Necrotizing Soft Tissue Infections: Wound Care & Surgical Management

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Dr. Matthew G. Stanwix
International and National Presentations

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International and National Presentations
Patient K.M.

- Trauma ED consult 40m h/o ETOH and DMI s/p fall head-first into ditch with impaled wooden stick into right forearm mobile wad
- No other injuries
- No ETOH ~48hrs
- Sent Home on Abx
- Came back 4 days later with severe pain
  - Especially on passive range of motion
Hospital Course

- Cultures → gram positive and gram negative rods
- Admitted to SICU → floor POD1
- ID consult → Switched to Unasyn, Clinda and Cipro
- Whirlpool daily with clorpactin change Q8h

What’s New in Necrotizing Soft Tissue Infections: NSTI?

- Ovid: 2005 – 2016 for NSTI, NF, Fournier’s, Gas Gangrene
  - English language, human: 1508
  - English, human, review articles: 215
  - Prospective studies: 36
    - 21 NSTI/NF/hyperbaric
    - 7 Staphylococcal/Vibrio epidemiology
    - 4 total/hyperbaric
  - Randomized trials: 1

NSTI: still - may be lethal

Overall mortality remains high:

- 74 retrospective reports 1980 - 2013
- 4507 patients

- Overall mortality – 22.6%
  - 1980-1999: mortality – 27.8%
  - 2000-2013: mortality – 21.2% (~24% RR reduction)

Unpublished data
Necrotizing Soft Tissue Infections
Is this name important?

• Literature commonly misuses the term NECROTIZING FASCIITIS for all necrotizing soft tissue infections
• So what?...
  • Incorrectly consolidates three tissue layers
  • Confounds the understanding of inherent resistance of each tissue layer and the pathophysiology of bacterial infection
  • Limits an in-depth understanding of both antibiotic and surgical approach

Today’s focus
1. Importance of nomenclature and implications of pathophysiology
2. Establishing the diagnosis
3. Important components of therapy and surgical coverage

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  BACTERIAL VIRULENCE GROWTH CHARACTERISTICS

… Presentation and severity of infection determined by a balance between these factors …

NSTI: bacterial pathogenesis

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
</table>
| Type 1: polymeric | - typically arise from a chronic, indolent source  
|        | - spread along fascial planes  
|        | - most common ~ 50-75 of NSTIs |
| Type 2: monomicrobial virulent Gm+, aerobic cocci | - pathophysiology related to toxin production +/- growth rate  
|        | - Streptococcus species  
|        | - CA-MRSA |
| Type 3: monomicrobial virulent Gm + or Gm – bacilli | - pathophysiology related to toxin production +/- growth rate  
|        | - Clostridia species  
|        | - Bacillus species  
|        | - Vibrio species  
|        | - Aeromonas species  
|        | - Eikenella species |

NSTI: Diagnosis

- Difficult to distinguish—takes experience
- Diagnosis is frequently delayed
- Cellulitis and abscess — most common admitting diagnoses
  - ~65-80% of patients

Wong CH. Crit Care Med. 2004;32:1535–41
Why is early diagnosis important?

- Delays in diagnosis associated with increased morbidity and mortality

Predictors of mortality in NSTI:
- Time to first debridement
- Extent of tissue involvement
- Failed organs on admission
- Inadequate first debridement
- Age > 60 years
- Bacteremia
- Elevated lactate

Symptoms and findings predictive of NSTI

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Laboratory values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pain disproportionate to findings on physical examination</td>
<td>1. White blood cell count &gt;14 x 10^9/L</td>
</tr>
<tr>
<td>2. Tense edema</td>
<td>2. Serum sodium concentration &lt;135 mEq/L</td>
</tr>
<tr>
<td>3. bullae</td>
<td>3. BUN &lt;15 mg/dL</td>
</tr>
<tr>
<td>4. Skin ecchymosis/necrosis</td>
<td>4. CRP &gt; 150 mg/L</td>
</tr>
<tr>
<td>5. cutaneous anesthesia</td>
<td></td>
</tr>
<tr>
<td>6. Systemic toxicity</td>
<td></td>
</tr>
<tr>
<td>7. Crepitence (not always present)</td>
<td></td>
</tr>
</tbody>
</table>

