

## Rapidly Progressing Necrotizing Fasciitis

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**N**ecrotizing fasciitis, a rare complication of wound infections that is often poorly defined, has been described in the literature since as early as the fifth century BCE.<sup>1</sup> Giuliano et al identified the bacteriology behind the disease and described the etiology based on a skin wound with "partial liquefaction of subcutaneous fat and adjacent deep fascia, with sparing of underlying muscle."<sup>2</sup> When associated with the perineal region in males, it often is referred to as Fournier's disease after the physician who first described the signs and symptoms.<sup>3</sup> There are no clear data to identify incidence; however, Green et al noted that between 1989 and 1991 approximately 5% to 10% of an estimated 10 to 15,000 cases of invasive group A streptococcal infections occurring annually progressed to necrotizing fasciitis.<sup>4</sup> Mortality rates vary but range from 13% to 76%.<sup>4-13</sup> In obstetrics, the risk for necrotizing fasciitis increases with episiotomy or other perineal trauma during delivery. Therefore, attention to risk factors and early signs of necrotizing fasciitis are warranted given the significant reduction in mortality associated with aggressive management.<sup>12,13</sup> We report a case of rapidly progressing necrotizing fasciitis in a young obstetrics patient who died despite swift and appropriate medical and surgical management.

### CASE PRESENTATION

#### Initial Presentation and History

A 19-year-old obese gravida 3 para 2 patient presented with onset of labor at 40 weeks and 2 days gestation as confirmed by first trimester ultrasound. Her prenatal course was remarkable for treatment of bacterial vaginosis, poor compliance to regular prenatal visits, and poor compliance to dietary restrictions given her body habitus. Her medical history was significant only for tobacco use, although she denied use of tobacco or other drugs during pregnancy. She was taking prenatal vitamins and denied any drug allergies.

After 12 hours of labor augmented with oxytocin, the patient progressed to complete dilation and effacement. Labor was complicated by maternal exhaustion and fetal bradycardia. Classical Kielland forceps delivery was performed for a deep transverse arrest of the

vertex in the right occiput transverse position and 0 to +1 station. A right mediolateral episiotomy was performed to minimize the risk of perineal laceration. A viable male infant was delivered with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively. Fecal contamination during labor and delivery was removed several times and the perineum was cleaned with sponges and povidone-iodine solution on multiple occasions. Inspection of the perineum and vagina after delivery revealed a partial third degree extension of the right mediolateral episiotomy and a left sulcus laceration. These were repaired without difficulty. The placenta was spontaneously delivered and estimated blood loss was 450 mL.

#### Two Days Postpartum

Two days postpartum, the patient complained of rectal and perineal pain with rigors. Evaluation was notable for a fever to 103.2° F (39.6°C), tachycardia (pulse, 139 bpm), and swelling and tenderness from the left labia to the buttocks unilaterally without discharge. Her blood sample showed a decreased hematocrit (23.1%) and an elevated leukocyte count ( $24.8 \times 10^3/\text{mm}^3$ ) with 73 segmented neutrophils and 20 banded forms.

A suspicion of chorioamnionitis prompted initiation of intravenous antibiotics (ampicillin, gentamicin, and clindamycin), and the patient was taken to the operating room for an incision and drainage of the right labia. Exploration of the perineum revealed breakdown of the episiotomy wound with a suppurative vaginal abscess extending from the hymenal ring to the ischial spine posteriorly and the periurethral area anteriorly. The wound was irrigated generously with 10%

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povidone-iodine solution and saline, then packed with sterile rolled gauze.

### **Clinical Course**

Initially, the patient showed some subjective improvement of what was thought to be an abscess. A second incision drainage performed 68 hours postpartum revealed brawny edema and necrotic tissue in and around the right labia. Postoperatively, the patient was slow to regain consciousness. She developed tachypnea, periorbital edema, and oliguria, at which point she was moved from the patient ambulatory care unit to the medical intensive care unit. A chest radiograph showed signs of acute respiratory distress syndrome. The patient was intubated, stabilized, and transferred to a tertiary facility for more definitive care.

On arrival at the tertiary facility, a Swan-Ganz catheter was placed to facilitate monitoring and fluid resuscitation. Penicillin was added to the patient's antibiotic regimen. After stabilization, surgeons débrided tissue from her lower abdominal wall to her buttocks. Despite this procedure, her respiratory status continued to deteriorate and the patient developed worsening acidosis that was unresponsive to dialysis. On postpartum day 4, she had multiple episodes of cardiac arrest. She was unresponsive to intervention after the fourth episode and died.

