CASE REPORT

Oesophageal rupture masquerading as STEMI

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SUMMARY

A 67-year-old man presented to the emergency department, with acute onset of chest pain. Based on ECG changes suggestive of ST elevation myocardial infarction (STEMI), he was taken emergently to the cardiac catheterisation laboratory for coronary angiography. There he was found to have only non-obstructive coronary disease. Subsequent physical examination and review of his chest radiograph revealed subcutaneous emphysema, and CT scan revealed a distal oesophageal rupture and pneumomediastinum. After stabilisation in the intensive care unit (ICU), he was taken to the operating room for thoracotomy, chest tube placement and stenting of his oesophagus. He survived the incident and, after several weeks of ICU stay, recovered to a large extent. His case highlights the importance of considering oesophageal rupture in the differential diagnosis for acute onset of chest pain.

BACKGROUND

Chest pain is a common symptom leading to emergency department visits. It is a complaint that attracts close scrutiny owing to the possibility of an underlying life-threatening condition.¹ The evaluation of patients with chest pain must initially be focused on early recognition of these life-threatening aetiologies. Delayed or missed diagnoses and medical interventions targeted towards incorrect diagnoses can lead to worse outcomes including significant morbidity or mortality. Important tools in the initial evaluation of patients with chest pain include ECG, chest radiograph and basic laboratory analyses. Of these, ECG is typically acquired very early in an encounter for chest pain owing to its non-invasive nature and ready availability. It serves essentially as a point of triage—patients with chest pain and ST elevation on ECG are given immediate consideration for reperfusion therapy via percutaneous coronary intervention (PCI) or, where PCI is unavailable, thrombolytic therapy. Early reperfusion therapy leads to better outcomes for patients with ST elevation myocardial infarction (STEMI) and is a performance measure recommended by the American College of Cardiology and American Heart Association.² For hospitals that perform PCI, the ‘door-to-balloon time’ for patients with STEMI is reported to the Centers for Medicare and Medicaid services and is tied to hospital quality assessment and reimbursement.³ The incidence of STEMI has been declining in recent years but remains relatively high. Annual incidence of STEMI was estimated at 50–77/100 000 in two recent studies of US populations.^{4 5} Oesophageal rupture, well-recognised as another life-threatening cause of chest pain, is less common

than STEMI, with an annual incidence estimated at 3.1 per million in one recent study of a European population.⁶ Moreover, only a minority of oesophageal ruptures are spontaneous, with most resulting from instrumentation.^{6 7} Diagnosis of spontaneous oesophageal rupture can be challenging owing to its relative rarity and oftentimes non-specific symptoms and signs—in fact, the diagnosis is often missed during initial evaluation.^{7–9} Early diagnosis of oesophageal rupture is critical, as delayed diagnosis leads to increased morbidity and mortality.⁷

In this case, we discuss a patient who presented to the emergency department with acute onset of chest pain and signs of haemodynamic instability. His initial ECG had ST elevation. The emergency department therefore activated the interventional cardiology team for emergent PCI. However, when his coronary angiography revealed no evidence of acute coronary occlusion, his diagnosis had to be rapidly reassessed. He was subsequently found to have oesophageal rupture; he was stabilised and taken emergently to the operating room for intervention, ultimately surviving the episode. His case is unique among chest pain presentations partly because of the relative rarity of spontaneous oesophageal rupture, but also because this diagnosis was preceded initially by a diagnosis of STEMI resulting in timely emergent cardiac catheterisation as per guidelines.

CASE PRESENTATION

A 67-year-old man with a history of alcohol abuse, tobacco abuse and remote cerebrovascular accident resulting in wheelchair dependence, was brought to the emergency department with acute onset of substernal chest pain. Although it was not realised during the initial patient interview at presentation, later discussion with the patient and his family revealed that he had consumed seven 12-ounce beers 2 days prior to presentation. Subsequently, he developed intractable nausea with roughly 20 episodes of vomiting dark brown and black material. During this episode of nausea, he acutely developed crushing, 10/10 substernal chest pain without radiation, prompting him to present to the emergency department.

