Barogenic rupture of oesophagus (Boerhaave syndrome) as diagnostic and therapeutic challenge requiring rapid and effective interdisciplinary cooperation – case report

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SUMMARY: We describe a 47-year-old male who was admitted to our centre from a local emergency unit with septic shock due to suspected Boerhaave syndrome. After the diagnosis was confirmed, the patient underwent emergency surgery. Postoperatively, the patient had symptoms of acute alcoholic delirium, and developed an oesophagomediastinal fistula as the most serious local complication. Successful conservative treatment enabled complete healing of the fistula, leading to patient recovery. No late complications like oesophageal stenosis were found at 6 months from discharge.

KEYWORDS: Boerhaave’s syndrome, mediastinitis, oesophageal fistula

INTRODUCTION

Boerhaave syndrome refers to spontaneous oesophageal rupture in normal oesophagus due to a sudden increase in intraluminal pressure during severe vomiting. In 1724, the disease was first described by a Dutch pathologist Hermann Boerhaave in a Dutch Adminal Jan van Wassenaer, who died of diffuse mediastinitis caused by oesophageal perforation following vomiting; the cause of death was diagnosed on autopsy [1]. Other causes of barotraumatic oesophageal rupture include a sudden increase in intraabdominal pressure, for instance, on lifting a heavy weight, straining, asthmatic exacerbation, seizures, or even laughing. Very rarely, Boerhaave syndrome may be a manifestation of inflammatory diseases of the oesophagus. Oesophageal wall tears occur usually in the distal one-third of the oesophagus, which is most susceptible to distension and lacks adjacent supporting structures [2,3]. The rarest tear localization is the cervical part of the oesophagus. Typical symptoms of oesophageal perforation are severe retrosternal and epigastric pain, dyspnoea, subcutaneous emphysema, and rapidly developing septic shock. Due to oesophageal wall disruption, gastric contents, saliva, or even bile enter the mediastinum as well as the left pleural cavity (70%), the right pleural cavity (20%), or both pleural cavities (10%). This leads to severe necrotizing mediastinitis and sepsis. The mortality rate is high and depends on the time from disease onset to treatment initiation; it reaches 90% in untreated patients and 40% in patients after appropriate surgery [1,7-9].

The most common radiographic findings include pneumothorax, pleural effusion, pneum mediastinum, and subcutaneous emphysema. Oesophagography with a water-soluble contrast can usually show oesophageal leakage.

Emergency surgery remains the treatment of choice although endoscopic procedures, e.g., stenting, have also been used [4-6]. Drainage could be used as the sole treatment [6]. Early repair is the method of choice for oesophageal perforation regardless of the cause [1,6,7]. Surgical methods include drainage of the mediastinum and pleural cavity through the left or right thoracotomy depending on rupture localization; resection of the thoracic oesophagus with salivary fistula, gastrostomy jejunostomy, and pleural drainage [3,10]; suture of the tear through thoracotomy with gastrostomy, feeding micro jejunostomy, and drainage of the mediastinum [10]; primary suture of the tear with an omental or pericardial flap [3,6,10]. Primary repair via a transhiatal access to the lower oesophagus with suture of the perforation, drainage of the pleural cavity, and gastrostomy/ jejunostomy are uniquely reported [4,5]. The rare occurrence of the disease, diagnostic difficulties, and insufficient personal experience of individual surgeons hinder establishing diagnostic and treatment standards for Boerhaave syndrome [2,4,5,8,9].

CASE REPORT

We report of 47-year-old male who was urgently admitted to our centre due to Boerhaave syndrome caused by severe vomiting after alcohol abuse. We describe in detail the clinical approach on admission, surgical treatment, perioperative course, and complications with their management.

