Necrotizing Fasciitis

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Necrotizing Soft Tissue Infections-Epidemiology

500 – 1,000 cases annually
Necrotizing Soft Tissue Infections - Epidemiology

- 500 – 1,000 cases annually
- 20-30% mortality rate
### Necrotizing Soft Tissue Infections - Epidemiology

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Necrotizing Soft Tissue Infections - Epidemiology

500 – 1,000 cases annually

20-30% mortality rate

4% increase in mortality every year of life

Time from admission to initial debridement most important variable determining mortality
Independent Predictors for Mortality

Retrospective reviews identify several factors:

- Time to first debridement
- Inadequate first debridement
- Extent of tissue involvement
- Age > 60 years
- Bacteremia
- # Failed organs on admission
- Elevated lactate

Bosshardt TL. Arch.Surg. 1996;131:846-52
Bilton BD. Am.Surg. 1998; 64:397-400
Necrotizing Soft Tissue Infection (NSTI) Tissue layers and infection

- Dermis and subcutaneous fat
  - Good resistance to bacterial invasion, proliferation
  - Infection: NECROTIZING CELLULITIS

- Fascia (deep or muscle)
  - Tentative blood supply, poor lymphatic drainage, and low resistance to bacterial invasion, growth, and spread
  - Infection: NECROTIZING FASCIITIS

- Muscle
  - Very good blood supply and good resistance to bacterial invasion and proliferation
  - Infection: MYOSITIS and MYONECROSIS
Determinants of Infection

Host vs Pathogen

HOST TISSUE RESISTANCE vs BACTERIAL VIRULENCE GROWTH CHARACTERISTICS

… Presentation and severity of infection determined by a balance between these factors …
Necrotizing Soft Tissue Infections - Risk Factors

- Risk Factors
  - Any condition causing a decrease in immune function
    - Diabetes and IVDA are most common.
  - Others include:
    - obesity,
    - peripheral artery disease
    - corticosteroid therapy
    - malnutrition
    - Smoking
    - chronic cardiac disease
    - chronic immunosuppression and cancer

Necrotizing Soft Tissue Infections - Risk Factors

- Risk Factors
  - NSAIDs
    - risk factor as use may initially mask the symptoms.

MOST COMMON

+/- trauma

Diabetes present in 18-60%

>50% cases in healthy individuals

Necrotizing Soft Tissue Infections—Signs and Symptoms

Review of 12 studies totaling 317 limbs found that erythema (73%), pain (63%) and edema were the most common physical exam findings.
Necrotizing Soft Tissue Infections—Signs and Symptoms

Study among 89 consecutive patients with necrotizing fasciitis

- Most common physical examination findings
  - erythema (100%)
  - pain out of proportion to physical findings (97.8%)
  - warm skin (96.6%)

Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO.

Necrotizing Soft Tissue Infections-
Signs and Symptoms

- 163 patients with NF
  - pain was present in all patients
  - erythema in (95%)
  - edema in (82%)
## Necrotizing Soft Tissue Infections - Diagnosis

Objective Criteria to Distinguish Necrotizing from Non-necrotizing Infection

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive Predictive Value (%)</th>
<th>Negative Predictive Value (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tense Edema</td>
<td>38</td>
<td>100</td>
<td>100</td>
<td>62</td>
</tr>
<tr>
<td>Gas on XR</td>
<td>39</td>
<td>95</td>
<td>88</td>
<td>62</td>
</tr>
<tr>
<td>Bullae</td>
<td>24</td>
<td>100</td>
<td>100</td>
<td>57</td>
</tr>
<tr>
<td>WBC &gt; 14 x 10⁹/L</td>
<td>81</td>
<td>76</td>
<td>77</td>
<td>80</td>
</tr>
<tr>
<td>Sodium &lt; 135 mmol/L</td>
<td>75</td>
<td>100</td>
<td>100</td>
<td>77</td>
</tr>
<tr>
<td>Chloride &lt; 95 mmol/L</td>
<td>30</td>
<td>100</td>
<td>100</td>
<td>55</td>
</tr>
<tr>
<td>BUN &gt; 15 mg/dL</td>
<td>70</td>
<td>88</td>
<td>88</td>
<td>71</td>
</tr>
</tbody>
</table>

Diagnosis of Necrotizing STI:

- “Hard signs” for the presence of a necrotizing process:
  - bullae
  - skin ecchymosis preceding skin necrosis
  - gas in tissues by exam or on radiographs
  - cutaneous anesthesia
    - present in 7-44% of cases
Necrotizing Soft Tissue Infections - Diagnosis

Diagnosis of Necrotizing SSTI:

- Suggestive signs:
  - pain disproportionate to examination
  - edema extending beyond skin erythema
  - systemic toxicity
  - progression of infection despite antibiotic therapy
Clinical Diagnosis!

