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- Continuing education calendar

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For Android devices, go to the Play Store and search for “UPMC EMS,” or download from https://play.google.com/store/apps/details?id=upmc.tdc.emsnavigator
Necrotizing Soft Tissue Infections

by Gina M. Howell, MD, and Matthew R. Rosengart, MD, MPH

Necrotizing soft tissue infections (NSTIs) are infrequent but aggressive and rapidly spreading infections with potential for high morbidity and mortality. Though commonly called necrotizing fasciitis, this name refers only to a subset of NTSIs that invade the fascial layers. The basic pathophysiology involves the invasion and subsequent spread of bacteria into the subcutaneous tissue, where they release enzymes and toxins causing local tissue ischemia and necrosis. Associated stimulation of the production of inflammatory cytokines promotes systemic toxicity, shock, multisystem organ dysfunction, and death.

The incidence of NSTI is approximately 500-1500 cases per year in the United States and is on the rise.1 Despite advances in medical care, mortality from NSTI remains as high as 35 percent.2 Early surgical debridement constitutes the mainstay of treatment,3-6 and has been shown in numerous studies to be the major determinant of survival1-6.

Microbiology

Three basic microbial subtypes of NSTI are described. Type I, or polymicrobial, NSTI is the most common form of disease, constituting 55 to 75 percent of all NSTI.4-7,9 These infections tend to occur in the perineal and trunk region, and are often diagnosed in patients with known risk factors or immune compromise. Type II infections are monomicrobial infections caused by Group A Streptococcus (GAS), either alone or in association with Staphylococcus aureus, and as such can be associated with toxic shock syndrome. Despite its notoriety in the lay press as “flesh-eating” bacteria, Type II infections are a much less common form of NSTI. These infections classically occur on the extremities, and are more likely to be diagnosed in otherwise young, healthy hosts. GAS can be an especially virulent pathogen, causing rapidly progressive disease due to a variety of unique virulence factors.10,11

Type III NSTI, also known as myonecrosis or “gas gangrene,” is primarily caused by Clostridium perfringens, and is much less common than types I or II NTSIs.

An emerging cause of NSTI is community-acquired methicillin-resistant S. aureus (CA-MRSA), which is present in cultures of one-quarter to one-third of cases of NSTI. This has important implications in the antibiotic management of NSTI, because many of the recommended antibiotic regimens may not provide adequate coverage for MRSA.

(Continued on page 4)
Lactate Helps to Improve Prehospital Triage
by Francis X. Guyette, MD

The Problem
Shock has been defined by the American College of Surgeons as “the presence of inadequate tissue perfusion and oxygenation” and, better still, by Gross as “the rude unhinging of the machinery of life.” Prehospital triage helps us to identify patients in shock who need of lifesaving interventions (LSIs) — tasks done in the field or hospital to prevent death. LSIs may include prehospital hemorrhage control, airway management, or fluid resuscitation. In addition, once the patient is brought to an emergency department, further action may be necessary to prevent death, including emergent operation, resuscitation with fluid or blood products, or definitive airway management. As prehospital providers, our role is to identify those patients who need LSIs and determine how to get them to the best care available.

Traditional trauma triage depends on vital signs and mechanism of injury, which often do not predict need for LSIs or injury severity. Mechanisms of injury are not accurate, and tend to send many uninjured people to the hospital while adding little benefit. A recent study suggests that a high heart rate (HR) and field hypotension (systolic blood pressure ≤ 90 mmHg) predicts an increased risk of death but may miss many serious injuries. Blood pressure and heart rate may change late in shock, and only when the body can no longer compensate for blood loss. Furthermore, patients with head injuries, the very old or very young, and those being treated with certain medications (for example, beta blockers) may not exhibit hypotension or tachycardia in response to traumatic injury.

Experience from Iraq and Afghanistan has taught us that very fit persons may be able to keep near normal heart rates and blood pressures despite life-threatening injuries. This type of “compensated shock” is not easily recognized, and may lead to triage of some patients away from specialized trauma centers and delayed resuscitation, which is strongly associated with death.

If paramedics cannot rely on mechanism of injury and vital signs, what other tools can help them to identify shock in trauma patients? One answer may be lactic acid, or lactate. Serum lactate is a byproduct of anaerobic metabolism; we have all felt that our muscles “burn” after hard exercise. Lactate is a circulating biomarker of poor organ oxygen supply or high demand, and is directly related to death in patients with sepsis, myocardial infarction, and trauma. Following lactate levels can help us to identify patients who are not getting enough oxygen to tissues, even when their vital signs are normal. A new generation of handheld, point-of-care (POC) lactate analyzers is now available for use in EMS. They work much like a standard glucometer.

