CASE REPORT

Oesophageal ischaemia: an uncommon cause of chest pain

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SUMMARY

A 79-year-old woman with a history of ischaemic heart disease and atrial fibrillation presented to hospital with severe chest pain. Blood tests showed an elevated D-dimer and a rise in troponin I. ECG showed right bundle branch block pattern and T wave inversion in leads V1 to V3, although these changes were present in old ECGs. A chest X-ray was done which was normal. Due to the nature and severity of her pain a CT aortic angiogram was done. This did not show any evidence of aortic dissection or a pulmonary embolism. The patient then had several episodes of haematemesis. An urgent oesophagogastroduodenoscopy was done which showed a circumferential, well demarcated area of blackened oesophageal mucosa. The patient was diagnosed with ischaemic damage to her oesophagus.

BACKGROUND

Ischaemic injury to the oesophagus is a rare but important differential diagnosis in a patient with chest pain and haematemesis. Chest pain can have a number of different causes. While cardiac and respiratory pathology are very common and should always be considered, gastrointestinal pathology must also be considered particularly if a patient also has gastrointestinal symptoms. In addition, this case illustrates that while cardiac pathology is the most common cause for a raised blood troponin level, a rise in troponin—especially if mild—can be due to non-cardiac pathology.

CASE PRESENTATION

A 79-year-old woman was admitted to hospital with severe chest pain. She had a medical history of myocardial infarction; previous percutaneous coronary interventions and placement of two coronary stents; hypertension, atrial fibrillation and aortic stenosis.

On admission, her regular medication included apixaban, aspirin, atorvastatin, ramipril and glyceryl trinitrate spray. Little of note was found on physical examination. An ECG was performed. This did not show any acute changes in keeping with myocardial ischaemia. A chest X-ray was done which was normal. Due to the nature of her pain and her description that it radiated through to her back, a CT angiogram of her aorta was done. This did not show a dissection or evidence of a pulmonary embolus. No oesophageal pathology was seen on the initial CT angiogram after reanalysis by a radiologist. Her blood tests did not show much

of note other than an elevated white cell count of 15.6 and a positive D-dimer of 281. An initial blood troponin I level was 16. This was repeated 3 hours later and was found to have risen to 95. In light of this raised troponin level and the absence of other findings, she was diagnosed as having a probable non-ST elevation myocardial infarction (NSTEMI). However, during her admission, the patient developed several episodes of coffee ground vomiting. Following these episodes of haematemesis, her blood urea level was seen to rise from 4.5 to 10.7 with no corresponding rise in creatinine. Due to this, an urgent oesophagogastroduodenoscopy (OGD) was performed. This showed a localised, circumferential and well demarcated region of black mucosa in the distal oesophagus.

This region of necrotic mucosa had an abrupt transition point and did not extend below the gastro-oesophageal junction. As a result of these findings, the patient diagnosed as having had an ischaemic insult to her oesophagus and was placed nil by mouth (NBM). She did not have any history of non-steroidal anti-inflammatory drug use or excessive alcohol ingestion.

INVESTIGATIONS

Blood tests

On admission

Haemoglobin 119 g/L (previous baseline 145).

White cell count $15.6 \times 10^9/L$.

Platelets $234 \times 10^9/L$.

International normalised ratio 1.0.

Troponin I 16 (rising to 95 when repeated).

D-dimer 281 ng/mL.

Cholesterol 5.7 mmol/L.

Glucose 6.2 mmol/L.

Urea 4.5 mmol/L (rising to 10.7 after episodes of haematemesis).

Chest X-ray on admission

Normal.

ECG

No acute changes.

Echocardiography

Echocardiogram showed left ventricular hypertrophy with good overall left ventricular function.

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Figure 1 Middle oesophagus above area of necrosis.

Also seen were: moderate aortic stenosis, mild mitral regurgitation and mild tricuspid regurgitation.

Percutaneous coronary angiography

Subsequent coronary angiography showed patent right coronary artery and patent cardiac stents.

CT aorta

No evidence of aortic dissection. No evidence of a pulmonary embolus. On reanalysis of the CT images by a radiologist, no radiological evidence of oesophageal pathology was seen at the time of admission. However, a CT angiogram is not the ideal imaging modality to identify oesophageal pathology.

Oesophageogastroduodenoscopy

Well demarcated circumferential area of localised oesophageal necrosis (figures 1-4).

Repeat OGD 7 days later showed complete resolution of oesophageal necrosis.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of a patient presenting to hospital with chest pain is extensive. It should include:

Cardiac pathology

- Acute coronary syndrome (including angina, unstable angina, STEMI and non-STEMI);

Figure 2 Lower oesophagus showing area of necrosis.

Figure 3 Lower oesophagus showing area of necrosis. Beyond the necrotic area can be seen the unaffected gastro-oesophageal junction.

- Pericarditis;
- Aortic dissection.

Respiratory pathology

- Pulmonary embolus;
- Pneumonia.

Gastrointestinal pathology

- Oesophageal pathology (including oesophagitis, oesophageal neoplasms and ischaemia);
- Gastric pathology (including gastritis, gastric neoplasms, gastric perforation and ischaemia);
- Duodenal pathology (including duodenitis, duodenal perforation and duodenal neoplasms).

Musculoskeletal pathology

- Fractures;
- Muscle tears and muscle strain;
- Pain secondary to neoplastic processes.

TREATMENT

The patient was made NBM and commenced on total parenteral nutrition.

Figure 4 Repeat OGD 1 week after initial OGD showing lack of necrosis in the lower oesophagus. OGD, oesophagogastroduodenoscopy.