Triad of

Pain

Skin Changes

Rapid Progression

= High Suspicion
Necrotizing Soft Tissue Infections—Progression

- Necrotizing infection
  - Early in disease process may present identical to cellulitis and erysipelas

- Fever may or may not be present.


Necrotizing Soft Tissue Infections-Progression

• With disease progression, systemic signs of *sepsis* may present:
  • hypotensive acidosis,
  • leukocytosis
  • Tachycardia
  • hypo or hyperthermia.

Necrotizing Soft Tissue Infections-Progression

- Variable depending on the size of
  - bacterial inoculum
  - organism involved
  - location of the infection
  - health of the patient.

Necrotizing Soft Tissue Infections—Progression

• Progression
  • Generally speaking, edema, erythema and necrosis progress slowly over a 2-4 day period.

• With group A streptococcal infection
  • progression rapid presenting with dark red bullae.

Necrotizing Soft Tissue Infections-Progression

- Level of suspicion should be heightened when
  - patients diagnosed with cellulitis have pain out of proportion to lesion.

- rapid progression of erythema and skin induration (>1 cm/hr) in spite of IV antibiotic treatment.

Necrotizing Soft Tissue Infections- Progression

• Blisters and Bullae
  • initially drain serosanguinous fluid then drain hemorrhagic fluid

• Crepitus
  • present when soft tissue in gas

• Pain dulls
  • cutaneous nerves destroyed

Later stage disease may present with a

- watery, grayish, foul smelling “dishwater pus” due to superficial fat and fascial necrosis

Necrotizing Soft Tissue Infections—Progression

Diagnosis often delayed due to similar presentation with cellulitis

- Compared 59 pts with NF to matched cohorts
  - NF group had
    - stronger complaint of pain
    - 5 fold increase in CRP levels
    - LRINREC scores were significantly higher.
# Necrotizing Soft Tissue Infections - Lab Studies

**(LRINEC) Score**

<table>
<thead>
<tr>
<th>Laboratory parameter, units</th>
<th>LRINEC points</th>
<th>Laboratory parameter, units</th>
<th>LRINEC points</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP, mg/L</td>
<td></td>
<td>Sodium, mmol/L</td>
<td></td>
</tr>
<tr>
<td>&lt;150</td>
<td>0</td>
<td>≥135</td>
<td>0</td>
</tr>
<tr>
<td>≥150</td>
<td>4</td>
<td>&lt;135</td>
<td>2</td>
</tr>
<tr>
<td>Total WBC, k/mm³</td>
<td></td>
<td>Creatinine, mg/dL</td>
<td></td>
</tr>
<tr>
<td>&lt;15</td>
<td>0</td>
<td>≤1.6</td>
<td>0</td>
</tr>
<tr>
<td>15-25</td>
<td>1</td>
<td>&gt;1.6</td>
<td>2</td>
</tr>
<tr>
<td>&gt;25</td>
<td>2</td>
<td>Glucose, mg/dL</td>
<td></td>
</tr>
<tr>
<td>Hb, g/dL</td>
<td></td>
<td>≤180</td>
<td>0</td>
</tr>
<tr>
<td>&gt;13.5</td>
<td>0</td>
<td>&gt;180</td>
<td>1</td>
</tr>
<tr>
<td>11–13.5</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;11</td>
<td>2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Necrotizing Soft Tissue Infections - Lab Studies

Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) Score

- The maximum cumulative score is 13.
- A score greater than or equal to 6
  - positive predictive value of 92% (95% CI, 84.3–96.0)
  - negative predictive value of 96% (95% CI, 92.6–97.9)
- The probability of necrotizing SSTI increased to more than 75% when the LRINEC score was greater than or equal to 8.
Necrotizing Soft Tissue Infections - Lab Studies

