Abdominal Wall Necrotizing Fasciitis: It Is Still "Meleney's Minefield"

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Necrotizing fasciitis is perhaps the deadliest form of soft-tissue infection, characterized by a fulminant course and a high mortality rate.[1] Meleney in 1924 gave the first modern description of this clinical entity when he introduced the term "acute hemolytic streptococcal gangrene."[2] The term necrotizing fasciitis, coined by Wilson in 1952, is perhaps the most accurate term describing the key features of this infectious process.[3] Although our understanding of the pathophysiology of necrotizing fasciitis continues to improve, physicians confronted with this clinical entity continue to thread through "Meleney's minefield," fraught with dangers of delayed or even missed diagnosis with catastrophic consequences. In this article, we present a case that we recently managed and highlight the deceptively benign initial presentation of necrotizing fasciitis.

CASE REPORT

A 66-year-old woman with poorly controlled diabetes mellitus was admitted for right loin pain for the previous 7 days associated with vomiting. There was no antecedent trauma to the affected area. She had previously consulted a general practitioner, who attributed her symptoms to urinary tract infection and prescribed a course of antibiotics and analgesics. She came to us when the right loin pain worsened and was admitted to the medical unit. On examination, her right loin was slightly tender and renal punch was positive. She had no urinary symptoms such as dysuria or hematuria. She was afebrile and her vital signs were stable. Blood and urine cultures taken at admission did not grow any organism. There was minimal erythema, warmth, or other sign of soft-tissue infection. Intravenous ceftriaxone and cloxacillin were started.

She developed a low-grade fever and her right flank pain progressively worsened. This was tender to palpation and was slightly warm. However, there was minimal erythema and there was no crepitus or other skin change. Computed tomographic scanning of the abdomen was performed for suspected pyelonephritis. This showed thickening of the lateral abdomen and chest wall, with gas in the soft-tissue planes. No other intraabdominal abnormalities were seen (Fig. 1). An emergency operative débridement was performed, and necrotizing fasciitis was noted with grayish necrotic fascia, lack of resistance of normally adherent deep fascia to blunt dissection, lack of bleeding of the fascia during dissection, and the presence of foul-smelling "dish-water" pus. Extensive wound débridement was performed (Fig. 2). Histologic examination of the resected tissue specimen confirmed the diagnosis of necrotizing fasciitis (Fig. 3). Tissue cultures taken at operation grew group B streptococcus. Antimicrobials were converted to high-dose penicillin and clindamycin. Her recovery was complicated by acute renal failure and respiratory failure. She required at total of seven wound débridements before finally achieving wound coverage with split-thickness skin grafting.

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DISCUSSION

Delayed recognition or diagnosis of necrotizing fasciitis with consequent delayed operative débridement has been shown in many clinical series to increase mortality.[1,4–10] This case illustrates the difficulty in early recognition of necrotizing fasciitis. The paucity of specific cutaneous signs early in the evolution of the disease makes early necrotizing fasciitis clinically indistinguishable from other soft-tissue infections.[1,7–11] Although it is common practice to initiate empirical antimicrobial treatment for soft-tissue infections such as erysipelas and cellulitis and expectantly await resolution of infection within the next 24 to 48 hours, adopting this approach in cases of necrotizing fasciitis is all too often disastrous.[1,11]

Our patient was admitted for right flank pain and was afebrile at admission. After failing to respond to antibiotics, a computed tomographic scan obtained for suspected renal abnormality revealed an unsuspected necrotizing soft-tissue infection, by which time extensive soft-tissue destruction had occurred. This case illustrates the difficulty (and sometimes impossibility) in the

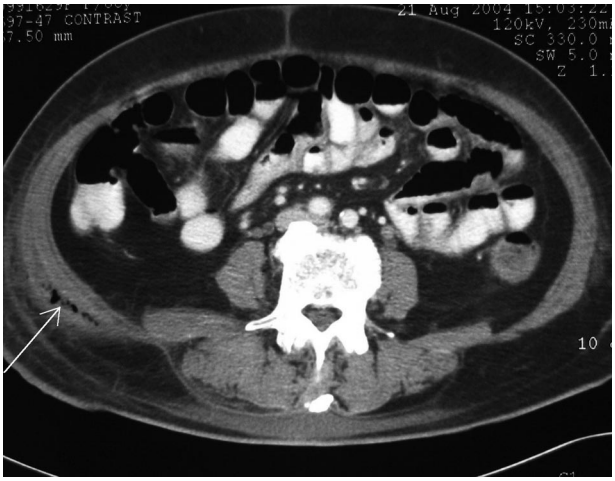

Fig. 1. Computed tomographic scan of the abdomen showing deep fascial thickening, swelling of the subcutaneous tissue, and fluid and gas (*arrow*) in soft tissue of the abdominal wall, features consistent with abdominal wall necrotizing fasciitis.

