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Case report

Spontaneous mediastinitis with multiple esophageal abscess in the esophagus

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A B S T R A C T

Acute mediastinitis (AM) is a rare and deadly disease without appropriate treatment. It is usually due to invasive medical procedures, being uncommon the spontaneous onset. We report a case of 49-year-old patient whose starting symptoms were dyspnea and oppressive epigastric pain.

1. Introduction

Mediastinitis is a rare and severe disease that is commonly misdiagnosed and require physicians to discard esophagus perforation.

2. Case report

We report the case of 49-year-old patient with medical history of emphysema, chronic liver disease due both to hepatitis C virus (HCV) and alcohol abuse (Child-Pugh, 6 points, Class A, with no evidence of portal hypertension), and acute severe alcohol related pancreatitis in 2009 and 2010. The patient initially presented to the Emergency Department (ED) with a 3 days-history of oppressive epigastric pain radiating into the left and right abdominal upper quarters and dyspnea. He also complaint of diarrhea for 2 weeks, after an increased alcohol consumption, but repeatedly denied any nausea or vomiting. Physical examination showed nothing except tachycardia (112 beats per minute), with no fever, and slight pain with the deep palpation at the epigastrium. Laboratory results showed increased inflammatory response, with leukocytosis (15,900/mm3) and neutrophilia (75.7%), and elevated C-reactive protein (6.93mg/dl). Transaminases and bilirubin were normal, except elevated GGT (124U/l). Pancreatic enzymes were slightly raised (amylase 239U/l and lipase 247U/l). Because of the abdominal symptoms, and the blood test results, Ciprofloxacin 400mg intravenous (iv) every 12 hours was empirically prescribed in the ED. The second day after hospital ward admission the patient got clinically worse, and suffered presyncope with hypotension and accused tachycardia and tachypnea, with no other symptoms, so a thoracic computerised tomography (CT) scan was done (Fig. 1) showing multiple esophageal abscess which surround the esophagus from the uppermost part of it to the gastroesophageal junction (about 20cm). Although the esophageal wall was poorly delimited, it seemed to be thickened on its middle third, and a collection of pus could be seen in the subserosa, leaving the mucosal membrane apparently unaffected. No gas bubbles could be seen. The abdominal CT revealed a pancreatic pseudocyst emanating from the tail of the gland suggestive of chronic pathology. After that, antibiotic treatment was changed into Meropenem 1g IV q8h, and he was referred to the thoracic surgery department. A right posterolateral thoracotomy was performed over the fifth intercostal space, finding localized collection of purulent material along all the posterior mediastinum which was drained. Surgical esophageal exploration was made, and methylene blue was instilled into the esophagus in an attempt to localize the injury, with no evidence of perforation. Blood cultures and purulent material cultures obtained from the pleura and mediastinum during the surgery were all negative, having received just two days of empirical antibiotic treatment with Ciprofloxacin and one day with Meropenem. Before the chest drainage tubes were removed, we prove the absence of any leakage after methylene blue swallow. During his hospital stay study was completed with an esophagogram, showing an esophageal wall with normal thickness and motility, with no evidence perforation (Fig. 2). Likewise, serology for cytomegalovirus (CMV) was negative, excluding it as a cause of esophageal mucosa damage. The following days the patient showed decrease of acute phase reactants and good clinical evolution including appropriate oral tolerance so he was discharged from the hospital 10 days after the surgery. Antibiotic treatment was completed on an outpatient basis, with Ertapenem 1g IV q24h for 7 days, and sequentially Cefixime 300mg q12h and Metronidazole 500mg q8h for 7 days more, completing one month of antibiotic therapy. Likewise a gastroscopy was made 3 weeks later with no sign of perforation in the

