Rare bowel emphysema with superior mesenteric artery syndrome after surgery

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ABSTRACT

Superior mesenteric artery syndrome, a rare cause of duodenal obstruction, occasionally requires surgery. Bowel emphysema might also require surgery and might be an ominous sign of a serious condition. We report the case of a 69-year-old Japanese man with left pneumothorax who was also diagnosed as having bowel emphysema and superior mesenteric artery syndrome simultaneously without serious infection after surgery for the pneumothorax. Following gastric decompression via a nasogastric tube, his general condition resolved quickly with no need for surgical intervention. Prompt and precise diagnosis by computed tomography and both adequate judgment and treatment can avoid surgery in such cases.

KEYWORDS

Bowel emphysema – Superior mesenteric artery syndrome – Computed tomography

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Background

Superior mesenteric artery syndrome, a rare cause of duodenal obstruction, occasionally requires surgery. Bowel emphysema might also require surgery and might be an ominous sign of a serious condition.

Case history

A 69-year-old Japanese man with a history of pulmonary emphysema, hypertension, hyperlipidaemia and left partial paralysis due to a cerebral infarction was admitted with the complaints of a 3-kg weight loss (body mass index 15 kg/m²), with a cough and chest pain persisting for one month. A physical examination revealed no abnormalities except for a non-respiratory sound in his left lung. Haematology laboratory results indicated slight anaemia and hypoalbuminaemia; his haemoglobin was 10.1 g/dl and his albumin was 3.0 g/dl. Chest x-ray and computed tomography (CT) confirmed large bullae in the right lung and pneumothorax of the left lung.

Video-assisted thoracic surgery revealed a hole producing massive air leakage at an apical pulmonary lesion. The fragile hole was sutured and then covered with a polyglycolic acid sheet and fibrin glue, but minor air leakage continued through the chest drainage tube. On postoperative day 2, CT was performed to investigate the cause of upper

abdominal distention and repeated vomiting upon the patient's attempt to consume a meal (Fig 1); it demonstrated extensive thoracic oesophageal, gastric and duodenal emphysema and obstruction of the third part of the duodenum. Careful evaluation of the CT images (Fig 2A,B) revealed the cause of the duodenal obstruction by the superior mesenteric artery (SMA), the so-called 'SMA syndrome' (SMAS). There were no signs of peritoneal irritation and the haematological findings revealed a slight inflammation; his white blood cell count was 9100 cells/ μ l and the C-reactive protein level was 1.0 mg/dl.

Gastrointestinal decompression by a nasogastric tube was successful for both the benign gastrointestinal emphysema and the SMAS. On postoperative day 5, CT revealed that the digestive tract emphysema had disappeared, without left lung air leakage. The patient's diet was carefully reintroduced without incident. His postoperative and post-discharge course was uneventful.

Discussion

Our patient's CT images revealed that the following two important events had occurred simultaneously. first, oesophageal, gastric, mediastinal and duodenal emphysema, with the emphysema stopping at the duodenum's third portion; and second, occlusion of the expanded duodenum by the SMA. St Peter *et al* demonstrated that in

Figure 1 Axial view of contrast-enhanced computed tomography through the patient's abdomen, which demonstrated that the third portion of the duodenum was occluded by the superior mesenteric artery and bowel emphysema.

emphysema of the bowel, the dissection of the gas from intraluminal compartments to the intramural compartments is due to increased intra-bowel pressure combined with a mucosal defect.¹ In our patient's case, we suspected that 'oesophagogastrroduodenal emphysema' occurred under the combination of (1) disruption of the mucosa leading to the air dissecting into the dietary tract

wall from the mediastinum emphysema due to the patient's pneumothorax surgery, (2) increased intra-bowel pressure caused by vomiting, and (3) obstruction of the third part of the duodenum. Oesophageal and mediastinal emphysema from surgery might be the trigger for gastroduodenal emphysema despite the postoperative suction of the thoracic drain using continuous negative pressure. SMAS itself might have been involved in the increased intra-bowel pressure, which is also one of the causes of gastroduodenal emphysema.

Generally, the gases of benign digestive tract emphysema are naturally absorbed; an exception is progressive disease due to a bacterial infection (eg, necrotising enterocolitis). Bowel emphysema caused by a bacterial infection was denied in our patient, because there were no signs of peritoneal irritation or abnormal data on blood collection. In cases of gastrointestinal decompression by a nasogastric tube, careful observation of the patient is necessary. We could not diagnose this patient as having SMAS before surgery, because he had no history of nausea or vomiting as a disorder of the passage of a meal. We could not identify his SMAS until we assessed his oesophagogastrroduodenal emphysema by CT.

Since Von Rokitsky first described SMAS in 1961, its true incidence has been widely debated.² Several reports suggested that SMAS is more frequent in individuals with a low body mass index and mesenteric fat loss,³ as in our patient; however, SMAS is not always associated with emaciation or thinness. SMAS can be divided into two types: the reversible type (transient) and the irreversible type (repeat or permanent) requiring surgery. The irreversible

Figure 2 Computed tomography coronal view. a) Mediastinal, oesophageal, gastric and duodenal emphysema (arrows). b) Stenosis at the third portion of the expanded duodenum by the superior mesenteric artery stenosis (arrow).

type has important anatomical problems caused by either (1) congenital or developmental factors, such as high insertion of the duodenum at the ligament of Treitz, a low origin of the SMA, a short ligament of Treitz, abnormal rotation of the intestines or compression of the duodenum's third part by peritoneal adhesion,² or (2) acquired factors (eg, adhesions due to surgery, duodenal stenosis due to inflammation such as pancreatitis and thickening of the root of the mesentery); displacement of the location of the Treitz ligament is also possible.

For reversible-type SMA (eg, a duodenal obstruction caused by gravity), interventions such as left lateral decubitus, knee-chest or prone position, or temporary gastric tube insertion, decrease the mesenteric pressure on the duodenum and alleviate the duodenal obstruction.⁴ Increasing mesenteric fat has generally been thought to prevent the compression of the duodenum by the SMA over the long term.⁵ In irreversible-type SMAS, the duodenal stenosis cannot be released at all or, if it is temporarily released, the symptoms recur. From the above considerations, SMAS should be first addressed with conservative therapy by decompressing the intestinal tract, improving nutrition (eg, by intravenous hyperalimentation) and

administering medication for intestinal peristalsis and appetite stimulation. If the patient's general condition worsens or does not improve after taking meals or conservative treatment, surgery such as Strong's procedure or duodenojejunal anastomosis should be considered. Recent reports described good results from laparoscopic duodenojejunal anastomosis.⁵ In any case, surgery should be performed only when it is clear that conservative treatment has failed.

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