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1. Ho G Jr, Tice AD, Kaplan SR. Septic bursitis in the prepatellar and olecranon bursae: an analysis of 25 cases. Ann Intern Med 1978;89:21–7.
2. Raddatz DA, Hoffman GS, Franck WA. Septic bursitis: presentation, treatment and prognosis. J Rheumatol 1987;14:1160–3.
3. Woods GL, Washington JA II. Mycobacteria other than *Mycobacterium tuberculosis*: review of microbiologic and clinical aspects. Rev Infect Dis 1987;9:275–94.
4. Benson CA, Ellner JJ. *Mycobacterium avium* complex infection and AIDS: advances in theory and practice. Clin Infect Dis 1993;17: 7–20.
5. Friedman BF, Edwards D, Kirkpatrick CH. *Mycobacterium avium–intracellulare*: cutaneous presentations of disseminated disease. Am J Med 1988;85:257–63.
6. Kelly PJ, Weed LA, Lipscomb PR. Infection of tendon sheaths, bursae, joints, and soft tissues by acid-fast bacilli other than tubercle bacilli. J Bone Joint Surg Am 1963;45:327–36.
7. Winter FE, Runyon EH. Prepatellar bursitis caused by *Mycobacterium marinum (balnei)*. J Bone Joint Surg Am 1965;47:375–9.
8. Kemper C, Havlir D, Haghighat D, Dube M, Bartok A, Deresinski S. Effect of ethambutol, rifampin, or clofazimine, given singly, on *Mycobacterium avium* bacteremia in AIDS [abstract PoB 3087]. VIII International Conference on AIDS/III STD World Congress; Amsterdam; July 1992.

Enterococcal Necrotizing Fasciitis Complicating Elective Hernia Repair in an Infant

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Necrotizing fasciitis is a rapidly progressive bacterial infection that causes local vasculitis, thrombosis, and tissue ischemia, accompanied by widespread necrosis of the deep dermal and superficial fascial layers [1]. The disorder is frequently associated with systemic toxicity and results in significant morbidity and mortality [2,3]. Necrotizing fasciitis may complicate various skin lesions, including traumatic wounds, surgical incisions [4], and the exanthem of primary varicella [5]. A number of bacterial pathogens cause necrotizing fasciitis, most notably the group A *Streptococcus*, and polymicrobial infections are common.

We describe the case of a 7-month-old boy who developed necrotizing fasciitis following elective hernia repair. Wound cultures were positive only for *Enterococcus faecalis*. Because this organism has not previously been reported as the sole etiologic agent of necrotizing fasciitis in any age group, the biologic and therapeutic implications of this finding are briefly discussed.

Case report. A previously healthy 7-month-old boy underwent elective bilateral herniorrhaphy and right hydrocelectomy without incident. That evening at home, his parents noted him to be fussy. During the night, he experienced repeated episodes of emesis and a fever of 40.0°C developed. By the following morning, the left incision had become erythematous and swollen. The infant was seen by a physician at a local hospital, where he was given vancomycin, clindamycin, and gentamicin intravenously while transport to Children's Hospital and Medical Center (Seattle) was arranged.

On arrival in the emergency department, the infant appeared lethargic, with labored respirations. Vital signs revealed a temperature of 38.1°C, pulse rate of 181 beats per minute, and respiratory rate of 62; his blood pressure was 103/54 mm Hg. Erythema and edema surrounding the left surgical incision extended to the left flank and umbilicus. No discharge was apparent. Initial laboratory studies included a leukocyte count of 18,700/mm^3, with 39% neutrophils and 19% band forms. His platelet count was 684,000/mm^3. His serum chemistries were remarkable only for a bicarbonate level of 17 mEq/L, and elevated blood urea nitrogen and creatinine levels of 25 and 1.4 mg/dL, respectively. A flat plate roentgenogram of the abdomen showed dilated loops of small bowel, compatible with ileus or an obstructive process.

