

Strangulated hernias secondary to generalized peritonitis: an old lesson relearnt

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INTRODUCTION

Generalized peritonitis can lead to collections of pus in recesses of the peritoneal cavity. External hernias and their associated sac of peritoneum provide a further recess where pus may collect and produce inflammation within the hernial sac.

This may lead to a hernia that becomes painful, tender and irreducible. Such an association has long been recognized and a number of cases have been reported as early as 1891 (Cronin and Ellis, 1959). This association, however, is often not recognized preoperatively. The authors

present two recently encountered cases of strangulated external hernias, secondary to generalized peritonitis, in which the peritonitis was only diagnosed at laparotomy after initial exploration of the hernia.

DISCUSSION

Cronin and Ellis (1959) reported five cases and reviewed another 27 in the published literature of patients with generalized peritonitis and pus in a hernial sac, misdiagnosed as strangulated hernias. Perforated appendicitis accounted for 16 (50%) of the cases, with perforated peptic ulcer the cause of peritonitis in three cases.

CASE REPORT 1

A 78-year-old woman presented to the accident and emergency department having collapsed at home. She was fully conscious and had no neurological deficit. Abdominal examination revealed a soft upper abdomen, suprapubic tenderness on deep palpation and a 4x5 cm hard, tender, irreducible swelling at the left mid-inguinal point. Investigations revealed a raised white cell count of 19.5×10^9 /litre and a markedly elevated C-reactive protein of 437 mg/ml. A plain abdominal film (Figure 1) showed several gas-filled loops of small bowel in the lower abdomen and faecal loading of the colon. Suspecting a strangulated hernia, she was taken to theatre for exploration of the left inguinal mass. An irreducible left femoral hernia was found, the hernial sac containing necrotic omentum with pus draining from within the peritoneal cavity. A midline laparotomy was performed, finding generalized peritonitis secondary to perforated appendicitis with approximately 300 ml of pus in the pelvis. An appendicectomy and thorough peritoneal lavage was performed. The hernia was repaired through the abdominal incision and the groin wound closed. Initially she recovered well, but then developed a metabolic acidosis with no obvious cause, a repeat laparotomy proving negative. Unfortunately she had a large perioperative stroke and died a few days later.

Figure 1. Plain abdominal film showing several gas-filled loops of small bowel in the lower abdomen (case 1).



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CASE REPORT 2

An 83-year-old man attended accident and emergency complaining of a 6-hour history of lower abdominal pain associated with nausea but no vomiting. He had a long-standing left inguinal hernia, which was usually easily reducible. On examination he was in considerable pain, he was dehydrated, had a mild pyrexia (37.3°C) and was tachycardic (pulse rate 110/min). Abdominal examination revealed tenderness and guarding in the lower abdomen and a tense, tender, irreducible left-sided inguinal hernia. Bowel sounds were markedly reduced. Investigations showed a white cell count within the normal range (4.1×10^9 /litre). An erect chest X-ray showed a large gas shadow under the left hemidiaphragm with the appearance of dilated bowel, and the appearance of free gas beneath the right hemidiaphragm (Figure 2). An abdominal film showed dilated loops of small bowel (Figure 3). Suspecting a strangulated inguinal hernia, with possible perforation of the containing bowel, he was taken to theatre. Exploration of the left inguinal region revealed a large direct inguinal hernia containing viable sigmoid colon, with no evidence of perforation. The sac contained bile, which continued to drain from the peritoneal cavity. The hernia was repaired and a midline laparotomy performed confirming a large amount of bile in the peritoneal cavity with associated peritonitis. A 2 cm perforated ulcer was found in the first part of the duodenum, which was closed and patched with omentum. Postoperatively he made a good recovery, and was discharged home with *Helicobacter pylori* eradication therapy.

Difficulty in distinguishing between peritonitis and a strangulated hernia was partly thought to be caused by the presence of a painful, irreducible hernia which the patient seeks help for, and partly by the ease with which the pain and distension of a late peritonitis can be interpreted as obstruction (Cronin and Ellis, 1959). Cronin and Ellis (1959) concluded that factors favouring the development of this complication are a large amount of fluid in the peritoneal cavity and delay in the treatment of the peritonitis.

