Unusual presentation of more common disease/injury

Gastric necrosis complicating acute gastric dilatation after Nissen fundoplication

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Summary

A woman in her 70s was referred for the management of complex intestinal fistulation, which had developed as a postoperative complication of laparoscopic Nissen fundoplication. Surgical treatment of a jejunocolic fistula and formation of a double-barrelled ileocolostomy was undertaken uneventfully. Routine postoperative nasogastric intubation was not undertaken. On the seventh postoperative day she had nausea and hiccupping. A chest x-ray demonstrated a massively dilated stomach which was decompressed with a nasogastric tube. The patient sustained a cardiac arrest and was resuscitated. A subsequent CT scan demonstrated acute gastric necrosis but the patient was judged too unwell for surgical intervention and died. Postmortem examination revealed emphysematous gastric necrosis. This case highlights the potentially fatal consequences of acute gastric dilatation following major abdominal surgery in patients who have previously had a Nissen fundoplication and are consequently unable to vomit.

BACKGROUND

Acute gastric dilation is a rare but well-recognised complication of major abdominal surgery, characterised by profuse vomiting. Nissen fundoplication procedures are undertaken to improve the competence of the lower oesophageal sphincter in patients with gastro-oesophageal reflux. In this case, acute gastric dilatation developed

following surgery for an intestinal fistula in a patient who had undergone previous Nissen fundoplication. The fundoplication prevented the postoperative vomiting usually associated with acute gastric dilatation, thus leading to fatal gastric necrosis as a result of the markedly increased intragastric pressure.

Figure 1 Chest radiograph demonstrating massively dilated stomach.

Figure 2 CT scan demonstrating the stomach decompressed with a nasogastric tube, oedema surrounding the left gastric vessels (arrow 1) and free air outwith the gastric wall (arrow 2).

Since Nissen fundoplication is a commonly undertaken surgical procedure, clinicians should be aware of the potential possibility of gastric necrosis complicating mechanical or functional intestinal obstruction in such cases.

CASE PRESENTATION

A woman in her 70s was referred for management of small bowel fistulation, which had developed as postoperative complication of a laparoscopic Nissen fundoplication, undertaken for the treatment of a large paraoesophageal hiatus hernia. During this operation, an unrecognised small bowel injury had occurred, necessitating return to theatre for a laparotomy for postoperative peritonitis. At surgery, a small bowel enterotomy was repaired and a proximal defunctioning stoma fashioned. However, this was in turn complicated by full-thickness abdominal dehiscence and fistulation of the small intestine proximal to the stoma, and the transverse colon into the open wound. After a 5-month period of inpatient treatment for abdominal sepsis and acute intestinal failure, and a further 4-month period of outpatient convalescence, the patient was readmitted for reconstructive surgery in order to restore intestinal continuity and reconstruct the abdominal wall defect.

Surgery was undertaken uneventfully. The jejunocolocutaneous fistula was resected and the distal small bowel and proximal transverse colon were exteriorised as a double-barrelled ileocolostomy. The abdominal wall was mobilised and repaired. Routine nasogastric intubation was not employed. The patient's initial postoperative recovery was apparently entirely uncomplicated, without evidence of fever, and with no haemodynamic or gastrointestinal disturbance. By the seventh postoperative day the patient was mobile, comfortable, eating and drinking satisfactorily, and her ileostomy was working normally.

The following day, however, the patient had nausea and hiccupping. There were no abdominal signs and she was haemodynamically stable but subsequently had a sudden onset of dyspnoea and central chest pain. A chest x-ray demonstrated a massively dilated stomach (figure 1). A diagnosis of acute gastric dilatation was made and a nasogastric tube was inserted to decompress the stomach. Intubation of the stomach resulted in drainage of several litres of heavily blood stained fluid. While the nasogastric tube was being inserted, the patient sustained an asystolic cardiac arrest, from which she was successfully resuscitated, albeit over a period of almost 10 min. Subsequent CT scan after transfer to intensive care demonstrated a

Figure 3 Micrograph of gastric wall demonstrating necrosis of the mucosa (A), represented by lack of staining within the nuclei due to cell death (arrow), congestion in the submucosa (B) and further areas of necrosis and haemorrhage within the gastric muscle coat (C).

thickened stomach, with gas present within the gastric wall, consistent with gastric necrosis (figure 2).