A 47-year-old male, unemployed, addicted to alcohol, was admitted to the Emergency Unit of the County Hospital in Trzebnica, Lower Silesia (35 km from our centre) with severe chest and epigastric pain and dyspnoea. The patient reported 3 weeks of binge drinking. Two days before hospitalization, severe upper abdominal pain occurred after dyspepsia, as reported by the patient. Vomiting increased on the following day. During an episode of vomiting, severe chest and upper abdominal pain occurred, followed by exacerbating dyspnoea. The patient was brought to a local Emergency Unit with a suspicion of acute coronary syndrome. Acute coronary syndrome was excluded, and the diagnostic workup was extended. An X-ray showed left pleural effusion
and mild subcutaneous emphysema. Oesophagography with water-soluble contrast showed leakage to the left pleural cavity on the level of the suprarenchymal part of the oesophagus. Left pleural drainage was introduced in the third intercostal space and closed after aspiration of the effusion. After a telephone consultation, the patient was sent to our centre. On admission, the patient was conscious, with no symptoms of circulatory or respiratory failure, with mild dyspnoea, no peritoneal symptoms, rumbling and diminished respiratory murmur over the left lung on auscultation, and moderate subcutaneous emphysema in the upper part of the thorax. An X-ray with contrast confirmed perforation of the lower oesophagus, large pneumothorax, and improperly placed pleural drain - inserted too high and curved downwards, preventing effective drainage. The patient was operated on immediately under general anaesthesia. The course of operation was as follows: upper median laparotomy - no free fluid in the peritoneal cavity; mobilization of the left colonic flexure and descending colon with the spleen, pancreas, and stomach (Maddox’s manoeuvre) with their displacement medially to get free approach to the oesophageal hiatus and the left diaphragm; dissection of the left triangular hepatic ligament and pulling the liver laterally to the right; dissecting the oesophagodiaphragmatic ligament, opening and dissecting the oesophageal hiatus. After entering the left pleural cavity through the hiatus, 2000 ml of gastric content was evacuated. A 5-cm tear in the left lateral part of the oesophageal wall was found. After placing a nasogastric tube, the perforation was closed with two-layer continuous suture. Then, under manual control, two drains were introduced to the pleural cavity so that the lower opening of the fluid drain was just above the diaphragm. The drain from the third intercostal space was removed. Then, Dor fundoplication was performed, followed by Witzel’s gastrostomy and pyloric dilation. The mediastinum was drained and the laparotomy wound closed. Immediately after the surgery, the patient had respiratory and circulatory failure, and was sent to an Intensive Care Unit for further treatment. Due to mediastinitis, aspirations pneumonitis, and rapidly developing septic shock, the patient required mechanical ventilation, medical circulation support, and broad-spectrum antibiotics. In the ICU, the patient developed symptoms of acute alcoholic delirium that required intensive sedation. On the 12th postoperative day, a control contrast oesophagography showed an esophageal fistula. Conservative treatment, including strict diet, total parenteral nutrition, oesophageal suction tube, and prolonged left pleural drainage with minimal suction pressure, led to healing of the fistula. Oesophagography, performed on the 21st postoperative day, did not show any leakage from the oesophagus. The patient was discharged home in good condition. Six months later, stenosis or other abnormalities were not found on radiologic and endoscopic examinations. According to the patient and his relatives, he quit drinking alcohol.

DISCUSSION

The case reported herein is an example of an efficient and effective interdisciplinary cooperation. Proper diagnosis and early referral to a specialized centre undoubtedly contributed to a favourable outcome. Considering the natural course of Boerhaave syndrome, direct correlation between the duration of symptoms, morbidity and mortality rates, and insufficient experience of local surgical units in oesophageal surgery, we agree with the decision to send the patient to our centre. In patients presenting with severe retrosternal pain and a history of vomiting, oesophageal rupture should always be included in the differential diagnosis. Standard radiography, including plain X-rays and water-soluble contrast oesophagography, usually lead to early diagnosis. Immediate surgery improves patients’ survival. Treatment delays dramatically increase morbidity and mortality, with up to 90% mortality after 48 hours from symptom onset [1,2,7,8,9]. In patients with distal oesophageal perforations, we recommend to approach the mediastinum through laparotomy and the oesophageal hiatus, perform primary closure of the tear, drain the pleural cavity, and place a feeding gastrostomy with pyloroplasty or perform pyloric diversion to prevent delayed gastric emptying. An extensive rupture of the oesophageal wall may, however, require thoracotomy and oesophagectomy without immediate restoration of gastrointestinal tract continuity [10].

CONCLUSION

Spontaneous oesophageal rupture (Boerhaave syndrome) occurs in normal oesophagus due to a sudden increase in intraoesophageal pressure. It should be taken into consideration in every patient who presents with severe retrosternal pain and a history of intense vomiting. This acute condition should be immediately diagnosed and managed. Chest X-ray may show pneumothorax, pleural effusion, pneumomediastinum, and/or subcutaneous emphysema, while oesophagography with water-soluble contrasts usually shows oesophageal leakage. Emergency surgery - early repair and drainage, is the treatment of choice for all oesophageal perforations. The prognosis is poor and the mortality rate is up to 90 % in untreated cases and 40% after appropriate surgery, respectively. Oesophagomediastinal fistulas are the most serious local complications of Boerhaave syndrome. Infrequent occurrence, diagnostic difficulties, and insufficient surgeons’ experience hinder establishing diagnostic and therapeutic standards for this condition.

REFERENCES:

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