Dobutamine stress echocardiography resulting in acute gastric dilatation and pneumoporta

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ABSTRACT

Acute gastric dilatation is a rare cause of gas within the hepatic portal vein, but one that is important to recognise as prompt decompression via a nasogastric tube is usually successful in resolving the situation. We report the rare case of a 68-year-old man with spontaneous acute gastric dilatation 50 minutes after a dobutamine stress echo that resulted in pneumoporta. The patient had a Nissen's fundoplication 18 months previously; patients with previous antireflux surgery or who have a degree of gastric outlet obstruction may be at increased risk of this unusual condition. Conservative management, with placement of a nasogastric tube was successful in resolving his symptoms.

KEYWORDS

Acute gastric distension – Portal vein gas – Nissen's fundoplication – Antireflux surgery – Gastric volvulus – Pneumoporta

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Pneumoporta secondary to acute gastric dilatation is a rare phenomenon, with two cases reported within the last 20 years.^{1,2} However, it is important to recognise, as prompt decompression via a nasogastric tube typically resolves the situation. We report the rare case of a 68-year-old man with spontaneous acute gastric dilatation 50 minutes after a dobutamine stress echo that resulted in pneumoporta.

Case presentation

A 68-year-old man presented with sudden onset of epigastric pain, nausea and distension. There were no obvious preceding factors, although he had undergone a dobutamine stress echocardiogram for investigation of atrial fibrillation and coronary artery disease 50 minutes earlier. The patient reported no other unusual activity, or abnormal diet prior to this event, and he had been swallowing normally. His past medical history included an open cholecystectomy and a laparoscopic Nissen's fundoplication for reflux and Barrett's oesophagus 18 months previously; since the operation, he had been otherwise well, with no history of gas bloating or aerophagia.

On examination, the patient was in discomfort, albeit with no pyrexia or cardiac compromise, and had a tense, distended abdomen, with tenderness and peritonism in the epigastric region. Urgent haematological investigation showed an elevated white cell count ($16.3 \times 10^9/L$; normal range $4.2\text{--}10.8 \times 10^9/L$) and increased levels of urea, at 11.3 mmol/L (normal range $2.5\text{--}6.6 \text{ mmol/L}$). His arterial blood gases indicated respiratory acidosis. He had a lactate level

of 1.1 mmol/L (normal range $0.4\text{--}1.4 \text{ mmol/L}$). An erect chest radiograph revealed an enlarged gastric bubble, and the passage of a nasogastric tube released a large amount of gas, with subsequent improvement in the patient's pain and distension. It was thought that the patient may have had an acute gastric outlet obstruction or a gastric volvulus, both of which improved with the passage of the gastric tube.

Urgent computed tomography confirmed gastric distension, despite the nasogastric tube and dilatation of the entire small bowel, with no obvious extrinsic compression or colonic pathology. Marked pneumatosis was seen within the stomach wall, with associated portal venous gas and air within the hepatic venules (Figures 1–3). Despite the radiological findings, the patient remained well in himself and was treated conservatively. He was kept nil-by-mouth, and given intravenous fluids and piperacillin/tazobactam. Gastroscopy performed 48 hours later showed mucosal inflammation throughout the stomach and duodenum, although there were no signs of ischaemia.

Following endoscopy, his diet was cautiously reintroduced and he remained asymptomatic, with his bowels opening normally. He was discharged 9 days after admission with complete resolution of his haematological disturbances, with a follow up gastroscopy to be performed at 4 weeks.

