Necrotizing soft tissue infections (NSTI)

MSS microbiology

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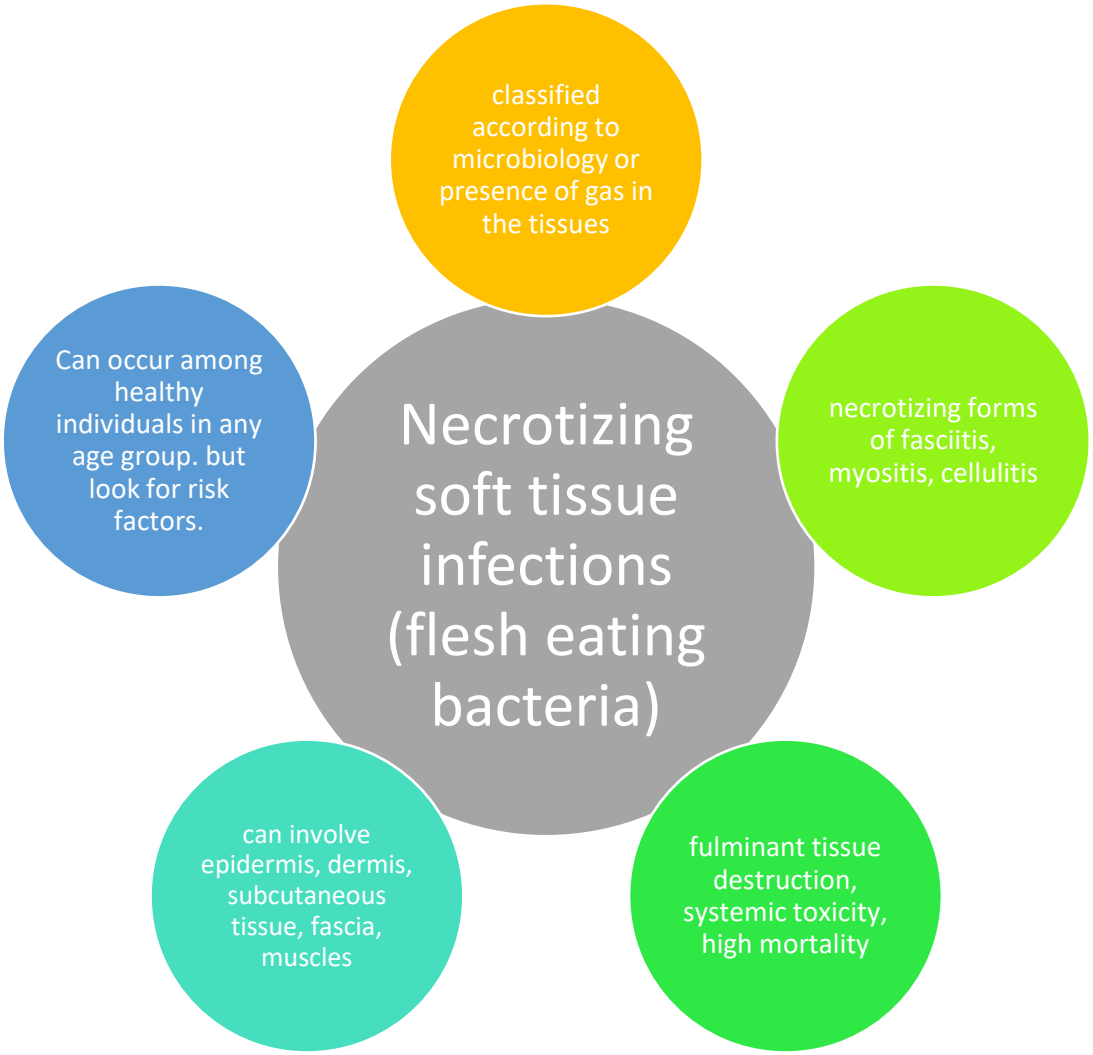


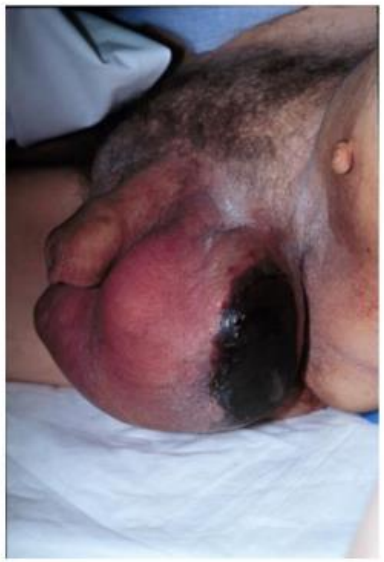
**Risk factors for NSTI:

