

Severe acute gastric dilatation causing respiratory failure

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ABSTRACT

Severe acute gastric dilatation occurring in the absence of bowel obstruction is uncommon. We report acute gastric dilatation developing postoperatively in a 79-year-old man, culminating in respiratory failure. On the third postoperative day following bilateral inguinal hernia repair, he developed abdominal distension with absent bowel sounds. Abdominal radiograph showed a grossly distended gastric shadow and small bowel dilatation. The patient's oxygen saturation then deteriorated suddenly and severely, necessitating intubation. He recovered well with conservative measures.

Keywords: acute gastric dilatation, postoperative complications, respiratory insufficiency

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INTRODUCTION

We report a case of severe postoperative gastric dilatation culminating into respiratory failure. The pathogenesis and risk factors of this condition are also discussed.

CASE REPORT

Our patient is a 79-year-old Indian man who presented to the Accident and Emergency Department with complaints of a painful groin swelling of a week's duration. His past medical history was hypertension with chronic renal impairment. Physical examination revealed that he was in congestive cardiac failure in addition to bilateral reducible inguinal hernias.

He was admitted to the medical unit for management of cardiac and renal failure. During the initial course of his stay, transthoracic echocardiography showed severely impaired left ventricular function with an ejection fraction of 20% and multiple segmental wall motion abnormalities, suggestive of diffuse coronary artery disease.

Renal ultrasonography showed bilateral chronic parenchymal disease.

He was treated with diuretics and was scheduled for elective bilateral inguinal hernia repair two weeks after his initial presentation. A bilateral inguinal hernia repair with mesh, haemorrhoidectomy and fistulectomy was performed under general anaesthesia with intermittent positive pressure ventilation and bilateral ilioinguinal nerve blocks. Operative findings were that of small bowel within the left hernia sac and omentum within the right hernia sac.

In the postoperative care unit, the patient became hypotensive necessitating insertion of a central venous catheter and low dose dopamine infusion to maintain a mean arterial pressure of at least 65 mmHg. This was weaned off rapidly by the first postoperative day in the high dependency unit. However, he experienced a further deterioration of renal function. There was no indication for initiation of haemodialysis at that time.

The patient was able to eat by the second postoperative day. However, on the morning of the third postoperative day, he developed abdominal distension with absent bowel sounds. Abdominal radiograph showed a grossly distended gastric shadow and small bowel dilatation (Fig. 1). He fasted and a nasogastric tube was inserted. Later that same evening, the patient's oxygen saturation deteriorated suddenly and severely. Air entry was reduced bilaterally but he was alert and responsive with stable haemodynamics. A chest radiograph showed massive gastric dilatation with diaphragmatic splinting. The tip of the nasogastric tube was noted to be in the oesophagus. Reinsertion of the nasogastric tube was attempted but failed.

Meanwhile, the patient suffered a further deterioration in oxygenation, necessitating endotracheal intubation. There were no clinical or radiological features suggesting pulmonary congestion or aspiration. Nasogastric tube insertion was successful after several attempts, and 1.8 litres

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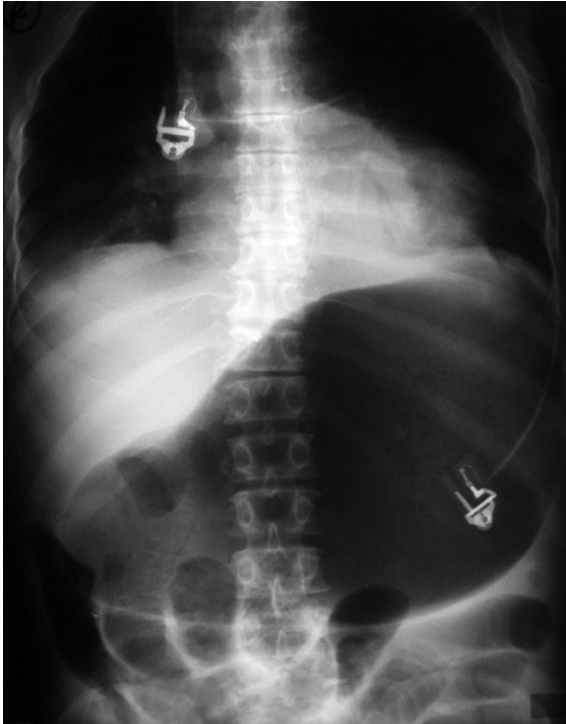


Fig. 1 Abdominal radiograph shows severe gastric dilatation.

of gastric fluid was drained. Initially, the patient required an FIO_2 (fraction of inspired oxygen) of 100% with a PEEP (positive end expiratory pressure) of 10 cm H_2O to maintain a $SpO_2 > 90$.