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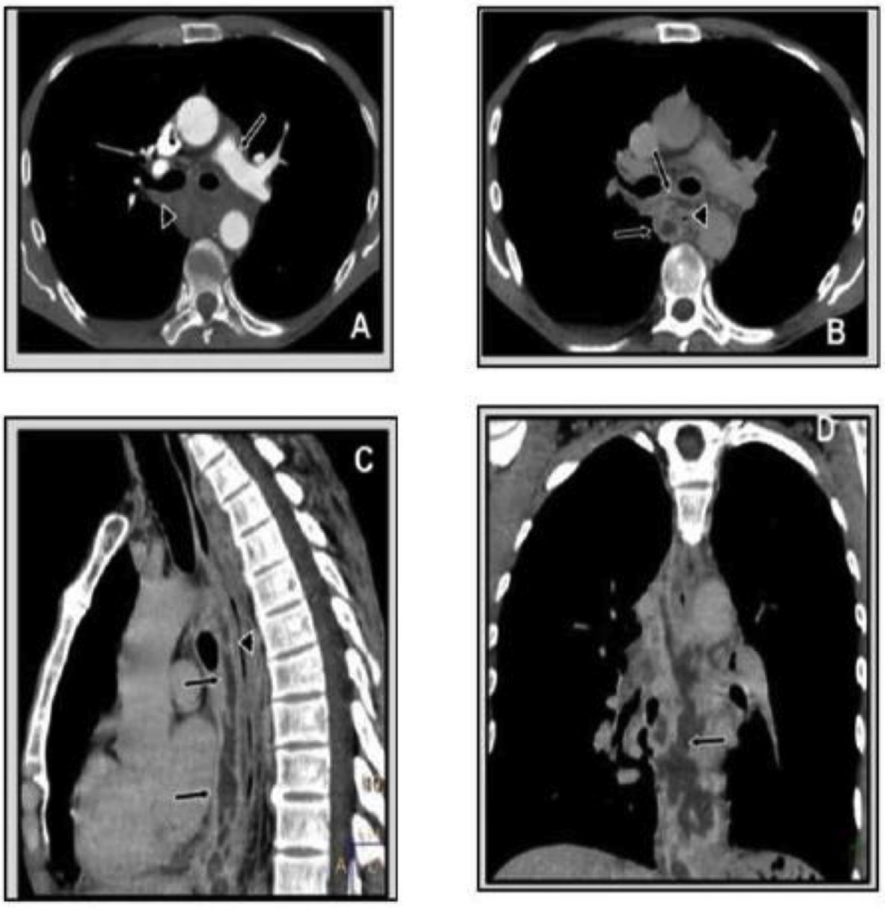

Fig. 1. Multiple oesophageal abscess which surround the oesophagus from the uppermost part of it to the gastroesophageal junction (about 20cm). Although the oesophageal wall was poorly delimited, it seemed to be thickened, and a collection of pus can be seen in the subserosa, leaving the mucosal membrane apparently unaffected. No gas bubbles can be seen. Several little lymph nodes in the upper mediastinum.

