Hernia
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CASE REPORT

Strangulated perforated hiatus hernia due to pyloric stenosis

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Abstract The first patient with strangulated hiatus hernia due to pyloric stenosis is reported. A 70-year-old male patient presented as an emergency with severe left-sided chest pain, tachycardia, tachypnea, dysphagia, and nausea but no vomiting. The diagnosis of strangulated hiatus hernia due to pyloric stenosis was suspected, because a CT scan done 24 h following a barium meal showed much retained barium in both infra- and supradiaphragmatic parts of the stomach. Recognition of this condition is important since absence of pneumoperitoneum should not delay the diagnosis. In this complication, the perforation is likely to be at the hiatus, not the fundus as occurs in other causes of strangulation. A gastric drainage procedure should be an essential part of treatment.

Keywords Hiatus hernia · Strangulation · Pyloric stenosis · Rolling hiatus hernia

Introduction

Unlike sliding hiatus herniae, the less common rolling (para-esophageal) variety is prone to strangulation and perforation. In spite of this, most are asymptomatic, and only a few case reports of strangulation have been documented [1, 2]. In some patients, strangulation occurs spontaneously, but more recently it has been seen following cardiac surgery, gastric banding, and laparoscopic Nissen fundoplication [3–5]. Gastric volvulus has also been documented, but we could find no report of strangulation and perforation caused by pyloric stenosis [6]. This report describes such a patient presentation and discusses its special features.

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Case report

A 70-year-old male presented as emergency with a 4-day history of severe retrosternal and left chest discomfort and worsening pain. He had been unable to swallow anything for the previous 12 h but had intermittent dysphagia for 4 weeks and was unable to swallow solids for 2 weeks. Although nauseated, he could not vomit. Twenty-four hours before admission, a barium meal requested by his general practitioner showed gastric distension with barium retention in both the abdominal and thoracic stomach although some contrast had gone through to the colon and rectum (Fig. 1a, b). His symptoms worsened markedly over the following 12 h, resulting in hospital admission. Prior to this, he was in very good health but had occasionally used antacids for epigastric discomfort over a 12-year period. He had no history of major trauma, hospitalization, or surgery.

He was an anxious, ill-looking, mildly dehydrated, elderly male with a pulse of 102/min, respiratory rate 28/min, and temperature 36.8°C. Abdominal examination revealed tenderness confined to the epigastrium; in the chest, there were decreased breath sounds at the left base. CT scan, performed 24 h after the barium meal, showed retained barium in the distal esophagus, and intrathoracic and infradiaphragmatic stomach, while some contrast had gone through to the small and large intestines (Fig. 2a, b). A diagnosis of pyloric stenosis with a rolling hiatus hernia was made. Emergency surgery through a left thoracoabdominal

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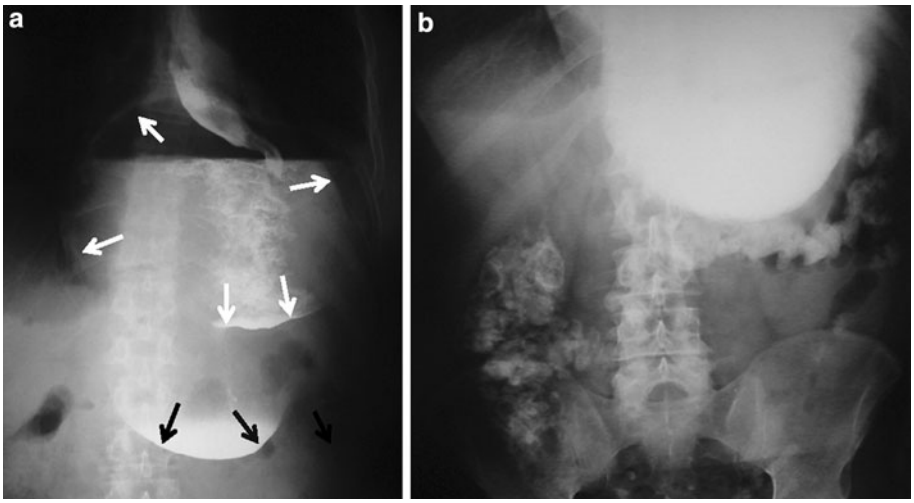

Fig. 1 Marked gastric distension in the chest (*white arrows*) and abdomen (*black arrows*) (**a**). Much barium is retained in the stomach after some has gone through to the colon and rectum (**b**)

