Case Reports

Successful Treatment of a Spontaneous Esophageal Rupture in an Elderly Patient: A Case Report

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Abstract

An 80-year-old woman was admitted to our hospital with severe chest and back pains after vomiting. Computed tomography (CT) of the chest revealed left-sided pneumothorax and pleural effusion. Some food was drained from an inserted chest tube, and we diagnosed spontaneous esophageal rupture (Boerhaave’s syndrome). A left thoracotomy was performed 7 hours after the onset of symptoms. A 3-cm perforation was discovered in the lateral wall of the distal esophagus. The perforation was repaired with a primary two-layered closure and covered with pericardial fat. The patient had a good postoperative course and was discharged 1 month after surgery. This case suggests the importance of early surgical treatment, even in elderly patients with spontaneous esophageal rupture.

(J Nippon Med Sch 2010; 77: 338-341)

Key words: spontaneous esophageal rupture, primary repair, old age

Introduction

Spontaneous esophageal rupture, or Boerhaave’s syndrome, originally described by Hermann Boerhaave in 1724, is a severe condition that easily progresses to sepsis. Therefore, early diagnosis and treatment are important for increasing the survival rate of patients. However, patients do not always present with the classic features, and treatment is sometimes delayed. We report a case of spontaneous esophageal rupture in an 80-year-old woman diagnosed early after symptom onset and treated with emergency surgery. We believe this case to be noteworthy because the patient recovered uneventfully after the operation despite her advanced age and the presence of other risk factors.

Case Report

An 80-year-old woman was admitted to the hospital with severe chest pain and back pain after vomiting. She had a history of surgery for meningioma and a history of hypertension, so had taken anticoagulant and antihypertensive medications for several years. She had never previously had symptoms of vomiting or weight loss and had no history of gastritis or gastric ulcer. On admission, vital signs were as follows: blood pressure, 154/90 mmHg; pulse rate, 118/minute;
Boerhaave’s Syndrome in Elderly Patient

Table 1 Laboratory examinations performed at our hospital revealed no abnormal findings

<table>
<thead>
<tr>
<th>WBC</th>
<th>7,100 /μL</th>
<th>AST</th>
<th>19 IU/L</th>
<th>Na</th>
<th>147 mEq/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBC</td>
<td>838×10⁶ /μL</td>
<td>ALT</td>
<td>14 IU/L</td>
<td>K</td>
<td>41 mEq/L</td>
</tr>
<tr>
<td>Hb</td>
<td>12.2 g/dL</td>
<td>LDH</td>
<td>200 IU/L</td>
<td>Cl</td>
<td>103 mEq/L</td>
</tr>
<tr>
<td>Ht</td>
<td>37.6 %</td>
<td>CPK</td>
<td>72 IU/L</td>
<td>BUN</td>
<td>183 mg/dL</td>
</tr>
<tr>
<td>Plt</td>
<td>21.9×10⁴ /μL</td>
<td>CKMB</td>
<td>1.5 mg/mL</td>
<td>CRE</td>
<td>1.09 mg/dL</td>
</tr>
<tr>
<td>CRP</td>
<td>&lt; 0.10 mg/dL</td>
<td>AMY</td>
<td>100 IU/L</td>
<td>TP</td>
<td>7.3 g/dL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T-BIL</td>
<td>0.3 mg/dL</td>
<td>Alb</td>
<td>4.9 g/dL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>D-BIL</td>
<td>0.1 mg/dL</td>
<td></td>
<td>Troponin test (-)</td>
</tr>
</tbody>
</table>

Fig. 1 Computed tomography (CT) image of the chest shows pneumothorax (arrow) and pleural effusion (arrowhead) in the left side of the chest.