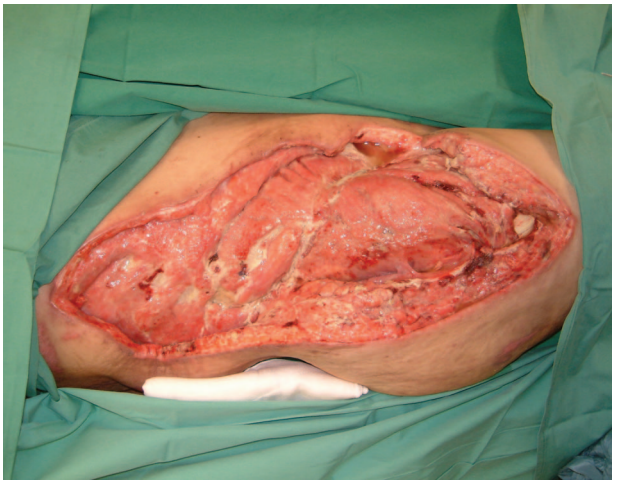

Fig. 2. Extensive soft-tissue loss noted after wound débridement.

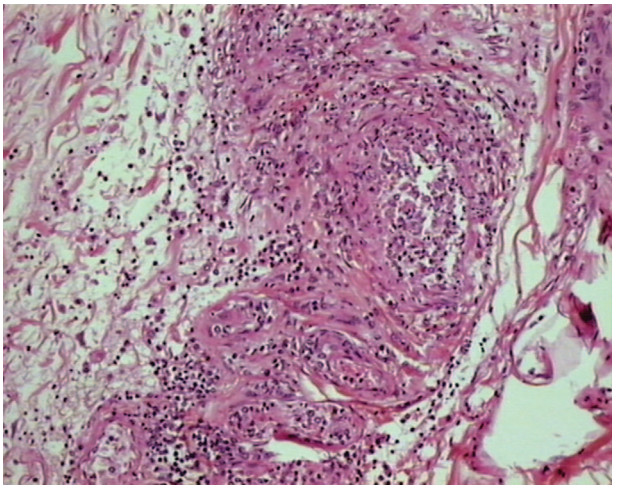

Fig. 3. Histologic section of tissue specimen showing characteristic findings of necrotizing fasciitis with necrosis of the superficial fascia, polymorphonuclear leukocyte infiltration of the deep dermis and fascia, thrombosis and suppuration of the veins and arteries coursing through the fascia, and microorganism proliferation within the destroyed fascia.

clinical recognition of early necrotizing fasciitis. Histologically, necrotizing fasciitis is characterized by angiothrombotic microbial invasion of the fascia.[12] As this process progresses, occlusion of perforating nutrient vessels to the skin causes progressive skin ischemia. An intermediate stage is characterized by multiple cutaneous blisters or bulla. This is a crucial early sign of skin ischemia and should raise the suspicion of necrotizing fasciitis. The so-called hard signs of necrotizing fasciitis become apparent only late in the evolution of the disease. Large hemorrhagic bullae, frank cutaneous gangrene, fluctuance, crepitus, and sensory and motor deficits are late signs seen when destruction of the underlying fascia is well established.[1,8–10]

Confounding this difficulty in recognizing early necrotizing fasciitis is the systemic manifestation of the disease. Although necrotizing fasciitis has classically been associated with pronounced systemic toxicity, it is clear from the recent literature that patients may appear to be quite well initially, with no fever, systemic toxicity, or hypotension.[1] In our previous review of 89 patients with necrotizing fasciitis, we found that only 47 (52.8 percent) patients were febrile at admission.[1] We postulated that widespread use of broad-spectrum antimicrobials may be responsible for this by blunting the systemic manifestation of the infection by reducing the systemic bacterial load. Recently, a variant form of necrotizing fasciitis, subacute necrotizing fasciitis, has been increasingly reported.[13–17] This form of necrotizing fasciitis has an indolent initial presentation, with minimal pain and systemic signs of sepsis, often festering for weeks. Sudden deterioration with rapid progression of infection usually follows later in the course of subacute necrotizing fasciitis. It affects predominantly immunocompromised hosts such as patients with diabetes mellitus or chronic liver disease. When operative débridement is eventually performed, extensive fasciitis reveals the true extent of the infection. The pathophysiology and causes of subacute necrotizing fasciitis are at this moment not well defined.[13–17]

Necrotizing fasciitis has been divided into distinct groups based on the microbiology of

infection.[18] Type I is polymicrobial infection involving synergistic infection by aerobic and anaerobic organisms, and type II necrotizing fasciitis is caused by group A streptococci, either alone or in combination with *Staphylococcus aureus*. Recently, as in this patient, there has been an increasing number of reports of necrotizing fasciitis caused by group B streptococci.[19] Horizontal transfer of DNA-encoding virulence factors from group A streptococci to group B streptococci has been postulated to be responsible for this and confers mutant strains of group B streptococci with increased invasiveness and virulence. The emergence of group B streptococci as causative organisms of necrotizing fasciitis is an area that warrants further intensive investigations.[19]

Antimicrobials are important in the management of necrotizing fasciitis. However, it is important to understand that although antimicrobials reduce the systemic bacterial load and the incidence of organ failure, they have very little effect on the primary site of disease.[1,8–10,18] The antibiotics therefore may ironically serve to temporarily mask the severity of the underlying infective process.[1] Progression of infection despite administration of antimicrobials is the rule in necrotizing fasciitis. This condition is characterized by an angiothrombotic microbial invasion and liquefactive necrosis.[12] Poor tissue penetration of antimicrobial enables the offending organisms to proliferate and flourish, with progressive tissue loss and necrosis. To address the primary disease, excision of all infected tissue is the only effective means of treatment.

