Diabetic Foot Infections

Background

* Annual incidence of foot ulcer among diabetics: 2.2% (5% among US veterans with diabetes) with lifetime incidence of 20-34%. Globally, > 9 million people develop a diabetic foot ulcer annually and more than half of these become infected1
* Lower extremity infections occur in diabetics due to a confluence of risk factors:
  + Hyperglycemia impairs multiple aspects of immune response (chemotaxis, adherence, opsonization, phagocytosis)
  + Vascular insufficiency – impairs wound healing, delivery of Abx
  + Neuropathy – Local trauma, delayed recognition
* Most diabetic foot infections develop as the result of neuropathic or ischemic wounds
  + Not all diabetic foot wounds are infected, but all will have bacteria if cultured

Microbiology

* Often polymicrobial
  + For mild infection main drivers are Gm + (MSSA, MRSA, GAS, CoNS)
  + Deeper, necrotic infections have more gram negatives and anaerobes
* *Pseudomonas aeruginosa* is occasionally recovered, however it often doesn’t need to be specifically treated
  + SIDESTEP trial of 586 patients with DFI compared ertapenem to pip-tazo x at least 5 days followed by up to 23 days of amox-clav. Favorable response was 94% in pip-tazo arm and 92% in ertapenem arm (seen even in patients who grew pseudomonas)2

Diagnosis

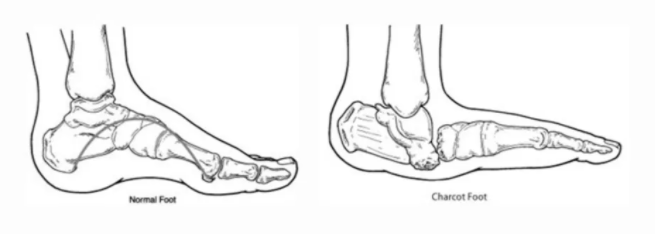
* Based on clinical manifestations (two or more) NOT microbiology:
  + Erythema, warmth, tenderness, swelling, induration, purulence
* Severity assessment & amputation risk: PEDIS (Perfusion, Extent, Depth, Infection, Sensation)
  + Mild: <2cm of surrounding erythema, no deep structures involved, no SIRS
  + Moderate: >2cm of erythema, deep structures involved, no SIRS
  + Severe: SIRS present
* Ankle-Brachial Index (ABI) often performed to evaluate for underlying bone involvement and PAD
* When infection is present culture from deep tissue is helpful, the role of superficial wound culture is limited

Osteomyelitis

* Factors associated with osteomyelitis:
  + Bone visible
  + Probe to bone
  + Ulcer >2cm
  + ESR >70
  + Ulcer duration >1-2 weeks
* For grossly visible bone osteomyelitis is assumed to be present. For other factors the next diagnostic steps are:
  + X ray
  + MRI if X ray is normal or inconclusive

Management

* Wound care – Perhaps the most important component of treatment in getting ulcers to heal
  + Local debridement of necrotic tissue – usually with sharp debridement but can use enzymatic, autolytic, and biological debridement. Larval therapy has been shown to increase duration of antibiotic free days as part of ‘wound hospice’3,4
  + Dressings to keep clean and moist (but not soaked) – no high-quality evidence for any specific dressing
  + Negative pressure wound therapy (Wound vac) may be a beneficial adjunct. Meta-analysis showed increased chance of wound healing (RR 1.4) and reduced risk of amputation (RR 0.33)5
* Antibiotics – Limited data to support any specific empiric regimen
  + For mild disease oral antibiotics for gram positives are usually enough (eg cefadroxil, cefalexin)
  + For moderate-severe disease usually use upfront IV antibiotics and add coverage for gram negatives and anaerobes (eg amp-sulbactam, Ceftriaxone +/- flagyl, Ertapenem)
  + If history of MRSA, add MRSA coverage (doxy or TMP/SMX for PO, Vanco or Dapto for IV)
  + If significant risk factors for pseudomonas can add pseudomonas coverage but generally not needed
  + Can narrow based on culture results
    - Often don’t need to treat every bacteria recovered (ie Enterococcus, CoNS)
  + Duration
    - If no osteomyelitis, treat until no longer clinically infected (1-2 weeks)
    - For osteomyelitis, it’s its own separate primer re Abx selection, IV v PO, but duration and role of rifampin remains unclear. Most would do at least 6 weeks but pilot study showed non-inferiority of 3 weeks6
* Surgery
  + Sharp debridement of necrotic soft tissue needed for severe and most moderate infections
    - Particularly important if abscess, joint involvement, necrosis, or crepitus
  + For bone involvement, debate over when bone resection and amputation are needed (likely needed for necrotic bone, but may not be necessary for infected but viable bone)
    - Appropriately, decision is often guided by patient preference

Charcot’s Foot

* Pathogenesis likely multifactorial with mechanical and vascular factors due to diabetic neuropathy and vasculopathy
* Development of Charcot’s increases likelihood of amputation by a factor of 6

References:

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