- Penetrating trauma.
- Recent surgery (GI, urogenital procedures)
- Immunosuppression. (esp. diabetes)

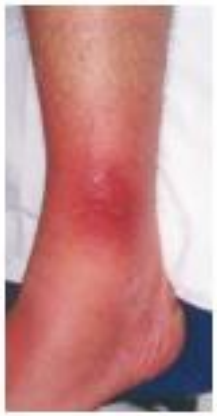
1. Necrotizing fasciitis

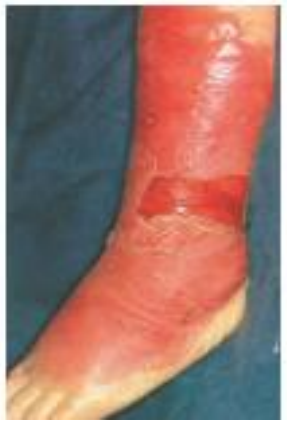
Layer infected	Muscle fascia and subcutaneous fat. (Muscle tissue is frequently spared due to its generous blood supply).
Etiology Type I: GI, urogenital flora Type II: skin flora	*Type I: polymicrobial* (anaerobe + enterobacteriacae + facultative anaerobic strep.) At least one anaerobe (Bacteroides, Clostridium, Peptostreptococcus) + Enterobacteriaceae (E.coli, Enterobacter, Klebsiella, Proteus) + facultative anaerobic streptococci (other than GAS). Fournier's gangrene (Necrotizing fasciitis of the perineum) can occur as a result of breach in the integrity of the gastrointestinal or urethral mucosa. (more common in men). *Type II: Monomicrobial* Caused by GAS, other beta-hemolytic strep., or staph. Infection with no clear portal of entry occurs due to hematogenous translocation of GAS from the throat to a site of blunt trauma or muscle strain. GAS strains with **M protein** 1 & 3 are associated with toxic shock syndrome in about 50% of cases.
Manifestations	Mostly involves the lower extremities (more in diabetes and peripheral vascular disease). Presents acutely over hours, and rarely sub-acutely over days. – Erythema, edema, warmth and severe pain – Fever and Crepitus – Skin bullae, necrosis, or ecchymosis (38%) Rapid progression to extensive destruction leading to systemic toxicity, limb loss, and/or death. Fournier gangrene may spread rapidly to the anterior abdominal wall and the gluteal muscles. Involvement may include the scrotum and penis.
Diagnosis	Clinical suspicion The overlying tissue can appear unaffected (difficult to diagnose) so, **Surgical exploration** is the only way to establish the diagnosis.(+ take samples for culture) **Gas in soft tissues** can be seen on imaging studies (clostridial infection or polymicrobial).
Treatment	Early and aggressive surgical exploration and debridement, with broad-spectrum antibiotic therapy and hemodynamic support. Mortality is 100% if you don't debride !!! Empiric abx: against G(+), G(-), and anaerobes (carbapenem + vancomycin for MRSA + Clindamycin for antitoxin activity
Prognosis	High mortality, even with optimal therapy

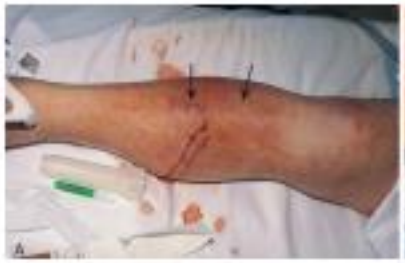
Fournier's gangrene in a patient with diabetes

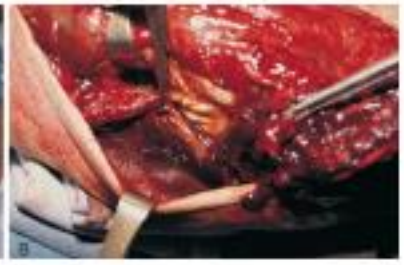


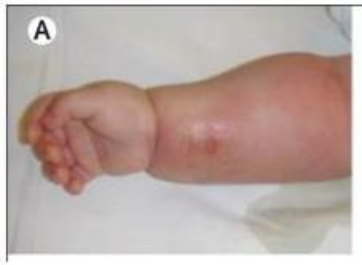
Necrotizing fasciitis of the perineum (Fournier's gangrene) can involve the scrotum. The infection can begin abruptly with severe pain and may spread rapidly.