- Initial lab workup of a suspected necrotizing infection should include:
  - CBC
  - serum albumin
  - electrolyte panel (including calcium)
  - BUN
  - liver function tests
  - PT and PTT.
  - ESR
  - CRP


D C Elliott, J A Kufera, and R A Myers

- Decreased
  - platelets
- Elevated
  - BUN
  - creatinine
  - bilirubin
  - blood lactate levels

Associated with Death!
Necrotizing Soft Tissue Infections - Lab Studies

Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) Score

• Caveats:
  – Not been prospectively validated in patients for whom the diagnosis of necrotizing SSTI is not apparent on initial history and physical examination.
  – It is also unclear if the LRINEC score can be applied to all age groups (the youngest patients were 13 and 27 years old in the study cohort).
Finding abnormalities that make up the LRINEC score in patients with SSTI should increase suspicion of a necrotizing infection such that further observation and evaluation should be considered.

Biopsy-Gold Standard for Diagnosis
Inflammatory cell infiltrate mostly PMNs
Tissue Diagnosis

Bacteria

May or may not be present
Vasculitis/Thrombosis

Seen in small vessels
Necrotizing Soft Tissue Infections - Biopsy Summary

Necrotic tissue

Bacteria

Inflammatory cell infiltrate

Vasculitis and Thrombosis
• NF
  • represents sustained injury from infection
Sustained tissue injury elicits an acute phase response
  - myokine IL-6 released from damaged tissue
  - IL-6 travels to the liver and affects the expression of over 1000 different genes.
Pathophysiology-Infection Provoked Acute Phase Response

- Most genes expressed make proteins involved in coagulation and inflammation leading to persistent tissue injury and sustained acute phase response.

**Hemostasis**

**Antimicrobial**

**PERSISTENT INJURY**
Pathophysiology-Infection Provoked Acute Phase Response

- Constant inflammatory state continues until immune system, antibiotics and/or surgery ends the infection.
Pathophysiology - Infection Provoked Acute Phase Response

- Without prompt treatment, tissue injury continues.

Hemostasis Antimicrobial
Pathophysiology: Infection Provoked Acute Phase Response

- Amplified acute phase causes development of SIRS, DIC, MSOF, and eventual death.
Pathophysiology-Infection Provoked Acute Phase Response

**IL-6**

**Hemostasis**

**Antimicrobial**

**CRP**
- rapid acute phase reactants which increases significantly in response to tissue from nec fasc

**SIRS**

**MSOF**

**DIC**

**Death**

**Systemic Complications**

**DIC**

**Death**

**SIRS**

**MSOF**
• During increasing hyperinflammatory state protein C and S become activated to buffer this effect.
Pathophysiology-Infection Provoked Acute Phase Response

- Hemostasis
- Antimicrobial
- CRP
- Ptn c
- Ptn s

- SIRS
- MSOF
- DIC
- Death
- Systemic Complications

- Longer the hyperinflammatory state persists, the more ptn c and s are consumed.
Pathophysiology - Infection Provoked Acute Phase Response

Hemostasis
Antimicrobial

CRP

Ptn c
Ptn s

SIRS
MSOF
DIC
Death
Systemic Complications

IL-6

As proteins consumed, ability to buffer coagulation decreases.
As this proceeds unchecked patients develop thrombus once they begin to consume their clotting factors.
Pathophysiology - Infection Provoked Acute Phase Response

- Hemostasis
- Antimicrobial
- CRP
- Ptn c
- Ptn s
- SIRS
- MSOF
- DIC
- Death
- Systemic Complications

• Hemorrhage occurs due to DIC
Necrotizing Fasciitis

Tissue Injury

Coagulation

Inflammation

Fibrin

Fibrinogen

Fibrin Clot Degradation

Clinically, continuous cascade leads to persistent tissue injury

Coagulation Inflammation Cycle
Necrotizing Fasciitis

Coagulation Inflammation Cycle

A hyperinflammatory environment ensues leading to an elevated CRP
Clinically this is recognized as SIRS/sepsis
Necrotizing Fasciitis

The consumption of protein C/S disrupts the balance between coagulation and inflammation.

The Coagulation Inflammation Cycle

- **Coagulation**
- **Inflammation**
- **Fibrin**
- **Fibrinogen**
- **Fibrin Clot Degradation**

**Protein C/S**

**SIRS/Sepsis**

**CRP**

Injury

The consumption of protein C/S disrupts the balance between coagulation and inflammation.
A hypercoaguable state develops evidenced by an increase in PT-INR.
Coagulation Inflammation Cycle

As the coagulation factors are consumed, the patient becomes hypocoaguable, as evidenced by an elevated INR.