Importance
High lactate identifies patients who have a higher risk of death, need for surgery, need for blood transfusions, and rate of ICU admission following ED presentation. Lactate may be added to vital signs and mechanism of injury to develop better triage criteria for trauma patients. In the near future, lactate levels may help direct transport to regionalized trauma centers for more aggressive early resuscitation. Ultimately, prehospital protocols might use lactate to determine who might benefit from treatment with fluids or medications.

The Evidence
Previous studies have demonstrated that lactate can predict severe bleeding after trauma and is associated with need for hospital admission, ICU admission, emergent intervention, and death. Trauma surgeons at the University of Alabama, Birmingham, note that standard blood pressure and heart rate monitoring underestimate the severity of hemorrhage. Among patients with systolic blood pressure under 110 mmHg, an increase in lactate was associated with the need for massive transfusion (more than six units of blood). They showed that lactate was better than blood pressure alone for the identification of shock.
In another study, trauma patients with high prehospital lactate were more likely to get admitted to the hospital and twice as likely to die even when their vital signs were normal. The authors concluded that lactate is better than all prehospital vital signs for the prediction of death and need for hospital admission.

Point-of-care lactate also has been used in a large air medical system to identify shock. In this study, 400 trauma patients underwent continuous vital sign monitoring and both prehospital and emergency department lactate sampling. Those patients with prehospital lactate levels >4 mmol/L had greater need for emergent operation, intubation, and vasopressors. Lactate better predicted death and surgery even when age, GCS, and initial vital signs were taken into account. Among the 265 patients with normal vital signs, those with lactates >4 mmol/L were five times as likely to need surgery (odds ratio [OR] 5; confidence interval [CI] 1.5–16.2) and 3.5 times as likely to die (OR 3.5; CI 1.3–9.7) compared with those with a lactate <4 mmol/L. Prehospital lactate strongly predicts death and the need for emergent surgery among trauma patients with normal vital signs.

The Next Step

Although lactate is better than vital signs alone for identifying critical trauma patients, we do not know if it is better than the overall result of current paramedic triage (vitals, mechanism, and gut feeling). EMS services in western Pennsylvania are part of a large multicenter study to evaluate the ability of prehospital lactate to triage patients that could not be otherwise identified by abnormal vital signs. In addition, a combination of lactate and vital-sign criteria may give advance warning in patients with life threatening hemorrhage. Lactate also may identify patients with more severe conditions who could potentially benefit from lifesaving interventions and new treatments.

Francis X. Guyette, MD, is an assistant professor of emergency medicine, University of Pittsburgh School of Medicine, and associate medical director, STAT MedEvac.

References
Diagnosis

The diagnosis of NSTI is often delayed due to the lack of recognition and the paucity of early clinical findings. One should utilize a high index of suspicion and maintain a low threshold for operative intervention in cases of suspected NSTI. This is particularly relevant in managing patients with known risk factors: diabetes mellitus, intravenous drug use, HIV, obesity, ethanol abuse, and recent trauma or surgery. However, a significant number of NSTIs occur in otherwise healthy, immunocompetent patients without these risk factors.

Clinical findings suggestive of NSTI include pain out of proportion to exam, tense edema, ecchymoses, bullae, crepitus, local anesthesia, systemic toxicity, and disease progression despite antibiotic therapy. Radiographic imaging in the form of plain film, CT, or MRI may reveal soft tissue gas, enhancement or thickening of involved fascia, and/or associated abscesses. However, soft tissue gas may be absent on x-ray in two-thirds of patients and on CT in a quarter of patients with documented NSTI; thus the absence of gas on imaging does NOT exclude the diagnosis of NSTI. Furthermore, surgical consultation and treatment should NOT be delayed in order to obtain imaging such as MRI. In many cases, the diagnosis of NSTI is best made on the basis of clinical exam and findings at surgical debridement.

Treatment

Rapid and simultaneous resuscitation, operative debridement, and antibiotics are critical. Most patients with NSTI should be rapidly triaged to a tertiary care center.

