Skin & Soft-Tissue Infections

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USAID PEER/Liberia ID curriculum
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Goals

• Review several major presentations of skin & soft tissue infection
• Note importance of rapid diagnosis in specific diseases
• This is not exhaustive list
75 YO F WITH WORSENING LLE PAIN X 72 HOURS

- Fever 102.2 F
- Hx: DM, HTN
- Meds: lisinopril, amlodipine, insulin
- Exam: tachycardic, sick not toxic, hot swollen tender red/purple LLE, pulse 2+, no crepitus
HOW TO THINK ABOUT ID CASES

Non-infectious causes

Opportunistic (i.e. HIV)

"Routine"

Gram Positives

Gram Negatives

Anaerobes

Viruses

Fungi

Parasites & Protozoa

"Weird" (i.e. seek references)

Other bacteria:
--Intracellular
--Acid Fast/Modified
--Spirochetes & other
GIVEN THIS, WHAT WOULD YOU START?

- A. Start vanco
- C. Start vanco & chloramphenicol
- D. Start vanco, ceftriaxone, metronidazole
- E. Start ceftriaxone
- F. Start gentamicin
GIVEN THIS, WHAT WOULD YOU START?

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- C. Start vanco & chloramphenicol
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- **E. Start ceftriaxone**
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ROUTINE SOFT-TISSUE INFECTION (“CELLULITIS”): HOW TO APPROACH

- **Probe into the DM hx.** People with A1Cs of 12 are in a very different risk category than those at 7!
- **Then** think about microbiology:
  - **Strep** is overwhelmingly most likely bug
  - **Staph** forms abscesses—it looks different
- Outpatient Tx: PCNs.
- Inpatient Tx: 1st gen cephalosporin (cefazolin)
“CELLULITIS” CON’T: HOW TO APPROACH

• Suppose the patient came in with severe pain
• Would you change your management?
• If so, how?
Necrotizing fasciitis

- “Pain out of proportion to exam”
- Usually Group A Strep
- Clostridium if wound involved
- Surgical emergency
- Clindamycin may have anti-toxin effect
WHAT IF HER FOOT LOOKED LIKE THESE PICTURES AND HER A1C WAS 11.6?
BAD DIABETIC FOOT INFECTION

- **A1Cs** tell the story
- Cells coated in sugar stop working through **non-enzymatic glycosylation**
  - “immune suppression”—neutrophils/MΦs cannot phagocytose
  - peripheral endothelium & nerve cell dysfxn
- **Polymicrobial** infection becomes a major consideration, esp **Pseudomonas, MRSA, GNRs**
- “Pain out of proportion” not as reliable an indicator
- **Broad spectrum coverage** is now appropriate: vanco plus pip-tazo (if avail); vanco/cipro/flagyl; maybe chloramphenicol
50 yo F with facial swelling x 48 hrs

- Erysipelas
- Strep pyogenes (Group A)
- Painful, swollen
- Purplish discoloration in lighter skinned pts (see next slide)
- Dx? Tx?
“Saint Anthony’s Fire”
Microbiology?
MSSA/MRSA

**Anti-MSSA**
- Dicloxacillin (po)
- Cephalexin (po)
- Oxacillin (IV)
- Nafcillin (IV)
- Cefazolin (IV)
- (+/- Fluoroquinolones)

**Anti-MRSA**
- TMP/SMX (po, IV)
- Doxycycline (po, IV)
- Vancomycin (IV not po)
- +/- clindamycin (po, IV)
- (+/- Fluoroquinolones)
THE RULES OF STAPH AUREUS

- #1: Staph *kills*
- #2: Staph *sticks*
- #3: Staph *goes everywhere*
- #4: Staph *recurs*
- #5: MRSA *probably* not more virulent than MSSA, only drug resistant (personal opinion)

Vanco: *not a great drug*...we use it *not because it’s better* but because it’s active against MRSA and we don’t have less toxic or less costly alternatives
Pt comes with complaint of painless rash

- What history questions do you want to ask?
- What test do you order to help rule in or rule out primary dx?
- Add’l tests?
- Anything else?
- Do you shake his hand?
High syphilis mortality in Liberia (2004 data)

Related: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6089383/#!po=10.2273
35 yo M

- Presenting with hair loss
- Not painful
- Not pruritic (itchy)
- Dx?
Tinea capitis

• “Ringworm of the scalp”
• A superficial mycotic (fungal) infection (aka “dermatophyte”)
• Fungi like hair follicles/shafts
• Leads to hair loss, poss permanent
• Tx: Griseofulvin po
Additional dermatophytes of scalp

- Tinea favosa (aka “favus”)
- Different species than that which causes tinea capitis
- But both diseases treated by griseofulvin
44 yo M with arm lesions

- Works as farmer
- No systemic illness, wt loss
- Treated once with antibiotics (can’t remember which) but no help
Sporotrichosis

- *Sporothrix schenckii* (fungus)
- Found in soil, thus farmers/gardners @ risk
-Usu cutaneous, but also
- Lung—can increase risk for TB or bacterial PNA from scarring
- Disseminated—seen in pts with advanced HIV, likes bone & brain
- Dx: culture, micro/path
- Tx: itraconazole or fluconazole
Two men, aged 45

- What is the differential dx?
Others
Mycetoma & Eumycetoma

- *Aka “Madura foot”*
- Many fungi & bacteria are causative
- Distinguished by color of discharge
- Mycetoma caused by *Actinomyces spp*
- Eumycetoma by fungi (aspergillus, exophiala, pseudallescheria, madurella, others)
Filariasis
(can also be podoconiosis)
35 yo M with ulcer

- Several weeks duration
- Also got abx but no help
- No other medical issues
Leishmaniasis

• Several *Leishmania* spp (*L. major*, *L. donovani*, *L. infantum*)
• This is cutaneous form ("cheese pizza" with heaped-up border)
• Can also present as disseminated cutaneous, mucocutaneous, and visceral (kala azar)
• Visceral (kala azar) common cause of splenomegaly
• Vector: sandfly
• Dx: culture, pathology, PCR becoming more common
Leishmaniasis con’t

• Always include in differential of HIV patient, esp with diffuse cutaneous leish, or in someone with HIV & wasting (dx: bone marrow)

• Tx: “pentavalent antimonials” e.g. Na⁺ stibogluconate (some probs with resistance), amphotericin, miltefosine (new), ?fluconazole
Sporothrix & Leishmania, micro
Patients (adult & pediatric) with skin lesions
Leprosy

Multi- vs. Paucibacillary
Lesions distinguished by…?
Tx?