Although aggressive surgical débridement is advised, a focused and clear aim is of paramount importance when performing the débridement. All necrotic fascia should be excised. This includes all superficial fascia that can be lifted off underlying deep structures such as muscles and tendons by blunt dissection. The overlying skin and subcutaneous tissue is a subject of some controversy. Some authors have advised excision of all soft tissue that can be lifted off deep structures.[4,6–8] Although this approach is safe in terms of clearance of infected tissue, it often results in large skin defects that are difficult to resurface, particularly in anatomically sensitive areas such as the hand. We adopt a more conservative approach when performing débridement. All fascia on the skin and some subcutaneous fat in the skin flap that can be lifted off the deep structures are meticulously excised. The remaining skin and subcutaneous fat are then progressively cut back to assess the quality of bleeding from the skin edges. This indicates the sufficiency of the blood supply mainly from the subdermal plexus as a random pattern skin flap. When adopting this approach, reexamination every 24 to 48 hours is crucial. Nonviable skin flap is further cut back until infection is controlled. When the wound bed is sufficiently clean, we use negative-pressure therapy (V.A.C. dressing; Kinetic Concepts, Inc., San Antonio, Texas) (continuous negative pressure of 40 to 100 mmHg) to encourage granulation before skin grafting and after skin graft to improve graft take.

The therapeutic challenge in the management of necrotizing fasciitis today is early recognition. This can dramatically reduce the mortality and morbidity of this condition. Clinical acumen and a high index of suspicion are the most important skills to acquire. In this respect, an early consultation with an experienced surgical team is important when patients are admitted to medical units for severe soft-tissue infections. However, as has often been demonstrated, early necrotizing fasciitis have been missed even by experienced surgeons. In the absence of diagnostic clinical findings, imaging modalities such as computed tomography and magnetic resonance imaging can be very helpful diagnostic adjuncts. Features of necrotizing fasciitis seen on computed tomographic scans include deep fascial thickening, enhancement, fluid, and gas in soft-tissue planes.[22–25] Magnetic resonance imaging of the affected areas has also been reported to be helpful in the early identification of necrotizing fasciitis. Features described to be distinct for necrotizing fasciitis include deep fascial thickening, deep fascial fluid collections, and hyperintense T2-weighted signal within the muscles.[20–22] Fascial enhancement has been described as a feature by some authors,[20,21] whereas other authors report the lack of fascial enhancement as a reliable indicator. However, although imaging has its role, it should not delay operative débridement. When the index of suspicion is high, the most appropriate action is to explore the wounds in the operating room. As mentioned previously, the clinical profile of early necrotizing fasciitis is difficult to recognize because of the paucity of specific cutaneous findings.[1,7–11] We studied the biochemical profile of patients with necrotizing fasciitis and compared these with patients with other severe soft-tissue infections and found that necrotizing fasciitis has a distinct biochemical profile. A diagnostic scoring system based on laboratory parameters routinely performed for all soft-tissue infections and readily available at admission (complete blood count, electrolytes, and C-reactive protein) was devised. Called the Laboratory Risk Indicator for Necrotizing

Fasciitis (LRINEC) score, this diagnostic adjunct is capable of distinguishing early cases of necrotizing fasciitis from other soft-tissue infections.[11]

Definitive diagnosis can often be made at operative exploration. The following are features of necrotizing fasciitis: the presence of grayish necrotic fascia, demonstration of a lack of resistance of normally adherent deep fascia to blunt dissection, lack of bleeding of the fascia during dissection, and the presence of foul smelling dish-water pus.[1,4–10] Intraoperatively, tissue should be taken for cultures and histologic examination. Histologic examination is important for confirming the diagnosis of necrotizing fasciitis. Histologic criteria for diagnosis of necrotizing fasciitis as described by Stamenkovic and Lew[12] can reliably identify even early cases of necrotizing fasciitis. This is important, particularly in cases where the operative findings were equivocal for early necrotizing fasciitis, as it determines the need for an early second look and repeat débridement.

CONCLUSIONS

Necrotizing fasciitis is a deadly disease that is clinically indistinguishable from other soft-tissue infection early in its evolution. A high index of suspicion and regular review for progression or worsening are important in patients in whom necrotizing fasciitis is suspected. Bulla or blistering of the skin are important diagnostic clues and should raise the index of suspicion. When in doubt, early exploration in the operating room is advisable. Modalities such as computed tomography or magnetic resonance imaging can be helpful diagnostic adjuncts in indeterminate cases but should not delay operative exploration in patients in whom the suspicion is sufficiently high. With a focused approach in the evaluation of soft-tissue infections, we hope that necrotizing fasciitis can be detected early in its evolution and survival improved.

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