Radiographic findings
- Presence of gas on imaging

Laboratory parameters aid in diagnosis

- 21 consecutive patients who proved to have NSTI

<table>
<thead>
<tr>
<th>Variable</th>
<th>No. of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hard signs</td>
<td></td>
</tr>
<tr>
<td>Bullae</td>
<td>7 (33%)</td>
</tr>
<tr>
<td>Crepitus</td>
<td>3 (14%)</td>
</tr>
<tr>
<td>Gas on x-ray</td>
<td>2 (9.5%)</td>
</tr>
<tr>
<td>SBP &lt;90 mmHg</td>
<td>3 (6.7%)</td>
</tr>
<tr>
<td>Skin necrosis</td>
<td>0</td>
</tr>
<tr>
<td>Other signs</td>
<td>0</td>
</tr>
<tr>
<td>Tense edema</td>
<td>12 (57%)</td>
</tr>
<tr>
<td>Pain out of proportion</td>
<td>10 (48%)</td>
</tr>
<tr>
<td>Violaceous discoloration</td>
<td>5 (24%)</td>
</tr>
<tr>
<td>Laboratory values</td>
<td></td>
</tr>
<tr>
<td>Admission WBC &gt; 15,400/μL</td>
<td>19 (90%)</td>
</tr>
<tr>
<td>Admission Na &lt; 135 mEq/L</td>
<td>19 (90%)</td>
</tr>
<tr>
<td>Admission WBC &gt; 15,400/μL or Na &lt; 135 mEq/L</td>
<td>21 (100%)</td>
</tr>
</tbody>
</table>

SBP = systemic blood pressure.

Buschardt TL. Arch Surg. 1996;131:845-52
Elliot RC. Ann Surg. 1996; 224:672-83
Elliott KD. Am Surg. 1996; 62:397-400
Childers BJ. Am Surg. 2002; 68:139-146

Laboratory parameters add to diagnosis

- Questionnaire of Chief Resident during evaluation

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Results of questionnaire</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Based on PE alone do you think patient has NSTI?</td>
<td>0</td>
</tr>
<tr>
<td>Based on PE alone do you think patient needs operative intervention?</td>
<td>17</td>
</tr>
<tr>
<td>Based on additional laboratory values do you think patient has NSTI?</td>
<td>18</td>
</tr>
<tr>
<td>Based on additional laboratory values do you think patient needs operative intervention?</td>
<td>19</td>
</tr>
</tbody>
</table>

- Mean time to OR: 10 hours (1 - 46)
- Mortality 25%


Treatment of NSTI – What really matters?

- OR as soon as the diagnosis is suspected
- Approach based upon tissues involved
  - Fasciitis: "drainage, irrigation, and debridement"
  - Necrotizing cellulitis: excision of non-viable tissues
  - Myositis, myonecrosis: excision of non-viable tissues
- Ensure adequate tissue perfusion and viability
  - Aggressive hydration
- Prevent fluid pooling or collections
- Dressing changes
- Re-evaluate/return to OR in 24 hours

May AK. Surg Infect. 2009; 10:467-499

Necrotizing Soft Tissue Infections

Anterior

Posterior
Necrotizing Soft Tissue Infections

Does time to re-debridement matter?

