Necrotising fasciitis secondary to a colocutaneous fistula

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ABSTRACT

Colocutaneous fistulae secondary to diverticular disease are rare, especially spontaneous fistulae. We report a case of a 74-year-old lady, with no previous history of diverticular disease, presenting with necrotising fasciitis of the anterior abdominal wall in the left iliac fossa, without any other symptoms. Urgent surgery was performed. An initial diagnostic laparoscopy demonstrated a perforated sigmoid diverticulum forming a fistula to the anterior abdominal wall. Following soft tissue debridement, a sigmoid colectomy was performed through a midline laparotomy.

Gastrointestinal pathology should be considered as a potential cause of abdominal wall necrotising fasciitis. Our approach of using laparoscopic visualisation to assess for intra-abdominal sources in this context (in the absence of preoperative imaging when imaging could delay treatment) has not been described before. To our knowledge, only two cases of abdominal wall necrotising fasciitis secondary to diverticular disease with a colocutaneous fistula have been reported in the English literature.

KEYWORDS

General surgery – Necrotising fasciitis – Colonic diverticulosis

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Colonic diverticular disease is increasingly common in the UK, especially in the elderly population.¹ Consequently, its complications, which include abscess, stricturing, perforation, fistula and haemorrhage, would be expected to increase as the proportion of the aging population rises. Colocutaneous fistulae in the context of diverticulitis are rare, presenting in about 1% of patients with diverticular disease.² The majority develop after previous bowel resection for diverticulitis³ and spontaneous colocutaneous fistulae are unusual.

We describe a case of a spontaneous colocutaneous fistula secondary to diverticular disease, with perforation, presenting as necrotising fasciitis (NF) of the anterior abdominal wall, as a first presentation of diverticular disease. To our knowledge, this presentation is exceedingly rare, with only two cases reported in the English literature.^{4,5}

Case history

A 74-year-old woman presented with a 3-day history of left-sided abdominal pain, general malaise and a black skin lesion on the left lower quadrant, which was rapidly increasing in size. There was no reported change in bowel habits. The only past medical history of note was a myocardial infarction five years before. At the time of presentation, she had a temperature of 37° and was haemodynamically stable. There was a large tender area of necrotic skin with crepitus to the left

lower quadrant of the abdomen (Fig 1), which was otherwise soft.

The patient's laboratory results revealed raised inflammatory markers (white cell count $43.4 \times 10^9/l$, C-reactive protein 432mg/dl), an acute kidney injury (creatinine 100µmol/l, urea 27.7mmol/l), deranged clotting (international normalised ratio 1.5) and metabolic acidosis. A diagnosis of NF was made, and it was decided to proceed to immediate surgery after adequate resuscitation with intravenous fluids and antibiotics. (Benzylpenicillin and clindamycin were started.)

No preoperative radiological imaging was obtained. At surgery, a diagnostic laparoscopy was initially performed as a potential causative intra-abdominal pathology was considered. A perforated sigmoid diverticulum was found, forming a fistula to the anterior abdominal wall in the left iliac fossa (Fig 2), but there was no peritoneal contamination. The 30cm x 30cm necrotic area in the anterior abdominal wall was debrided; there was no involvement of musculature (Fig 3). A midline laparotomy incision was then made and a sigmoid colectomy performed. The debrided area on the left, which was felt to potentially require further debridement, precluded an optimally sited left-sided stoma. It was therefore decided to form a loop ileostomy in the right iliac fossa.

Following surgery, the patient had a short stay on the intensive care unit. Pathological examination of specimens

Figure 1 Large area of necrotic skin in the left iliac fossa

Figure 2 Perforated sigmoid diverticulum forming a fistula to the anterior abdominal wall in the left iliac fossa

Figure 3 Left iliac fossa wound following debridement of necrotic tissue

confirmed the diagnoses of diverticular disease with perforation and NF. Cultures from the wound grew mainly *Escherichia coli*, *Bacteroides* spp and *Streptococcus porcinus*. Intravenous antibiotics were changed to piperacillin/tazobactam and metronidazole according to sensitivities. The debrided wound remained healthy and no further surgery was required. The patient made good progress clinically and was started on negative pressure wound therapy 11 days post-operatively, with continued improvement of the wound (Fig 4). Her care has been transferred to the regional plastic surgery service for skin grafting.

