TREATMENT OF DIABETIC FOOT INFECTION

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a multidisciplinary diabetic foot care team

- Infectious diseases specialist
- Endocrinologist
- Nephrologist
- Surgeon
- Orthopedics
- Vascular surgeon or interventional cardiologist
- Psychiatrist or psychologist
- Plastic surgeon
- Prosthetics
- Nurse dressing
- Microbiologist
Diabetic foot ulcers may have multiple causes
Neuropathy

Motor
- Muscle wasting
- Foot weakness
- Postural deviation

Sensory
- ↓ nociception
- ↓ Proprioception, Unawareness of foot position
- Stress on bones & joints
- Plantar pressure
- Deformities, stress and shear pressures
- Callus formation

Autonomic
- Reduced sweating
- A-V Shunt* open Permanent
- Dry skin
- Increase foot Blood flow
- Fissures and cracks
- Bulging foot veins, Warm foot

Trauma

Infection

*Shunts: blood vessels that bypass capillaries and lead directly from arteries to veins
“Then how are blood vessels affected?”

- High blood sugar expedites **atherosclerosis** giving peripheral vascular disease (reduction of blood supply to the foot).
- The delivery of essential nutrients and oxygen to the foot is compromised leading to anaerobic infections and tissue necrosis.
Acute trauma: abrasions and burns occur often due to the absence of nociception. Poor wound healing makes ulcerations more likely occur.

Chronic trauma: reduced motor function results in a high arch. Together with decreased proprioception, this creates classical deformed foot shapes (explained later). These result in bony prominences which, when coupled with high mechanical pressure on the overlying skin, results in ulceration.
Assessment Some Common Foot Deformities

Claw toes

Nail deformity

Charcot foot deformity
• Diabetes also is associated with **immunological perturbations**
• especially reduced polymorphonuclear leukocyte function
• impaired humoral
• impaired cell-mediated immunity
Clinicians should evaluate a diabetic patient presenting with a foot wound at 3 levels:

- the patient as a whole,
- the affected foot or limb
- the infected wound
• local and **systemic inflammatory responses** to infection may be diminished in patients with peripheral neuropathy or arterial insufficiency
### History

- burning, tingling, numbness of the foot and nocturnal leg pain indicate cutaneous *sensory deficits*
- Note that in \(~35\%\) of patients who are asymptomatic, neuropathy can be detected by examination

### Examination

- Inspect deformities such as claw toes, hair loss, muscle atrophy and a high medial longitudinal arch (giving prominent metatarsal heads)
- Test for reduced power and reflexes that are evidence of muscular motor deficits.
- Test sensation by skin pinprick (spinothalamic tracts), proprioception and vibration (dorsal columns)
“So how do we know how well the blood is flowing?”

- History: claudication (calf pain after walking a specific distance) that is relieved by rest. However, this is uncommon in people with diabetes due to the concomitant neuropathy.

- Examination: Palpate the foot for temperature (cool in PVD); palpate the dorsalis pedis pulse and, if absent, the posterior tibial pulse. Test for Bergers angle (at which leg turns white) and reactive hyperaemia (leg turns bright red on declining back to the ground).
Structural abnormalities and deformities lead to bony prominences which are associated with high mechanical pressure on the overlying skin. This results in ulceration, particularly in the absence of a protective pain sensation and when shoes are unsuitable. Ideally, the deformity should be recognised early and accommodated in properly fitting shoes before ulceration occurs.

Common abnormalities / deformities include:

i. Callus
ii. Bunion
iii. Hammer toes
iv. Claw toes
v. Charcot foot
vi. Nail deformities

Note: It is vital to inspect the patients shoes as part of the assessment!
• The initial assessment should also include an evaluation of the patient’s social situation and psychological state
• which may influence his or her ability to comply with recommendations and appear to influence wound healing
assess a diabetic patient presenting with a foot infection

• Infection in foot wounds should be defined clinically by the presence of inflammation or purulence, and then classified by severity
• This approach helps clinicians make decisions about which patients to hospitalize to send for imaging procedures
• whom to recommend surgical interventions
How to Classify Infection.
<table>
<thead>
<tr>
<th>Clinical Manifestation of Infection</th>
<th>PEDIS Grade</th>
<th>IDSA Infection Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms or signs of infection</td>
<td>1</td>
<td>Uninfected</td>
</tr>
</tbody>
</table>

Infection present, as defined by the presence of at least 2 of the following items:

- Local swelling or induration
- Erythema
- Local tenderness or pain
- Local warmth
- Purulent discharge (thick, opaque to white or sanguineous secretion)

Local infection involving only the skin and the subcutaneous tissue (without involvement of deeper tissues and without systemic signs as described below). If erythema, must be >0.5 cm to ≤2 cm around the ulcer.