Discussion

This case describes two unusual acute surgical conditions; first, acute gastric distension; and, second, gas within the

Figures 1–3 Transverse, coronal and sagittal computed tomography images showing gastric distension with gas within the hepatic portal veins. Sagittal image shows air within the stomach wall.

portal vein. Acute gastric distension can occur after eating or enteral feeding, following abdominal surgery (classically following splenectomy) or following small bowel obstruction or ileus; rarely, it occurs secondary to peptic ulceration or air swallowing. It has been postulated that ‘gas-bloating’ (the inability to belch following fundoplication) may also

be implicated in the development of progressive acute gastric dilatation, which could be exacerbated by an intestinal obstruction affecting antegrade propulsion.^{3–5} It is thought that raised intraluminal pressure results in gastric pneumatosis, with resultant absorption of intraluminal air into the portal circulation.^{6,7}

Acute gastric dilatation following stress echocardiogram has not previously been reported, and the short time frame between the intervention and the onset of symptoms suggests a causal relationship. Minor non-cardiac side effects following dobutamine are self-limiting and reported in up to a quarter of patients. These include: nausea; vertigo; anxiety; flushing; urgency; and allergic reactions.⁸ Two scenarios can be entertained: first, the patient may have had increased air swallowing (from feelings of anxiety or nausea) during the test, leading to gas bloating and gastric distension; or, second, the dobutamine itself was directly or indirectly responsible for the pneumoporta. In other words, dilatation of the gastric arteries secondary to dobutamine (which predominately affects β receptors) may have resulted in increased gastric flow and engorgement of the gastric mucosa, which, coupled with the previous Nissen’s fundoplication, could have led to increased gas bloating. Alternatively, the patient may have had areas of asymptomatic microvascular disease and the increased β -sympathetic stimulation resulted in areas of localised gastric ischaemia.

Acute gastric dilatation presents with upper abdominal pain and concurrent distension,⁹ and may be accompanied by signs of cardiovascular compromise, metabolic acidosis or lactate acidosis on blood gas analysis. Plain films and cross-sectional radiological imaging can confirm the presence of gross gastric distension; the latter may show evidence of gas within the luminal wall, suggesting localised ischaemia. Immediate management should consist of prompt gastric decompression via the placement of a nasogastric tube, which also decreases the risk of aspiration. This may be difficult in the presence of gastric volvulus (Brochart’s triad, or the inability to pass a nasogastric tube, epigastric pain and intractable retching) and, as such, may require endoscopic or operative intervention.^{10,11} Conservative management for an acute gastric dilatation is advocated, with the use of gastroscopy to ensure that there is no gastric necrosis, which can also predispose to hepatic portal vein gas.¹² Repeat endoscopic assessment should be performed if there is mucosal inflammation to ensure that it has returned to normal.

Pneumoporta has traditionally been considered an ominous radiological sign and is typically associated with mesenteric infarction. As a result, it is associated with a mortality rate of up to 90%. Other causes include necrotising enterocolitis, pneumatosis intestinalis, perforated peptic ulcer, trauma, intestinal dysmotility, severe gastrointestinal inflammation and iatrogenic causes (ie operative intervention). Intestinal mucosal damage leads to air entering the venules that connect to the portal vein, with a resultant accumulation of gas within the portal vein and, ultimately, the liver.

Radiology provides the mainstay of diagnosis; plain abdominal films may reveal a branching radiolucency

extending up to 2 cm beneath the liver capsule.⁶ This classical finding is due to the centrifugal portal blood flow causing in gas in the peripherally of the liver. This is in contrast to pneumobillia, in which gas collects centrally as a result of centripetal movement. However, intrahepatic portal gas is difficult to detect on conventional radiological investigation, and thus computed tomography is advocated. This technique has a high specificity, can assess the extra-hepatic portal vein and splanchnic vasculature, and is able to detect the underlying pathology.¹³ Ultrasonography can also be used to detect bubbles within the portal vein.^{6,7,15}

Treatment of pneumoportia depends on the underlying causative mechanism, with operative intervention being advocated in unstable patients when the diagnosis is uncertain or there is a possibility of intestinal ischaemia.⁶ Stable patients with a more benign underlying mechanism can be managed expectantly.

Conclusions

This case is unusual, in that the patient presented with an acute gastric distension related to dobutamine stress echocardiography and previous antireflux surgery. This had a combined effect of localised gastric ischaemia and raised intra-luminal pressure, causing a very large amount of portal air. Conservative management was successful and the patient made a complete recovery.

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