Figure 4 Improvement of wound with negative pressure wound therapy

Discussion

NF is a rapidly progressive soft tissue infection involving fascia and subcutaneous tissue. A recent 8-year review quoted a 30-day mortality rate of 27% despite treatment.⁶ Abdominal wall NF usually occurs as a result of bacterial invasion following local trauma or as a postoperative wound complication,⁴ more commonly in the presence of underlying risk factors such as diabetes, liver cirrhosis, advanced age and states of immunosuppression.

Abdominal wall NF secondary to sigmoid diverticular perforation is a rare occurrence. A review of the English literature reveals reports of six cases.^{4,5,7-10} However, only two of these involve a colocutaneous fistula,^{4,5} as in our case.

While perforation of diverticulitis in the abdomen usually leads to localised pericolic abscess formation or generalised peritonitis, in the presence of a colocutaneous fistula, perforation into the abdominal wall tissue can lead to spreading soft tissue infection.¹¹ In the other cases without a fistula, NF is believed to have occurred secondary to peritonitis caused by perforated diverticulitis. Conn *et al* report a case of perforated sigmoid diverticulum and abdominal wall NF but this is in the context of a strangulated femoral hernia.¹² A few cases of perforated diverticulitis presenting as NF of the lower limbs have also been described, when the infection is thought to track deep to the inguinal ligament along the psoas muscle or through the femoral canal to enter the thigh, although other mechanisms of spread have been suggested as well.¹⁵

Early in the process, NF can be difficult to diagnose as clinical findings are non-specific. These include erythema, swelling and severe pain or tenderness. Skin changes such as crepitations and necrosis appear in later stages of the disease.¹⁴ In some of the six cases we reviewed, plain films demonstrating subcutaneous emphysema^{4,7} or computed tomography (CT) showing gas in the abdominal wall⁹ supported the diagnosis of NF. Nevertheless, NF spreads very rapidly and it is therefore important that once recognised,

radiological imaging (whether to confirm the diagnosis or to establish the underlying cause) does not delay debridement. In our case, the diagnosis was made on clinical grounds based on the skin changes and we proceeded to urgent surgery.

In the presence of abdominal wall NF, it is important that the possibility of an intra-abdominal source is borne in mind when planning surgery. Unless this is dealt with concomitantly, the soft tissue infection cannot be treated adequately. This is exemplified in the case described by Harrison, where an initial conservative excision of soft tissue necrosis failed to heal.⁸ The patient therefore returned to theatre for an exploratory laparotomy to deal with the diverticular perforation, which was initially not suspected. In most of the previously documented cases, the patients were reported to have gastrointestinal symptoms preceding (or associated with) the skin manifestations.^{4,5,7,8,10} Our patient had no signs or symptoms suggesting intra-abdominal pathology and was not known to have diverticular disease. On the other hand, she did not have any significant risk factors for the development of soft tissue infection and this prompted us to consider an underlying disease process.

When suspected, knowledge of the nature of such underlying pathology is certainly helpful in planning surgery. Lipsit and Lewicki performed a contrast study to confirm their suspicion of a perforated viscus causing subcutaneous emphysema⁷ while Mahler *et al* had the benefit of CT confirming a colocutaneous fistula prior to their patient developing NF.⁵ Kawamoto *et al* also described the use of CT to establish the source of NF preoperatively based on initial plain films showing dilated bowel loops suggesting obstruction.⁹ However, radiological imaging may not be appropriate in emergency scenarios. As we proceeded to immediate surgery without prior imaging to prevent impending clinical deterioration, a diagnostic laparoscopy was undertaken to guide our plan of action (ie simple debridement vs debridement and laparotomy) and this in fact established the cause of NF in our patient. This approach has not been used in previously reported cases.

Conclusions

To our knowledge, this is the third case of abdominal wall NF secondary to perforated diverticular disease with a colocutaneous fistula that has been reported in the English

literature. It is unusual for intra-abdominal sources to be responsible for abdominal wall NF but a high index of suspicion should be maintained to avoid missing underlying pathology, especially when there is no history of trauma or there are no convincing risk factors for spontaneous NF, as was the case in our patient.

Prompt recognition and aggressive debridement with antibiotic cover are key to the successful management of NF. As time is of the essence, if no preoperative imaging is available, laparoscopic visualisation may be helpful in establishing an intra-abdominal diagnosis at the time of surgery to help in the planning of subsequent management. In the series of cases we reviewed, this approach has not been described before.

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