Exclude other causes of an inflammatory response of the skin (eg, trauma, gout, acute Charcot neuro-osteoarthropathy, fracture, thrombosis, venous stasis).

Local infection (as described above) with erythema > 2 cm, or involving structures deeper than skin and subcutaneous tissues (eg, abscess, osteomyelitis, septic arthritis, fasciitis), and

No systemic inflammatory response signs (as described below)

Local infection (as described above) with the signs of SIRS, as manifested by ≥2 of the following:

- Temperature >38°C or <36°C
- Heart rate >90 beats/min
- Respiratory rate >20 breaths/min or PaCO₂ <32 mm Hg
- White blood cell count >12 000 or <4000 cells/μL or ≥10% immature (band) forms

Abbreviations: IDSA, Infectious Diseases Society of America; PaCO₂, partial pressure of arterial carbon dioxide; PEDIS, perfusion, extent/size, depth/tissue loss, infection, and sensation; SIRS, systemic inflammatory response syndrome.

Ischemia may increase the severity of any infection, and the presence of critical ischemia often makes the infection severe. Systemic infection may sometimes manifest with other clinical findings, such as hypotension, confusion, vomiting, or evidence of metabolic disturbances, such as acidosis, severe hyperglycemia, and new-onset azotemia [29, 43, 44].
need to be hospitalized

- all patients with a severe infection,
- selected patients with a moderate infection with complicating features eg, severe peripheral arterial disease
- lack of home support
- any patient unable to comply with the required outpatient treatment
- failing to improve with outpatient therapy
obtain specimen(s) for culture

- obtained specimens for culture prior to starting empiric antibiotic therapy if possible
- Cultures may be unnecessary for a mild infection in a patient who has not recently received antibiotic therapy
• sending a specimen for culture that is from deep tissue
• obtained by biopsy or curettage after the wound has been cleansed and debrided
• We suggest avoiding swab specimens, especially of inadequately debrided wounds, as they provide less accurate results
<table>
<thead>
<tr>
<th><strong>Do</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Obtain an appropriate specimen for culture from almost all infected wounds</td>
</tr>
<tr>
<td>Cleanse and debride the wound before obtaining specimen(s) for culture</td>
</tr>
<tr>
<td>Obtain a tissue specimen for culture by scraping with a sterile scalpel or dermal curette (curettage) or biopsy from the base of a debrided ulcer</td>
</tr>
<tr>
<td>Aspirate any purulent secretions using a sterile needle and syringe</td>
</tr>
<tr>
<td>Promptly send specimens, in a sterile container or appropriate transport media, for aerobic and anaerobic culture (and Gram stain, if possible)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Do not</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Culture a clinically uninfected lesion, unless for specific epidemiological purposes</td>
</tr>
<tr>
<td>Obtain a specimen for culture without first cleansing or debriding the wound</td>
</tr>
<tr>
<td>Obtain a specimen for culture by swabbing the wound or wound drainage</td>
</tr>
</tbody>
</table>
antibiotic regimen for a diabetic foot infection

• We recommend that clinically uninected wounds not be treated with antibiotic therapy

• select an empiric antibiotic regimen on the basis of the severity of the infection and the likely etiologic agent
• For **mild to moderate infections** in patients who have not recently received antibiotic treatment,
• therapy just targeting aerobic **GPC** is sufficient
• For most severe infections
• starting broad-spectrum empiric antibiotic therapy, pending culture results and antibiotic susceptibility data
• Empiric therapy directed at *Pseudomonas aeruginosa* is usually unnecessary except for patients with risk factors for true infection with this organism
• definitive therapy be based on
• the results of an appropriately obtained culture and sensitivity testing of a wound specimen
• as well as the patient’s clinical response to the empiric regimen
• **parenteral therapy** for all severe, and some moderate, DFIs, at least initially

• with a switch to **oral agents** when the patient is systemically well and culture results are available

• Clinicians can probably use highly bioavailable oral antibiotics alone in most mild, and in many moderate, infections

• and topical therapy for selected mild superficial infections
• For **severe infections**, and for more **extensive, chronic moderate infections**

• it is safest to promptly commence therapy with a broad-spectrum regimen

• The agent(s) should have activity against GPC, as well as common gram-negative and obligate anaerobic organisms to ensure adequate tissue concentrations
Clinicians must also consider covering ESBL-producing gram-negative isolates, especially in countries in which they are relatively common.
Methicillin-Resistant S. aureus.

