Boerhaave Syndrome Due to Excessive Alcohol Consumption: Two Case Reports

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Case report

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Abstract

Background

Spontaneous esophageal rupture, or Boerhaave syndrome, is a fatal disorder caused by an elevated esophageal pressure derived from forceful vomiting, and subsequent presentation of chest pain, dyspnea, and shock.

Case presentation:

We present two cases of Boerhaave syndrome that were both triggered by excessive alcohol consumption and quickly detected in the emergency room. The first patient complained of severe chest pain, nausea, and vomited on his arrival: he was diagnosed with Boerhaave syndrome complicated with mediastinitis from the computed tomography (CT) and esophagogram findings. An emergency operation was successfully performed, where a 3-cm tear was found on the left-posterior wall of the distal esophagus. The patient subsequently suffered from anastomotic leakage but was discharged 41 days later. The second patient reported severe chest pain, nausea, vomiting, and hematemesis on his arrival: he was suspected of having Boerhaave syndrome without mediastinitis based on the CT findings. The symptoms gradually disappeared after a conservative treatment. Upper gastrointestinal endoscopy performed on the 9th day revealed a scar on the left wall of the distal esophagus. The patient was discharged 11 days later. In addition to the varying severity between the cases, the second patient was also differently diagnosed with Mallory-Weiss syndrome.

Conclusion

Emergency clinicians must accurately distinguish Boerhaave syndrome from Mallory-Weiss syndrome as they both have similar history and symptoms. CT can be a valuable and useful modality to detect any severity of Boerhaave syndrome.

Background

Boerhaave syndrome was first reported by Hermann Boerhaave in 1724 as a case of oesophageal rupture by vomiting after a large meal [1]. It is induced by increased oesophageal pressure followed by straining such as retching, vomiting, weightlifting, childbirth, or defecation [2]. It is crucial to correctly diagnose Boerhaave syndrome because Mallory-Weiss syndrome has the same causation but usually differs in the site and depth of laceration, required treatment, and prognosis. Generally, it is difficult to promptly diagnose, and cases can have fatal consequences if early intervention is not achieved [3].

Case Presentation
Case 1

A 45-year-old man, who had hypertension, dyslipidemia, and habitual drinking, came to our hospital with complaints of severe chest pain, back pain, bilateral shoulder pain, and vomiting. He had drunk alcohol heavily for a day, and felt ill after drinking. Three and a half hours before arrival, he had felt discomfort in his chest which was followed by vomiting. Two hours and 20 minutes prior to his arrival, he felt nauseated and then described a feeling of sudden stiffness of his whole upper body. On the arrival, he was alert, and his blood pressure was at 139/98 mmHg, pulse rate at 64/min and regular, body temperature at 36.5 °C, respiratory rate at 20/min, and oxygen saturation at 95%. He was 173 cm tall and weighed 73.1 kg. No abnormalities other than cold sweat and epigastric tenderness were noted on the physical examination. Chest radiography of the lateral view (sitting) showed several areas of free-air just below the diaphragm (Fig. 1). Contrast-enhanced computed tomography (CT) showed free-air, bilateral pleural effusion, and a dilated esophagus/stomach (Fig. 2). With these findings, an esophageal rupture with mediastinitis was definitive and he was diagnosed with Boerhaave syndrome. A subsequent esophagogram further confirmed the diagnosis. Nine hours after the onset of the rupture, an emergency operation was performed as follows. Through a left thoracoabdominal incision, closure of the perforation and drainage of the mediastinum and thoracic/abdominal cavity were performed in succession. The penetrating laceration was approximately 3 cm in length vertically on the left-posterior wall of the distal esophagus and patched with his omentum. Although the patient suffered a complication of anastomotic insufficiency on the 16th day, it healed and he was given a liquid diet on the 35th day, and he was discharged on the 41st day. Upper gastrointestinal endoscopy was performed on the 98th day, and a scar from the suture was observed in the same location as the previous perforation (Fig. 3).

Case 2

A 27-year-old previously healthy man came to our hospital with complaints of vomiting, hematemesis, pyrosis, severe chest pain, and epigastralgia. He had been drinking alcohol heavily until 14 hours before his arrival. Six hours before the arrival, he had felt epigastralgia and nauseated followed by retching and diarrhea. Three and a half hours prior to his arrival, he started to feel pyrosis, had intermittent chest pain, and had an episode of hematemesis. On the arrival, he was alert, and his blood pressure was at 110/62 mmHg, pulse rate at 96/min and regular, body temperature at 37.4 °C, respiratory rate at 18/min, and oxygen saturation at 97%.
He was 172.3 cm tall and weighed 53.5 kg. Epigastric tenderness and retrosternal pain with forced respiration were noted on the physical examination. Although chest radiography showed no findings, plain CT revealed small areas of free-air in the mediastinum (Fig. 4). At first, Mallory-Weiss syndrome was suspected because of hematemesis, but CT findings suggested Boerhaave syndrome. No hematemesis recurred during his hospitalization and his condition was followed up by esophagogram and CT scan. Upper endoscopy was performed on the 9th day, and a linear scar was observed on the left wall of the distal esophagus including the esophagogastric junction (EGJ) (Fig. 5). The injury healed with a conservative treatment including rest, nothing by mouth, and preventive antibiotics and he was discharged on the 11th day.

