stable first MTPJ. Plantar medial ulcers (Figure 1) are commonly recognized as being associated with medial column overload and moderate to severe hallux valgus. Similarly, the central-medial ulceration (Figure 2) is associated with medial column overload and mild to moderate hallux valgus. Hallux limitus in either functional or structural forms may also contribute to formation of medially positioned lesions.

Different from medial shifted ulcers, the plantar central metatarsal wound (Figure 3) is classically associated with a rigidly plantarflexed first metatarsal, cavus foot type, stable medial column, and internal hip/femoral rotation. Although hallux malleus may be coexistent with the plantar central ulcer the primary ulcerative force is exerted by the rigidity of the first metatarsal (typically hallux malleus causes ulceration of the distal hallux or dorsal hallux interphalangeal joint).



Figure 1. Plantar medial ulcer.



Figure 2. Central-medial ulceration.



Figure 3. Plantar central metatarsal wound.

PROCEDURE SELECTION FOR PLANTAR FIRST METATARSAL HEAD ULCERS

Several procedural options exist for plantar first metatarsal ulcers (Table 2). The selection of surgical procedures to curatively heal plantar first metatarsal ulcers is patient-centered and involves consideration of several factors including: vascular and metabolic status, functional level, bone quality, patient compliance, patient age, presence of infection, and postoperative social support. In general, osteotomy is reserved for younger and more active patients; whereas joint resection is indicated for older, more sedentary patients, and for cases with concomitant osteomyelitis.

Curative healing of central metatarsal head ulceration is achieved by alleviating the pressure of a rigidly plantarflexed first metatarsal. Soft tissue procedures such as the Jones teno-suspension and peroneus longus lengthening are ineffective in the presence of a rigid deformity and do not endure progressive neuropathy. Osseous procedures are reliably curative and a proximally based dorsiflexory wedge osteotomy is preferred to preserve first MTPJ function. Joint preservation procedures are also primarily recommended for plantar medial and central-medial first metatarsal ulcers. Procedure selection is based upon presenting deformity, severity, and patient compliance and support.

When joint resection is indicated, preserving as much bone as possible is the principle towards safeguarding postoperative function. This surgical principle is common among recommended joint resection procedures for first MTPJ ulcer sites, and is based upon maintaining tendon function and tension by preserving lever arm (osseous) length. Towards achieving this goal, minimal resection of the hallux proximal phalanx base or of the first metatarsal head is recommended for the Keller and mini-Mayo procedures, respectively.

When performing the Keller procedure, postoperative function is enhanced when the dorsal to plantar base resection is 1-2 mm distal to the concavity of the proximal phalanx base (Figure 4). This level of resection usually equates to 20% of the proximal phalanx's length. When carrying out the mini-Mayo procedure, preservation of postoperative function is maximized when the oblique dorsal distal to plantar proximal head resection is carried out just proximal to the dorsal articular cartilage (Figure 5). The sesamoids are undisturbed unless osteomyelitis is present.

Use of fixation for joint resection procedures allows both early weight bearing in a protective/padded shoe or boot and enhances functional outcome. An intramedullary pin is recommended fixation for Keller and mini-Mayo

Table 2

FOCAL PROCEDURE OPTIONS FOR PLANTAR FIRST METATARSAL HEAD ULCERS*

Plantar Central	Elevation first metatarsal osteotomy DFWO Mini-Mayo (Partial first metatarsal head excision)
Plantar Central-Medial	Distal first metatarsal osteotomy -Austin -1st MTPJ Decompression Keller
Plantar Medial	Distal first metatarsal osteotomy -Austin First MTPJ arthrodesis Proximal first metatarsal osteotomy -CBWO Lapidus Keller

*Additional procedures such as gastrocnemius recession or TAL may be indicated.

procedures postoperatively to allow for stabilization and controlled fibrosis of the first MTPJ for 2-3 weeks postoperatively. A large diameter intramedullary pin (2 mm or larger) is utilized and especially useful for diabetic neuropathic patients in whom fixation failure (pin fracture) is common. The pin is advanced proximally across the nearest 1 or 2 subchondral plates to ensure stabilization. For Keller procedures, the pin is usually advanced only into the metatarsal (Figure 6A). It may be advanced across the first metatarsal-medial cuneiform joint in obese and less compliant patients (Figure 6B). For mini-Mayo procedures, this fixation is advanced across the first metatarsal-medial cuneiform joint.

Preoperative education and cautious postoperative management of the neuropathic patient improves the likelihood of successful outcomes. Weekly office visits with serial radiographs is recommended until fixation is removed. Physical therapy is typically prescribed 3 weeks postoperatively for gait stabilization, balance training, strengthening, and flexibility. Once edema has subsided, the patient is allowed to transition into supportive shoes.



Figure 4A. Preoperative Keller arthroplasty radiograph.



Figure 4B. Postoperative Keller arthroplasty radiograph. Note minimal resection (20%) of the proximal phalanx base, which is performed 1-2mm distal to the articular cartilage. Minimal resection maintains osseous length which optimizes tendon function. This procedure is recommended when osteotomy or arthrodesis with internal fixation is not a reconstructive option.



Figure 5A. Preoperative mini-Mayo (partial first metatarsal head resection) radiograph.



Figure 5B. Postoperative mini-Mayo (partial first metatarsal head resection) radiograph. The dorsal distal to plantar proximal resection is initiated just proximal to the dorsal articular cartilage. This procedure is recommended when first metatarsal DFWO is not indicated. The sesamoid complex is not disturbed unless osteomyelitis is present.



Figure 6A. Towards preventing pin fracture, use of a large diameter (2 mm) intramedullary pin is recommended fixation for the neuropathic patients. Typically this is advanced proximally across one intact cortex.



Figure 6B. Advancing the pin proximally into the medial cuneiform is recommended for patients who are obese and have difficulty remaining nonweight bearing. This more proximal advancement is also preferred fixation for the mini-Mayo procedure.



Figure 6C. Advancing the pin proximally into the medial cuneiform.

CONCLUSION

A slight change in position of plantar first metatarsal ulcers is functionally meaningful and prompts the observant clinician to more closely evaluate the patient's contributing biomechanics. In addition to ulcer location, comprehensive examination is necessary to exact ulcerative causing biomechanics. This examination approach directs the offloading surgeons' procedure selection for successful curative healing of plantar first metatarsal diabetic ulcers.

REFERENCE

1. Armstrong DG, Lavery LA, Harkless LB. Validation of a diabetic wound classification system. The contribution of depth, infection, and ischemia to risk of amputation. *Diabetes Care* 1998;21:855-9.