- 64 patients with NSTI at USC-LAC over 6 years
- Practice algorithms by 2 different services
  - Short duration (24-48 hrs) vs Extended duration (>48 hrs) until second debridement
  - Short duration associated with lower AKI and mortality

<table>
<thead>
<tr>
<th>Table 4. Comparison of Outcomes in Patients with Necrotizing Soft Tissue Infection Subjected to SED vs EBD*</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Patients</td>
</tr>
<tr>
<td>RRT at admission</td>
</tr>
<tr>
<td>Initial休重</td>
</tr>
<tr>
<td>RRT at 48 hrs post admission</td>
</tr>
<tr>
<td>AKI</td>
</tr>
<tr>
<td>AKI at 48 hrs post admission</td>
</tr>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Shock</td>
</tr>
<tr>
<td>Mortality</td>
</tr>
</tbody>
</table>


NSTI: AB therapy based upon presentation

Non-rapidly progressive NSTI –
- Polymicrobial or less virulent pathogens
- Possible MSSA

Rapidly progressive NSTI –
- Highly virulent pathogens due to toxin production
- Gram positive
  - Gram positive cocci – (Type 2) Group A strep, Co-MRSA
  - Gram positive bacilli – (Type 3) clostridia, bacillus
- Gram negative (Type 3)
  - Vibrio species
  - Aeromonas species
  - Eikenella species

- Dual coverage with antitoxin agents may improve outcome
- Gm +: Clindamycin / Linezolid
- Gm -: Tetracycline class
Surgical Reconstruction

• Wait!!!!
  • Patient must be fully stable
  • Full granulation or healthy
  • Wounds must be pristine
  • Nutrition optimized
  • Follow reconstructive ladder

The Reconstructive Ladder

- Free tissue transfer
- Local tissue transfer
- Tissue expansion
- Skin grafts
- Delayed primary closure
- Primary intention
- Secondary intention
Wound Care-Healing by Secondary Intention

- If Moist-Keep Dry
- If dry-Keep Moist
- Don’t be afraid to debride/open
- Don’t pack tight!

Secondary Intention:

ROLE OF MACROPHAGES IN WOUND HEALING

- Oxygen radicals: $H_2O_2$, $O_2^-$, $OH^-$, Nitr oxide
- Phagocytosis: Enzymes, collagenase, elastase
- Growth factors: PDGF, TGF-
- Cytokines: TNF-$\alpha$, IL-$\beta$, IFN-$\gamma$
- Enzymes: collagenase, arginase
- Prostaglandins: PGE$_2$

History of NPWT

- Application of negative pressure to a sealed, draining wound was developed in the 1950s
- Negative pressure wound therapy as a method for treatment of wounds was developed simultaneously in the United States and Germany in the late 1980s
- In the US, Vacuum Assisted Closure® (V.A.C.*) Therapy was developed by Morykwas and Argenta at Wake Forest University in the mid-1990s. It was cleared by the Food and Drug Administration in 1995.
- Currently V.A.C.* Therapy is the form of NPWT most widely used
NPWT

• Uses subatmospheric pressure for wound healing by secondary or tertiary intention
• Works by deformational forces
• Used to prepare wounds for closure, removal of infectious material and fluid, reduction of edema, promotion of granulation tissue formation
• Used in patients with chronic, acute, traumatic, subacute and dehisced wounds, partial thickness burns, diabetic or pressure ulcers, flaps, and grafts

The Reconstructive Ladder

Primary Intention
Skin Grafts

The Reconstructive Ladder

- Free tissue transfer
- Local tissue transfer
- Tissue expansion
- Skin grafts
- Delayed primary closure
- Primary intention
- Secondary intention
These have an excellent blood supply, are typically designed in a V- or Y-shaped manner, and can often be readvanced in the case of recurrence (Fig. 6).

Dr. Michael Christy
Stage 1: Subcutaneous Tissue Expansion, HADM Inlay & Pedicled ALT Flap
Microvascular Surgery

- State of the art reconstruction
  - Allows for reconstruction not previously available
  - Results much improved
  - Amputations Avoided
  - Defects Filled
  - Lives saved
  - Only done at large academic centers
  - Dedicated centers with trained surgeons

- Microvascular reconstruction
  - Head and Neck
  - Breast
  - Lower Extremity
  - Transplantation!

Lower Extremity
Head and Neck

Congenital Cheek Deformity
Thank you

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My wife, Jessie, and I on a cleft lip/palate medical mission in Vietnam

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