

PURULENT PERICARDITIS AS THE FIRST MANIFESTATION OF ESOPHAGEAL CARCINOMA

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Abstract

Introduction: Purulent pericarditis secondary to esophago-pericardial fistula is a serious complication that has been previously reported in patients with esophageal cancer treated with radio/chemotherapy and esophageal stenting. However, the presence of esophago-pericardial fistula as the first manifestation of advanced carcinoma of the esophagus is exceedingly infrequent. We report the case of a 61-year-old male who presented with sepsis, cardiac tamponade and septic shock who was found to have an esophago-pericardial fistula secondary to squamous carcinoma of the esophagus. Emergency pericardiocentesis was performed with subsequent hemodynamic improvement. The drained pericardial fluid was purulent in nature and cultures were positive for *Streptococcus anginosus*. A CT scan followed by upper gastrointestinal endoscopy with tissue biopsy confirmed the diagnosis of squamous cell carcinoma of the esophagus. A self-expanding covered stent was endoscopically placed to exclude the fistula and restore the esophageal lumen. In this report, we discuss some aspects related to the diagnosis and management of this serious clinical entity.

Keywords: purulent pericarditis, esophagopericardial fistula, esophageal carcinoma.

INTRODUCTION

Advanced esophageal carcinoma may be associated with penetrating esophageal ulcers and/or perforation with subsequent formation of fistulas communicating with the airways, the mediastinum, or the pericardial cavity. More than half of fistulas affect the trachea and main bronchi. . Most cases of purulent pericarditis secondary to perforated esophageal cancer have been reported in patients who were previously treated with radiotherapy, chemotherapy, or esophageal stenting¹⁻³. On the contrary, the formation of an esophago-pericardial fistula as the first manifestation of esophageal cancer is exceptionally rare³⁻⁵. We present the case of a male patient who presented with purulent pericarditis and cardiac tamponade due to an esophago-pericardial fistula caused by an esophageal squamous cell carcinoma.

CLINICAL CASE

A 61-year-old male patient with a past medical history of smoking, obesity, and arterial hypertension presented with productive cough, vomiting, low-grade fever, dysphagia, odynophagia, anorexia, and slowly progressive weight loss over several weeks. He was initially diagnosed with lower respiratory tract infection and received a course of outpatient antibiotics and bronchodilators. However, a week later the patient presented with shivering and dyspnea with significant clinical deterioration. Upon admission, he was diaphoretic and tachypneic with a tympanic temperature of 38.5°C and blood pressure of 85/50 mmHg. Initial chest x-ray did not show acute pulmonary pathology (figure 1).

His arterial blood gas analysis showed alkalemia (pH 7.5), normoxemia (PO₂ 74.8 mmHg), hypocapnia (PCO₂ 31.1 mmHg),



Figure 1

Chest x-ray in posteroanterior projection. There is slight enlargement of the cardiac silhouette and absence of pleuropulmonary lesions.

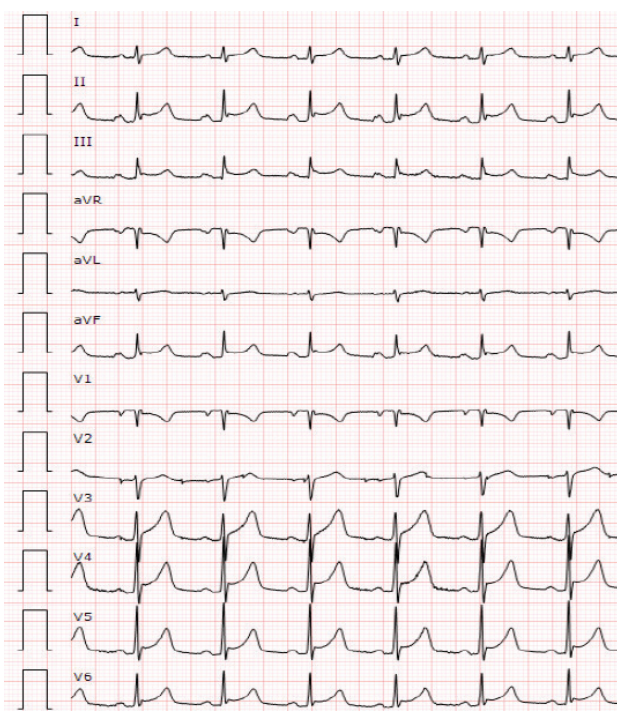


Figure 2

Electrocardiogram. Diffuse concave ST segment elevations and diminished voltages are present.