## **DISCUSSION**

### **Necrotizing Fasciitis in Obstetric Patients**

Although necrotizing fasciitis is a rare finding, cases are reported in the literature, and the entity is well known in obstetrics because of increased mortality associated with pelvic infections.<sup>8,11</sup> Nonobstetric literature discusses various risk factors including age, diabetes, prior radiation therapy, immunosuppression, atherosclerosis, obesity, alcoholism, malignancy, and renal disease.<sup>5,7,13,14</sup> The obstetric population generally is not impacted by these comorbid conditions, but pregnant women who do have these conditions are at particular risk. Gallup et al<sup>15</sup> reviewed cases of diagnosed necrotizing fasciitis in their hospital system and noted that the majority of their obstetric patients who developed the disease had concomitant obesity, diabetes, hypertension, and/or heart disease. McHenry et al<sup>16</sup> also note that the altered immune function of pregnancy typically results in a more aggressive course of the disease.

The fecal contamination and pelvic tissue injury typical of the birthing process puts women with immune-compromising comorbidities at risk for postpartum infection. In addition, operative vaginal delivery has been associated with an increase in traumatic compli-

cations.<sup>17</sup> Nevertheless, in cases of fetal bradycardia, arrest of descent, and maternal exhaustion (as with this patient), assisted vaginal delivery has been associated with reduced morbidity in comparison to caesarean section.<sup>18</sup> Furthermore, although the routine use of episiotomies is no longer recommended,<sup>19</sup> even ardent opponents of episiotomy use acknowledge that they may be indicated when expeditious delivery is deemed appropriate.<sup>20</sup>

### **Pathogenesis**

The exact cause of the "flesh-eating" nature of the pathogens involved in necrotizing fasciitis is unclear but is thought to be associated with the release of endogenous cytokines and bacterial toxins.<sup>4</sup> In 1931, Meleney first characterized the synergistic effects of gram-negative and gram-positive bacteria in the propagation of the infection.<sup>21</sup> Using improved technology, Giuliano et al<sup>2</sup> separated patients into the type I (anaerobic and facultative anaerobic bacteria [non-group A streptococcus]) and type II (group A streptococcal organisms) infections. Based on their findings, they recommend penicillin G for the group A streptococcal pathogens and additionally an aminoglycoside and clindamycin for anaerobic coverage in type I infections. Although antibiotics are not thought to independently reduce mortality<sup>8</sup> (in contrast to surgical débridement), they do appear to decrease recovery time.<sup>22,23</sup>

Necrotizing fasciitis develops from a localized infection. It is unclear why some infections spread to the underlying fascia but not others. Once in the fascia, it appears that the toxins that are released aid in the rapid destruction of tissue and the spread of bacteria. When the infection involves the superficial and deep fascia, the overlying, healthy-appearing skin becomes undermined, edematous, and indurated. Often, thrombosis of cutaneous vasculature occurs as the infection spreads, with resultant overlying focal necrotic areas of skin. Gangrene and other frank cutaneous changes usually are not apparent until 4 to 5 days from onset.<sup>24</sup> In addition, destruction of nerves makes the once hyperesthetic skin now hypoesthetic or anesthetic. Spread of the bacteria into the blood results in sepsis and, eventually, multi-organ system failure and death.

### **Clinical Presentation and Diagnosis**

Early recognition and rapid surgical intervention are essential to survival; time to presentation, however, varies greatly. Initially, patients may have little more than local induration, edema, and marked tenderness, and their condition may be mistaken for local trauma or endometritis. A review of 11 different studies

(116 cases) showed an average time from presentation to surgical exploration of 5.4 days (range, 1–15 days).<sup>5,6,9,10,11,14,21,22,25–29</sup> In the present case, the progression of disease was more rapid than usual, with death occurring within 4 days, thus underscoring the need for early identification and the high mortality rate despite aggressive treatment.

High fevers, as observed with this patient, should never be attributed merely to localized infection. Necrotizing fasciitis should be suspected in patients with puerperal wound complications that do not respond to initial débridement and antibiotic coverage within the first 24 to 48 hours, and immediate exploratory surgery should be performed under adequate anesthesia.<sup>8</sup> Differential diagnosis of necrotizing fasciitis includes cellulitis, brown recluse spider bites, phycomycoses, erysipelas, hydradenitis suppurativum, furuncles, impetigo, staphylococcal scalded skin syndrome, cutaneous diphtheria, anthrax, and disseminated infection with *Pseudomonas aeruginosa*, *Neisseria meningitidis*, and *Candida*.<sup>25</sup>

Examination is performed digitally or with a blunt instrument; by passing through the wound or an incision in the suspect area, one can probe the tissue for signs of necrotizing fasciitis. Any lack of resistance in the subcutaneous tissue is suggestive of a disruption of the fascial plane by the spreading bacteria. There is usually an absence of bleeding; the most common clinical finding is a “dishwater” discharge,<sup>8,25</sup> most likely the result of partial liquefaction of subcutaneous fat.<sup>26</sup>

Although subcutaneous exploration is not appropriate in all cases of perineal inflammation, Livengood and Addison<sup>25</sup> note that when subcutaneous edema and induration are present, exploration is warranted. Cultures and radiographic findings may help to diagnose this disease. However, examination suggestive of spreading infection is sufficient enough to warrant immediate treatment; delay for more involved diagnostic tools also may have lethal consequences.