• many studies have demonstrated the increasing role of MRSA in DFI (10-30%)
Factors noted to increase the risk for infection with MRSA

• prior long-term or inappropriate use of antibiotics
• previous hospitalization
• long duration of the foot wound
• the presence of osteomyelitis
• nasal carriage of MRSA
• history of MRSA infection
The patient has a history of previous MRSA infection or colonization within the past year.

The local prevalence of MRSA is high enough (perhaps 50% for a mild and 30% for a moderate soft tissue infection)

The infection is sufficiently severe
<table>
<thead>
<tr>
<th>Infection Severity</th>
<th>Probable Pathogen(s)</th>
<th>Antibiotic Agent</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (usually treated with oral agent[s])</td>
<td><em>Staphylococcus aureus</em> (MSSA); <em>Streptococcus spp</em></td>
<td>Dicloxacillin</td>
<td>Requires QID dosing; narrow-spectrum; inexpensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clindamycin(^b)</td>
<td>Usually active against community-associated MRSA, but check macrolide sensitivity and consider ordering a “D-test” before using for MRSA. Inhibits protein synthesis of some bacterial toxins</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Cephalexin</strong>(^b)</td>
<td>Requires QID dosing; inexpensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Levofloxacin(^b)</td>
<td>Once-daily dosing; suboptimal against <em>S. aureus</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Amoxicillin-clavulanate</strong>(^b)</td>
<td>Relatively broad-spectrum oral agent that includes anaerobic coverage</td>
</tr>
<tr>
<td></td>
<td>Methicillin-resistant <em>S. aureus</em> (MRSA)</td>
<td>Doxycycline</td>
<td>Active against many MRSA &amp; some gram-negatives; uncertain against streptococcus species</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Trimethoprim/sulfamethoxazole</strong>(^b)</td>
<td>Active against many MRSA &amp; some gram-negatives; uncertain activity against streptococci</td>
</tr>
<tr>
<td>Disease Severity</td>
<td>Pathogens</td>
<td>Treatment Options</td>
<td>Summary</td>
</tr>
<tr>
<td>------------------</td>
<td>-----------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Moderate (may be treated with oral or initial parenteral agent[s]) or severe (usually treated with parenteral agent[s])</td>
<td>MSSA; <em>Streptococcus</em> spp; Enterobacteriaceae; obligate anaerobes</td>
<td>Levofloxacin&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Once-daily dosing; suboptimal against <em>S. aureus</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cefoxitin&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Second-generation cephalosporin with anaerobic coverage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ceftriaxone</td>
<td>Once-daily dosing, third-generation cephalosporin</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Ampicillin-sulbactam</strong>&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Adequate if low suspicion of <em>P. aeruginosa</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Moxifloxacin&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Once-daily oral dosing. Relatively broad-spectrum, including most obligate anaerobic organisms</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Ertapenem</strong>&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Once-daily dosing. Relatively broad-spectrum including anaerobes, but not active against <em>P. aeruginosa</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tigecycline&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Active against MRSA. Spectrum may be excessively broad. High rates of nausea and vomiting and increased mortality warning. Nonequivalent to ertapenem + vancomycin in 1 randomized clinical trial</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Levofloxacin&lt;sup&gt;b&lt;/sup&gt; or ciprofloxacin&lt;sup&gt;b&lt;/sup&gt; with clindamycin&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Limited evidence supporting clindamycin for severe <em>S. aureus</em> infections; PO &amp; IV formulations for both drugs</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Imipenem-cilastatin</strong>&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Very broad-spectrum (but not against MRSA); use only when this is required. Consider when ESBL-producing pathogens suspected</td>
</tr>
</tbody>
</table>
IDSA Guideline for Diabetic Foot Infections • CID 2012;54 (15 June) • e151

**Pseudomonas aeruginosa**

**Vancomycin**

**Daptomycin**

**Linezolid**

**MRSA**

- **Piperacillin-tazobactam**
  - Used for broad-spectrum coverage of MRSA and other gram-negative bacteria. Effective against both MSSA and MRSA.