During the course of his evaluation, erythema was seen to advance along the patient's left anterior and lateral abdominal wall onto the chest wall. He was taken to the operating room where he underwent exploratory laparotomy through the left herniorrhaphy incision site. Initial enlargement of that incision revealed an extensive area of purulence and necrosis within the subcutaneous tissues. The underlying muscle appeared viable. No free fluid was found in the peritoneal cavity. Inspection of the entire large and small bowel uncovered no evidence of injury or perforation. Extensive debridement of the left lower abdominal subcutaneous tissues and fascia was performed, and frozen-section biopsy specimens were obtained. Aerobic and anaerobic specimens of fluid and tissue for culture were collected intraoperatively and placed in appropriate transport media. The wound was packed with moist gauze. The baby tolerated the procedure well. Postoperatively, he received vancomycin, penicillin, clindamycin, and gentamicin. The patient was immediately transported to a nearby hospital to begin hyperbaric oxygen (HBO) therapy, according to a protocol established

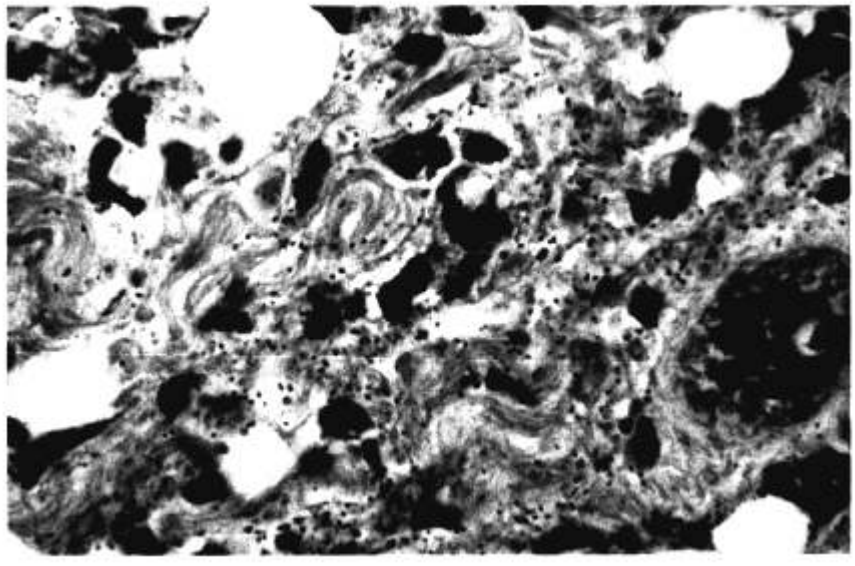

FIGURE 1. Biopsy of pelvic subcutaneous fibrofatty tissue demonstrating necrosis, widespread neutrophilic infiltrate, and numerous enterococci in pairs and short chains. Sample was formalin-fixed and stained with hematoxylin and eosin. Magnification = 1000×.

by the surgical department at our institution. He received a total of five treatments, each at 2.4 atmospheres for 2 hours, separated by 12-hour intervals. Between treatments, the patient returned to the intensive care unit, where he experienced hypotension requiring dobutamine and dopamine infusions. On the third postoperative day, after completion of HBO therapy, inotropic support was weaned, and the patient was extubated successfully.

Biopsies demonstrated necrosis within the fibrofatty connective tissue, marked neutrophilic infiltration, and a single morphologic type of gram-positive cocci in pairs and short chains (Figure 1). Wound (aerobic and anaerobic) and urine cultures yielded only *E. faecalis*, whereas blood cultures proved sterile. After susceptibility testing, antibiotic coverage was narrowed to ampicillin and gentamicin. The wound was closed, using minimal tension, on the fifth postoperative day. The patient received a total of 10 days of parenteral antibiotic therapy before discharge. He has experienced no long-term sequelae.

Discussion. Enterococci are a component of the normal human bowel flora and a leading cause of nosocomial infection [6]. The urinary tract, abdominal and pelvic wounds, and the bloodstream are the most common sites from which enterococci are isolated. Although enterococci may gain access to these sites by direct inoculation, through an indwelling catheter, or through perforation of or injury to the intestinal mucosa, occasionally there exist no obvious predisposing factors [7]. The etiology in such cases may lie in the ability of enterococci to translocate across normal, intact, gastrointestinal epithelium into mesenteric lymph nodes and the bloodstream, as has been demonstrated in animal studies [8,9].

A true pathogenic role for enterococci in abdominal and pelvic soft tissue infections has been questioned. In the majority of cases, enterococci are identified as part of polymicrobial cultures that include a variety of other anaerobic and facultative gut flora [10]. In animal models, *E. faecalis* is known to act synergistically with otherwise avirulent organisms (*Bacteroides*, *Fusobacterium*, anaerobic cocci) to produce skin necrosis, subcutaneous abscesses, intraabdominal abscesses, or peritonitis [11–13]. In these studies, however, *E. faecalis* failed to establish disease when inoculated as a single agent. Some mixed enterococcal soft tissue infections or abscesses can be effectively treated with antibiotic regimens lacking in vitro activity against enterococci, further supporting the concept of a synergistic rather than primary role for this organism [14,15]. Indeed, enterococci may be isolated in as great a frequency from clinically noninfected surgical wounds as from clinically infected ones [16].