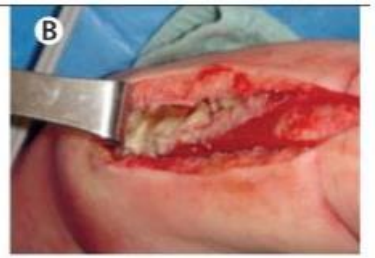




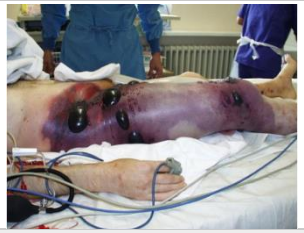




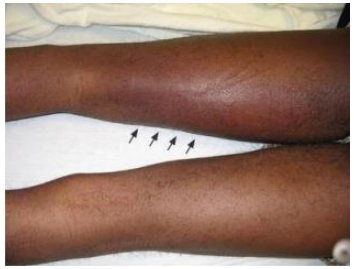
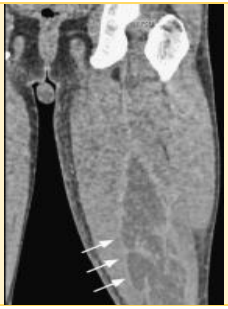




2. Clostridial myonecrosis (gas gangrene)

Layer infected	Muscle tissue mainly
Epidemiology	Clostridium species are commonly found in soil, marine sediments, and human and animal intestinal tracts. (Widespread coz they form endospores).
Pathogenesis	1. Penetrating wounds (most common) with vascular compromise such as knife wounds create an anaerobic environment that is ideal for proliferation of clostridia. 2. Contiguous spread from an area of trauma 3. Hematogenously from the GI tract.
Etiology	Traumatic gas gangrene is mostly caused by **C. perfringens** (spontaneous gangrene is caused by the more aerotolerant C. septicum). α-toxin, inserts into the plasma membrane of cells, producing gaps in the membrane that disrupt normal cellular function. Shock may be attributable to both direct and indirect effects of alpha and theta toxins. In food poisoning, the enterotoxin is produced during the transition phase from vegetative cells to spores and released in the small intestine when the cells undergo the terminal stages of sporulation.
Manifestations	Pain at a site of traumatic injury together + signs of systemic toxicity. C. perfringens causes gas formation in the soft tissue. Clostridial food poisoning has a short incubation period, abdominal cramps, course lasts less than 24 hours.  
Diagnosis	– Manifestations should be suggestive – **Crepitus** in the soft tissue is the most sensitive and specific finding on clinical examination. – Radiographic studies can help. – Blood cultures should be obtained.
Treatment	– **Debridement and excision**, with amputation necessary in many cases. – Water soluble antibiotics (penicillin) alone are not effective (muscles are ischemic, so no penetration). Antibiotics with excellent in vitro activity against C. perfringens include penicillin, clindamycin, tetracycline, chloramphenicol, metronidazole. – Give a booster tetanus vaccine (for patients who didn't receive it in 5 years). – Use of hyperbaric oxygen (HBO) ?
Prognosis	Patients with bacteremia and hemolysis have a likelihood of progressing to shock and death. Mortality is highest for patients in shock at the time of diagnosis.

3. Pyomyositis (a purulent infection)

Layer infected	skeletal muscles
Epidemiology	Common in the tropics. (Increasing frequency in temperate regions).
Pathogenesis and risk factors	Hematogenous spread, with abscess formation Risk factors include immunodeficiency (particularly HIV infection), trauma, injection drug use, concurrent infection.
Etiology	90% of tropical cases → S. aureus 75% of temperate cases → S.aureus
Manifestations	Mostly in the lower extremity. Pyomyositis can be divided into three clinical stages: Stage 1: Crampy local muscle pain, swelling, and low-grade fever. Stage 2: Fever, exquisite muscle tenderness, and edema. (10-21 days after the initial onset). Stage 3: Systemic toxicity, the affected muscle is fluctuant. Complications of S. aureus bacteremia: septic shock, endocarditis, pneumonia, pericarditis, renal failure,... 
Diagnosis	MRI is the most useful tool for diagnosing, defining the site(s) of infection, and for ruling out other entities. Cultures of specimens and/or blood to determine the pathogen. 
Treatment	Stage 1: antibiotics alone. Stage 2 or 3: both antibiotics and drainage. (Most patients present at stage 2)

4. Diabetic foot infections

<table>
<tr><td>Layer infected</td><td colspan="3">Can infect all layers reaching to the bone.</td></tr>
<tr><td>Risk factors</td><td colspan="3">Neuropathy, peripheral vascular disease, and poor glycemic control.</td></tr>
<tr><td>Pathogenesis</td><td colspan="3">Sensory neuropathy: diminished perception of pain and temperature.
Autonomic neuropathy: diminished sweat secretion → dry, cracked skin that facilitates entry of pathogens.
Motor neuropathy: results in foot deformities, which lead to pressure-induced soft tissue damage.