Table 1: Group A Streptococcus Virulence Factors

<table>
<thead>
<tr>
<th>Virulence Factor</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>M protein</td>
<td>Surface proteins. Facilitate attachment to host cells, inhibit phagocytosis. M1 and M3 are most virulent subtypes.</td>
</tr>
<tr>
<td>Streptolysin O, S</td>
<td>Beta-hemolysis.</td>
</tr>
<tr>
<td>Streptokinase</td>
<td>Activates plasminogen.</td>
</tr>
<tr>
<td>Exotoxins</td>
<td>Damages endothelium, causes loss of microvascular integrity, stimulates release of cytokines. Exotoxin A may be important mediator of toxic shock syndrome.</td>
</tr>
<tr>
<td>Superantigens</td>
<td>Directly stimulates T-cell activation, causing massive release of systemic cytokines.</td>
</tr>
<tr>
<td>Hyaluronidase</td>
<td>Degrades hyaluronic acid.</td>
</tr>
</tbody>
</table>

Table 2: The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) Score

<table>
<thead>
<tr>
<th>Variable</th>
<th>Partial Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-reactive protein (mg/L)</td>
<td>0, 4</td>
</tr>
<tr>
<td>WBC (cells/mm3)</td>
<td>&lt;15, 15-25, &gt;25</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>&gt;13.5, 11-13.5, &lt;11</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>&gt;135, &lt;135</td>
</tr>
<tr>
<td>Creatinine (mcg/L)</td>
<td>&lt;141, &gt;141</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>&lt;10, &gt;10</td>
</tr>
</tbody>
</table>

Add partial scores for total LRINEC score; sum of >6 has a high correlation with necrotizing infection, with a positive predictive value of 92 percent and a negative predictive value of 96 percent.
**Surgery**

The most important determinant of survival in NSTI is prompt surgical debridement. Several studies have shown that delay of debridement beyond 24 hours after presentation is associated with increased mortality. Radical excision of all devitalized tissue should be performed until healthy, bleeding tissue is encountered. Surgical findings consistent with NSTI include: gray necrotic fascia, loss of resistance to blunt finger dissection (i.e., the “finger test”), lack of bleeding tissue, and the presence of foul-smelling “dishwater” fluid. Serial debridements spaced 12 to 36 hours apart are generally the rule, because infections are rarely eradicated after the initial debridement. Initially, as with all infected wounds, they should be left open and packed with wet-dry dressings. Once infection is controlled, the patient may benefit from negative-pressure wound therapy to assist with granulation and wound closure, though larger wounds may ultimately require skin grafting.

**Antibiotics**

Impaired delivery of antibiotics to necrotic infected tissue can limit their effectiveness locally, but antibiotics remain critical in restricting bacterial spread and ameliorating systemic sepsis. Empiric broad-spectrum antibiotic therapy directed against Gram-positive cocci, Gram-negative rods, and anaerobes should be instituted immediately. There is no single regimen that has been advocated in the literature to date, and many single and multidrug regimens have shown to be efficacious.

Special consideration should be given to antimicrobial coverage of GAS and MRSA. Many would advocate the addition of clindamycin to any empiric regimen, especially for coverage of GAS. Because MRSA appears to be evolving as a significant pathogen in NSTI, empiric therapy should include vancomycin, daptomycin, or linezolid.

Regardless of the initial regimen, antibiotic therapy should be appropriately tailored to final microbiological speciation and antibiotic sensitivity. Current guidelines recommend continuing treatment until no further surgical debridement is necessary and the patient’s physiology has normalized — typically a 10- to 14-day course.

**Hyperbaric Oxygen Therapy**

Hyperbaric oxygen (HBO) therapy has been proposed as an adjunct to surgery and antibiotics in the treatment of NSTI. The technique involves the administration of 100 percent oxygen at a pressure greater than 1 atmosphere absolute (ATA), resulting in a dramatic increase in oxygen tension. Typical HBO therapy involves administration at 2 to 3 ATA for 90 minutes three times in the first 24 hours, then twice daily thereafter, though no standard regimen has been established.

The proposed benefits of HBO include suppression of clostridial α-toxin production and generalized bacterial growth, enhancement of leukocyte-killing activity and antibiotic effects, promotion of tissue repair and wound closure, and bacteriocidal effects on anaerobic organisms.

Despite the physiologic rationale behind its use, clinical human studies are limited mostly to case series and retrospective analyses, and the data from these are inconsistent. However, many authors argue that the purported theoretical benefits and relatively few major risks may support its use as an adjunct to standard therapy.

**Table 3. Recommended Antibiotic Regimens for NSTI**

<table>
<thead>
<tr>
<th>Monotherapy Agents</th>
<th>Multidrug Regimens</th>
</tr>
</thead>
<tbody>
<tr>
<td>imipenem/cilastatin</td>
<td>penicillin or cephalosporin</td>
</tr>
<tr>
<td>meropenem</td>
<td>PLUS amikacin or fluoroquinolone</td>
</tr>
<tr>
<td>ertapenem</td>
<td>PLUS clindamycin or metronidazole</td>
</tr>
<tr>
<td>piperacillin-tazobactam</td>
<td>add vancomycin, linezolid, or daptomycin for MRSA coverage if indicated</td>
</tr>
<tr>
<td>tigecycline</td>
<td>add protein synthesis inhibitor in severe or rapidly progressive infections (clindamycin, linezolid)</td>
</tr>
</tbody>
</table>
Intravenous Immunoglobulin

