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

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