The experimental data suggesting a low virulence potential for enterococci as sole agents of severe soft tissue infections are supported by the paucity of cases in the pediatric or adult clinical literature. The enterococci are commonly isolated only from compromised soft tissues such as decubiti, diabetic ulcers, or burns; de novo cellulitis is not seen. In case series of necrotizing fasciitis, enterococci have been listed as contributors to polymicrobial etiologies [1,17], but, to our knowledge, *E. faecalis* has never before been reported as the only culturable isolate. Although antibiotic pretreatment precludes a categorical assertion that *Enterococcus* alone produced necrotizing fasciitis in our patient, the gram stain, the histologic and culture findings from extensive purulent fluid and tissue, the concurrent positive urine culture, and the lack of intestinal perforation indicate a primary role. Inoculation of the wound site with enterococci may have occurred from contaminated urine in this diapered infant.

Enterococci produce a number of virulence factors of potential relevance to necrotizing fasciitis. Enterococcal gelatinase [18] and hyaluronidase [19] could theoretically facilitate spread through soft tissue planes, although these hypotheses have never been tested. Many strains harbor a plasmid-encoded cytolysin, which is associated with a 10-fold-lower LD_{50} following intraperitoneal injection [20] and increased tissue damage in an endophthalmitis model in rabbits [21].

The notion that *Enterococcus* may be more than a passive bystander in the pathogenesis of abdominal or pelvic necrotizing fasciitis has important therapeutic implications. Enterococci are relatively resistant to penicillin and other β-lactam antibiotics, including cephalosporins, on the basis of low-affinity penicillin-binding proteins [22]. For this reason, combination drug therapy with ampicillin or vancomycin plus an aminoglycoside is widely recommended for serious infections or bacteremia [23]. Acquired high-level aminoglycoside and vancomycin resistance among enterococcal isolates is an emerging problem requiring individualized management at given institutions [24]. A further reason to employ specific antienterococcal therapy is the observation that soft tissue infection is an important source for development of enterococcal bacteremia [25]. When necrotizing fasciitis develops in the abdominal or pelvic region, gram-stain identification alone of chaining gram-positive cocci should not prompt narrowing of antibiotic coverage toward the more common group A or anaerobic streptococcal etiologies.

Finally, the use of adjunctive HBO therapy in necrotizing fasciitis caused by a facultative aerobe such as *Enterococcus* may seem superfluous. Nevertheless, we have employed HBO with apparent beneficial effect in therapy of group A streptococcal necrotizing fasciitis complicating primary varicella, [26] as have other investigators treating aerobic and mixed necrotizing fasciitis infections [17,27]. The potential mechanisms by which HBO might exert a therapeutic benefit include improved host leukocyte function [28] or wound healing [29] when oxygen tension is increased in vascularly compromised tissues. Prompt recognition, complete surgical debridement, parenteral antibiotics, and aggressive supportive care, however, remain the cornerstones of successful management of these dangerous infections.