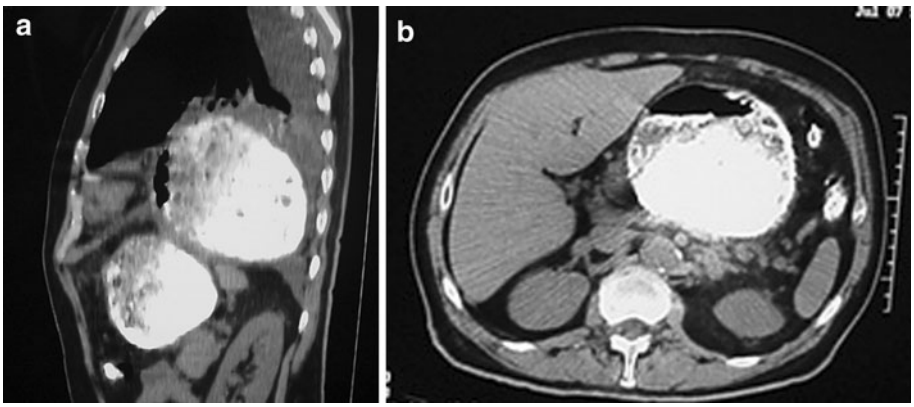

Fig. 2 CT showing gastric distension both above and below the diaphragm with little barium passed into the intestines after 24 h (**a**, **b**)

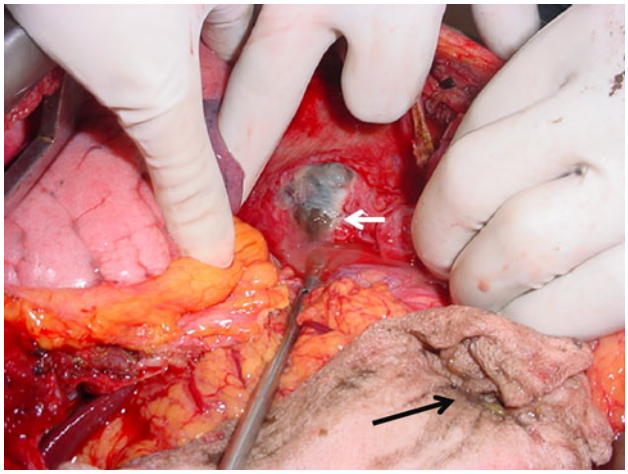

Fig. 3 A 3 cm gangrenous, perforated area at the level of the hiatus (*white arrow*). Note the leaked gastric contents on the swab (*black arrow*)

incision revealed distended but viable stomach both above and below the diaphragm. At the level of the hiatus, a gangrenous ring was noted with a 3 cm perforation antero-laterally with spillage of gastric contents (Fig. 3). The perforation was repaired and the ischemic ring inverted. The pylorus was scarred and fibrotic, likely due to his 12-year history of ulcer symptoms, but no mass was present. A Heineke-Mikulicz pyloroplasty was done through the scarred pylorus. The stomach was easily reduced into the abdominal cavity, the diaphragm repaired, and the hiatus tightened with interrupted, nonabsorbable sutures. Because of gangrenous stomach and spillage of gastric contents, mesh repair at this site was not considered a good option. He recovered uneventfully, was able to eat after 72 h, and go home on the 7th post-operative day.

Discussion

Although we could find no previous case report on gastric strangulation and perforation due to pyloric stenosis in a hiatus hernia, a report by Axon et al. [7] suggests that, in their first patient, gastric outlet obstruction might have been the etiologic factor rather than the previous intrathoracic Nissen's fundoplication as they reported. In most patients of perforation in strangulated hiatus hernia, the rupture occurs in the fundus or body of the stomach [1, 8, 9]. In our patient (and Axon's first patient) the perforation occurred at

a ring of ischemic stomach strangulated at the hiatus. Perforation at this site with subdiaphragmatic leakage is unlikely to have free intraperitoneal air since the gastric air bubble preceding perforation in pyloric stenosis would have been in the chest. Neither Axon et al.'s nor our patient had free intraperitoneal air, and pneumoperitoneum need not be a feature of this type of perforation.

When pyloric stenosis is the etiology, the stomach is distended both above and below the diaphragm, but the limitation of the hiatal opening serves as a strangulating force at this specific site. As in our patient, the most severe ischemia and perforation are likely to be at this site with viable stomach proximally and distally in the early stages. Later on, gangrene may progress to involve other parts of the stomach as in the case of Axon et al. If diagnosed and explored early, it might be possible to simply repair the perforation and ischemic area rather than having to do extensive gastric resection.

In addition, we feel that it is important to identify pyloric stenosis as the etiologic factor if it is present. Failure to do a pyloroplasty or drainage procedure in such cases could theoretically result in failure of both the gastric repair and the hiatal reconstruction. Thus, recognition of pyloric stenosis in these patients could be a guide to both the likely site of perforation as well as the need for a gastric drainage procedure. Retained barium after 24 h with gastric distension both in the abdominal and thoracic portions of the stomach should alert one to the diagnosis of strangulated hiatus hernia, especially in a very ill patient with chest pain, dysphagia, and nausea. Vomiting is not a feature since strangulation at the hiatus does not allow propulsion of gastric contents up the esophagus. Moreover, the absence of pneumoperitoneum should not delay the diagnosis when patients with a hiatus hernia and pyloric stenosis present with severe abdominal or retrosternal pain.

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