Protein C/S ↓  PT-INR ↑
Necrotizing Fasciitis

Tissue Injury

Coagulation Degradation

Fibrinogen

Fibrin

Fibrin Clot

DIC

SIRS/Sepsis

↑CRP

↑D-Dimer

↓Protein C/S

↑PT-INR

↑PT

- INR

↓Protein C/S

If left untreated, DIC ensues, as evidenced by elevated D-dimer.
Several ways to break this cycle:
- Surgery and antibiotics to stop the tissue injury

Coagulation Inflammation Cycle

- ↑PT - INR
- ↓Protein C/S
- ↑PT-
- DIC
- ↑CRP
- ↑D-Dimer
- Surgery
- Antibiotics
- SIRS/Sepsis
- Necrotizing Fasciitis
- Fibrin Clot Degradation
- Fibrin
- Fibrinogen
- Tissue Injury
- Coagulation
While not specifically studied in nec fasc, Steroids have been well studied in sepsis and have been shown to be effective in treating shock associated with severe infection, due to their ability to help end the hyperinflammatory state.
Vitamin K, while not as well studied, has also been found to help treat the coagulopathy that develops by restoring vitamin K dependent clotting factors, mainly protein C.
Necrotizing Soft Tissue Infections - Imaging

- Radiographs not normally indicated in the work up in necrotizing fasciitis.
  - In clostridial myonecrosis
    - gas in the muscle bellies

Necrotizing Soft Tissue Infections—Imaging

- CT scan
  - may be more sensitive than plan radiographs for
    - air in the soft tissues
    - findings
      - gas within the superficial fascia distributed linearly
      - predominance of fascial involvement.

• CT scans of 20 patients with biopsy proven necrotizing fasciitis
  • 80% had asymmetric subcutaneous fat or fascial stranding
  • 55% had superficial or deep tracking of air
  • 35% had a loculated abscess
• Looked at CT scans before surgery in patients with suspected necrotizing fasciitis.
  • 25 diagnosed with necrotizing fasciitis based on pathology
  • All 25 of these patients had enhanced soft tissues consistent with inflammation and necrosis.
  • 9 had subcutaneous gas and 7 had fluid collections.
  • CT
    • 100% sensitive and 81% specific
    • PPV 76%
    • NPV 100% in identifying soft tissue infections
MRI differentiates cellulitis from necrotizing soft tissue infection
  • distinguished surgically proven necrotizing fasciitis and cellulitis in all 33 patients
  • On MRI
    • both cellulitis and NF show decreased T1 signal and increased T2 signal in subcutaneous tissue
    • NF shows increased T2 signal in the fascia itself.
Main drawbacks of MRI is the length of time needed for the study.

Should not be used if it slows down time to surgical treatment.
Necrotizing Soft Tissue Infections - Bacteriology

Streptococcal

Group A strep (*Strep. pyogenes*)

- incubation period 1-3 days
- fulminant course due to streptolysin / hemolysins / hyaluronidase
- “flesh-eating” bacteria – streptococcal pyrogenic exotoxin (*speA, speB, speC*)

Feingold DS *Arch Dermatol* 1996; 132:67-70
Necrotizing Streptococcal Cellulitis
Streptococcal

Group B strep (*Strep. Agalacitiae*)

a. More common in pts with altered resistance
   • diabetes, cancer, neonatal, etc

b. Short incubation period
Necrotizing Soft Tissue Infections - Bacteriology

Clostridial species

a. incubation period 1-2 days

b. very fulminant course due to toxins
   - *C. perfringens* – 20 known exotoxins

c. local gas, brownish discharge, high fever, high mortality

d. *C. perfringens, C. septicum*

Stevens DL *Clin Inf Dis* 1997; 25:S160-64
Necrotizing Soft Tissue Infections - Bacteriology

Gram negative bacillary

E. coli, Kleb, Proteus, others

- incubation period 7-14 days
- fever (FUO), local symptoms, sepsis
- may be mixed
Necrotizing Soft Tissue Infections - Bacteriology