Discussion

A comparison of the two cases is shown in Table 1. Both were equally distressed on the arrival, but the clinical course was more complicated in case 1 compared to case 2. The initial diagnoses of both cases were symptom based involving vomiting, pain in the lower thorax, and mediastinal emphysema [4].
Table 1
Comparison of the reported cases

|                  | Case 1                  | Case 2                  |
|------------------|-------------------------|-------------------------|
| Age (years)      | 45                      | 27                      |
| Physique (body mass index) | 24.4                | 18.0                    |
| Symptoms         | Vomiting, chest pain, back pain, bilateral shoulder pain | Vomiting, chest pain, epigastralgia, hematemesis, pyrosis |
| Severity         | Distressed              | Distressed              |
| Trigger          | Excessive drinking from the previous day | Excessive drinking from the previous day |
| Hematemesis      | –                       | +                       |
| Operation        | +                       | –                       |
| Location         | On the left posterior wall of the distal esophagus | On the left wall of the distal esophagus |
| First endoscopy  | Day 68                  | Day 9                   |
| Period of hospitalization | 41 days             | 11 days                 |
| Estimated time from onset to rupture | 1.2 hours         | 2.5 hours               |
| Estimated time from rupture to operation room | 9 hours           | (No operation) |

Esophagography, upright chest X-ray, and chest CT scan are useful for the correct diagnosis of Boerhaave syndrome [5, 6]. In cases similar to case 1, X-rays are more appropriate in patients with major hydropneumothorax or pneumomediastinum, but if these are minor, as in case 2, CTs are preferred owing to recent improvements in imaging quality. In case 2, conservative treatment was revealed that if the inflammation of the rupture was localized in the mediastinum, non-operative choices could be successful [3].

The mechanism of Boerhaave syndrome is caused by a lack of coordination between the upper and lower esophageal sphincters, resulting in a transmural tear of the distal esophageal wall caused by increased intragastric pressure which is transmitted to the esophagus during vomiting [7]. In both presented cases, the left wall of the distal third of the esophagus was torn, which was more likely to occur due to anatomical reasons such as the thin muscular layer, nerve and vascular entrance, and lack of support for the surrounding connective tissue [7].
Several reports have described diagnostic errors which regarded esophageal rupture as Mallory-Weiss syndrome [8–10]. Commonly, massive hematemesis with less pain indicates Mallory-Weiss syndrome and less hematemesis with more pain indicates Boerhaave syndrome, despite both disorders having an onset of vomiting after drinking. Hematemesis was observed in case 2, but the endoscopic findings showed a scar only in the esophagus, not in the stomach, namely Mallory-Weiss’s lesions were not observed at all (Fig. 5).

The etiologies of both Boerhaave and Mallory-Weiss syndromes are similar, with the result of abnormally elevated intraluminal cardio-esophageal pressures during vomiting. Some predecessors have mentioned the relationship between the two syndromes [11–17], and some of them have identified Boerhaave syndrome as an extension of Mallory-Weiss syndrome [12–17], but this may be disputed for two reasons: the differences in the common sites (left distal esophagus and gastric cardia, respectively) and scarcity of reported transitional cases. Our review of accessible “transitional cases” [14–17] shows, except for one case which might have a real transitional lesion [17], that they had both several lacerations that extended into the submucosa at the gastric cardia and a single laceration through all layers at the distal esophagus separately. Thus, the two syndromes are primarily different, and many of the “transitional cases” may be considered to have occurred independently in the same individual. Describing pathophysiology, the Mallory-Weiss’s lacerations may mainly involve the part of the gastric cardia including EGJ by repeated retching, while the Boerhaave’s lacerations may occur all at once in the weakest part of the esophageal wall based on the idea that the muscular layer initially tears before the mucosal layer when an explosive force is applied [18].

In summary, we report two cases of Boerhaave syndrome with different severities. Severity may vary greatly among the patients with this disorder. Plain CT can detect any severity of this syndrome quickly and easily, and is recommended as a preliminary test if the environment permits. Although the initial cause is the same as that of Mallory-Weiss syndrome, the nature of the two syndromes is distinctive, and correct diagnosis is essential for positive patient outcomes.

**Abbreviations**

CT  
computed tomography; EGJ:oesophagogastric junction

**Declarations**

**Ethics approval and consent to participate**

This report was prepared in accordance with the ethical standards of the institutional ethics committee and with 1964 Helsinki Declaration.

**Consent for publication**
Written informed consents were obtained from the patients for publication of this report.

**Availability of data and materials**

Not applicable

**Competing interests**

The authors declare that they have no competing interests. This manuscript has not been published and is not under consideration for publication elsewhere. Additionally, all of the authors have approved the contents of this paper and have agreed to the journal’s submission policies.

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**Author’s contributions**

All authors have significantly contributed to the paper: YH contributed to the whole conception and design, literature review, and manuscript writing and correction. SY, HA, TN and NH contributed to management of the patients in the emergency room and checked the manuscript. TH contributed to Discussion as an esophagus specialist.

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Figures
Figure 1

Chest X-ray of the lateral view (sitting) on arrival in case 1. Several areas of free-air (arrows) just below the diaphragm are evident.

Figure 2

Contrast-enhanced CT on arrival in case 1 (A,B: Transverse section, C: Sagittal section). Free-air (arrows) around the distal esophagus, the lesser curvature of the stomach, and the para-aortic area of the
abdomen is observed. Bilateral pleural effusion (arrowheads), and the dilated esophagus/stomach (dotted-line arrows) are also observed.

Figure 3

Upper endoscopy on day 98 from the first visit in case 1. A scar (arrows) after the suture is observed on the left-posterior wall of the distal esophagus 38-41 cm from the incisors.

Figure 4
Plain CT on arrival in case 2 (A: Transverse section, B: Sagittal section). Small free-air (arrows) is observed in the mediastinum around the esophagogastric junction and around the boundary between the middle and lower thoracic esophagus.

![Image of CT scan showing small free-air around esophagogastric junction.]

**Figure 5**

Upper endoscopy on day 9 from the first visit in case 2. A linear scar (arrows) is observed on the left wall of the distal esophagus including the esophagogastric junction.