Gastric perforation secondary to incarcerated hiatus hernia: an important differential in the diagnosis of central crushing chest pain

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Gastric perforation in association with incarceration of a hiatus hernia rarely features on a list of differential diagnoses of acute chest pain. A patient presented to the emergency department with acute chest pain characteristic of myocardial ischaemia. Several risk factors for ischaemic heart disease (IHD) were present. Investigations revealed normal cardiac enzymes and normal electrocardiography both initially and at 90 mins. A chest radiograph demonstrated the presence of a hiatus hernia. The patient was diagnosed with, and treated for, unstable angina. A troponin T test at 12 h post-admission was normal. The patient's clinical condition continued to deteriorate. The source of her pain was found to be gastric perforations in association with an incarcerated hiatus hernia. Her postoperative course was complicated by pulmonary and intra-abdominal sepsis necessitating admission to the intensive care unit where she remained for 23 days. This case highlights the challenge that non-cardiac chest pain presents to the acute care physician. Patients who present with risk factors for and symptoms consistent with a diagnosis of IHD may have non-cardiogenic pathology which can be life-threatening.

A 49-year-old woman presented to the emergency department with severe central crushing chest pain radiating to her left shoulder. It was associated with shortness of breath and nausea. The pain was exacerbated by exertion. Intravenous morphine was required to provide adequate analgesia. The patient had suffered similar episodes of chest pain over the previous month.

Several risk factors for ischaemic heart disease (IHD) were present. These included obesity, hypercholesterolaemia, hypertension and smoking (40 pack years). There was also a strong family history of IHD among first degree relatives.

The patient also described dysphagia with solid food which she attributed to her gastro-oesophageal reflux disease for which she had been receiving ranitidine.

Her past medical history included chronic obstructive pulmonary disease, fibromyalgia and depression. Prescription medications included amitriptyline, fluoxetine, diazepam, zopiclone, ranitidine, bendroflumethiazide and sublingual glyceryl trinitrate spray.

On examination she was afebrile. Her pulse was 90 beats/min and blood pressure was 144/89 mm Hg with no difference between limbs or delay in pulse radiation distally. Peripheral oxygen saturation was 98% and respiratory rate was 20 breaths/min. Cardiovascular and respiratory examinations were unremarkable. Her abdomen was soft with mild tenderness in the epigastrium but

no guarding or rebound. Bowel sounds were present and normal.

Haematological investigations revealed a haemoglobin concentration of 12 g/dl, white cell count $14.2 \times 10^9/l$ (neutrophilia $12.26 \times 10^9/l$) and a normal coagulation profile. Blood biochemistry was normal. D-dimer was 0.13 µg/ml and amylase 41 units/l. Initial and 90 min cardiac enzymes were normal.

Arterial blood gas analysis on 28% inspired oxygen via face mask and humidifier showed type II respiratory failure with a compensated respiratory acidosis giving a pH 7.42, P_{O_2} 7.2 kPa, P_{CO_2} 7.3 kPa, HCO_3^- 31.9 mmol/l and base excess of +9.3 mmol/l. Blood

Figure 1 Oral contrast swallow (written consent obtained from patient).

lactate and glucose concentrations were within normal limits.

A 12 lead electrocardiograph (ECG) showed normal sinus rhythm with a rate of 90 beats/min and no acute ischaemic changes. An erect postero-anterior chest radiograph showed a hiatus hernia; there was no mediastinal widening and both lung fields were normal. An abdominal radiograph was unremarkable.

The patient had no previously documented evidence of IHD. Given her history and risk factors she was admitted according to the hospital's chest pain protocol to the medical unit. The initial diagnosis was unstable angina. She was treated with low molecular weight heparin (LMWH), clopidogrel, metoprolol and atorvastatin. A 12 h troponin T value was <0.01 ng/ml.

Incarceration of her hiatus hernia was then considered as a potential diagnosis. A contrast swallow radiograph was performed (fig 1).

The oral contrast swallow test showed a large hiatus hernia. No contrast passed into the stomach and a fluid level was noted to T1; this was consistent with an incarcerated hiatus hernia.

A surgical consult was requested. Endoscopy demonstrated patches of necrosis in the posterior gastric wall. A laparotomy was performed. Surgeons noted three ischaemic perforations in the posterior wall of the gastric fundus. A partial gastrectomy with resection of the proximal half of the stomach was performed and a feeding jejunostomy placed.

Postoperatively she was transferred to the intensive care unit for ventilator and inotropic support.

DISCUSSION

Acute chest pain is a common presenting symptom, accounting for an estimated 2.4–6% of adult emergency department attendances.^{1,2} In one study, 54.5% of patients had neither clinical nor ECG evidence of an acute coronary syndrome, but 12.5% were categorised as having other potentially life-threatening pathologies.²

Differentiation of ischaemic from non-ischaemic chest pain can be difficult. The differential diagnosis of acute chest pain is extensive.³ This patient had several risk factors for IHD. The pain was characteristic of myocardial ischaemia, but the normal cardiac enzymes and normal ECG made this diagnosis less likely. The chest radiograph findings raised the possibility of an incarcerated hiatus hernia.

The clinical presentation of a para-oesophageal hernia may be incidental or a catastrophic and life-threatening

emergency. This patient had ischaemic gastric perforations. Perforation of a gastric ulcer in the context of a paraoesophageal hernia has a mortality rate >60%. Risk factors for perforation include old age, hernial size and incarceration.⁴ Such serious sequelae have resulted in a trend towards early surgical treatment of paraoesophageal hernias once diagnosed.⁵ This approach has been questioned recently since the pooled annual risk of requiring emergency surgery for life-threatening complications is only around 1%.⁶

The initial diagnosis of unstable angina resulted in this patient receiving both LMWH and clopidogrel. This had a significant impact on the perioperative management of the case. Intraoperatively, intravenous infusions of platelets and fresh frozen plasma were required. The use of blood products is not without risk.

Incarceration of a hiatus hernia rarely features on a list of differential diagnoses of acute chest pain. This case highlights the importance of maintaining a high

index of suspicion of non-cardiac, life-threatening causes of chest pain. Patients who present with risk factors for and symptoms consistent with a diagnosis of IHD may have non-cardiogenic pathology which can be life-threatening. Such patients pose diagnostic dilemmas for all practitioners involved in the assessment and treatment of acute chest pain.

CONTRIBUTORSHIP

Dr D Trainor managed the case, carried out a literature search and wrote the article; Dr M Duffy managed the case and edited the article; Mr A Kennedy performed the surgery; Dr P Glover edited the article; Dr B Mullan is senior author and guarantor.

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