Peripheral artery disease impairs blood flow → no healing of ulcers and infections.
Hyperglycemia impairs neutrophil function → reduced host defenses.
Trauma in patients with these risk factors precipitates slowly healing wounds and secondary infections.</td></tr>
<tr><td>Etiology
(Most are polymicrobial)</td><td>Superficial infections:
(Cellulitis and infected ulcers in antibiotic-naïve individuals).

Due to aerobic G(+) cocci:
– S.aureus
– Strep. Agalactiae
– Strep. Pyogenes
– Coagulase-negative staph.</td><td>Deep, chronic infections:
(And previously treated with antibiotics)

Polymicrobial:
The previous organisms +
– Enterococci
– Enterobacteriaceae
– Pseudomonas aeruginosa
– Anaerobes</td><td>Wounds with extensive local inflammation and necrosis:
(Malodorous drainage, gangrene, signs of systemic toxicity)

All previous pathogens + anaerobes:
– Anaerobic streptococci
– Bacteroides
– Clostridium</td></tr>
<tr><td>Manifestations</td><td colspan="3">Localized superficial skin erythema, warmth, swelling, and tenderness +/- pus at the site of a preexisting lesion.

Or infection of the skin and deeper structures that has spread beyond the site of local trauma, can extend to joints, bones, and blood.

Osteomyelitis can occur in a diabetic foot wound with/without evidence of local infection.

<table>
<tr><th>Infection severity</th><th>Clinical manifestations of infection</th></tr>
<tr><td>Uninfected</td><td>Wound lacking purulence or any manifestations of inflammation.</td></tr>
<tr><td>Mild</td><td>Presence of ≥2 manifestations of inflammation (purulence, or erythema, pain, tenderness, warmth, or induration), but any cellulitis/erythema extends ≤2 cm around the ulcer, and infection is limited to the skin or superficial subcutaneous tissues; no other local complications or systemic illness.</td></tr>
<tr><td>Moderate</td><td>Infection (as above) in a patient who is systemically well and metabolically stable but which has ≥1 of the following characteristics: cellulitis extending >2 cm, lymphangitic streaking, spread beneath the superficial fascia, deep-tissue abscess, gangrene, and involvement of muscle, tendon, joint or bone.</td></tr>
<tr><td>Severe</td><td>Infection in a patient with systemic toxicity or metabolic instability (eg, fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, severe hyperglycemia, or azotemia).</td></tr>
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Foot ischemia may increase the severity of any infection, and the presence of critical ischemia often makes the infection severe.</td></tr>
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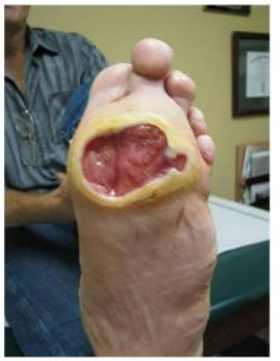
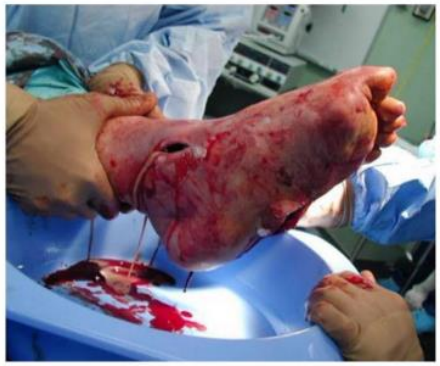
Diagnosis	<u>Clinical</u> 1. Determine the extent and severity. (Location, involving skin, subcutaneous tissue, muscles, tendons, bone) If the bone was visible, there's a very high chance of osteomyelitis. 2. Identify predisposing factors Do a neurologic evaluation to see sensory loss as well as a vascular evaluation. 3. Assess the microbial etiology: samples for culture (aspirate from an abscess or curettage from ulcer base). Do cultures only if suspicion is high for infection (positive cultures can be due to colonizing harmless pathogens).  • Fig. 1. Large forefoot ulcer in patient with diabetes.
Treatment	Wound management: Debridement of callus and necrotic tissue, wound cleansing, and relief of pressure on the ulcer. Antimicrobial therapy: Empiric abx are selected based on the severity and the likelihood of resistant organisms. Surgery: Consultation with a surgeon is important for moderate-severe cases.  Fig. 3. Diabetic foot with abscess undergoing surgical irrigation and debridement. (Courtesy of William DeCarbo, D.P.M., Columbus, OH.)