Boerhaave’s syndrome in a case of acute exacerbation of chronic obstructive pulmonary disease managed with non-invasive ventilation: a case report

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Abstract

Barotraumatic esophageal rupture (Boerhaave’s syndrome) is a rare and life-threatening disease, described as a Non-Invasive Ventilation (NIV) complication in a very small series. We report the case of a 72-year-old man admitted to the Emergency Department (ED) for severe dyspnea in a Chronic Obstructive Pulmonary Disease (COPD) stage Gold III. After NIV treatment, the patient suffered esophageal perforation with mediastinal and pleural contamination; emergency surgical treatment was successful for the esophageal repair, but the patient developed a fatal septic shock 12 days after surgery. Among NIV complications, few cases concern esophagogastric perforation: our case report describes an uncommon clinical situation treated with a successful damage control surgery approach.

Introduction

Barotraumatic perforation of the esophagus (Boerhaave’s syndrome) usually results in a left postero-lateral esophageal perforation, followed by contamination of the mediastinal cavity, chemical mediastinitis, emphysema, and local inflammation. If untreated, Boerhaave’s syndrome can rapidly evolve into pleural contamination and secondary sepsis due to the translocation of infected mediastinum content.1

Non-Invasive Ventilation (NIV) is a fairly safe method used as first-line ventilation support in patients with respiratory failure. Among NIV complications, few cases concern esophagogastric perforation: our case report describes an uncommon clinical situation treated with a successful damage control surgery approach.

Case Report

A 72 years old man, suffering a Chronic Obstructive Pulmonary Disease (COPD - stage Gold III) acute relapsing, was admitted to our Emergency Room (ER) for severe dyspnea. He claimed to suffer only from hypertension, no cardiac failure or other chronic diseases were detected in the anamnestic recap.

A bedside chest X-Ray showed left pleural effusion with radiological signs of pneumonia (Figure 1).
Respiratory acidosis - evidenced by arterial blood gas test (pH 7.27, pCO2 59 mmHg, pO2 55 mmHg) - and the severity of clinical presentation suggested the Emergency Physician start a respiratory treatment with Non-Invasive Ventilation. Lab tests showed leukocytosis (16.4 x10³/ul) and an elevated value of C-Reactive Protein (CRP) of 8 mg/dL.

After an initial clinical improvement, the patient suffered a vomiting episode in the Emergency Department and started suddenly getting hypotensive, and tachycardic and became unconscious. The clinical evaluation evidenced neck emphysema and the absence of respiratory sound from the left hemithorax; the ultrasound evaluation evidenced missing left pleural sliding. After our surgical survey, we decided on an anterior chest tube placement, and free air with gastric-like liquid was drained from the pleural cavity. Then, we decided on additional imaging, performing an oral-contrast Computed Tomography (CT-Scan): it showed a tear in the left thoracic distal thoracic part of the esophagus, with an important gastric distension (Figure 2).

The patient was immediately admitted to the Operating Room (OR) after antibiotics and antifungal prophylactic treatment were started: a left thoracotomy was performed by a thoracic surgeon and a 3-cm-length esophageal perforation was confirmed. Despite local perforation and contamination, esophageal mucosa looked well vascularized and therefore surgeon decided on a thoracic and mediastinal cavity toilett and a primary repair of the laceration.

The patient was admitted to Intensive Care Unit (ICU) for post-operative treatment. On the fifth Post-Operative Day (POD), an oral-contrast CT-Scan was performed: it revealed a good surgical resolution of the perforation without pleural effusion and the absence of any leakage from the esophageal suture (Figure 3).

However, concomitant pneumonia and consequent septic shock led the patient to Multi-Organ-Failure (MOF) and death on the 12th POD.

