

Severe Necrotizing Fasciitis of the Abdominal Wall Secondary to Colon Perforation in a Child

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We discuss the successful saving of an 18-month-old boy with necrotizing fasciitis of the abdominal wall secondary to colon perforation and peritonitis. The patient underwent emergency surgery with exploratory laparotomy, repeated procedures of debridement, and reconstructive abdominal wall surgery with skin graft. He recovered 6 months after admission. We also address the issue of the closure of large abdominal wall defect and the importance of alimentation in this patient group. We discuss the need to recognize necrotizing fasciitis as a potential complication of intra-abdominal disease, as once necrotizing fasciitis occurs, mortality is more likely. We conclude that recognition and aggressive surgical debridement is mandatory. [*J Chin Med Assoc* 2008;71(5):259–261]

Key Words: necrotizing fasciitis, split thickness skin graft

Introduction

Necrotizing fasciitis (NF) is a life-threatening infection and necrosis of the skin, subcutaneous tissue, deep fascia and muscle, with a fulminant course and a high mortality rate. NF may be a complication of minor soft tissue infection, or it can occur after a trauma or surgical procedure. Intra-abdominal pathology such as necrotizing enteritis, appendicitis or abscesses may spread to the abdominal wall and result in abdominal wall NF.^{1,2} We report a case of severe abdominal wall NF with massive abdominal wall defect following necrotizing colitis in a young boy. The importance of aggressive surgical management and control of the disease are highlighted.

Case Report

This boy was in good condition after birth until the age of 18 months when he had an episode of bloody mucous diarrhea and fever. He was managed in the hospital, where colonoscopy demonstrated multiple ulcers in the colon. Pathologic findings of the mucosa revealed chronic inflammation and focal hemorrhage.

Stool culture revealed normal flora. Ten days later, the patient was discharged in good condition.

Three months later, he was hospitalized again because of high fever, vomiting and diarrhea. Pneumonia, demonstrated on chest film, and enteritis were diagnosed initially. Three days after admission, he developed severe sepsis with hypotension requiring fluid resuscitation, as well as generalized ecchymosis, especially at bilateral abdominal wall and extremities. Disseminated intravascular coagulopathy developed soon after. He was also noted to have abdominal distension. Therefore, exploratory laparotomy was performed when the patient was stabilized on the same day. Laparotomy disclosed severe colitis with a perforation hole in the ascending colon near the ileocecal junction. Simple closure of the perforation and ileostomy was performed. However, ecchymosis and swelling of the abdominal wall did not decrease (Figure 1A). Gangrenous change of some fingers and toes also occurred (Figure 1B). The necrosis of the abdominal wall progressed, finally resulting in a 15 × 10-cm defect in the left and a 10 × 8-cm defect in the right abdomen (Figure 2). During this period, the patient re-entered the operation room several times for checking of abdominal wall bleeding and debridement.



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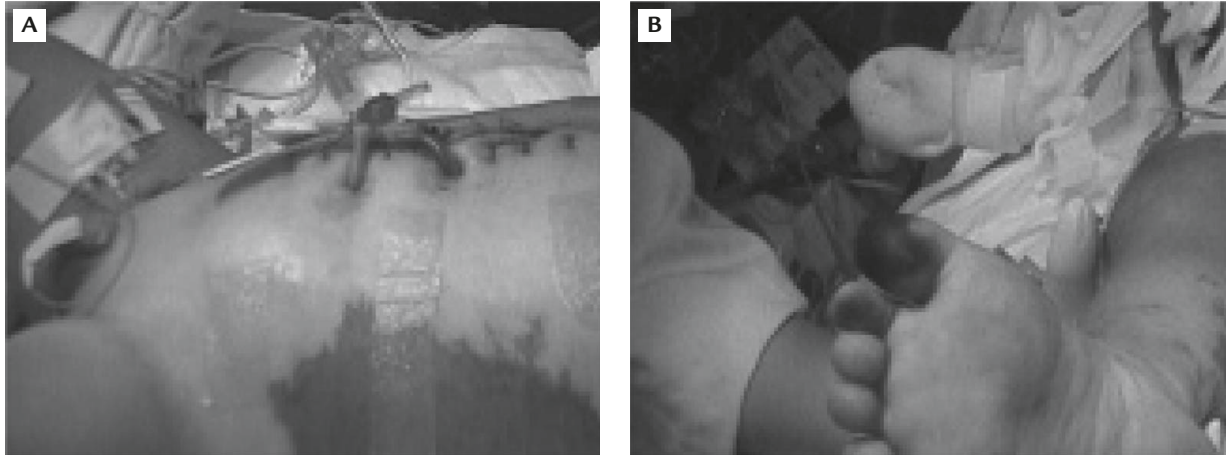


Figure 1. (A) Large area of ecchymosis and necrosis of the left side of the abdominal wall. (B) Ecchymosis and necrosis of the leg and toes.



Figure 2. Exposure of the intestines after full-layer necrosis of the abdominal wall. The Mersilene mesh was partially removed.