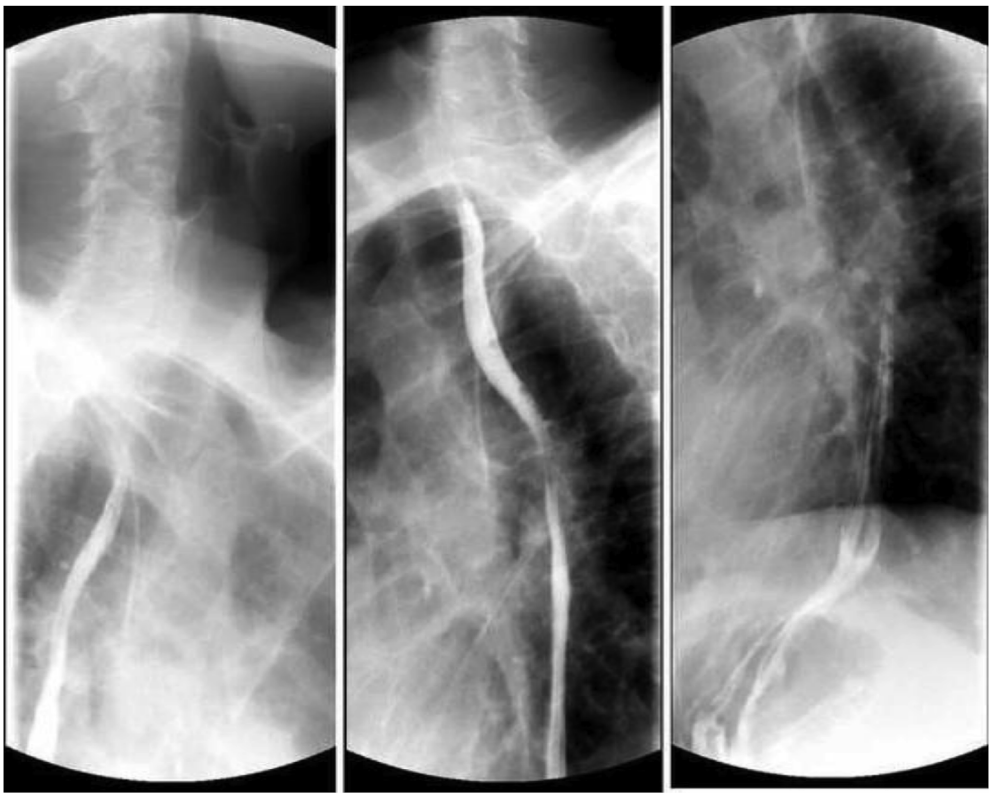

Fig. 2. Image of esophagogram (from left to right, cervical, thoracic and abdominal portion), showing a oesophageal wall with normal thickness and motility, with no evidence perforation.

esophageal exploration, and being our patient finally diagnosed with spontaneous mediastinitis.

3. Comment

Esophageal perforation is a rare condition (3.1 per 1,000,000 per year) that can be caused by direct inoculation, lymphatic or hematogenous dissemination, or spread of local infection. AM is mainly produced by iatrogenic effect as a consequence of thoracic and esophageal surgery [4], and also after endoscopic studies or orotracheal intubation [2,3]. Sometimes, AM appears as a result of rupture of tracheobronchial structure or esophagus perforation, which may produce the dissemination of anaerobic or mixed aerobic-anaerobic bacterial flora into the mediastinum [2]. When it is not associated to cardiac or thoracic surgery, is commonly misdiagnosed and can be fatal if not promptly treated [1]. In this case, without any evidence in the patient's medical history, of esophageal perforation risk factors, an angio-CT was made because of the high suspicious of pulmonary embolism. After the diagnosis and a correct treatment based on surgery and broad spectrum antibiotic, all medical studies were made in order to look for esophageal perforation, including discarding CMV esophagitis [2–5]. Considering that all the results get from the analysis were negative, we could classify this case as a spontaneous mediastinitis. Being the mediastinitis a rare cause of chest pain [6–8], the spontaneous mediastinitis is even more exceptional, and that is what makes our case significant. Another remarkable fact, is the clinical presentation of this case [6–8], where the initial symptoms were epigastric pain and dyspnea, which make the diagnosis of this illness ever more complicated. Therefore, this diagnosis could be taken into account when other causes of chest pain or epigastric pain were reasonable discarded. Boerhaave Syndrome is the most usual cause of spontaneous esophageal perforation, so this diagnosis should be considered in case of a sudden increased of intraesophageal pressured, such as vomiting, sneezing or weight lifting.

In our patient, although he had alcoholism as an esophageal perforation risk factor, medical history did not suggest preceding swallow of foreign bodies, neither vomiting or previous episodes of increased esophageal pressure, which could explain a Boerhaave Syndrome. Even tough, although any clinical test made did not prove esophageal injuries, the finding of esophageal wall thickening in the CT (Fig. 1C), could suggest a spontaneous perforation of it. Another remarkable issue, is that, in the case of presume a Boerhaave Syndrome, the vast majority of them occur in the lower third of the esophagus. In our case, the esophagus thickening outlined in the CT was located on the middle third, which would be an uncommon location. In this kind of patients, an exhaustive study should be made in order to exclude esophageal perforation based on CT, esophagogram and upper endoscopy of the esophagus.

In conclusion, non-iatrogenic AM is a life threatening pathology which requires clinical suspicion when presented as chest pain with no fever and requires an early medical-surgical approach.

Disclosures

The authors declare that the have no conflict of interest.

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