Discussion

NIV has become a common treatment for acute and chronic respiratory failure; in comparison with invasive mechanical ventilation, Non-Invasive Ventilation reduces procedural complications and mortality. Although generally well tolerated and safe, NIV has been associated with some adverse effects: patients should be carefully considered before starting treatment to reduce protentional severe complications. According to M. Carron’s review,4 aerophagia and gastric insufflation occur in 5% to 30–40%. During NIV, air can be inflated into the stomach and gastric distension compresses the lungs through the diaphragm: consequently, reduction in pulmonary compliance might require higher ventilation pressure. Therefore, gastric insufflation facilitates vomiting and aspiration: barotrauma with gastric and esophageal perforations has been described.

First described by Dr. Herman Boerhaave in 1724, Boerhaave’s syndrome is an uncommon life-threatening disease still challenging in diagnosis and treatment.5 It is determined by barotrauma related

Figure 1. Chest X-Ray after ER admission.

Figure 2. CT-Scan after oral contrast.

Figure 3. Post-Operative Chest X-Ray demonstrating esophageal leakage surgical resolution.
to a rise in esophageal pressure — usually by forceful emesis — and this condition has high mortality and morbidity if not promptly detected and treated. Boerhaave’s syndrome classic presentation is Mackler’s triad: vomiting, followed by chest pain and subcutaneous emphysema due to esophageal rupture. Mortality exceeds 90% if not treated and 40% even with appropriate surgical treatment, primarily for septic complications after mediastinal and pleural contamination.

The laceration is a left postero-lateral longitudinal and transmural tear in 90% of the cases, involving the distal part of the thoracic esophagus: mucosal injury in this region typically is longer and extends beyond the muscular layer. Mediastinum and one or both pleural cavities could be contaminated by gastric material and it is potentially a source of critical septic complications.

Treatment of Boerhaave’s syndrome can be divided into three possible approaches, looking at the patient’s general condition, esophageal damage, and timing for diagnosis: conservative, using antibiotics medications and percutaneous drainage, endoscopic, with esophageal stents, and surgical. The surgical standard technique is based on a left thoracotomy with firstly a mediastinal/pleural toilette and then a primary esophageal repair. The suture could be or not be followed by fundic reinforcement.6

Early diagnosis and appropriate treatment are fundamental, with intravenous antibiotics as soon as possible. The chest X-Ray with oral contrast is a classic diagnostic tool, but actually, we prefer torso oral-contrast CT-Scan and endoscopic imaging before surgery. In addition, lung ultrasound (US) has emerged recently as a simple non-invasive bedside procedure with better sensitivity and specificity7 than chest X-Ray for pleural effusion and pneumothorax. This imaging technique is important not only for the diagnosis of lung, pleural, or mediastinal disease but also as a follow-up in patients with chest tube placement and prior thoracic surgery.8

Our case report, to our acknowledge, is a presentation of a very uncommon condition and only two other cases of Boerhaave’s syndrome have been published until now. The first one was a 68-year-old female presented with a clinical picture of community-acquired pneumonia and exacerbation of asthma: after a first non-invasive and then invasive ventilation treatment, a tension pneumothorax developed, and an emergency decompression of the chest revealed gastric contents in the left hemithorax.2 In the second case, the authors describe a case of a 50-year-old man presented with atypical Boerhaave’s syndrome, in which the diagnosis was made several days following presentation in the ED for dyspnea and treatment with NIV, by observing a large pleural effusion at chest X-Ray. In the first clinical case, the patient was treated with the same operative approach as we did; in the second case, due to a hemodynamically stable condition, the physicians performed non-surgical management with endoscopic and medical treatments.

Conclusions

Surgeons should be aware of complications needing surgery or some medical treatments, especially when performed in an emergency department setting. NIV may cause gastric distention and vomiting: emesis against a closed cricopharyngeus may increase esophageal pressure causing rare lacerations. Early diagnosis and appropriate multidisciplinary (radiologic-endoscopic-surgical) treatment are key points for the patient’s prognosis in Boerhaave’s Syndrome.

References

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