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Esophagopleural Fistula and Empyema Secondary to Esophageal Rupture Resulting From Ligation of Esophageal Varices: A Case Report

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ABSTRACT

The treatment of esophageal varices usually requires invasive procedures for the administration of sclerotherapy and ligation. Among the main related complications are pleuropulmonary location, esophageal fistula toward the pleura, and the development of pleural infection (empyema). In rare cases, the condition is caused by esophageal perforation.[10,12] We present here a report on a patient with left esophagopleural fistula and empyema as complications of sclerotherapy and the ligation of esophageal varices.

Keywords: esophagopleural fistula, esophageal perforation, empyema, upper endoscopy

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INTRODUCTION

Esophagopleural fistula is a rare complication caused by esophageal rupture that can result from transmural disruption of the esophagus. The most common cause of esophageal rupture is Boerhaave syndrome, being observed in up to 15% of cases, common in the lower left border, and usually associated with endoscopic procedures, especially esophageal dilation, ligation of esophageal varices, and sclerotherapy. Other, rarer causes include surgical and external trauma, malignancy, foreign bodies, and the ingestion of caustics. The mortality range is 10-20%, with timely treatment being the strongest predictor of survival.[10] Most cases (54%) of complicated pleural infection or empyema, classified as exudate according to Light's criteria, are caused by pneumonia. A pH of less than 7.2 indicates the need for a drainage tube in patients with effusion, especially in the context of high levels of lactate dehydrogenase (LDH) and glucose less than 60 mg/dL. Esophageal perforation is found in patients with a history of esophageal tumor or reflux, chest pain, and fever. The presence of food particles may also suggest esophageal perforation. Routine testing should include protein, LDH, culture, pleural fluid cytology, and pH. Characteristically, when thoracentesis is unsuccessful, there are markers in the pleural fluid, such as amylase, that point to or confirm the diagnosis of esophageal perforation.[1,7]

CLINICAL CASE

This case report focuses on a married 63-year-old male patient working as a janitor, originally a resident of Zacatecas, without significant family history but with a history of chronic tobacco consumption (20 packs/year). The patient had suffered illness associated with alcohol consumption from the age of 20 and been diagnosed with liver disease, and he had been in treatment for chronic alcohol consumption for 3 years, receiving 20 mg propranolol every 12 hours. His history also included previous hospitalization for WHO grade 4 anemia secondary to upper gastrointestinal bleeding with ligation of esophageal varices diagnosed in December 2022, a report of esophageal varices grade III based on endoscopy, and transfusion of blood products without apparent complications.

The illness described in this report began one day after the patient's discharge from previous hospitalization, presenting with coughing fits productive of yellowish expectoration, dyspnea, fever, and diaphoresis. After being treated by a private doctor with unspecified doses of amikacin and amoxicillin and experiencing no improvement in his condition, the patient went to the emergency room. His vital signs on admission were BP 112/72 mmHg, HR 89 bpm, RR 26 rpm, temperature 36.8°C, and oxygen saturation of 80% on room air. A physical examination showed decreased amplexion and amplexation movements in both hemithoraxes but predominantly in the left, decreased vesicular murmur in the ipsilateral hemithorax, decreased voice transmission and dullness to percussion throughout the left hemithorax, and massive pleural effusion syndrome. Accordingly, laboratory studies (Table 1) and a simple chest tomography was requested.

