A 55-year-old lady presented to the Emergency Department with intermittent retrosternal chest pain of 1-week duration. She also gave a history of cough and recurrent vomiting for the past few days. She had a 10-year history of type 2 diabetes mellitus and 2-year history of dyslipidaemia.

Baseline clinical examination and investigations (full blood count and biochemistry screen) were normal except for a random blood sugar of 355 mg/dL along with a glycosylated haemoglobin value of 8.6.

A 12-lead electrocardiogram (ECG) revealed T-wave inversion in leads V2-V6. Cardiac enzymes (creatine kinase-MB, troponin) were normal. Chest radiogram showed no evidence of cardiomegaly or pneumomediastinum or subcutaneous emphysema.

The patient initially underwent a cardiology evaluation for suspected Acute Coronary Syndrome (ACS). A two-dimensional echocardiography showed normal left and right ventricular function. Coronary Angiogram was normal except for mild plaquing in proximal segment of left circumflex artery. There were no dynamic changes in ECG or cardiac enzymes on serial followup. However, her chest pain persisted despite adequate cardiac treatment, for which she was referred to our side.

On revisiting the history, she stated that she had a 1-month history of dysphagia, more to solids than to liquids and recurrent vomiting episodes. She underwent an upper gastrointestinal endoscopy, which revealed an extrinsic compression in the oesophagus from 33 - 36 cm. Barium swallow study showed an extrinsic circumferential compression in the lower 1/3rd of thoracic oesophagus with smooth sloping margins and significant luminal narrowing [Figure 1].
The radiological possibilities considered at this stage were oesophageal duplication cyst, cystic leiomyoma or a cystic schwannoma. Biopsy of the lesion could not be performed because of its close proximity with the left atrium and therefore we proceeded with an endoscopic ultrasound-guided FNAC. Microscopy revealed a predominant inflammatory infiltrate along with numerous multinucleate giant cells, foam cells, red blood cells and nuclear debris, which was highly suggestive of a contained perforation of the oesophagus. The patient was treated with intravenous antibiotics in the intensive care unit and a repeat CT was done after 2 weeks, which showed significant decrease in the size of the lesion [Figure 3].

After 1 month of followup, the patient was totally asymptomatic and reported a significant weight gain of 5 kg. The repeat CT after 1 month showed complete resolution of the lesion [Figure 4], thus confirming the diagnosis of a contained perforation of the oesophagus, which mimicked an intramural tumour of the oesophagus and presented as chest pain.

DISCUSSION

Oesophageal perforation is usually iatrogenic due to endoscopic procedures such as oesophageal dilatation for strictures and achalasia. In about 15% of the cases, there is a spontaneous rupture with no known pre-existing pathology of the oesophagus commonly due to repeated vomiting or severe retching which causes an increase in intra-abdominal pressure. Spontaneous rupture of the oesophagus, first described by Hermann Boerhaave in 1724, was originally called Boerhaave’s syndrome.

Chest pain is regarded as the cardinal symptom of oesophageal perforation and is present in more than 70% of patients with a full thickness perforation of intrathoracic oesophagus. A missed diagnosis that was first identified at autopsy has been reported in 17% of cases. The pain associated with oesophageal perforation is usually sudden in onset with radiation to the back or to the left shoulder. In about 25% of the patients, this pain is followed by vomiting and shortness of breath. The triad of vomiting, chest pain and subcutaneous emphysema is known as the Mackler triad.

Diagnosis can be difficult mainly due to non-specific symptoms, and requires a high index of suspicion. In our case, the patient was initially misdiagnosed to have ACS, because of the patient’s chest pain and ECG findings.

Treatment of oesophageal perforation depends on the aetiology, site and duration, underlying oesophageal disease, size of perforation and the general health status of the patient. Algorithm for management of spontaneous oesophageal perforations, adopted from suggestions made by Shenfine and Griffin, is shown in Figure 5.

Small contained perforations tend to seal without sequelae as in our case. Patients with perforation that is confined to the mediastinum and minimal clinical signs of sepsis can also be treated by nonsurgical methods. However, perforations that affect the pericardium, pleura or peritoneum require rapid surgical intervention. As the delay between perforation and treatment increases, the prognosis worsens due to the development of sepsis and progressive organ failure. In our case non-operative management was chosen, because it was a small contained perforation with no evidence of mediastinal contamination.

CONCLUSION

All cases presenting as chest pain should not be assumed to be of cardiac origin. While evaluating a patient with chest pain we must broaden our differentials, to include rare causes like oesophageal perforation which can be fatal if missed.
REFERENCES