On physical examination at presentation, he was afebrile with a heart rate of 123, blood pressure of 80/58, respiratory rate of 22 and O₂ saturation of 85% on room air. He was a thin man with increased work of breathing. He was grunting with pain and intermittently clutching his chest. His physical examination was notable for dry mucus membranes, tachypnoea with clear lung fields, tachycardia with regular rhythm and normal S1/S2, normoactive bowel sounds without tenderness to

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Figure 1 ECG obtained in the emergency department.

palpation over his abdomen, and cool lower extremities with barely palpable dorsalis pedis pulses.

In the emergency department, peripheral intravenous access was obtained and 2 L of normal saline were bolused with subsequent recovery of blood pressure to around 110 over 60.

INVESTIGATIONS

Basic labs, ECG and chest radiograph were obtained.

Prior to the patient's laboratories and chest radiograph becoming available for viewing, his ECG was noted by his emergency department physician to have ST segment elevation in some of the inferior, anterior and lateral leads (figure 1). Given the patient's chest pain and ST elevation, and in accordance with guidelines for early reperfusion therapy, the emergency department immediately activated the cardiac catheterisation lab for emergent PCI.

Coronary angiography revealed 50% stenosis of the mid-portion of the 1st obtuse marginal branch of the left circumflex artery but was otherwise unremarkable. Notably, the patient continued to have 10/10 substernal chest pain during and after coronary angiography. Therefore, aortography was performed, but revealed only calcification of the aortic arch and descending aorta. During his catheterisation, additional data from the emergency department became available for viewing including the patient's laboratories and chest radiograph. Laboratories were notable for a white cell count of 17 000 with neutrophilic predominance, sodium 132 and potassium 2.8, anion gap of 18 and lactate of 9.0.

A portable, upright, anteroposterior chest radiograph that had been obtained but was not available for viewing prior to catheterisation revealed subcutaneous emphysema predominately in the right cervical and supraclavicular areas, with subtle tracking into the mediastinum (figure 2). Follow-up physical examination after catheterisation was notable for crepitus in the right cervical, supraclavicular and thoracic areas.

CT of the neck, chest, abdomen and pelvis was obtained with gastrograffin swallow. This imaging revealed extensive subcutaneous emphysema throughout the neck soft tissue (figure 3A), extensive pneumomediastinum and bilateral pleural effusions. Additionally, a longitudinal defect in the oesophagus was noted with contrast and air extravasation into the mediastinum directly visualised (figure 3B).

TREATMENT

The patient was supported in the intensive care unit (ICU) with intravenous fluids, pressors, antibiotics and mechanical

ventilation, then was taken emergently to the operating room where he underwent bilateral chest tube placement, flexible endoscopy (which confirmed a 5 mm anterior oesophageal perforation 7 mm above the gastro-oesophageal junction), WallFlex stent placement for exclusion of the oesophageal perforation, percutaneous gastrostomy tube placement and left-sided video-assisted thoracoscopic surgery with irrigation of the left hemithorax.

OUTCOME AND FOLLOW-UP

The patient required several weeks of mechanical ventilation including tracheostomy, but was ultimately weaned from the ventilator and from supplemental oxygen altogether. He had difficulty swallowing after extubation, and his postoperative course was complicated by oesophageal stricture requiring dilation. However, he is now able to safely ingest food and has returned to a state of health similar to that prior to his presentation.

DISCUSSION

Oesophageal rupture presenting with acute onset of chest pain

This patient's presenting history was classic for effort rupture of the oesophagus, or Boerhaave's syndrome, which results from an increase in intra-oesophageal pressure combined with

Figure 2 Anteroposterior chest radiograph obtained in the emergency department.