### Management

Most researchers have concluded that complete surgical débridement is essential for survival.<sup>30,31</sup> Rouse et al<sup>13</sup> showed a decrease in mortality from 92% to 60% in nonobstetric patients when antibiotic and surgical intervention occurred within 12 hours, and similar results have been noted in other retrospective studies.<sup>6,8,9,12,16,27</sup> Débridement should be wide and expansive, with excision of tissue until the skin and subcutaneous tissue can no longer be separated from the deep fascia.<sup>24,28</sup> Aggressive fluid resuscitation is encouraged and, in some cases, steroids have been used.<sup>22,32</sup> Hy-

perbaric therapy<sup>6,33</sup> also has been advocated; it is thought that the increased oxygen tension in the wound reduces the virulence of anaerobic pathogens and enhances the microbicidal action of leukocytes, but it is unclear whether there is any added benefit in practice.<sup>24</sup> Some clinicians also have advocated diverting colostomies in postpartum obstetric patients with perineal infections to reduce the risk of further fecal contamination to the wound,<sup>4,14,22</sup> which is thought to be a cause of increased morbidity and mortality in perineal and perianal infections.<sup>8</sup>

Survivors of these perineal necrotizing fasciitis infections typically have prolonged hospital courses. A review of necrotizing fasciitis in obstetric patients showed a mean stay of 31 days.<sup>14,16,21,26,27,32,34</sup> They often require extensive skin grafting and delayed skin closure,<sup>12</sup> but long-term morbidity is minimal. Wound management is similar to the management of burns.<sup>27</sup>

### CONCLUSION

Necrotizing fasciitis, although uncommon, has a highly virulent course and significant mortality rate. Postpartum women seldom have risk factors commonly associated with the disease, but its course is more aggressive in obstetric patients because of their immunosuppressed status. Management centers on early identification, mainly by clinical examination, and wide excisional débridement of the affected tissue. Necrotizing fasciitis is rarely seen in obstetrics, and thus postepisiotomy complications and puerperal pelvic wounds often are treated as the more common hematomas or superficial infections. Because of significantly increased mortality associated with delay to definitive treatment in necrotizing fasciitis, wounds not responding to initial débridement and wide-spectrum antibiotic therapy should be further explored until fasciitis can be ruled out. Any doubt should prompt wide débridement of tissue as discussed. Delay usually has devastating consequences, but appropriate and timely intervention improves the likelihood of survival with low morbidity. **HP**

### REFERENCES

1. Descamps V, Aitken J, Lee MG. Hippocrates on necrotizing fasciitis [letter]. *Lancet* 1994;344:556.
2. Giuliano A, Lewis f, Hadley K, Blaisdell F. Bacteriology of necrotizing fasciitis. *Am J Surg* 1977;134:52–6.
3. Fournier JA. [Gangrene foudroyante de la verge.] [Article in French.] *La Semaine Medicale* 1883;3:345–8.
4. Green R, Dafoe D, Raffin T. Necrotizing fasciitis. *Chest* 1996;110:219–29.
5. Pessa M, Howard R. Necrotizing fasciitis. *Surg Gynecol Obstet* 1985;161:357–61.
6. Gozal D, Ziser A, Shupak A, et al. Necrotizing fasciitis.

