Acute Gastric Dilation as a Rare Complication of Incisional Hernia Repair 30 Years after Open Anti-Reflux Surgery

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Abstract

Acute gastric dilation is a rare but potentially fatal complication of all forms of abdominal surgery, but more so following fundoplication type anti-reflux procedures. In extremis it can progress to gastric necrosis and perforation but can easily be remedied by early insertion of a nasogastric tube for gastric decompression. This case demonstrates the need to consider acute gastric dilation as a cause of abdominal pain in a patient with a history of anti-reflux surgery 30 years previously.

ABBREVIATIONS

OGD: Oesophagastroduodenoscopy; USS: Ultrasound Scan; CT: Computerised Tomography; ITU: Intensive Therapy Unit

CASE PRESENTATION

A 63 year old male presented with a two day history of central and epigastric abdominal pain associated with a lump over a midline laparotomy scar. He described tenderness with a burning sensation worse when laying flat and after eating. His abdomen was distended though he had been opening his bowels and passing wind normally and had not been vomiting. He had recently been investigated for assumed gastritis with a normal OGD and normal USS with no evidence of gallstones. His past surgical history was an open repair of a hiatus hernia and an open fundoplication 30 years previously. He was a non-smoker, drank 30 units per week and took omeprazole and co-codamol.

On examination he was reported to have a 7-8cm irreducible incisional hernia above the umbilicus which was tender to palpation. Bowel sounds were present and he was not pertionitic. The abdominal radiograph showed no dilation and the erect chest radiograph showed no free air. Blood tests including venous gas were all normal. The following day, at operating, a hernia sac containing only omentum was identified. There was no evidence of bowel in the hernia sac or any evidence of infarcted bowel. As the defect was small the hernia was reduced and the sac closed with 0 nylon interrupted sutures.

The following day the patient developed some minor abdominal pain over the operation site but was otherwise well and was kept in for observation.

In the early hours of the following morning the patient described feeling something pop in his abdomen. He was nauseated but did not vomit. When he was seen on the morning ward round he was distended and whilst having passed wind he had not yet opened his bowels. A CT scan of the patients abdomen was requested which revealed a residual defect in the abdominal wall, into which some omental fat had prolapsed. There was marked dilation of the stomach, which on the scan measured 16x24x18cm. The small bowel was dilated to the point of the hernia site, beyond which it returned to normal caliber. Several attempts were made to pass a nasogastric tube on the ward but were unsuccessful. The patient returned to theatre for a midline laparotomy where it was found the stomach was massively dilated, tense and severely congested. The small bowel was dilated to the point of the terminal ileum and the colon was distended to the sigmoid. There was a band of omentum stuck in the right iliac fossa bed but there were no obstructing mass lesions identified. The omentum was freed and due to a degree of pre-operative respiratory compromise and the concern of post-operative abdominal compartment syndromes the decision was taken to leave the abdominal cavity open with a negative pressure dressing. A nasogastric tube was placed and the patient transferred to ITU post operatively. Three days later the patient returned to theatre where early adhesions were divided and the abdomen irrigated with saline. There were multiple small bowel loops but no obstruction and no transition point. The large bowel was normal. The abdomen was closed with loop nylon and after a further 24 hours on ITU the patient returned to the ward where he made a steady recovery and was discharged.

DISCUSSION

Acute gastric dilation is a rare complication of abdominal
surgery and when progressed to the point of ischaemic perforation can have disastrous consequences. After review of the available literature no cases have been reported following open fundoplication, and only one recent case following benign disease (a nephrectomy) [1]. Since the introduction of proton pump inhibitors surgical management of gastroeosophageal reflux disease has reduced. Laparoscopic fundoplication procedures, now reserved for patients with medication resistant reflux, those with respiratory complications from reflux, or those with Barretts oesophagus are still performed. The consensus is that during open anti-reflux surgery accidental damage to the vagus nerve can lead to denervation of the stomach which in turn can lead to atony. This sequence of events can lead to acute gastric dilation, although this is more frequently in the shorter term post operatively rather than the longer term. In true acute gastric dilation the progression of symptoms is rapid and prompt treatment is necessary. Whilst the initial symptoms can be vague, patients are seen to vomit in greater than 90% of cases [2]. Progressive abdominal distension associated with discomfort are also common but can begin with varying degrees of intensity. Treatment in the acute situation consists of intubating the stomach with a nasogastric tube to deflate the upper gastrointestinal tract and remove the excess fluid. Surgical management is often reserved for unstable patients, or those with intercurrent bowel obstruction or small bowel ischaemia [3].

CONCLUSION

This case highlights the importance of prompt decompression of the stomach in patients who have had recent abdominal surgery with increasing pain, on a background of anti-reflux surgery. Although attempts were made by the ward nurses to intubate the stomach, had this been successful a laparotomy might have been avoided. The diagnosis of acute gastric dilation should be born in mind in patients following abdominal surgery and trauma particularly if they have had previous upper abdominal surgery.

REFERENCES