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1. Umbert IJ, Winkleman RK, Oliver GF, Peters MS. Necrotizing fasciitis: a clinical, microbiologic and histopathologic study of 14 patients. J Am Acad Dermatol 1989;20:774–81.
2. Sudarsky LA, Lachinger JC, Coppa GF, Spencer FC. Improved results from a standardized approach in treating patients with necrotizing fasciitis. Ann Surg 1987;206:661–5.
3. Sawin RS, Schaller RT, Tapper D, Morgan A, Cahill J. Early recognition of neonatal abdominal wall necrotizing fasciitis. Am J Surg 1994; 167:481–4.
4. Farrell LD, Karl SR, Davis PK, Bellinger MF, Ballantine TVN. Postoperative necrotizing fasciitis in children. Pediatrics 1988; 82:874–9.
5. Falcone PAS, Pricolo YE, Edstrom LE. Necrotizing fasciitis as a complication of chickenpox. Clin Pediatr 1988;27:339–43.
6. Emori TG, Gaynes RP. An overview of nosocomial infections, including the role of the microbiology laboratory. Clin Microbiol Rev 1993;6:428–42.
7. Landry SL, Kaiser DL, Wenzel RP. Hospital stay and mortality attributed to nosocomial enterococcal bacteremia: a controlled study. Am J Infect Control 1989;17:323–9.
8. Alexander JW, Boyce ST, Babcock GF, et al. The process of microbial translocation. Ann Surg 1990;212:496–512.
9. Wells CL, Jechorek RP, Erlandsen SL. Evidence for the translocation of *Enterococcus faecalis* across the mouse intestinal tract. J Infect Dis 1990;162:82–90.
10. Dougherty SH. Role of *Enterococcus* in intra-abdominal sepsis. Am J Surg 1984;148:308–12.
11. Onderdonk AB, Bartlett JG, Louis T, Sullivan-Seigler N, Gorback SL. Microbial synergy in experimental intraabdominal abscess. Infect Immun 1976;12:22–6.
12. Matlow AG, Bohnen JMA, Nohr C, Christou N, Meakins J. Pathogenicity of enterococci in a rat model of fecal peritonitis. J Infect Dis 1989;160:142–5.
13. Brook I. Effect of *Streptococcus faecalis* on the growth of *Bacteroides* species and anaerobic cocci in mixed infection. Surgery 1988;103: 107–10.
14. Willey SH, Hindes RG, Eliopoulos GM, Moellering RC. Effects of clindamycin and gentamicin and other antimicrobial combinations against enterococci in an experimental model of intraabdominal abscess. Surgery 1989;169: 199–202.
15. Bartlett JG, Onderdonk AB, Louis T, Kasper DL, Gorbach SL. A review: lessons from an animal model of intraabdominal sepsis. Arch Surg 1978;113:853–7.
16. Horvitz RA, Von Graevenitz A. A clinical study of the role of enterococci as sole agents of wound and tissue infections. Yale J Biol Med 1977;50:391–5.
17. Hirn M. Hyperbaric oxygen in the treatment of gas gangrene and perineal necrotizing fasciitis: a clinical and experimental study. Acta Chir 1993;570:S6–S36.
18. Makinen PD, Clewell DB, An F, Makinen KK. Purification and substrate specificity of a strongly hydrophobic extracellular metalloendopeptidase ("gelatinase") from *Streptococcus faecalis* (strain OG1–10). J Biol Chem 1989;264:3325–34.
19. Jett BD, Huycke MM, Gilmore MS. Virulence of enterococci. Clin Microbiol Rev 1994;7: 462–78.
20. Ike Y, Hashimoto H, Clewell DB. Hemolysin of *Streptococcus faecalis* contributes to virulence in mice. Infect Immun 1984;45:528–30.
21. Jett BD, Jensen HG, Nordquist RE, Gilmore MS. Contribution of the pAD1-encoded cytolysin to the severity of experimental *Enterococcus faecalis* endophthalmitis. Infect Immun 1992;60:2445–52.
22. Williamson R, LeBouguenec C, Gutmann L, Horaud T. One or two low affinity penicillin-binding proteins may be responsible for the range of susceptibility of *Enterococcus faecium* to benzylpenicillin. J Gen Microbiol 1985;131:1933–40.
23. Murray BE. The life and times of the *Enterococcus*. Clin Microbiol Rev 1990;3:46–65.
24. Moellering RC. Emergence of *Enterococcus* as a significant pathogen. Clin Infect Dis 1992; 13:1173–8.
25. Malone DA, Wagner RA, Myers JP, Watanakunakorn C. Enterococcal bacteremia in two large community teaching hospitals. Am J Med 1986;81:601–6.
26. Brogan TV, Nizet V, Waldhausen JHT, Rubens CE, Clarke WR. Group A streptococcal necrotizing fasciitis complicating primary varicella: a series of fourteen patients. Ped Inf Dis J 1995;14:588–94.
27. Riseman JA, Zamboni WA, Curtis A, Graham DR, Konrad HR, Ross DS. Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. Surgery 1990;108:847–50.
28. Hohn DC, MacKay RD, Halliday B, Hunt TK. Effect of oxygen tension on microbicidal function of leukocytes in wound and in vitro. Surg Forum 1976;27:18–20.
29. Kivisaari J, Niinikoski J. Effect of hyperbaric oxygenation and prolonged hypoxia on the healing of open wounds. Acta Chir Scand 1975;141:14–9.