Table 1. Laboratories at admission

Blood cytometry		Clinical chemistry	
Hemoglobin	10.4 g/dL	Total proteins	7.1 g/dL
Hematocrit	33.2%	Albumin	3.1g/dL
VCM	73 femtoliters	Glucose	202 mg/dL
HCM	22.9 pg/cell	Urea	62 mg/dL
Platelets	461 thousand/mm3	BUN	29.3 mg/dL
Leukocytes	24.6 mil/uL	Creatinine	1.0 mg/dL
Neutrophils	22.3 mil/uL (91.1%)	Chlorine	99 mmol/L
Coagulogram		Potassium	5.0 mmol/L
T.P.	17 sec	Sodium	130 mmol/L
INR	1.31	DHL	219 u/L
TPT	24.2 sec	PCR	208 mg/L

Simple tomography (Figure 1) of the chest in coronal section showed air in the distal third of the esophagus, mediastinum, and left pleural cavity, a massive left pleural effusion in the axial section of the lung window with air density in the esophageal tract extending from the distal third to the pleural cavity, and an air-fluid level that causes pulmonary atelectasis.

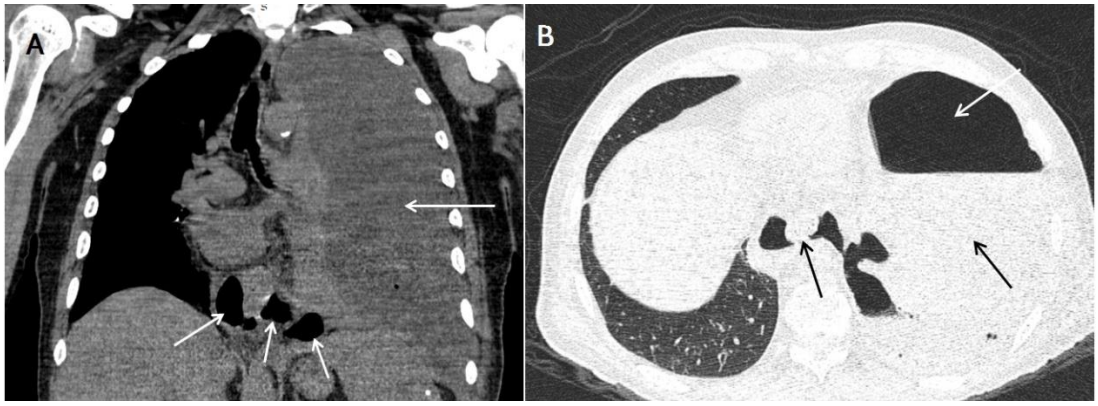

Figure 1. Simple tomography of the chest: (A) coronal projection in the mediastinal window showing the presence of air in the distal third of the esophagus, pneumomediastinum, and left pneumothorax; (B) axial projection in pulmonary window showing air-fluid level with left pulmonary atelectasis.

A left endopleural probe was positioned to manage the massive pleural effusion and sample the pleural fluid for analysis (Table 2). The patient met Light's criteria for exudate, and, given the increase in amylase in the pleural fluid, esophageal perforation was suspected. An esophagogastroduodenal series was performed with oral barium contrast that showed transfer of contrast to the pleura (Figure 2). Upper endoscopy revealed engorged varicose cords of purplish color in the esophagus with a tortuous course extending to the lower third (Figure 3).

Table 2. Pleural fluid study	
Aspect	Milky
Density	1.01
pH	7.0
Glucose	290 mg/dL
Proteins	1.42 g/dL
DHL	7,793 u/L
Cholesterol	12.5 mg/dL
Triglycerides	263 mg/dL
Amylase	9,361 u/L

