

Necrotizing Fasciitis Developing from a Pressure Injury

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Case Description

Introduction

- Necrotizing fasciitis (NF) is a surgical emergency characterized by fulminant muscle fascia and subcutaneous fat destruction, signs of systemic toxicity, and a significant risk of mortality.¹
- It is a rare type of necrotizing soft tissue infection (NSTI) with approximately 700-1150 cases a year in the United States, and an increasing incidence in past decades.^{2,3}
- NF is divided into polymicrobial (type I) and monomicrobial infections (type II). Gram-positive Staphylococcus aureus and streptococci strains are the most common causes of type I while gram-negative and anaerobic bacteria cause type II.¹
- Deep pressure injuries (PI) always involve necrotic tissue. STIs often develop from them, but NF is a rare complication.⁷ NF is typically due to trauma or postoperative infections. It most commonly involves the extremities and escalates within hours.¹
- Risk factors: diabetes mellitus, obesity, cirrhosis, advanced age, trauma or surgery, cardiovascular disease, delay in therapy.^{3,4}
- Initial presentation is cellulitis; may appear nonspecific, benign. It rapidly progresses into systemic toxicity with fever, lethargy, & disorientation. Other features are extreme pain disproportional to clinical findings, crepitus, bullae, induration, skin necrosis.^{1,4}
- Diagnosis (dx) can only be confirmed through direct exploration of fascial planes and tissue in the operating room (OR).^{1,8}
- Differential dx: cellulitis, gas gangrene, toxic shock syndrome, pyoderma gangrenosum, pyomyositis, deep vein thrombosis.1,4,8

Diagnostic Results



- Day 2 cultures revealed bacteremia with Proteus mirabilis, Bacteroides fragilis. S. constellatus.
- Figure 1 shows the computed tomography (CT) results of a midline/ left gluteal sacral ulcer with soft tissue swelling and a large amount of gas tracking from the surface wound to the deep soft tissue (raising possibility of NF).

History & Vitals

- A 63-year-old bedbound, minimally verbal African General appearance: American male with a past medical history of bilateral basal ganglia stroke in 2017 managed with daily therapeutic apixaban 5 mg, deep vein thrombosis, sacral decubitus ulcer, hypertension managed on lisinopril 40 mg, and uncontrolled diabetes mellitus presented to the emergency department (ED) from a nursing facility for fever and unwitnessed fall from bed.
- No known allergies
- Family history unremarkable
- Social history: patient has been living in a skilled nursing facility since his stroke in 2017
- Review of symptoms: unattainable
- Vitals: Temperature: 38.4°C
 - Pulse: 136 beats per minute
 - Blood Pressure: 140/88 mm Hg
 - Respiratory Rate: 20 breaths per min
 - Pulse Oximetry: 95% on room air

Initial Physical Exam

- Patient lying in bed; appears quadriplegic but when prompted, mobilizes his extremities. - Appears apathetic, nonverbal.
- Intermittent coughing.

Skin:

- Warm, clammy, diaphoretic to touch. - Unstageable sacral decubitus ulcer: Persistent nonblanchable deep red discoloration, induration, fullthickness skin/tissue loss of 5x10cm length & 3cm deep. Extent of damage cannot be confirmed because it is obscured by slough and eschar.
- Neurologic exam:

 - Awake. Lethargic, but arousable. Following commands. - Motor strength: 2/5 in bilateral extremities - Grip strength: 3/5 in bilateral hands
 - Sensory: decreased to touch and pain prick
 - Cranial nerves grossly intact
- Remainder of exam was within normal limits.



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Management

- Does not appear in acute distress or pain.

History of Present Illness

Dav 1: Pt was admitted for COVID-19 and sepsis secondary to a urinary tract infection as evidenced by leukocytosis, lactic acidosis, tachycardia, & lethargy. Treated (tx) with cefepime 1 gram every 8 hours. Placed on intravenous fluids (IVF) & Paxlovid. Wound care began management of his sacral ulcer.

Hospital Course

- Dav 2: + blood cultures and fever not resolving. Held apixaban when hemoglobin (Hgb) decreased from 10.7 to 7.0 g/dL
- Dav 3: Hgb 6.4. Hgb 9.4 post two units of packed red blood cells (pRBC). Etiology of anemia unclear, but leading dx was gastrointestinal blood loss. Ordered abdomen and pelvis CT scan, esophagogastroduodenoscopy (EGD), and occult blood.

Patient Outcome

- Dav 4: STAT debridement & drainage of soft tissue necrosis; confirmed NF in the OR, no osteomyelitis. Pt tolerated it well.
- Dav 5: discontinued cefepime. Started piperacillin/tazobactam 12.5g every 12H and vancomycin 1g every 12H
- Dav 9: repeat CT demonstrated healing debrided ulcer

Discussion

- NF poses significant mortality & morbidity, particularly in pts with chronic disease, most notably diabetes. Mortality rate without debridement is nearly 100%, but with proper tx, it ranges from 14-35%.^{1,5,6} Sequalae includes limb loss, multi-organ failure, septic shock, and shorter survivor life span.^{1,3,7}
- NF is a rare complication of deep PI. Limited studies have documented the incidence of NF from a PI.⁷
- Due to the ambiguous presenting features and rarity, NF is often incorrectly dx, especially in the early stages.⁷ One study showed only 14% of pt were properly dx on admission.
- Sequalae from basal ganglia stroke complicated early NF dx.
- Figure 3 discusses an overview of dx and tx of suspected NF.⁸

Conclusion

- NF is a rare, rapidly progressing complication of deep pressure injuries, especially in bedridden patients.
- · It requires early surgical intervention to minimize mortality.
- Due to the high M&M and frequent delays in dx, clinicians must maintain a high level of suspicion and keep NF on their differential in the context of a patient with a PI.