Gastric volvulus with diaphragmatic hernia presenting with unexplained weight loss: a delayed diagnosis

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ABSTRACT
Gastric volvulus (GV) is a rare condition that presents with epigastric pain, retching and at times, vomiting. There are two types of GV: organoaxial and mesenteroaxial. We report the case of a 49-year-old woman with chronic mesenteroaxial volvulus with left-sided diaphragmatic defect. She presented with significant weight loss over a period of two years, with nonspecific symptoms of heartburn, occasional mild epigastric pain and anorexia. The patient was diagnosed with barium meal and contrast-enhanced computed tomography imaging. She was treated with open repair of diaphragmatic defect and anterior gastropexy. She was asymptomatic and had gained weight at the six-month follow-up.

Keywords: anterior gastropexy, diaphragmatic hernia, eventration, gastric volvulus

INTRODUCTION
Gastric volvulus (GV) is an uncommon condition. While chronic GV presents with nonspecific upper abdominal or chest pain, acute GV presents with epigastric pain, retching and occasionally, vomiting. Torsion occurs along the stomach’s longitudinal axis (organoaxial GV) in about two-thirds of GV cases and along the vertical axis (mesenteroaxial GV) in one-third of the cases. We present a patient with chronic mesenteroaxial GV who presented with significant weight loss and a long history of nonspecific symptoms.

CASE REPORT
A 49-year-old Bengali woman presented to us with a weight loss of 10 kg and anorexia for the past two years. She also had symptoms of heartburn and occasional mild epigastric pain for the past five years. The patient has been hypertensive for the last five years, and her condition has been controlled with enalapril and hydrochlorothiazide. Faecal occult blood test, gastroduodenoscopy and physical examination were normal except for malnourishment and a thin built (weight 36 kg). Barium meal radiography revealed a mesenteroaxial GV with a left-sided diaphragmatic hernia (Fig. 1), which was confirmed on contrast-enhanced computed tomography (CT) imaging (Figs. 2 & 3).

The patient was explored through a left subcostal incision, which showed that the left dome of her diaphragm was thinned out (Fig. 4) and had herniated into the left thorax. Laxity of the gastric attachments was also observed. The left dome of the diaphragm was plicated with Prolene stitches, and anterior gastropexy was performed (Fig. 5). The fundus and antral region of the patient’s stomach were stitched to the anterior abdominal wall with a few interrupted, non-absorbable stitches. The nasogastric tube was removed on the third day, and the patient was put on an oral diet. She was discharged on Day 6. At the six-month follow-up, the patient was asymptomatic and had gained 2 kg. A postoperative barium meal showed the stomach in a normal position (Fig. 6).
DISCUSSION

GV was first described by Berti in 1866.\(^3\) In 1904, Borchardt described the classic triad of GV, which is severe epigastric pain, retching with vomiting and the inability to pass a nasogastric tube.\(^2\) There are two types of GV: organoaxial GV, where torsion occurs along the stomach's longitudinal axis; and mesenteroaxial GV, where torsion occurs along the vertical axis. Organoaxial GV usually occurs acutely and is associated with a diaphragmatic defect, whereas mesenteroaxial GV is partial (torsion < 180°), recurrent and not associated with a diaphragmatic defect.\(^9\) The associated diaphragmatic hernia in organoaxial GV can be due to the failure of muscularisation (congenital eversion of the diaphragm), traumatic disruption of the diaphragm (traumatic hernias), failure of fusion of embryonic components (various other congenital hernias), or phrenic nerve dysfunction (acquired eversion of the diaphragm).

In congenital evagination, the dome of a hemidiaphragm fails to muscularise, resulting in a membrane-like thinning of the diaphragm. These patients usually present early in the paediatric age group with respiratory symptoms or features of acute GV, both of which require urgent operative intervention. In contrast, congenital evagination rarely presents in adulthood and is mostly clinically silent, thus not requiring any surgical intervention. Symptomatic patients usually present with respiratory insufficiency or symptoms related to displaced abdominal organs. Acquired evagination occurs due to trauma to the phrenic nerve resulting from birth injury, external trauma, neoplastic or inflammatory processes and operative injury. In contrast, diaphragmatic defects due to other congenital or traumatic hernias are complete, and the herniated abdominal contents are either in direct communication with the chest cavity or covered with a peritoneopleural sac.

Our patient did not provide any history of birth trauma and trauma to the abdomen or chest. Intraoperatively, we found that the left dome of the diaphragm was thinned out and elevated, with normal peripheral muscular attachments resembling evagination. This is in contrast to other types of diaphragmatic hernias like Bochdalek hernia or Morgagni hernia, where localised defects are observed in the diaphragm as a result of failed fusion of embryonic components. A traumatic hernia usually has a defect in the diaphragm extending from the oesophageal hiatus with no true hernial sac. There were no clinical symptoms or CT findings suggestive of any neoplastic, infectious or degenerative diseases of the mediastinum or neck giving rise to phrenic nerve palsy in our patient. Our case is more likely to be congenital evagination of the diaphragm, a type of diaphragmatic hernia.\(^1\)

The symptoms of acute GV are a sudden onset of severe epigastric pain, retching and occasionally, vomiting, whereas chronic GV patients present with nonspecific upper abdominal discomfort, chest pain, gastric bleeding and at times, postprandial abdominal pain. Strangulation obstruction from acute GV leads to gastric necrosis and sepsis, and if not immediately recognised and treated, can be fatal. Chronic GV, when suspected based on a patient's history, is usually diagnosed with a barium meal. CT imaging is also diagnostic and aids in diagnosing the presence of herniation of other organs into the chest. Chronic GV is usually treated by repair of the diaphragmatic hernia and fixation of the stomach below the diaphragm. However, in cases of severe gastric torsion and herniation into the chest, gastropexy of the antrum to the anterior abdominal...
Fig. 4 Operative photograph shows the diaphragmatic defect (arrows).

Fig. 5 Operative photograph shows the diaphragmatic repair (black arrow) and anterior gastropexy in the process of fixing the fundus to the diaphragm (white arrow).

Fig. 6 Postoperative barium meal radiograph shows the surgical correction after one month.

Various surgical procedures, such as the plication of the diaphragm either by thoracotomy, laparotomy or minimal access and the reinforcement of suture repair with mesh, have been described in the eventration of the diaphragm. The hernial contents were easily reduced in our patient, and the thinned out left diaphragm could be easily plicated without tension with Prolene sutures approximating the healthy edges. We did not excise any part of the thinned-out diaphragm.

The diagnosis in our case was delayed due to the patient’s chronic atypical symptoms. However, adequate repair of the diaphragmatic hernia and gastropexy at the fundus and antrum were performed, which ameliorated her symptoms. In addition, a postoperative barium meal confirmed repair of the patient’s stomach. Chronic gastric volvulus can present with atypical nonspecific symptoms, thereby delaying diagnosis and management. Repair of the associated diaphragmatic defect and anterior gastropexy provide a simple and durable solution to this problem.

ACKNOWLEDGEMENT
Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

REFERENCES