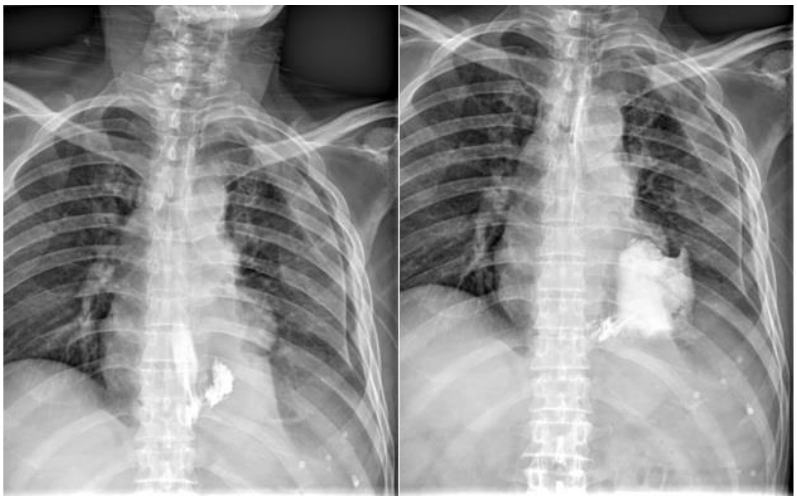

Figure 2. Esophagogastroduodenal series with oral contrast

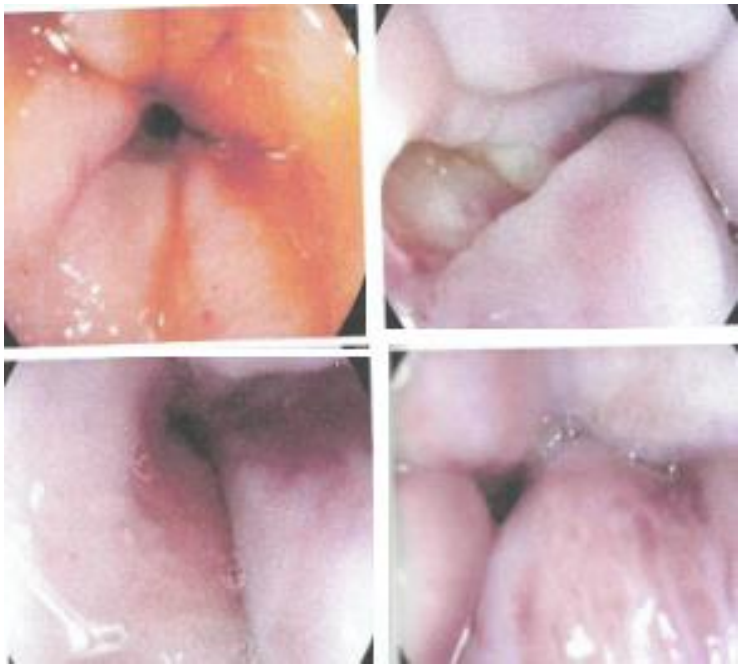

Figure 3. Panendoscopy: at 35 cm from the dental arch, an 8 mm solution of continuity was observed with perilesional fibrin along with an engorged venous cord adjacent to the lesion and erosion in the mucosa.

CONCLUSION

Some 60 causes of pleural effusion have been documented.[1] An etiological diagnosis of exudate (inflammatory origin) can be made using Light's criteria.[2] The indications for drainage are glucose < 60 mg/dL or DHL > 1,000 U/L. The typical clinical presentation of empyema involves the presence of cough, dyspnea, expectoration, fever, chest pain, and clinical findings of pleural effusion. The diagnosis is made based on puncture and study of pleural fluid as well as ultrasound, more specifically, contrast-enhanced tomography. The enhancement and separation of pleurae (split pleura) are also characteristic.[3,4] Further, elevated levels of amylase in the pleural fluid are suggestive of esophageal perforation but can also indicate pancreatitis, malignancy, or tuberculosis.[7] Esophageal perforation is iatrogenic in 60% of cases, in particular, associated with endoscopic procedures, especially esophageal dilation, ligation of varicose veins, and sclerotherapy. This condition is an important cause of the contamination of the surrounding spaces and sepsis. The indirect indications in contrast-enhanced tomography include the presence of pleural effusion, pneumomediastinum, subcutaneous emphysema, hydrothorax, pneumothorax, and atelectasis. The mainstay of treatment following a diagnosis of esophageal perforation with extensive pleural or mediastinal contamination is initial surgery (primary closure), with 1-3% of patients requiring surgery because of complications (perforation, mediastinitis, empyema, fistula, or hemorrhage).[10]

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