

Case report

Gastric necrosis due to type II paraesophageal hernia in obese patient with gastric band: rescue sleeve gastrectomy

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Obesity is a risk factor for the development of paraesophageal hernia (PEH). The treatment of PEH depends on its subtype. Type I PEH is the displacement of the gastroesophageal junction into the thoracic cavity through the hiatus. In type II PEH, the gastroesophageal junction remains in its normal position but the gastric fundus migrates to the thoracic cavity. Type III PEH is a combination of types I and II. In type IV PEH, some abdominal organs, in addition to the stomach, exit through a hernia sac into the thoracic cavity. Some patients with type II PEH will be asymptomatic, and air bubbles in the thoracic cavity might be the only diagnostic finding in such cases. If gastric strangulation occurs, urgent surgical intervention is indicated. We report a case of gastric strangulation and ischemia associated with previous gastric banding in an obese patient with type II PEH.

Case report

The patient, a 36-year-old man, had attempted body building and weight gain with herbal drugs because he weighed 50 kg (body mass index [BMI] 15.9 kg/m²) 10 years earlier. Within a short period, his weight had increased to around 130 kg. Even after discontinuing the herbal drugs, his weight increased to 200 kg (BMI 63 kg/m²); however, it returned to 120 kg with diet and exercise. Subsequently, his weight had increased again, and a laparoscopic adjustable gastric band was applied 18 months earlier when he weighed 178 kg (BMI 56 kg/m²). No postoperative complications developed. The patient then started exercise maneuvers that caused an increase in intra-abdominal pressure.

The patient had presented to another hospital 4 days before the present index admission with dyspnea, abdominal pain, and distension. A diagnosis was not made after physical examination and laboratory studies. Treatment with analgesics and some antifatulent medications were used, but his symptoms became more severe, leading to hematemesis. Thereafter, the patient was transferred to our hospital.

On examination, the patient appeared acutely ill with general weakness and dyspnea. His blood pressure was 120/70 mm Hg, pulse 136 beats/min, temperature 37°C, respiratory rate 24 breaths/min. Respiratory sounds could

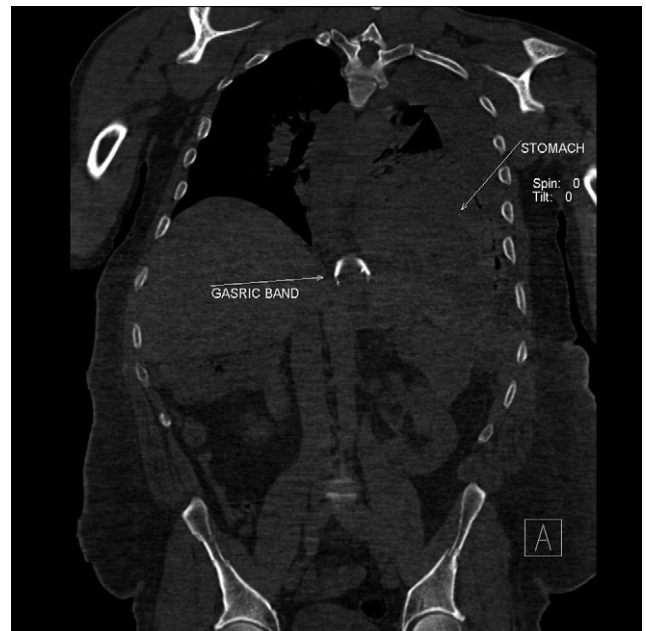


Fig. 1. Computed tomography scan showing gastric band and gastric herniation.

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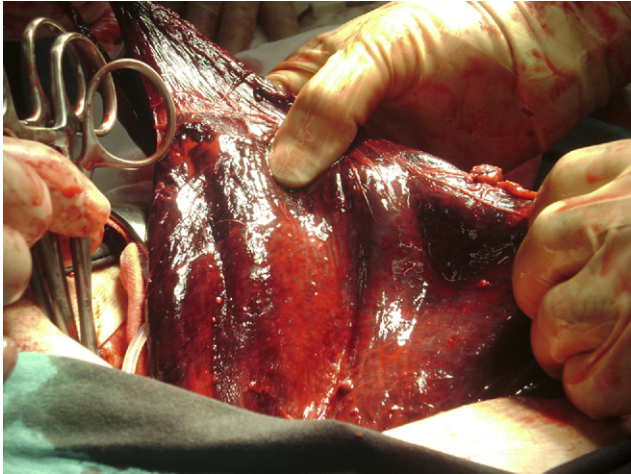


Fig. 2. Gastric necrosis within greater curvature.

not be heard on auscultation of the left hemithorax. No abnormal physical findings suggestive of abdominal disease were present, although melena and hematemesis were detected. His laboratory findings were normal, except for an elevated white blood cell count of $15,800 \text{ cells/mm}^3$. His BMI was 31.9 kg/m^2 .

Thoracic and abdominal computed tomography studies revealed a collapsed left lower lobe and herniation of the stomach into the left hemithorax. The mediastinum was shifted to the right, and a massive pleural effusion in the left hemithorax was detected. Air bubbles in the gastric wall corresponding to necrotic changes were clearly seen on the computed tomography scan. The gastric band was also visible on the cardia at its expected position (Figs. 1 and 2).

An emergent laparotomy was performed, with the diagnosis of gastric necrosis and type II PEH. Laparotomy was achieved using a midline incision. At exploration, the gastric band was found in its expected place, and the stomach had visibly herniated into the left thoracic cavity through the esophageal hiatus (type II strangulated PEH), consistent with the computed tomography findings. Sero-hemorrhagic fluid was observed in the left hemithorax. After hiatal dilation, the stomach was retracted into the abdominal cavity. The greater curvature of the stomach was necrotic. Intra-gastric hemorrhage was detected in the nasogastric tube. The gastric band was removed, and the necrotic areas in the greater curvature were resected vertically by stapler, completing a sleeve gastrectomy. The diaphragm was repaired by primary closure, and a thoracic drainage tube was inserted into the left hemithorax. The patient required 4 U of red blood cells. The patient was discharged uneventfully on the seventh postoperative day.

Discussion

The frequency of gastric band placement has been increasing. It is preferred by some bariatric surgeons, because

it is less invasive and has a lower risk of complications. However, gastric banding still has risks of complications that can be fatal if not treated. The most frequent complications are esophagitis, band migration, pouch and esophageal dilation, gastric perforation, necrosis, and volvulus [1–3].

Gastric necrosis is a complication related to gastric banding and resulting from ischemia of the stomach proximal to the band. Volvulus and necrosis related to gastric banding has been previously reported [4–6]. Diaphragmatic rupture, which can occur during the placement of the band, is 1 of the causative factors for the development of hiatal hernia [7].

We believe that the main cause for PEH developing in the present patient was related to the exercise maneuvers that increased the intra-abdominal pressure. A collective effect of an elevated pouch pressure and intra-abdominal pressure during exercise might have caused the PEH.

Laparoscopic sleeve gastrectomy is an acceptable procedure after removal of the band for inadequate weight loss or band complications [8]. In our patient, we performed a stapled sleeve gastrectomy as resection of the necrosed parts of the stomach, because the lesser curvature appeared to be viable.

Disclosures

The authors have no commercial associations that might be a conflict of interest in relation to this article.

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