Figure 3 (A) Representative image from chest CT. The white arrow points to an area where there is extensive pneumomediastinum. (B) Representative image from chest CT. The white arrow points to an area where there is extravasation of oral contrast from the oesophagus.

negative intrathoracic pressure caused by straining or vomiting. Oesophageal rupture is recognised as a life-threatening cause of acute chest pain, yet its diagnosis is not always made promptly. In one case series of 51 patients presenting to a tertiary care facility who were ultimately found to have atraumatic oesophageal rupture, the initial diagnosis was correct in only 33% of patients.⁹ Moreover, the average time to correct diagnosis was 24 h. In most patients whose initial diagnosis was incorrect, their symptoms were attributed to pneumonia, spontaneous pneumothorax, myocardial infarction, pulmonary embolism or other gastrointestinal tract pathologies. Oesophageal rupture allows spillage of air and gastrointestinal contents into the mediastinum and pleura, leading to sepsis, respiratory compromise and, ultimately, shock. Rapid diagnosis is important—one review of 726 patients showed that treatment delays of more than 24 h were associated with a doubling of mortality.⁷

Key historical and examination findings pointing towards the diagnosis are chest pain, vomiting and subcutaneous emphysema.^{1 8} However, this combination (termed ‘Mackler’s triad’) is only present in about 14% of cases of oesophageal rupture. Other common but non-specific presenting features are dyspnoea, dysphagia, fever, tachycardia, and tachypnoea. The upright chest radiograph is a simple and sensitive method to evaluate for oesophageal rupture—75–90% of patients with

oesophageal rupture have abnormalities on chest radiograph, including pneumomediastinum, mediastinal widening, hydro-pneumothoraces and pleural effusions. Once the diagnosis is suspected, oesophagram or CT of the chest and abdomen with a water-soluble contrast agent is used for confirmation. In this patient’s case, CT was preferable because it allowed assessment of the extent of pneumomediastinum, more detailed assessment of the lungs and pleura, and because the patient needed to continue lying flat in the hours after his femoral cardiac catheterisation.

Management of oesophageal rupture should always include consultation with a specialist—although contained ruptures can sometimes be safely managed conservatively, endoscopic and/or surgical intervention is most often required.⁸

ST elevation on ECG in the absence of myocardial infarction

This patient’s ECG shows ST elevation in leads II, III, aVF and V2–V6, without reciprocal ST depressions. The lack of reciprocal ST changes could have been an indication of vasospasm or pericarditis not suggested by his presentation, but should have been considered. In hindsight, the ECG is not typical for STEMI. Yet multiple factors contributed to his ultimately being taken emergently for coronary angiography. Neither his recent binge drinking nor his chest X-ray results were known at the time of initial interview and review of the ECG. Additionally, in the age of ‘door-to-balloon times’ for use as a quality measure, patients such as this, with chest pain and ST elevation on ECG, are universally considered for emergent PCI or, where PCI is unavailable, thrombolytic therapy. The emergency department is the first line of defence for recognition of STEMI and activation of the catheterisation laboratory, and the implications of missing a STEMI are great in terms of patient outcomes, and hospital grading and reimbursement.

It is important to remember though, that a number of processes other than acute myocardial infarction can cause ST elevation on ECG. Their recognition is important to avoid unnecessary exposure to coronary angiography, or worse, empiric thrombolytic therapy. Alternative aetiologies of ST elevation are reviewed in detail by Wang *et al*¹⁰ and include normal variation (particularly in young men), early repolarisation, left ventricular hypertrophy, left bundle branch block, acute pericarditis, hyperkalaemia, Brugada syndrome, pulmonary embolism, cardioversion and Prinzmetal’s angina.

Learning points

- ▶ Oesophageal rupture is a well-known life-threatening aetiology of chest pain, but its diagnosis is challenging and often missed on initial presentation.
- ▶ Symptoms and physical signs of oesophageal rupture are mostly non-specific. Subcutaneous emphysema on examination and/or chest radiograph, when present, helps to differentiate oesophageal rupture from other causes of chest pain.
- ▶ The upright chest radiograph is a simple and relatively inexpensive test with good sensitivity for oesophageal rupture—typical findings can include pneumomediastinum, mediastinal widening, hydropneumothoraces and pleural effusions.
- ▶ There are numerous aetiologies for ST elevation on ECG other than myocardial infarction, highlighting the necessity for careful history-taking, physical examination and ancillary studies to elucidate the diagnosis in patients presenting with chest pain.

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