Figure 5 Repeat OGD 1 week after initial OGD showing lack of necrosis in the lower oesophagus. OGD, oesophagogastroduodenoscopy.

A course of intravenous broad spectrum antibiotic tazocin (piperacillin/tazobactam) was given and high-dose intravenous proton pump inhibitor omeprazole was administered.

OUTCOME AND FOLLOW-UP

The patient improved once being placed NBM, given intravenous omeprazole and commenced on antibiotics. Her chest pain gradually subsided and she did not have any further episodes of haematemesis. After she settled, a percutaneous coronary angiogram was performed. This showed patent coronary arteries and patent coronary stents.

A repeat OGD was performed 7 days later. This showed no evidence of oesophageal necrosis but did show mild reflux oesophagitis. Following her repeat OGD she was allowed to eat and drink and commenced on oral omeprazole. She was discharged home and will be followed up in the outpatient gastroenterology clinic.

DISCUSSION

Acute oesophageal ischaemia or acute oesophageal necrosis is a very rare clinical entity with estimated prevalence of up to 0.2% in autopsy series.^{1,2} In endoscopy series, the prevalence of acute oesophageal necrosis ranges from 0.001% to 0.2% of cases.^{2,3}

Endoscopically, the condition is characterised by a marked and striking diffuse circumferential black appearance of the oesophageal mucosa. This can affect various lengths of the oesophagus. In case series, the distal oesophagus is the region most commonly affected. In classic descriptions of the condition, there is an abrupt transition between necrotic mucosa and normal mucosa at the gastro-oesophageal junction⁴ (figure 5).

The disease was first described by Goldenberg *et al* in a case report published in 1990. It was later classified as a syndrome in its own right by Gurvits *et al* in 2007.^{5,6} Hence in some texts, it is referred to as Gurvits syndrome. It is a disease which predominantly affects men with male to female ratio of 4:1.⁷ The most common presentation is with features of upper gastrointestinal haemorrhage commonly haematemesis or coffee ground vomiting and melaena (70% of patients). Other symptoms may include dysphagia, abdominal pain or chest pain.⁷ Causes of acute oesophageal necrosis are largely multifactorial. Patients tend to be affected by a combination of ischaemic insult,

compromised mucosal defences and corrosive reflux injury of gastric contents.⁴

In case reports, infections, gastric volvulus, hyperglycaemia and diabetic ketoacidosis, an underlying malignancy, ischaemic phenomena and use of broad-spectrum antibiotics have all been linked to acute oesophageal necrosis.^{8–12}

Significant comorbidities including cardiovascular disease, diabetes mellitus and renal disease are important risk factors that may predispose patients to the development of acute oesophageal ischaemia.

The main stay of treatment should involve keeping patients NBM and administering high-dose intravenous proton pump inhibitors. There is a role for nutritional supplementation in these patients. The rationale behind this being that adequate nutrition can help promote mucosal healing. Case reports advocate the use of total parenteral nutrition due to the potential risk of the insertion of a nasogastric tube causing a perforation of the necrotic segment of oesophagus. Broad-spectrum antibiotics have also been documented to have been used in these cases with the aim of guarding against the potential of sepsis if perforation occurs. Volume expansion with intravenous fluids and treatment of underlying illnesses and comorbidities should also form a part of management. A follow-up oesophagogastroduodenoscopy may be indicated after a patient is stabilised to re-examine the extent of the disease and aid the early identification of mucosal breakdown or other complications. While a repeat endoscopy is not an essential part of management, it can be useful to correlate the appearance of the oesophageal mucosa to the patient's clinical symptoms. With prompt and appropriate supportive care, resolution of endoscopic findings occurs in most patients.⁹ However, estimated mortality rate ranges from 13% to 35%. This is often due to the patient's underlying diseases with only 6% mortality directly attributable to complications of acute oesophageal necrosis.^{9,10,13,14}

When the diagnosis of acute oesophageal necrosis was made, there was some uncertainty as to whether this episode of acute

Patient's perspective

I had been feeling unwell for 3 to 4 weeks, not wanting to eat or drink. When having my tablets with water I kept getting an awful pain similar to heartburn but a lot more severe. I also had a lot of nausea. I just felt generally awful.

Learning points

- ▶ Consider that chest pain may be caused by gastrointestinal pathology.
- ▶ Haematemesis in any patient with chest pain should prompt an urgent oesophagogastroduodenoscopy (OGD). It is the opinion of the authors that an OGD should still be done promptly in the context of confirmed haematemesis and chest pain even if a patient has had a recent cardiac event. The caveats to this being the presence of cardiac arrhythmias or haemodynamic instability.
- ▶ Although very rare, oesophageal ischaemia should be considered as a potential cause of chest pain with associated haematemesis. This is true in all patients but especially those who are elderly, have risk factors for atherosclerosis and have thrombophilia.

oesophageal ischaemic injury was caused by a transient haemodynamic compromise secondary to an NSTEMI.

A coronary percutaneous coronary intervention was done by the cardiology team. This did not identify any significant new narrowing of the patients coronary vessels. Her previously inserted coronary stents were patent. However, the most likely underlying cause for this episode remains an underlying NSTEMI precipitating the acute oesophageal necrosis.

Contributors AS was the first doctor from the gastroenterology team who came in contact with the patient and had done all initial work and discussion with Consultant gastroenterologist BR. AA then took over from AS regarding continuing care, organising investigations and daily follow-up while the patient in the hospital including liaising with cardiology team and organising repeat endoscopy.

Competing interests None declared.

Patient consent Obtained.

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