Pathogenic gram negative bacteria

- Vibrio vulnificus - shellfish
- Aeromonas hydrophila – fresh water
- Pasteurella multocida – dog/cat bites
- Eikenella corrodens – human bites

- Treat with tetracycline class + beta-lactam
Necrotizing Soft Tissue Infections - Bacteriology

Mixed aerobic / anaerobic

a. incubation period 10-14 days
b. local pain, edema, purplish discoloration
c. Meleny’s progressive cutaneous gangrene
d. Fournier’s gangrene
e. Necrotizing fasciitis
Necrotizing Soft Tissue Infections - Bacteriology

Others – “high-risk” for unusual or resistant pathogens

a. Special exposures
   – Bites and environmental

b. Nosocomial
   – LOS > 4d, AB use, high APACHE II

c. Chronic
   – Previous AB use, altered tissue resistance
The “Eagle effect”

- 1952 – Harry Eagle demonstrated failure of cell-wall agents in *S. pyogenes* myositis model with high inoculum
- Clindamycin >> erythromycin > penicillin
- Believed related to stationary phase of growth and PBP
- Also demonstrated in *C. perfringens* and *S. aureus* models
- Retrospective human studies suggest outcome from *S. pyogenes* infection better with clindamycin

Protein synthesis-inhibiting antibiotics

- Shown to decrease production of toxins, superantigens, and enzymes from:

  **Gram positive:**
  - *S. aureus*
  - *S. pyogenes*
  - *C. perfringens*

  **Clindamycin (linezolid)**

  **Gram negative:**
  - *Vibrio sp*
  - *Aeromonas sp*
  - *Pasteurella sp*

- No prospective human studies

Zimbelman J Ped Infect Dis J 1999; 18:1096-1100
Antibiotic Rx for necrotizing SSTI due to virulent pathogens:

Group A strep (*Strep. pyogenes*)

- incubation period 1-3 days
- fulminant course due to streptolysin / hemolysins / hyaluronidase
- “flesh-eating” bacteria – streptococcal pyrogenic exotoxin (*speA*, *speB*, *speC*)
- High dose penicillin + clindamycin (2.4 g/d)

Feingold DS *Arch Dermatol* 1996; 132:67-70
Antibiotic Rx for necrotizing SSTI due to virulent pathogens:

Clostridial species *(C. perfringens, C. septicum)*

a. incubation period 1-2 days

b. very fulminant course due to toxins
   • *C. perfringens* – 20 known exotoxins

c. local gas, brownish discharge, high fever, high mortality

d. penicillin (24 million U/day) or carbapenems + clindamycin (2.4 g/d)
Antibiotic Rx for necrotizing SSTI due to virulent pathogens:

Highly virulent gram negative bacteria:

- *Vibrio vulnificus*
- *Aeramonas sp.*

- Both associated with contaminated water exposure
- Both highly virulent with fulminate course
- Antibiotic Rx –
  - 3rd generation cephalosporins, imipenem/meropenem, and ciprofloxacin/oflaxacin – in combination with
  - Tetracycline/minocycline
Necrotizing Soft Tissue Infections - Management

- Surgical Management
  - After patient anesthetized, perform follow-up exam
    - important due to rapid progression
  - some areas such as the posterior aspects of the thigh and trunk are difficult to examine while the patient is awake due to pain.
Necrotizing Soft Tissue Infections - Management

- Surgical Management
  - The zone approach is often used during surgical debridement.
  - Zone I:
    - Area clearly defined as necrosis presenting with induration and erythema.

Remove all necrotic tissue

Intra-op frozen sections to determine margin
Necrotizing Soft Tissue Infections - Management

• Surgical Management
  • The zone approach is often used during surgical debridement.
  • Zone II:
    • reactionary zone
    • zone surrounding the area of necrosis and presents with induration and erythema.

Remove all necrotic tissue

Intra-op frozen sections to determine margin
Necrotizing Soft Tissue Infections - Management

- Surgical Management
  - The zone approach is often used during surgical debridement.
  - Zone III is considered healthy tissue.

Remove all necrotic tissue
Intra-op frozen sections to determine margin
Necrotizing Soft Tissue Infections-Management

• Surgical Management
  • Completely debride all **necrotic** tissue in zone I

• Make exploratory incisions in zones II and III. Get ahead of the disease!