A repeat chest radiograph done the following day showed evidence of a right aspiration pneumonia for which intravenous piperacillin/tazobactam was started. He made remarkable progress after gastric decompression and was extubated four days later. A frusemide infusion was started to induce and maintain diuresis to which there was a good response. There was subsequent resolution of ileus.

DISCUSSION

Gastric dilatation is commonly encountered post-abdominal surgery secondary to postoperative ileus. This is commonly self-limiting and rarely leads to serious complications. Nonetheless, severe gastric dilatation can occur as a result of gastric outlet obstruction from conditions such as gastric volvulus, superior mesenteric syndrome and post fundoplication or post gastric bypass surgery^(1,2).

It is unusual, in several aspects, that our patient developed severe postoperative acute gastric dilatation. There was no clinically-apparent cause of gastric or bowel obstruction in our patient, and no evidence of incarceration or strangulation of hernia contents. There was also minimal handling of bowel during surgery. In addition, the temporal sequence of events made it unlikely that gastric insufflation

(during induction or extubation) contributed to gastric dilatation. There was also no pharmacological cause of gastrointestinal ileus.

Although nasogastric decompression was initiated quickly at the onset of symptoms, there was difficulty in proper positioning of the tube, and this allowed the progression of gastric dilatation. In view of pre-existing impaired cardiac function, this culminated in respiratory compromise and a need for ventilatory support. The deterioration in respiratory status was multifactorial with a major contribution from diaphragmatic splinting secondary to severe acute gastric dilatation. Lesser contributing factors included an aspiration pneumonia (likely contributed by the gastric dilatation) and cardiac failure.

We conducted a MEDLINE search from 1985 to 2004 using keywords of "acute", "gastric", "stomach" and "dilatation". In all, there were more than 20 reports of acute gastric dilatation occurring in the absence of gastric or bowel obstruction. We reviewed 14 reports available in English⁽³⁻¹⁷⁾. Several pertinent points can be derived from a review of these cases. Firstly, there is a bimodal distribution in age: one group consisted of individuals aged 20 years or below, while the other group were older patients aged above 50 years. Secondly, there was no gastrointestinal obstruction that precluded acute gastric dilatation. Thirdly, the majority of patients presented with typical features of upper gastrointestinal obstruction, such as upper abdominal distention, pain and vomiting. The onset of symptoms was usually rapid (in hours).

Hmouda et al⁽³⁾ reported a case where a ventilated patient presented with atypical symptoms of cardiorespiratory symptoms of hypotension, sinus bradycardia and complete atrioventricular block. All these signs resolved spontaneously with gastric decompression and relief of intra-abdominal pressure. In our patient, the degree of gastric distention was severe enough to compromise respiration, but there was no haemodynamic or circulatory insult. Lastly, most of the younger patients had pre-existing eating disorders, and an episode of food binging preceded the occurrence of acute gastric dilatation. Others had underlying muscular dystrophies or mental retardation from various causes⁽⁷⁻¹⁷⁾.

Reported precipitating events in older patients include surgery for benign gynaecological surgery, anorexia and *Staphylococcus aureus* bacteraemia. They also had comorbidities of diabetes mellitus or hypertension. The aetiology remains elusive. Several authors have postulated that visceral muscle involvement or neuromuscular incoordination predisposes to abnormal gastric emptying and progressive gastric dilatation⁽¹⁰⁾. In patients who

have underlying diabetes, autonomic neuropathy may lead to vagal dysfunction and gastroparesis^(6,18). This predisposes them to abnormal gastric emptying and distention. These patients may be asymptomatic when healthy⁽¹⁸⁾.

It is possible that our patient had autonomic neuropathy which manifested postoperatively, and led to acute and severe gastric dilatation. In addition, although clinically asymptomatic, the deterioration in renal function contributed to the development of an adynamic ileus. The problem is likely to recur during future episodes of anaesthesia. We would recommend performing rapid sequence induction, instead of bag and mask ventilation which would predispose to gastric insufflation and distention.

Early diagnosis and prompt nasogastric decompression appears to offer the best outcome for gastric dilatation. Delayed treatment can lead to gastric necrosis from excessive gastric wall distention, and subsequently to perforation and peritonitis. Clinicians should be alerted to the possibility of such a condition when susceptible patients groups present with acute upper abdominal pain, distension and gastric outlet obstruction.

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