An unusual diaphragmatic hernia with gastric perforation and sub-acute presentation

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ABSTRACT

Gastric perforation into the thoracic cavity through a diaphragmatic hernia is rare but, when it occurs, patients present in severe distress, with mortality approaching 50%. We present a unique case in which a fibrotic reaction between the stomach and the parietal pleura led to a subacute presentation upon perforation. The extra time that this afforded led to more effective multidisciplinary team management and ultimately an excellent outcome for the patient.

KEYWORDS

Laparoscopic surgery – Diaphragmatic hernia – Stomach disease

Accepted 21 May 2016

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Introduction

Large, spontaneous diaphragmatic hernias are extremely rare in adults. Subsequent incarceration and perforation is even rarer, but can be life-threatening.¹ The diagnosis is often difficult and a delay in diagnosis frequently results in increased morbidity and mortality.² We report such a case, made unique by the fact that the patient presented sub-acutely despite perforation of his herniated stomach.

Case report

A 70-year-old gentleman was admitted to a district hospital with left-sided, pleuritic chest pain, increasing breathlessness and mild epigastric discomfort. His past medical history was unremarkable and he denied any recent trauma.

On examination, he had epigastric and left chest-wall tenderness; he had a heart rate of 105 beats/minute and a respiratory rate of 22 breaths/minute, but otherwise his observations were stable and he looked well. Chest x-ray showed left-sided opacification consistent with consolidation.

Laboratory testing revealed a C-reactive protein (CRP) of 407 mg/L (normal range: <10mg/L) and a white cell count (WCC) of $17.7 \times 10^9/L$ (normal range: $4\text{--}11 \times 10^9/L$). An arterial blood gas showed PO₂ of 8.59 kPa, PCO₂ of 4.43 kPa, pH of 7.44 and lactate of 1.5mmol/L. The CRP and WCC remained elevated after 3 days of intravenous antibiotic treatment for a presumed empyema secondary to pneumonia. For this reason, computed tomography (CT) was ordered, which showed what was thought to be a large hiatus hernia. An oesophago-gastro-duodenoscopy was performed at this stage. This showed no evidence of perforation

or necrosis and the scope was passed into the duodenum without difficulty. A nasogastric tube was also placed to decompress the stomach.

The patient was transferred to a tertiary centre as it was felt that there was a high risk of strangulation and perforation. After transfer, the patient remained clinically stable. However, he developed cyclical pyrexia and his inflammatory markers remained elevated, despite ongoing intravenous antibiotic treatment, and the haemoglobin dropped to 81g/L (normal range: 130–180g/L). For this reason, a repeat CT scan was performed. What was initially thought to be a hiatus hernia was in fact a diaphragmatic hernia. The fundus of the stomach had herniated through the left hemidiaphragm, occupying most of the left hemithorax and collapsing nearly the entire left lung (Fig 1). There was also evidence of perforation of the stomach fundus and communication with the extra-pleural space. However, the patient's clinical picture remained stable throughout. Multidisciplinary team discussion concluded that urgent reduction and repair, after nutritional optimisation, was required.

During laparoscopy, the team found the stomach fundus herniating up through the centre of the left hemidiaphragm. The stomach had become incarcerated at the level of the diaphragm and had perforated, contaminating the cavity with necrotic debris and gastric contents. Cardiothoracic input was sought at this stage and it was considered that a solely transabdominal approach was appropriate.

It would appear that the inflammatory reaction caused by the stomach over a period of time had thickened the parietal pleura to such an extent that, when perforation had occurred, the lung parenchyma was protected by a thick layer of fibrotic tissue. The stomach fundus was reduced

back to the abdominal cavity with difficulty and the necrotic edges excised from the residual fundus to allow primary closure (Fig 2). The fibrotic cavity was washed out and a drain placed transabdominally into the thoracic cavity. The diaphragmatic defect measured 3cm × 3cm and was too rigid for primary closure, so was patched with a biological mesh and secured with sutures (Figs 3 and 4). Postoperatively, the patient was managed in the intensive care unit and commenced on liquid enteral nutrition, supplemented with parenteral nutrition. A follow-up CT scan at 12 months

Figure 1 Coronal computed tomography image showing defect in centre of the left hemidiaphragm and collapse of the left lung

Figure 2 Primary closure of the stomach after removal of the necrotic fundus

Figure 3 Biological mesh patching the diaphragmatic defect

Figure 4 Intraoperative video showing removal of gastric contents from thoracic cavity, visualisation of diaphragmatic defect and patching with biological mesh

showed close to complete reinflation of the lung and no reherniation. The patient has not suffered any on-going sequelae of the perforation or surgical repair.

Discussion

Diaphragmatic hernias are typically divided into congenital and acquired types and diagnosis is often established using a combination of plain film radiograph, contrast swallow and CT scans. Congenital diaphragmatic hernias are due to a defect in diaphragmatic development between the fourth and twelfth week of gestation. These defects are most often diagnosed during the neonatal or perinatal period, although some are diagnosed incidentally in adulthood.⁵ The two most common congenital diaphragmatic hernias are defects in the posterolateral diaphragm (Bochdalek) and anterior diaphragm (Morgagni).⁴ In the present case, it was felt that the location of the defect, in the centre of the left hemidiaphragm, was not consistent with a late-presenting congenital hernia.

Excluding hiatus hernia, by far the most common cause of adult-onset diaphragmatic hernia is trauma, the usual

mechanisms being motor accidents and penetrating injuries. However, in this case, the patient did not report any recent or past episodes of significant trauma. Spontaneous diaphragmatic hernias are much rarer than either congenital or traumatic types. There were 28 detailed reports in the world literature between 1956 and 2009, of spontaneous rupture of the diaphragm, in which coughing was the preceding event for 32% of patients, physical exercise in 21%, vaginal delivery in 14%, vomiting in 7% and massage in 4%.⁵ Since this literature review, additional cases have been reported. The preceding events for these cases were pilates,⁶ sneezing,⁷ squatting⁸ and vaginal delivery.⁹ The most common symptoms were abdominal pain, nausea, vomiting and dyspnoea.

Subsequent incarceration and perforation of diaphragmatic hernias is an uncommon but potentially life-threatening set of events. The associated mortality of gastric perforation into the thoracic cavity is extremely high, reported at 42–56%.¹⁰ What makes the present case unique is the patient's lack of serious symptoms. There is no other case reported of gastric perforation into the thoracic cavity in which the patient presents subacutely.

The subacute presentation was hugely beneficial, allowing the patient to be nutritionally and haemodynamically optimised prior to surgery. The additional time also meant that a multidisciplinary team meeting could take place to

reach a consensus regarding definitive treatment. This led to an excellent outcome for the patient, avoiding the serious morbidity and mortality that is so prevalent in cases of this type.

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