• Perform the finger sweep test
  • Run a finger between the fascia and the subcutaneous tissue in a forward sweeping manner

• Necrotic tissue in the subcutaneous tissue will peel away from fascia

• Use wound vac for dressings

Necrotizing Soft Tissue Infections - Management

• Resuscitate the patient in shock
• Physiologic Support (O2, Fluids)
• Begin broad-spectrum antibiotic coverage
• Early aggressive surgical debridement
  – Obtain gram stain and culture
  – Histology, if necessary
• Repeat debridement every 24-48 h as necessary
• Adjust antibiotic therapy based on culture
• Nutritional support (enteral preferred)


Necrotizing Soft Tissue Infections-Management

- Surgical debridement is mainstay of therapy
- Aggressive EARLY incision or debridement of all involved tissues
- Average number of débridements 3-4 / patient
- Primary Closure of wounds once tissue improves
- STSG or flap coverage most commonly used for coverage of tissue defects
- Studies suggest that early and aggressive surgical therapy can reduce mortality to < 10%

Bilton BD. *Am. Surg.* 1998; 64:397-400

Bosshardt TL. *Arch. Surg.* 1996; 131:846-52
Adjuvants

- IGG
- Steroids
IGG

- Experimental data on streptococcal toxins suggest that normal polyspecific immunoglobulin given intravenously may inhibit T cell proliferation.
- May bind to a toxin itself neutralizing.
- Neutralizing mitogenic and cytokine-inducing activities of group A streptococcal superantigens.
- Also causes down regulation of TNF-a and IL-6.
• Steroids
  • not specifically studied in nec fasc
  • well studied in sepsis
  • effective in treating shock associated with severe infection, due to ability to help end the hyperinflammatory state
• 47 patients in both treatment and control group

• Hospital mortality 8.5% (4 of 47) treatment group vs. 40.4% (19 of 47) in the control group (P < .001).

• Sepsis-Related Organ Failure Assessment score decreased in all patients in the treatment group, none developing progressive organ failure.

• All patients in the treatment group weaned off vasopressors, a mean of 18.3 ± 9.8 h vs. 54.9 ± 28.4 h in the control group (P < .001).
Treatment

- **Steroids**
  - Inhibit T cell activation
  - Inhibit cytokines

- **Four case reports**
First and ONLY Report of this Transmission

Case report
- 47 y/o male surgeon with hx of right THA
  - In-hospital exposure to index patient while assisting in hip disarticulation in patient with necrotizing fasciitis
  - 10 day later developed necrotizing infection at site of chronic tinea capitis in right foot with ecchymosis migrating to the medial thigh
    - 10 mg IV dexamethasone given followed by 4 mg given q6 hrs for 48 hours
How to Avoid Transmission?
Transmission of Group A *Streptococcus* Limited to Healthcare Workers with Exposure in the Operating Room

Rebecca E. Chandler, MD; Lore E. Lee, MPH; John M. Townes, MD; Randy A. Taplit, MD
Transmission of Group A *Streptococcus* Limited to Healthcare Workers with Exposure in the Operating Room

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Prolonged intraoperative exposure

GAS aerosolized in OR setting
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Prolonged intraoperative exposure

GAS aerosolized in OR setting
Index patient: 34 yo M LE nec fasc, taken to OR for debridement
Index patient: 34 yo M LE nec fasc, taken to OR for debridement

48 h postop HCW 1 (resp therapist): + GAS pharyngitis
Index patient: 34 yo M LE nec fasc, taken to OR for debridement

48 h postop HCW 1 (resp therapist): + GAS pharyngitis
Transmission through aerosols:
Open wounds
Secretions
Transmission through aerosols:
Open wounds
Secretions
Transmission through aerosols:
Open wounds
Secretions

Nosocomial Transmission of Invasive Group A Streptococcus from Patient to Health Care Worker

Mark D. Lacy and Kim Horn
Infectious Disease Service, Flagstaff Medical Center, Flagstaff, Arizona
The best treatment places healthcare workers at risk

Protect your oropharynx!
Summary

- **HARD** signs = necrotizing infection and require OR
  - Bullae, cutaneous anesthesia, ecchymosis, tense edema, gas

- Early and aggressive surgical debridement improves outcome with achievable mortality of < 10%

- *Empiric surgical exploration if in doubt!*

- **Protect your oropharynx**

- **Consider Steroids or IGG in “Toxic” patient (Strep)**

- **Transfer patient AFTER initial debridement**