- **Vancomycin MICs for MRSA are generally lower than those for MSSA.**

- **When used >2 wk:** Requires serial monitoring of CPK levels due to the risk of rhabdomyolysis.

- **Expensive; increased risk of toxicities:**

**Vancomycin:**

- Gradually increasing TID/QID dosing is useful for broad-spectrum coverage. P. aeruginosa is an uncommon pathogen in diabetic foot infections except in special circumstances (2).

- Once-daily dosing requires serial monitoring of CPK levels.
<table>
<thead>
<tr>
<th>Infection Severity</th>
<th>Probable Pathogen(s)</th>
<th>Antibiotic Agent</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MRSA, Enterobacteriaceae, <em>Pseudomonas</em>, and obligate anaerobes</td>
<td>Vancomycin(^c), ceftazidime, cefepime, *piperacillin-tazobactam(^b), aztreonam,(^b) or a carbapenem(^b)</td>
<td>Very broad-spectrum coverage; usually only used for empiric therapy of severe infection. Consider addition of obligate anaerobe coverage if ceftazidime, cefepime, or aztreonam selected</td>
</tr>
</tbody>
</table>
Teicoplanin versus vancomycin for diabetic foot infection

- **Similar effect** of teicoplanin compared to vancomycin on clinical and microbiological cure.
- RR of **nephrotoxicity** was reduced by 34% when using teicoplanin.
- **Cutaneous rash, red man syndrome** and total adverse events were also less common with teicoplanin than vancomycin.
Advantages of Teicoplanin as Compared with Vancomycin

- Less nephrotoxicity
- Fewer anaphylactoid reactions
- Requires less monitoring
- More convenient to administer
  - Once daily iv bolus
  - Im injection
- Available for OPAT
Advantages of OPAT

• More beds available for other patients
• Savings of costs related to stay in hospital
• Less risk of nosocomial infection
• Quality of life for the patient and his/her family
• Parenteral dosing for most infections
  – Loading dose of 400 bid for 3 doses
  – Then 400 mg od
diagnose and treat osteomyelitis

- DFO may be present in up to 20% of mild to moderate infections and in 50%–60% of severely infected wounds

- Noninfectious neuro osteoarthropathy (Charcot foot) is sometimes difficult to distinguish from DFO, and they can coexist
• doing a **PTB test** for any DFI with an open wound
• obtaining **plain radiographs** of the foot
• but they have relatively low sensitivity and specificity
• using serial plain radiographs to diagnose or monitor suspected DFO
• For a diagnostic imaging test for DFO, we recommend using **MRI**
• **leukocyte or antigranulocyte scan**, preferably combined with a bone scan
• the **most definitive way** to diagnose DFO is by the combined findings on bone **culture and histology**

• When bone is debrided to treat osteomyelitis, we suggest sending a sample for culture and histology
differentiate osteomyelitis from cellulitis

• Taking together clinical and laboratory findings
  • ulcer depth >3 mm or **CRP** >3.2 mg/dL
  • ulcer depth >3 mm or **ESR** >60 mm/hour)
• Any ulcer with either a positive PTB test (ie, palpable hard, gritty bone) 
• or in which bone is visible is likely to be complicated by osteomyelitis 
• an adequate blood supply to the affected foot when an ulcer 
• especially if it is deep, does not heal after at least 6 weeks of appropriate wound care and off-loading.
Management of Diabetic Patients With Osteomyelitis of the Foot

• If the plain radiograph has changes suggestive of osteomyelitis (cortical erosion, active periosteal reaction, mixed lucency, and sclerosis), treat for presumptive osteomyelitis,

• preferably after obtaining appropriate specimens for culture (consider obtaining bone biopsy, if available).
• If the radiographs show no evidence of osteomyelitis

• treat the patient with antibiotics for up to 2 weeks if there is soft tissue infection, in association with optimal care of the wound and off-loading

• Perform repeat radiographs of the foot 2–4 weeks after the initial radiographs.
• If these repeat bone radiographs remain normal but **suspicion of osteomyelitis remains**:

• Where the depth of the wound is decreasing and the PTB test is negative, osteomyelitis is unlikely.
• Where the wound is not improving or the PTB test is positive, 1 of the following choices should be considered:

• Additional imaging studies, preferably MRI. If results are negative, osteomyelitis is unlikely.

• **Bone biopsy** for culture and histology.