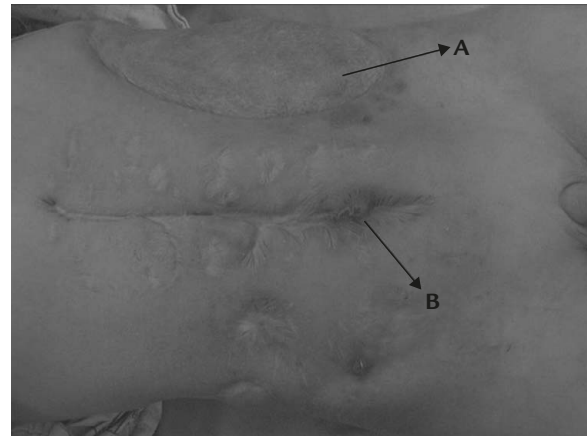


Figure 3. The final appearance of the abdomen. There is a large ventral hernia (A) on the left side. There is also chronic infection (B) of the wound at the lower midline.

The abdominal wall defects were temporarily closed with Mersilene mesh. Another 2 perforations of the small intestine were noted during surgery. Polymicrobial growth of organisms, including *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Proteus mirabilis*, *Escherichia coli*, *Morganella morganii* and yeast, were repeatedly cultured from the patient's abdominal wall.

Sepsis was successfully controlled with amikacin, maxipime and amphotericin B. Nutrition was maintained by total parenteral nutrition until oral intake was tolerated. Further debridement, revision of the wounds and ileostomy were carried out later. Over the following 4 months, multiple reconstructive procedures were initiated to close the abdominal wall defect. Some of the defects were covered by a local rotational flap, but the remaining larger areas of defect needed split thickness skin graft (STSG) coverage.

Six months after the onset of disease, the patient was discharged from hospital with an ileostomy at the right lower abdomen, several enterocutaneous fistulas

on the left upper abdomen, and 2 small skin defects in the middle, with chronic infection due to underlying mesh. Most of the defects were well covered with STSG.

More than 10 operations, during a whole year, were carried out to manage the perforated intestines and necrosis of the abdominal wall (Figure 3). The patient had normal activity and satisfactory growth in the following 3 years. The fingers and toes that were gangrenous initially were auto-amputated at the most distal parts and had normal function. An abdominal bandage was recommended because of large ventral hernia. We plan to continue management of this problem in the following years.

Discussion

NF is infrequent and is usually fatal in infants and children. There are some reports of childhood NF

resulting from appendicitis, intra-abdominal abscess, omphalitis, balanitis and mammitis.^{3,4} Predisposing factors vary with age. Diabetes, immunocompromised status,⁵ and taking nonsteroidal anti-inflammatory drugs⁶ have been implicated as predisposing factors in adults. In children, varicella infection,⁷ malnutrition, and immunosuppression due to leukemia are associated with NF. In our patient, NF resulted from a combination of shock and septic emboli secondary to a perforated colon; the latter also caused gangrene of the fingers and toes.

The importance of early diagnosis of NF is strongly emphasized. The clinical symptoms and signs, such as erythematous rashes and other signs of sepsis, are important for differential diagnosis.⁸ Intra-abdominal disease such as perforated colon or appendix should always be considered as the source of abdominal wall necrotizing colitis. Sonograms and magnetic resonance imaging may be helpful in the diagnosis of NF, although the findings may be nonspecific.⁹ In many cases, it is difficult to distinguish early NF from cellulitis since fever, skin rashes and other clinical findings are common symptoms of infection and sepsis. In our case, the signs of peritonitis were masked by the coexisting pneumonia. As a result, early diagnosis was difficult. Once extensive ecchymosis of the skin developed, we had little means to stop the process. NF has been divided into 2 types.^{10,11} Type I usually involves the abdominal wall, perineal and groin areas and postoperative wounds. It is always caused by Gram-positive or Gram-negative polymicrobial growth and anaerobes. Type II infection is usually caused by *Streptococcus pyogenes* infection. Our case was a typical type I infection. Once blood culture and wound culture have been obtained, broad spectrum antibiotics should be given.

Aggressive surgery and debridement are usually required in combination with antibiotic therapy to limit the spread of infection. In our patient, repeated debridement and skin grafts were carried out, and these may have been the keys to success in avoiding widespread infection. After extensive debridement, the covering and closure of the wound became a major problem. The wound was initially covered with Mersilene mesh. This mesh caused many problems because it adhered firmly to nearby tissue and intestines and was difficult to remove later. If we had used silastic sheets at the beginning, the problem would have been minimized. For long-term coverage, a local rotational flap was tried, although it was insufficient because of the large defects. Eventually, the wound was mainly covered with STSG, which was taken from the patient's scalp. Reconstruction of large abdominal wall defects remains a challenging

problem. STSG was used directly to close the abdominal defect and worked well, although treatment of the patient's ventral hernia will be a challenge in the future.

Other adjunctive wound therapies such as hyperbaric oxygen therapy have been proposed for improving the outcome. We have had no experience in using hyperbaric oxygen therapy in a critical young patient, although its application in the management of NF is supported in some reports.^{12,13} The proposed mechanisms of this therapy are the bactericidal effect on anaerobes and improved tissue oxygenation.

In conclusion, NF is a potential complication of intra-abdominal pathology. Despite the high mortality, it is important to maintain prompt resuscitation and supportive therapy with fluids. Aggressive surgical debridement, appropriate antibiotic therapy and hyperalimentation seem to be the cornerstone of effective therapy.

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