A further series of 13 patients presenting with strangulated hernias

Figure 2. Plain erect chest X-ray showing dilated small bowel under the left hemidiaphragm and free gas beneath the right hemidiaphragm (case 2).



Figure 3. Plain abdominal X-ray showing dilated loops of small bowel in the left upper quadrant of the abdomen (case 2).



associated with generalized peritonitis found that in nine of these patients the peritonitis was not recognized preoperatively (Ekwueme, 1973). Increased awareness of this association, and a conscious effort to exclude it, resulted in correct preoperative diagnosis in the other four patients.

In only one of these cases was the peritonitis secondary to appendicitis, reflecting the relatively lower incidence of appendicitis in Uganda where these cases occurred (Ekwueme, 1973). In two of the cases the peritonitis was secondary to acute pancreatitis, the first time an association between acute pancreatitis and strangulated hernia had been reported (Ekwueme, 1973). Four of the five patients with purulent peritonitis, in whom a separate incision had been made to explore the hernia, developed a wound infection postoperatively. Ekwueme (1973) suggests that a formal repair of the groin hernia through a separate incision is only justifiable where the associated peritonitis is not of the purulent type.

Chary (1977) reported delayed abscess formation in pre-existing inguinal hernias in three patients who were recovering from peritonitis. Each patient had a reducible inguinal hernia at initial laparotomy, which was not repaired, and the hernia subsequently became tender and irreducible postoperatively. At exploration, a closed hernial sac containing pus was found, with no communication with the peritoneal cavity. Occlusion of the neck of the sac by debris and fibrin appeared to be an important contributing factor in the development of the abscess (Chary, 1977). Chary (1977) concluded that the differential diagnosis between abscess formation in the hernial sac and strangulation is difficult, and the hernia should be explored in all such cases.

Peritonitis is a major complication in continuous ambulatory peritoneal dialysis (CAPD) patients. Additionally, the increased intra-abdominal pressure caused by CAPD may result in abdominal wall hernias

developing. Differentiating between peritonitis and strangulation of an abdominal hernia in a CAPD patient may be difficult, and a low threshold for surgical exploration of any tender swelling in these patients should be adopted (Engeset and Youngson, 1984). Inguinal abscess formation secondary to recurrent peritonitis has also been reported in a CAPD patient (Chang et al, 1999).

Strangulated hernia in association with generalized peritonitis has also been reported in two children who had primary bacterial peritonitis (Cameron, 1991).

CONCLUSION

A patient with generalized peritonitis may occasionally present with a painful, tender, irreducible hernia secondary to pus within the hernial sac. Focusing solely on the patient's hernia can lead the clinician to overlook the associated peritonitis. An increased awareness of this association, and a conscious effort to exclude it, can lead to correct diagnosis preoperatively.

A laparotomy is best performed to deal with the intraperitoneal lesion, and the associated groin hernia repaired by a preperitoneal approach through the laparotomy incision (Ekwueme, 1973). Abscess formation may occur postoperatively in a patient with a hernia recovering from peritonitis. Distinguishing an abscess from a strangulated hernia in such a patient can be difficult and such a hernia should always be explored. **HM**

- Cameron BH (1991) Two cases of primary bacterial peritonitis presenting with a tender hernia. *Aust NZ J Surg* **61**: 794–6
- Chang YP, Chen TW, Chen CP, Ng YY, Yang WC (1999) Inguinal abscess in a CAPD patient secondary to inguinal hernia and repeated episodes of peritonitis: a case report. *Perit Dial Int* **19**(2): 177–8
- Chary S (1977) Delayed abscess of hernial sac secondary to peritonitis. *J R Coll Surg Edinb* **22**(4): 282–4
- Cronin K, Ellis H (1959) Pus collections in hernial sacs. An unusual complication of general peritonitis. *Br J Surg* **46**: 364–7
- Ekwueme O (1973) Strangulated external hernia associated with generalised peritonitis. *Br J Surg* **60**: 929–33
- Engeset J, Youngson GG (1984) Ambulatory peritoneal dialysis and hernial complications. *Surg Clin North Am* **64**(2): 385–92