Gastric outlet obstruction with an elevated serum pancreatic lipase secondary to an infraumbilical hernia

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ABSTRACT
We report the case of a 68-year-old woman who presented with symptoms and signs of gastric outlet obstruction with a history of a ventral hernia. Clinical examination revealed a large ventral hernia with visible peristalsis of the herniated viscera. Initial serum biochemistry revealed a markedly elevated lipase level and deranged renal function. Computed tomography demonstrated an infraumbilical hernia with herniation of the stomach through the ventral defect and distortion of the pancreatic anatomy. The hernia was reduced operatively and repaired, leading to an uneventful recovery.

KEYWORDS
Gastric outlet obstruction – Hernia – Ventral – Umbilical

Accepted 6 February 2013; published online XXX

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Case history
A 68-year-old woman presented to our emergency department with a 7-day history of worsening upper abdominal pain of acute onset associated with profuse vomiting and an abdominal wall swelling. On questioning, it was discovered she had been unable to tolerate any oral intake for four days and had been passing only minimal flatus for the last two prior to presentation.

Our patient had a ventral hernia, which had been present for over 40 years. On two previous occasions, she had attended emergency departments with similar presentations that had resolved spontaneously. Investigations on these occasions were limited to blood testing, on which elevated amylase and lipase levels were noted, leading to a diagnosis of chronic pancreatitis. Her hernia was never repaired owing to her travelling schedule.

On abdominal examination, a minimally tender and irreducible infraumbilical ventral hernia with visible peristalsis (Figs 1 and 2) was evident. The patient was also dehydrated although not hypotensive. Biochemical screening revealed a serum lipase level of 2,103 mmol/l as well as hypokalaemia (3.3 mmol/l) and impaired renal function (estimated glomerular filtration rate 37 ml/min).

Computed tomography (CT) with oral contrast was performed to delineate the anatomy. The axial images identify a ventral defect of 8 cm diameter, giving rise to an 18 cm diameter hernia sac located in the infraumbilical abdominal wall. In the hernia sac was distal stomach (distended and containing contrast) along with omentum and transverse colon. Contrast had entered the distended herniated stomach but was not moving beyond this point. The pancreatic...
anatomy was distorted owing to the traction but no inflammatory changes were evident (Figs 3 and 4).

Following resuscitation, our patient underwent a laparotomy. At operation, a ventral defect caudal to and separate from the umbilicus was noted. On opening the hernia sac, there was no evidence of gastric or colonic ischaemia, or perforation. There was also no evidence of acute or chronic pancreatitis. Reduction of the viscera and a simple hernia repair were performed.

Discussion

Gastric outlet obstruction secondary to gastric herniation is a rare event, with pyloric strictureting being far more common. Reported sites of gastric herniation include diaphragmatic, incisional, epigastric, umbilical and inguinal defects.1,2 Among these, the midline epigastric and umbilical hernias are the rarest with fewer than ten cases reported in the literature; these defects also seem to present almost exclusively with gastric outlet obstruction.1,3 Our case is the first reported of an infraumbilical ventral hernia containing stomach and causing gastric outlet obstruction.

In our case, the diagnosis of an obstructed hernia was evident clinically although the nature of the herniated viscera was not. CT with oral contrast confirmed our clinical findings and delineated the anatomy (Figs 3 and 4). Contrast-enhanced CT is known to be useful in diagnosis and management of intestinal obstruction including that of the gastric outlet.1,4 Our patient was also noted to have an elevated serum pancreatic lipase level. The specificity of the assay for pancreatic lipase excludes an elevated gastric lipase due to trauma to the stomach. We suspect that our patient had this derangement as a result of traction on the pancreas itself without overt clinical or radiological pancreatitis. Similar events have been described previously but have involved clear clinical pancreatitis.1,3
Like the majority of cases described in the literature, our patient underwent a surgical reduction and repair of the hernia, and remains well in follow-up. While this seems to be the treatment of choice, at least one patient has been managed conservatively for the obstruction when the perioperative mortality risk was deemed to be too high.\(^2\)

**Conclusions**

Our case report highlights that any part of the alimentary canal may be obstructed in any hernia regardless of the location. Surgical repair is the usual treatment but contrast CT may well prove useful in planning this. We are also reminded that high lipase levels do not necessarily equate to clinical pancreatitis.

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