Necrotizing fasciitis: an unusual cause

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INTRODUCTION

Necrotizing fasciitis is a polymicrobial synergistic infection that spreads rapidly along fascial planes with secondary involvement of muscle, subcutaneous tissue and skin leading to extensive tissue destruction and death (Heitmann et al, 2001). The usual causes include surgery, trauma and perineal infections.

Necrotizing fasciitis has been rarely reported in conjunction with large bowel pathology (Groth and Henderson, 1999; Sy et al, 2001). This article presents a case with an unusual cause.

DISCUSSION

Necrotizing fasciitis is defined as a polymicrobial synergistic infection that spreads rapidly along fascial planes with secondary involvement of muscle, subcutaneous tissue and skin. The aetiological factors include trauma, surgery and perineal infections (Cushieri et al, 1995; Groth and Henderson, 1999; Sy et al, 2001). Known risk factors include diabetes mellitus, immunosuppression, excessive use of non-steroidal anti-inflammatory drugs, malnutrition and intravenous drug abuse. A detailed literature search, however, has not revealed any record of this condition complicating a perforated duodenal ulcer. The condition often presents with minimal clinical signs, giving a false sense of security (Hung et al, 1996). If left untreated there can be rapid spread of the infection with massive subcutaneous necrosis. Septic shock rapidly ensues and death from multiorgan failure can occur.

Computed tomography may show fascial thickening, fat infiltration, focal fluid collection, soft tissue gas, muscle involvement and intra-abdominal extension (Wysoki et al, 1997). Management is aggressive volume resuscitation, intravenous broad-spectrum antibiotics, radical surgical debridement and secondary wound closure as appropriate (Bilton et al, 1998). The prognosis is poor with high mortality.

This case was atypical not only in its aetiology but also because of the absence of any underlying risk factors in the patient and serves to highlight the need for a high index of suspicion and aggressive management. HM

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CASE REPORT

A 76-year-old woman was admitted through the accident and emergency department with a paraumbilical lump of 2 weeks' duration and a sinus which was discharging foul-smelling pus since that morning. One month before this, she was treated for abdominal pain with diarrhoea and vomiting by her GP with loperamide. On examination, she was febrile but was stable. Abdominal examination revealed a 10 cm x 5 cm fluctuant paraumbilical swelling with a sinus discharging foul-smelling pus. The surrounding skin was erythematous with patchy blackened areas. The provisional diagnosis at this stage was of an abdominal wall abscess with a possible colocutaneous fistula.

Investigations revealed leucocytosis (19.6×10^9/litre). Erect chest and supine abdominal radiographs were normal. An urgent computed tomography scan with oral contrast revealed an anterior abdominal wall abscess, oedematous mesentery and sigmoid diverticular disease. There was also evidence of some fat necrosis and free gas in the omentum but no obvious breach in the peritoneum or communication with underlying bowel.

The patient was taken to surgery and the abscess cavity was deroofed. The contents had the typical appearance of 'dishwater pus'. There was gross necrosis of the anterior rectus sheath and radical debridement was carried out with excision of the affected rectus sheath, part of the rectus abdominis muscle and the overlying necrotic skin. It was then decided to proceed with formal laparotomy, which showed an inflammatory mass involving the omentum and transverse colon adherent to the anterior abdominal wall and right liver edge. On careful dissection of this mass, a 5 mm perforated chronic duodenal ulcer became apparent. This was repaired and a thorough peritoneal lavage carried out. Reconstruction of the abdominal defect with healthy anterior rectus sheath was achieved with some difficulty after extensive mobilization. The skin and subcutaneous tissue were left open and packed loosely with proflavine gauze to facilitate free drainage.

Microbiology of the pus swab revealed anaerobes, diptheroids and streptococcus D (Elliott et al, 2000). Postoperatively, the patient was managed on antibiotics and regular wound cleansing under aseptic conditions which involved general anaesthesia on two further occasions. Primary closure of the wound was performed 3 weeks later and the patient made a slow but uneventful recovery and was discharged on triple therapy for *Helicobacter pylori* eradication. Histopathological examination of the skin and necrotic tissue showed dermal thrombi, necrosis in the dermis with the underlying fat and skeletal muscle showing evidence of inflammation with abscess formation, thereby confirming the diagnosis of necrotizing fasciitis (Walter and Talbot, 1996).