• **Empiric treatment**: Provide antibiotic therapy (based on any available culture results, and always covering at least for S. aureus) for another 2–4 weeks and then perform radiography again.
Table 10. Approach to Treating a Patient With Diabetic Foot Osteomyelitis

When to consider a trial of nonsurgical treatment

- No persisting sepsis (after 48–72 h if on treatment)
- Patient can receive and tolerate appropriate antibiotic therapy
- Degree of bony destruction has not caused irretrievable compromise to mechanics of foot (bearing in mind potential for bony reconstitution)
- Patient prefers to avoid surgery
- Patient comorbidities confer high risk to surgery
- No contraindications to prolonged antibiotic therapy (eg, high risk for *C. difficile* infection)
- Surgery not otherwise required to deal with adjacent soft tissue infection or necrosis

When to consider bone resection

- Persistent sepsis syndrome with no other explanation
- Inability to deliver or patient to tolerate appropriate antibiotic therapy
- Progressive bony deterioration despite appropriate therapy
- Degree of bony destruction irretrievably compromises mechanics of foot
- Patient prefers to avoid prolonged antibiotics or to hasten wound healing
- To achieve a manageable soft tissue wound or primary closure
- Prolonged antibiotic therapy is relatively contraindicated or is not likely to be effective (eg, presence of renal failure)
### Table 11. Suggested Route, Setting, and Duration of Antibiotic Therapy, by Clinical Syndrome

<table>
<thead>
<tr>
<th>Site of Infection, by Severity or Extent</th>
<th>Route of Administration</th>
<th>Setting</th>
<th>Duration of Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soft-tissue only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>Topical or oral</td>
<td>Outpatient</td>
<td>1–2 wk; may extend up to 4 wk if slow to resolve</td>
</tr>
<tr>
<td>Moderate</td>
<td>Oral (or initial parenteral)</td>
<td>Outpatient/inpatient</td>
<td>1–3 wk</td>
</tr>
<tr>
<td>Severe</td>
<td>Initial parenteral, switch to oral when possible</td>
<td>Inpatient, then outpatient</td>
<td>2–4 wk</td>
</tr>
<tr>
<td>Bone or joint</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No residual infected tissue (eg, postamputation)</td>
<td>Parenteral or oral</td>
<td>...</td>
<td>2–5 d</td>
</tr>
<tr>
<td>Residual infected soft tissue (but not bone)</td>
<td>Parenteral or oral</td>
<td>...</td>
<td>1–3 wk</td>
</tr>
<tr>
<td>Residual infected (but viable) bone</td>
<td>Initial parenteral, then consider oral switch</td>
<td>...</td>
<td>4–6 wk</td>
</tr>
<tr>
<td>No surgery, or residual dead bone postoperatively</td>
<td>Initial parenteral, then consider oral switch</td>
<td>...</td>
<td>$\geq$ 3 mo</td>
</tr>
</tbody>
</table>

### Table 12. Signs of a Possibly Imminently Life-Threatening Infection

- Evidence of systemic inflammatory response
- Rapid progression of infection
- Extensive necrosis or gangrene
- Crepitus on examination or tissue gas on imaging
- Extensive ecchymoses or petechiae
- Bullae, especially hemorrhagic
- New onset wound anesthesia
- Pain out of proportion to clinical findings
- Recent loss of neurologic function
- Critical limb ischemia
- Extensive soft tissue loss
- Extensive bony destruction, especially midfoot/heel
- Failure of infection to improve with appropriate therapy

In clinical settings with less advanced healthcare available, infection severity may make an infection limb-threatening.

...drainage (Figure 1). Prompt and adequate surgical debridement, including limited resections or amputations, can decrease the likelihood that a more extensive procedure will be needed [227, 254]. The progressive development of gas within the foot, especially in the presence of bone, can rapidly lead to irreparable tissue damage.

Various publications suggest that there are between 2 to 4 compartments in the foot; the 4 in the plantar, 3 in the medial, lateral, and central plantar and deep plantar [255, 256]. The key element of any surgical approach is to decompress all the affected compartments.
Debridement is the removal of necrotic and dead tissue in order to enhance healing.

