wound debridement and exploration. After completion, emergent air transport arranged to burn center for wound care. Patient is intubated and prior to transport received gentamicin, Zosyn, and vancomycin.

57 y/o M presents to ED with GI upset x6 days followed by expanding blister on arm x3 days. Arm presents with weeping blisters, skin breakdown, edema, and generalized jaundice noted. VS are BP 81/42, HR 140, RR 22. Labs are lactate 10.9, WBC 50.3 (left shift), BGL 664. Emergent air transport to surgical destination requested. Prior to transport patient received vancomycin and Invanz - during transport 3L NS infused and Phenylephrine started.

These three case studies have one diagnosis in common: the condition popularly known as flesh-eating bacteria. This unfortunately is a mischaracterization since the bacteria do not "eat" the flesh. Clinically, necrotizing cellulitis or fasciitis is a bacterial infection which leads to aggressive tissue destruction and systemic shock. Without equally as aggressive treatment, necrotizing infection will lead to death. The key to a positive patient outcome is rapid recognition of the bacterial process, swift initial interventions, followed by critical care transport to a specialty center (where possible) for long-term management.

Cellulitis vs Fasciitis

Cellulitis is a localized superficial infection to the skin and adjacent soft tissues (Ratner, 2015, p. 1323). Fasciitis is an infection of the deeper tissues with progressive destruction of the muscle fascia and subcutaneous fat. Air and/or fluid blisters may form, skin sloughing occurs, tissue necrosis is visible, and systemic signs will likely be present. Surgical exploration is the only standard for distinguishing how

NCME Up-2-Date

Necrotizing Soft Tissue Infections

October2017

NCME Case Studies and Introduction:

28 y/o M presents to ED with 1 day history of arm rash and blister. Wound was lanced with a razor blade by the patient PTA. Wound is now swollen with expanding margins around the arm, and patient is febrile with rigor, and considerable pain. Vitals are BP 123/83, HR 121, T 104.4F. Labs reveal lactate 3.6, WBC 12.1, CT imagining shows SQ air. Emergent air transport arranged for surgical destination. Patient received Tylenol, clindamycin, Ancef, vancomycin, 2L NS prior to transport..

71 y/o F presents to rural ED after 3 day history of worsening thigh rash with developing necrotic margins. ED labs reveal lactate 1.6, WBC 17, CT imaging shows SQ air in thigh. From ED patient sent to local OR for

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involved the infection is. Anesthesia, or loss of sensation of the infected area, and flu-like symptoms may precede necrosis and aid in the determination of fasciitis (Stevens & Baddour, 2017). More aggressive treatment is required for necrotizing fasciitis than cellulitis, and will be the area of focus for this UpToDate.

Pathogenesis

Necrotizing fasciitis is classified as two types based on the predominant bacterial source. Treatment course is similar for both.

Risk factors: diabetes (most significant), PVD, immunocompromised, post-surgical wounds

Common locations: lower extremities (diabetics and/or PVD patients), cervical spine (dental/pharyngeal origin), perineum (Fournier's gangrene), umbilicus and genitalia in neonates (often MRSA-caused)

Type I

Type I, a synergistic polymicrobial infection of aerobic and anaerobic bacteria. Members include, bacteroides, clostridium, with non-group A streptococci, and enterobacteriaceae (e.g. E. coli).

Type II

Type II, a monomicrobial infection most commonly by group A Streptococcus (GAS).

GAS can lead to streptococcal toxic shock syndrome (STTS). A patient with STTS will develop hypotension and renal failure within four to eight hours of the skin signs (Stevens & Baddour, 2017). Highest mortality rate exists with STTS.

Pathophysiology

Tissue destruction is thought to occur as the result of a cascade of reactions after initial tissue insult. Heat-producing exotoxins released by the bacteria can become "superantigens" leading to specific and prolific cytokine production which contributes to shock, infarction, organ failure, and death (Stevens, 1996).

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Treatment

- Early and aggressive surgical exploration and debridement (at most immediate facility possible)
- Early broad-spectrum empiric antibiotic therapy, suggestions:
  - o A carbapenem or βL/βLI +
  - o Clindamycin +
  - o Vancomycin
  - o Tailored antibiotic therapy is ideal once bacterial types are determined
- Aggressive hemodynamic support (sepsis protocol)
- Hyperbaric oxygen therapy for long-term management (Mechem & Manaker, 2016)

Clinical Pearls

- Immediate surgical intervention above all other testing
- Antibiotics without debridement is futile (Anaya & Dellinger, 2007)
- Lab findings are nonspecific but commonalities include:
  - o ↑ WBC with left shift
  - o ↑ CK, lactate, creatinine
  - o Coagulopathy
  - o Blood cultures are not necessarily helpful
- CT/MRI can reveal extent of muscle tissue involvement and presence or absence of gas

Prevention and Prognosis

Proper wound care once tissue injury identified.
Early recognition of simple skin infection with close monitoring.
Type II (GAS) is highly contagious, and prophylactic exposure therapy is warranted for those in close contact with a GAS patient.

Mortality

- Type I = 21%
- Type II = 14-34% (STTS patients have highest mortality)

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