Fig. 2 A perforation on the left lateral wall of the distal esophagus. A gastric tube inserted from the mouth is visible (arrow).

body temperature, 35.2°C; and SpO₂ 93.4% (O₂ 12 L). At first, acute myocardial infarction was suspected because a minimal ST wave change was observed on electrocardiography (ECG) performed during the ambulance ride to the hospital. Laboratory examinations, including a troponin T test, performed at our hospital revealed no abnormal findings (Table 1). The ECG findings obtained at our hospital were not typical of acute myocardial infarction. Physical examination revealed diminished breath sounds over the left lung. Computed tomography (CT) of the chest revealed left-sided pneumothorax and pleural effusion and marked dilatation of the stomach by food (Fig. 1). A chest drainage tube was inserted, and some food was drained. These results confirmed a rupture of the esophagus, and an emergency left thoracotomy was performed 7 hours after symptom onset. A 3-cm perforation was discovered in the left lateral wall of the distal esophagus (Fig. 2). The perforation was repaired with a two-layered closure and was covered with pericardial fat (Fig. 3). In the 11 days after the surgery, laboratory examinations showed no abnormalities. Oral intake was started on postoperative day 16. The patient had no postoperative complications and was discharged 1 month after the surgery.
Discussion

Spontaneous esophageal rupture is uncommon but is associated with significant mortality, with reported mortality rates of 8% to 60% [1]. Successful management depends on early diagnosis and prompt treatment, as the morbidity and mortality rates rise rapidly after the first 12 to 24 hours [2]. Brinster et al. have reported that delays in treatment of more than 24 hours after perforation can result in a doubling of the mortality rate from 14% if diagnosed early to 27% if diagnosed late [3]. However, the condition is rare, and many physicians may be unfamiliar with its features. Consequently, the diagnosis of spontaneous esophageal rupture may be initially missed or delayed. The most common misdiagnosis is perforated ulcer, followed by myocardial infarction, pulmonary embolism, dissecting aneurysm, and pancreatitis [4]. In our case, the patient complained of severe chest and back pains, and ECG findings obtained during the ambulance ride to the hospital suggested myocardial ischemic changes. Therefore, cardiovascular disease, including acute myocardial infarction, was initially suspected. However, a chest CT revealed pneumothorax and pleural effusion, and spontaneous esophageal rupture was correctly diagnosed without any critical delays.

Spontaneous esophageal rupture is believed to occur through the transmission of increased intra-abdominal pressure to the esophagus against the closed glottis and in many instances is associated with violent retching and vomiting, which cause a sudden increase in intraesophageal pressure [5]. Most cases follow bouts of heavy eating and drinking [6]. In the present case, marked dilatation of the stomach from the presence of food was observed on the CT scan, and an episode of heavy eating and vomiting was mentioned by the patient’s family. Marked dilatation of the stomach was only due to heavy eating and drinking because the patient had no history or symptoms of stenosis of the duodenum or other parts of the small intestine. Accordingly, the clinical course of the present case was thought to be typical for spontaneous esophageal rupture.

Multiple treatments for esophageal rupture have been described, ranging from conservative measures to extensive surgery. Conservative management, including the restriction of oral intake, placement of a nasogastric tube, and the administration of intravenous antibiotics, is usually performed when the esophageal rupture is small and does not necessitate drainage [7]. We have previously reported the successful management of an esophageal rupture using conservative therapy in a 41-year-old man [8]. We had decided to treat him conservatively on the basis of the following factors: a stable general condition without sepsis, the limitation of the esophageal disruption to the mediastinum, and early diagnosis.

Surgical treatment is usually selected in cases with rupture into the pleural cavity because the contamination must be cleaned with thoracic drainage as soon as possible. Various surgical
techniques to repair esophageal defects have been described and include primary repair, reinforced primary closure with either tissue or mesh, T-tube drainage, exclusion, diversion, and intraluminal stenting, and resection. When an esophageal perforation is closed within the “golden” period, meaning the first 12 hours, postoperative leakage is thought to be unlikely. On the other hand, postoperative leakage has been reported to occur in 83% of cases treated more than 24 hours after onset. In the present case, we were able to treat the patient within 7 hours of onset. Therefore, we selected a primary two-layered closure and patching with pericardial fat as the repair method.

The mean age of patients with esophageal rupture is 50 to 60 years, and cases in patients older than 80 years are rare. We could not find any reports of successful surgical treatment of esophageal rupture in a patient older than 80 years.

In conclusion, the prognosis of spontaneous esophageal rupture depends on early diagnosis and appropriate management. Our case suggests the importance of early surgical treatment, even in elderly patients.

References


(Received, August 25, 2010)
(Accepted, October 16, 2010)