The patient remained haemodynamically unstable and became rapidly unresponsive to inotropic support. She was judged too unwell to survive surgical intervention and died the following day. Subsequent postmortem examination confirmed the cause of death as emphysematous gastric necrosis (figure 3).

INVESTIGATIONS

Chest radiograph demonstrated a massively dilated stomach (figure 1). A CT scan demonstrated the stomach decompressed with a nasogastric tube, oedema surrounding the left gastric vessels (figure 2, arrow 1) and free air out with the gastric wall (figure 2, arrow 2).

DISCUSSION

This case illustrates that a relatively common surgical procedure (Nissen fundoplication) can substantially increase the risk of death following acute gastric dilatation, which is itself an uncommon complication of abdominal surgery and critical illness.

Acute gastric dilatation is a potentially fatal condition if not diagnosed and treated promptly. Although acute gastric dilatation most commonly develops in the postoperative period, it may also develop as a result of anorexia nervosa, bulimia, trauma, electrolyte disturbances and diabetes mellitus.¹⁻⁴ The pathophysiology of acute gastric dilatation remains unclear but it has been suggested that reflex inhibition of gastric motor nerves with unopposed parasympathetic activity may lead to gastric dilatation, similar to that seen in colonic pseudo-obstruction. The majority of patients (90%) present with profuse vomiting⁵ followed by abdominal pain.⁴ Respiratory embarrassment

due to splinting of the diaphragm and aspiration of gastric contents may occur and early placement of a nasogastric tube is essential in order to prevent these complications. Delayed diagnosis and treatment have been associated with very high mortality.⁴

Nissen fundoplication was first described in 1956 as for the treatment of gastro-oesophageal reflux disease.⁶ Although there are a variety of variants, the procedure involves reconstruction of the lower oesophageal sphincter by compressing the cardia and distal oesophagus with a portion of the gastric fundus. The aim of the procedure is to ensure that when intragastric pressure rises, so does the pressure on the oesophagogastric junction, thus preventing or reducing the reflux of gastric contents into the oesophagus.⁷

The combination of Nissen fundoplication and postoperative acute gastric dilatation is thus potentially lethal, as not only does the Nissen fundoplication prevent the cardinal feature of acute gastric dilatation (vomiting) but the inability to vomit further increases the intragastric pressure. This results in a vicious cycle in which increased intragastric pressure increases the competence of the lower oesophageal sphincter, resulting in further increases in intragastric pressure because of the inability to vomit. Embarrassment of gastric venous return occurs once the pressure within the stomach exceeds that of gastric venous drainage (14 mm Hg),⁸ leading to venous congestion, reduced arterial flow, gastric ischaemia and, ultimately, full-thickness necrosis of the stomach, as occurred in the present case.

We have been able to identify a single case of gastric necrosis reported previously following mechanical small bowel obstruction in a patient who had undergone Nissen fundoplication 14 years earlier.⁹

The present case indicates that mechanical intestinal obstruction is not necessary for sufficient intragastric hypertension to cause gastric necrosis in patients who have had a previous Nissen fundoplication, and that the same complications can occur simply as a consequence of disordered postoperative intestinal motility.

Although the routine use of nasogastric tubes in patients undergoing abdominal surgery is not supported by the findings of a systematic review,¹⁰ postoperative nasogastric drainage or drainage gastrostomy should be considered in patients who have undergone previous Nissen fundoplication if gastric necrosis is to be prevented.

Learning points

- ▶ Acute gastric dilatation remains a preventable cause of postoperative mortality.
- ▶ The cardinal signs of acute gastric dilation may be absent in patients who have undergone previous Nissen fundoplication.
- ▶ Routine use of nasogastric tube or drainage gastrostomy should be considered in all patients with a previous Nissen fundoplication following major abdominal surgery.

Competing interests None.

Patient consent Not obtained.

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