

CARDIAC TAMPONADE COMPLICATING SPONTANEOUS ESOPHAGEAL RUPTURE

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Rupture of the esophagus is usually secondary to trauma or intrinsic esophageal disease, such as stricture and neoplasm. Spontaneous rupture (Boerhaave's syndrome) is uncommon, and usually follows violent vomiting in the elderly due to incoordination between the cardia and cricoid muscle, and presents with acute chest or upper abdominal pain with shock. It has a high mortality.¹ The condition can be complicated by mediastinitis, with pericarditis and pleural effusion. Cardiac tamponade has been reported as a complication with traumatic rupture of the esophagus due to sharp objects² and after esophagoscopy,³ but there have been no previous reports of cardiac tamponade with spontaneous rupture.

Case Report

An 80-year-old woman was admitted to hospital with acute onset of severe central chest pain, which was preceded by vomiting in the previous 24 hours. She was previously healthy, apart from osteoarthritis of both knees, but was not under any treatment. On examination, she looked ill, was thin, and had a sinus tachycardia of 130/min., BP 100/60, temperature 37.6°C, and respiratory rate of 40/min. Her chest was clear, heart sounds normal, and there were no added sounds. Abdominal examination was normal and CNS was intact.

Investigations revealed a hemoglobin of 13.6 g/dL, WBC $11.2 \times 10^9/L$ (neutrophils 82%, lymphocytes 15%), and platelets $370 \times 10^9/L$. Urea and electrolytes, liver function tests and cardiac enzymes were normal. ECG showed sinus tachycardia but no other abnormality. There was pneumomediastinum and a small left pleural effusion on chest x-ray. A ruptured esophagus was suspected. Esophagogram with gastrografin revealed a leak in the lower third of the esophagus, confirming the diagnosis. The patient was admitted to the Intensive Care Unit. Arterial and central venous pressure lines were inserted. She was started on oxygen, intravenous fluids, antibiotics

and nasogastric suction. She was sedated and ventilated. The patient subsequently became stable, arterial blood gases were normal on FIO₂ 60%, central venous pressure was 10 mm Hg and heart rate 110/min. Sinus rhythm and urine output were normal.

Ten hours later, the heart rate was observed on the monitor to accelerate over a few minutes to 140/min., the blood pressure dropped to 70/50, the oxygen saturation decreased to 84%, and the central venous pressure rose to 20 mm Hg. The patient appeared irritable. She had a feeble pulse with no paradox. Venous waves in the neck could not be seen. Breath sounds were reduced and the heart sounds were muffled. Right ventricular infarction and cardiac tamponade were suspected. ECG showed sinus tachycardia and no other abnormality. An urgent echocardiogram revealed the presence of a moderate pericardial effusion, with right atrial and right ventricular diastolic collapse diagnostic of cardiac tamponade. A chest x-ray done while awaiting pericardiocentesis showed an increase in the cardiac silhouette.

Immediate percutaneous pericardiocentesis through subxiphoid approach was performed under local anesthesia, with echocardiographic guidance. Using #5F pigtail catheter, 100 mL of serous fluid was drained. The blood pressure rose immediately to 100/60, the central venous pressure decreased to 12 mm Hg, and the heart rate decreased to 110/min. Subsequently very little fluid drained, and the catheter was taken out. An echocardiogram the next day showed minimal pericardial fluid, with normal filling pattern. Biochemical analysis of the fluid confirmed a transudate. Cultures were negative and there was no evidence of malignancy. The patient remained stable thereafter, with no recurrence of tamponade. Follow-up radiological studies showed no leakage from the esophagus. The patient was discharged in good health after six weeks. Two years after her illness, she remains alive and well.

Discussion

This report shows that cardiac tamponade can complicate spontaneous rupture of the esophagus. Pericarditis with pericardial effusion results from the spillage of esophageal contents in the mediastinum. However, rupture can occur into the pericardial sac. In this

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case, the pericardial effusion resulted from a sympathetic reaction. Although the amount was small, the rapid accumulation led to cardiac tamponade. Clinical detection of cardiac tamponade may be difficult in patients on ventilators. A paradoxical pulse may not be present, and venous waves may be difficult to see. A high index of suspicion is therefore required. Cardiac tamponade should be suspected in these patients if there is sudden hemodynamic deterioration with hypotension, acceleration of heart rate, a rise in central venous pressure, and a reduction in urine output.

As patients with esophageal rupture are already critically ill, 2D echocardiography should be done to rule out this reversible lethal complication. It is the diagnostic method of choice, can be arranged expeditiously, and can guide percutaneous pericardiocentesis, which is a highly

effective therapeutic measure.⁴

In conclusion, cardiac tamponade can complicate spontaneous rupture of the esophagus. As the condition has a high mortality, every effort should be made to detect and treat reversible adverse complications.

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