Debridement is undertaken to:

- Remove callus in neuropathic foot to lower plantar pressure
- Assess the true dimension of the ulcer
- Drain exudate and remove dead tissue to render infection less likely
- Take a deep swab for culture
- Encourage healing and restore a chronic wound to an acute wound aids
- granulation tissue formation and reepithelialization

Forcep and a scalpel is the usual technique by cutting away of all slough and non-viable tissue.
• Debridement can usually be undertaken as a clinic or **bedside procedure** and without anesthesia, although patients who do not have a loss of protective sensation
• Debridement may be relatively contraindicated in wounds that are primarily ischemic
• We generally prefer **sharp debridement** (with scalpel, scissors, or tissue nippers) to other techniques
• Other methods of debridement include autolytic dressings
• and biological debridement with maggots
### Table 13. Questions to Ask When Dealing With Nonresponse or Recurrence

<table>
<thead>
<tr>
<th>Question</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Is there a failure of wound healing?</td>
<td></td>
</tr>
<tr>
<td>- Is the patient adhering to the wound care regimen?</td>
<td></td>
</tr>
<tr>
<td>- Has the wound been adequately debrided?</td>
<td></td>
</tr>
<tr>
<td>- Has the wound been appropriately dressed?</td>
<td></td>
</tr>
<tr>
<td>- Has the wound been adequately off-loaded?</td>
<td></td>
</tr>
<tr>
<td>- Is there unidentified or untreated ischemia?</td>
<td></td>
</tr>
<tr>
<td>- Is the lesion malignant?</td>
<td></td>
</tr>
<tr>
<td>- Is there undiagnosed or improperly treated infection?</td>
<td></td>
</tr>
<tr>
<td>Is there a failure of infection to respond?</td>
<td></td>
</tr>
<tr>
<td>- Is there unidentified or untreated limb ischemia?</td>
<td></td>
</tr>
<tr>
<td>- Is there unidentified necrotic soft tissue or bone?</td>
<td></td>
</tr>
<tr>
<td>- Is there an undrained abscess?</td>
<td></td>
</tr>
<tr>
<td>- Has the wound been adequately debrided?</td>
<td></td>
</tr>
<tr>
<td>- Is there osteomyelitis that has not yet responded?</td>
<td></td>
</tr>
<tr>
<td>- Is there an untreated or an unidentified pathogen?</td>
<td></td>
</tr>
<tr>
<td>- Is there an antibiotic delivery problem?</td>
<td></td>
</tr>
<tr>
<td>- Is there an antibiotic nonadherence issue?</td>
<td></td>
</tr>
<tr>
<td>- Have all metabolic aberrations been corrected?</td>
<td></td>
</tr>
</tbody>
</table>
Off-loading Pressure.

- The choice of off-loading modality should be based on the wound’s location
- the presence of any associated PAD
- the presence and severity of infection
- and the physical characteristics of the patient and their psychological and social situation.

Dr. Khorvash
• Redistribution of pressure off the wound to the entire weight-bearing surface of the foot (“off-loading”).

• While particularly important for **plantar wounds**, this is also necessary to relieve pressure caused by dressings, footwear, or ambulation to any surface of the wound.
Air cast (walking brace)
A bivalved cast with the halves joined together with Velcro strapping. The cast is lined with 4 air cells which can be inflated with a hand pump to ensure a close fit. The cast can be removed easily by patients to check their ulcers and before going to bed.

Scotch cast boot
A simple, removable boot made of stockinette, soffban bandage, felt and fibreglass tape.

Total contact cast
It is a close-fitting plaster of paris and fibreglass cast applied over minimum padding. It is very efficient method of redistributing plantar pressure, and should be reserved for plantar ulcers that have not responded to other casting treatments.
the effects of NPWT

- increased local blood flow
- formation of granulation tissue
- Decrease oedema
- Angiogenesis
- Reduce infection rate and decreased bacterial colonization
- Faster wound healing results in an overall decrease in hospitalization
- avoids the additional morbidity of chronic wounds
(Biograft) is placed within the wound and covered by a clear, vapor permeable, plastic dressing. Continuous suction is kept out of the wound (arrows).
CASE PRESENTATION

Mr. 41 years old with a history of uncontrolled diabetes

described the patient with swelling and a wound on the left foot, with a yellow discharge. He has received treatment under the program + tangerine for 22 days. The wound has not healed, and the foot is exposed to infection. He has received antibiotic treatment from Dr. Khorvash.

Under the program + tangerine for 22 days. The wound has not healed, and the foot is exposed to infection. He has received antibiotic treatment from Dr. Khorvash.

در تاریخ ۸۰.۹/۱۹ با تورم و زخم ۷×۷ سانتی‌متر می‌باشد. 
در زوار چربی زرد یک رنگ فراوان بر روی پودر و گانگری پزشکی شده است.

در برابر نسوج نگریزه و تخیله کف پا و فاصلات موی
dr.khorvash