Chest pain is one of the most common complaints expressed by patients presenting to the emergency department, and any initial evaluation should always consider life-threatening causes. Esophageal rupture is a serious condition with a high mortality rate. If diagnosed, successful therapy depends on the size of the rupture and the time elapsed between rupture and diagnosis. We report on a 41-year-old woman who presented to the emergency department complaining of left-sided chest pain for two hours.

**Key Words:** Chest pain • Coronary artery disease • Esophageal rupture • Misdiagnosis

**INTRODUCTION**

The complaint of acute non-traumatic chest pain presents one of the most difficult diagnostic challenges in emergency medicine. Patients with this complaint account for approximately 5% of all emergency department (ED) visits. Given that coronary artery disease (CAD) is the leading cause of death worldwide, rapid identification of an acute myocardial infarction is the foremost consideration in the emergency physician’s differential diagnosis. Patients with acute coronary syndrome (ACS) often have symptoms indistinct from those of other emergent conditions. The complaint of chest pain can be observed in patients with an aortic dissection, pulmonary embolism, pneumothorax, pericarditis or esophageal rupture. Delay in treatment and diagnosis of any of these conditions leads to an increase in morbidity and mortality. However, rupture of the esophagus is extremely rare, and early symptoms of the disease are similar to those of emergency diseases of the chest and abdomen. Given the undifferentiated nature of chest pain as a symptom, proper diagnosis and subsequent treatment is often delayed, which in some cases results in an unfavorable outcome.

**CASE REPORT**

A 41-year-old woman presented to the ED two hours following the onset of dull central chest pain that radiated into the interscapular area. The patient stated that she had been experiencing a runny nose, congestion, sore throat, cough, and vomiting after meals for the last few days. She had no underlying esophageal disease or risk factors for CAD. Baseline clinical examination and investigations (full blood count and biochemistry screen) were normal. Chest radiogram demonstrated right pleural effusion, but no radio-opacity was detected and there was no evidence of pneumomediastinum or subcutaneous emphysema (Figure 1). A 12-lead electrocardiogram (ECG) revealed prominent ST segment depression and T wave inversion in leads V1-V6. Cardiac enzymes (creatine kinase, creatine kinase-MB, troponin) were all in the normal range. The patient was hospital-
ized in the cardiology department with suspected ACS. A two-dimensional echocardiography showed normal left and right ventricular function. Cardiac enzymes and ECG follow-up remained unchanged after 6 hours. However, six hours after the onset of the symptoms, the hemoglobin level had dropped from 13 g/dl to 11 g/dl. The patient experienced increased nausea and vomiting. Because of the persistent chest pain, chest computed tomography (CT) was performed. A small amount of extraluminal air in the surrounding mediastinum and mediastinal fluid with pleural effusion in the lower esophagus were observed (Figure 2A, B). Also, pulmonary embolism and aortic dissection were excluded by computed tomography. The patient was promptly transferred to our surgery room. On the fifth day of hospitalization, the patient had no complications, and a follow-up CT scan of the chest revealed an absence of abcess formation and a significant decrease in extraluminal air in the mediastinum. Follow-up 3 months after discharge showed the patient to be recovering with no complaints.

**DISCUSSION**

Since its original description by Boerhaave, the term “spontaneous rupture of the esophagus” has been used almost routinely in the literature to include all perforations involving the entire thickness of the esophageal wall, whenever perforation was associated with forceful or prolonged emesis. It clarifies the factors concerned in spontaneous rupture of the esophagus: increased intraluminal pressure, preexisting esophageal diseases, and neurogenic causes of perforation. Each of these factors can cause spontaneous rupture independent of the other, but they commonly are combined in an individual patient.5 Spontaneous esophageal rupture is a dangerous and often fatal condition because it may rapidly progress to severe mediastinitis, sepsis, and multiple organ failure. The classic history of vomiting followed by severe chest pain, subcutaneous emphysema and shortness of breath is not always seen. A recent case series
showed that 12-50% of patients have no history of vomiting. Patients with esophageal perforation may complain of dyspnea, cough, fever, and abdominal pain. Physical examination may reveal diminished breathing sounds, heart sounds with audible crepitus or subcutaneous air in the thorax or neck. The majority of patients will have chest radiographic abnormalities. We initially misdiagnosed the present case as ACS because of the patient’s chest pain and ECG findings such as ST-segment depression and T wave inversion in leads V1-V6. We considered that the ECG changes mimicking angina pectoris in this case were probably caused by rapid expansion of the gastric tube. Previous reports of ECG changes mimicking myocardial ischemia or infarction from cardiac compression by extra-cardiac causes include a gastric tube located retrosternally after esophageal reconstruction. The mechanism of ST-T changes may be explained as follows. During cardiac systole, a recoil force produced by ejection of blood into the aorta thrusts the left ventricle against the chest wall in the anterior direction. A large volume of air in the reconstructed esophagus increases the space between the sternum and the heart, resulting in more dynamic cardiac movement. The heart moved toward the sternum during systole and away from it during diastole. Therefore, in the anterior chest leads, the ECG changed in accordance with the cardiac cycle. Differences between esophageal rupture and acute coronary syndrome are shown in Table 1.

Confirmatory diagnostic methods include esophageal contrast radiography, CT scans, or endoscopy. Chest CT is an efficient diagnostic method for esophageal ruptures because it can reveal pneumomediastinum, pneumothorax, and pleural effusion. Treatment of esophageal rupture depends on the etiology, site, duration, underlying esophageal disease, size of perforation and the health status of the patient. Small perforations tend to seal without sequelae. Perforation of the cervical esophagus can be managed conservatively in most cases. Also, patients with perforation that is confined to the mediastinum and minimal clinical signs of sepsis can be treated by nonsurgical methods. Perforations of the lower side of the esophagus that affect the pericardium, pleura, or peritoneum require rapid surgical intervention. With an increased delay between perforation and treatment, the prognosis worsens, owing to the establishment of sepsis and progressive organ failure. We suggest rapid CT scan of the chest whenever esophageal perforation is suspected. In our case, operative management was chosen, based on the fact that the patient’s general condition was impaired.

In conclusion, it is important to consider the possibility that patients who complain of chest pain following vomiting may have esophageal perforation. All patients with undifferentiated chest pain should be questioned about antecedent vomiting and have their chest CT reviewed before medical treatment is initiated. The present case is intended to emphasize that chest pain due to esophageal disease may be overlooked, though this type of case does not frequently occur.

REFERENCES