- Arch Surg 1986;121:233-5.
7. Francis K, Lamaute H, Davis J, Pizzi W. Implications of risk factors in necrotizing fasciitis. *Am Surg* 1993;59:304-8.
  8. Stone H, Martin J. Synergistic necrotizing cellulites. *Ann Surg* 1972;175:702-11.
  9. Stephenson H, Dotters D, Katz V, Droegemueller W. Necrotizing fasciitis of the vulva. *Am J Obstet Gynecol* 1992;166:1324-7.
  10. Schorge J, Granter S, Lerner L, Feldman S. Postpartum and vulvar necrotizing fasciitis. *J Reprod Med* 1998;43:586-90.
  11. Oh C, Lee C, Jacobson J. Necrotizing fasciitis of perineum. *Surgery* 1982;91:49-51.
  12. Bilton B, Zibari G, McMillan R, Aultman D, Dunn G, McDonald J. Aggressive surgical management of necrotizing fasciitis serves to decrease mortality: a retrospective study. *Am Surg* 1998;5:397-400.
  13. Rouse TM, Malangoni MA, Schulte WJ. Necrotizing fasciitis: a preventable disaster. *Surgery* 1982;92:765-70.
  14. Nolan TE, King LA, Smith RP, Gallup DC. Necrotizing surgical infection and necrotizing fasciitis in obstetric and gynecologic patients. *South Med J* 1993;86:1363-7.
  15. Gallup DG, Freedman MA, Meguir RV, et al. Necrotizing fasciitis in gynecologic and obstetric patients: A surgical emergency. *Am J Obstet Gynecol* 2002;187:305-10.
  16. McHenry CR, Pitrowski JJ, Petrinic D, Malangoni MA. Determinants of mortality for necrotizing soft-tissue infections. *Ann Surg* 1995;221:558-63.
  17. Gei AF, Belfort MA. Forceps-assisted vaginal delivery. *Obstet Gynecol Clin North Am* 1999;26:345-70.
  18. Bashore RA, Philips WN Jr, Brinkman CR 3rd. A comparison of the morbidity of mid-forceps and cesarean delivery. *Am J Obstet Gynecol* 1990;162:1428-35.
  19. Larsson PG, Platz-Christensen JJ, Bergman B, Wallsterson G. Advantage or disadvantage of episiotomy compared with spontaneous perineal laceration. *Gynecol Obstet Invest* 1991;31:213-6.
  20. Klein MC. Reducing perineal trauma and pelvic floor relaxation: an evidence-based approach. *Clin Fam Pract* 2001;3:365-83.
  21. Kovalcik P, Jones J. Necrotizing perineal infections. *Am Surg* 1983;49:163-6.
  22. Golde S, Ledger W. Necrotizing fasciitis in postpartum patients: a report of four cases. *Obstet Gynecol* 1977;50:670-3.
  23. Janevicius R, Hann S, Batt M. Necrotizing fasciitis. *Surg Gynecol Obstet* 1982;154:97-101.
  24. Wilkerson R, Paull W, Coville FV. Necrotizing fasciitis: review of the literature and case report. *Clin Orthop* 1987;216:187-92.
  25. Livengood CH 3rd, Addison WA. Necrotizing fasciitis in obstetrics and gynecology. *Postgrad Obstet Gynecol* 1987;7:1-6.
  26. Shy KK, Eschenbach DA. Fatal perineal cellulitis from from an episiotomy site. *Obstet Gynecol* 1979;54:292-8.
  27. Majeski J, Alexander J. Early diagnosis, nutritional support, and immediate extensive débridement of improve survival in necrotizing fasciitis. *Am J Surg* 1983;145:784-7.
  28. Wilson BL. Necrotizing fasciitis. *Am Surg* 1952;18:416-31.
  29. Rowan JA, North RA. Necrotizing fasciitis in the puerperium. *Am J Obstet Gynecol* 1995;173:241-2.
  30. Pryor JP, Piotrowski E, Seltzer CW, Gracias VH. Early diagnosis of retroperitoneal necrotizing fasciitis. *Crit Care Med* 2001;29:1071-3.
  31. Urschel JD. Necrotizing soft tissue infections. *Postgrad Med J* 1999;75:645-9.
  32. Sutton G, Smirz L, Clark D, Bennett J. Group B streptococcal necrotizing fasciitis arising from an episiotomy. *Obstet Gynecol* 1985; 66:733-6.
  33. Yuen JC, Feng Z. Salvage of limb and function in necrotizing fasciitis of the hand: role of hyperbaric oxygen treatment and free muscle flap coverage. *South Med J* 2002;95:255-7.
  34. Lowthian J, Gillard L. Postpartum necrotizing fasciitis. *Obstet Gynecol* 1980;56:661-3.

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*Phy & Neilson : CVC Guidewire Complication : pp. 41-43, 50*

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(from page 43)

- 1989;12:111-3.
6. Loesberg A, Taylor FC, Awh MH. Dislodgment of inferior vena caval filters during "blind" insertion of central venous catheters. *AJR Am J Roentgenol* 1993;161:637-8.
7. Stuart RK, Shikora SA, Akerman P, et al. Incidence of arrhythmia with central venous catheter insertion and exchange. *JPEN J Parenter Enteral Nutr* 1990;14:152-5.
8. Wang LP, Einarsson E. A complication of subclavian vein catheterisation. Extravascular knotting of a guidewire. *Acta Anaesthesiol Scand* 1987;31:187-8.
9. Kjeldsen L. Transvenous misplacement and loop formation of spring guide wire. *Anaesthesia* 1987;42:216-7.
10. Schweiger J, Fuhrman T. Vascular access and cannulation. In: Murray M, Coursin D, Prough D, editors. *Critical care medicine: perioperative management*. Lippincott-Raven, 1997:71-85.