Intravenous immunoglobulin (IVIG) is another adjunctive measure that has been studied in patients with NSTI, primarily in those with GAS or staphylococcal infections. Patients with NTSI are deficient in antibodies recognizing streptococcal cell wall-attachment proteins, which theoretically would be replaced with pooled IVIG.10 Some small studies suggest a mortality benefit for patients with NTSI treated with IVIG. While it is difficult to form solid conclusions, some experts support the use of IVIG as an adjunct to surgery and antibiotics in critically ill patients with NSTI secondary to streptococcal or staphylococcal species.

Conclusion

Though relatively uncommon, NSTIs are rapidly progressive soft tissue infections that threaten both life and limb of affected patients. Clearly, the major determinant of outcomes is the rapidity with which the disease is diagnosed and surgical debridement performed. Early, aggressive surgical debridement, broad-spectrum antibiotics, and resuscitation form the cornerstone of management. Most of these patients should be triaged to a tertiary care facility that can provide the surgical and critical care resources necessary to manage them. Adjunctive therapies such as HBO, IVIG, and others may have real promise and the potential to improve outcomes.

Gina M. Howell, MD, is a surgical resident, and Matthew R. Rosengart, MD, MPH, is an associate professor of surgery and critical care medicine at the University of Pittsburgh School of Medicine.

References

**CALENDAR OF EVENTS**

**Continuing Education Classes**

<table>
<thead>
<tr>
<th>Name</th>
<th>Date</th>
<th>Time</th>
<th>Location</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cadaver Lab</td>
<td>May 31</td>
<td>6 to 8 p.m.</td>
<td>Ross West View EMS 5325 Perry Highway Pittsburgh, PA 15229</td>
<td>None</td>
</tr>
<tr>
<td>AMLS Provider Course Two days</td>
<td>May 31 &amp; June 1</td>
<td>8 a.m. to 4 p.m. (Both days)</td>
<td>Murrysville Medic One 3237 Sardis Road Murrysville, PA 15668</td>
<td>UPMC/CMC Command providers $15.00 all other providers: $50.00</td>
</tr>
<tr>
<td>AMLS Provider Course Four days</td>
<td>June 6</td>
<td>8 a.m. to 4 p.m.</td>
<td>Unionton Firemen's Ambulance 84 N. Beeson Blvd. Uniontown, PA 15401</td>
<td>UPMC/CMC Command providers $5.00 per discipline all other providers: $45.00 per discipline</td>
</tr>
<tr>
<td>Advanced Burn Life Support</td>
<td>June 27</td>
<td>8 a.m. to 4 p.m.</td>
<td>UPMC Mercy Sr. M. Ferdinand Clark Auditorium 1400 Locust St. Pittsburgh, PA 15219</td>
<td>$25</td>
</tr>
<tr>
<td>Fire Fighter Rehab</td>
<td>July 18</td>
<td>6 to 9 p.m.</td>
<td>Unionton Firemen's Ambulance 84 N. Beeson Blvd. Uniontown, PA 15401</td>
<td>None</td>
</tr>
<tr>
<td>Advanced Burn Life Support</td>
<td>Sept. 14</td>
<td>8 a.m. to 4 p.m.</td>
<td>UPMC Mercy Sr. M. Ferdinand Clark Auditorium 1400 Locust St. Pittsburgh, PA 15219</td>
<td>$25</td>
</tr>
</tbody>
</table>

**Advanced Trauma Life Support 2011**

<table>
<thead>
<tr>
<th>Date</th>
<th>Date</th>
<th>Time</th>
<th>Location</th>
</tr>
</thead>
</table>
| June 18-19       | July 19-20       | Nov. 12-13            | Dec. 3-4
| June 19 (re-verification) | July 19 (re-verification) | Nov. 13 (re-verification) | Dec. 4 (re-verification) |

For more information about ATLS courses, email maleylj@upmc.edu or call 412-647-8115.

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Consider the opportunity to earn continuing education credits by reading *Trauma Rounds* and completing the corresponding continuing education test. After reading, log on to [http://www.upmc.com/Services/EmergencyMedicine/prehospital-care/Pages/TraumaRounds.aspx](http://www.upmc.com/Services/EmergencyMedicine/prehospital-care/Pages/TraumaRounds.aspx). On the *Trauma Rounds* website, you can print the test and mail the completed version back to UPMC, or you can take the test online through the Pennsylvania Department of Health’s online testing program.