slightly diminished O₂ saturation (93%), and hiperlactacemia (2.8 mmol/L, range 0.0-1.8). Blood tests revealed leukocytosis (20.47x10³/μL) with neutrophilia (88.4%), anemia (hemoglobin 8.9 g/dL), thrombocytosis (529x10³/μL), elevation of transaminases AST/GOT (827 IU/L, range 15-37), ALT/GPT (616 IU/L, range 16-62), elevated serum creatinine (2.01 mg/dL, range 0.4-1.3), and urea (70 mg/dL, range 12-44), elevated C reactive protein (33.6 mg/dL, range <0.3), and procalcitonin (7.83 ng/mL, normal value <0.5), with normal ultra sensitive troponin I and

total bilirubin. His electrocardiogram showed sinus tachycardia, with low QRS voltage, and slight concave elevation of the ST segment in anterior and inferior leads (figure 2).

The patient developed septic shock with hypotension, oliguria, and acute renal failure. Aggressive fluid resuscitation was administered and vasopressor support was initiated. Transthoracic echocardiography was performed, which demonstrated preserved biventricular function and moderate pericardial effusion with collapse of the right heart chambers, inferior vena cava plethora and respiratory changes of the mitral E velocity >25% and tricuspid E velocity >50%, suggestive of cardiac tamponade.

Ultrasound-guided pericardiocentesis was performed through via the apical approach, draining about 600 ml of purulent fluid with immediate hemodynamic improvement. The microbiological study of the pericardial fluid showed the presence of gram-positive polymorphonuclear cocci that grew positive for *Streptococcus anginosus*.

Chest CT scan demonstrated thickening of the esophageal wall at the level of the middle third and inflammatory changes in the periesophageal and subcarinal spaces with gas bubbles and distension of the proximal esophagus (figure 3). Pericardial drainage was maintained for 5 days, and the antibiotic treatment was discontinued after 15 days as advised by the Infection Diseases Team.

Given the high suspicion of esophageal carcinoma that may have fistulized into the pericardial cavity, an upper gastrointestinal endoscopy was performed, demonstrating an ulcerative lesion at the level of the middle third of the esophagus with obliteration of the esophageal lumen. A tissue biopsy was obtained, and a covered self-expanding esophageal stent was implanted (Figure 4). The histopathological study confirmed the diagnosis of squamous cell carcinoma. The patient continued to improve clinically, and he was discharged 16 days after admission. The patient is currently undergoing cancer staging studies for further management of his neoplastic process.

DISCUSSION

The presence of purulent pericarditis and cardiac tamponade as the first manifestation of esophageal carcinoma is exceptionally rare³⁻⁵. These are typically described in men with advanced squamous cell carcinoma, although cases associated with small cell carcinoma have been also described⁶. The neoplastic lesion is usually located at the lower third of the esophagus where the anterior wall of the esophagus is in proximity with the pericardial sac³⁻⁵. The esophago-pericardial fistula can be complicated with the development of purulent pericarditis that very frequently causes cardiac tamponade¹⁻³.

Patients usually present with symptoms of acute pericarditis such as fever, epigastric pain, substernal pain, and dyspnea. Symptoms of esophageal cancer such as dysphagia, odynophagia, cough, and weight loss have also been reported³⁻⁶. The clinical diagnosis is usually established after the occurrence of symptoms of cardiac tamponade, which can evolve very rapidly^{4,5}.

In terms of diagnostic workup, blood tests consistently

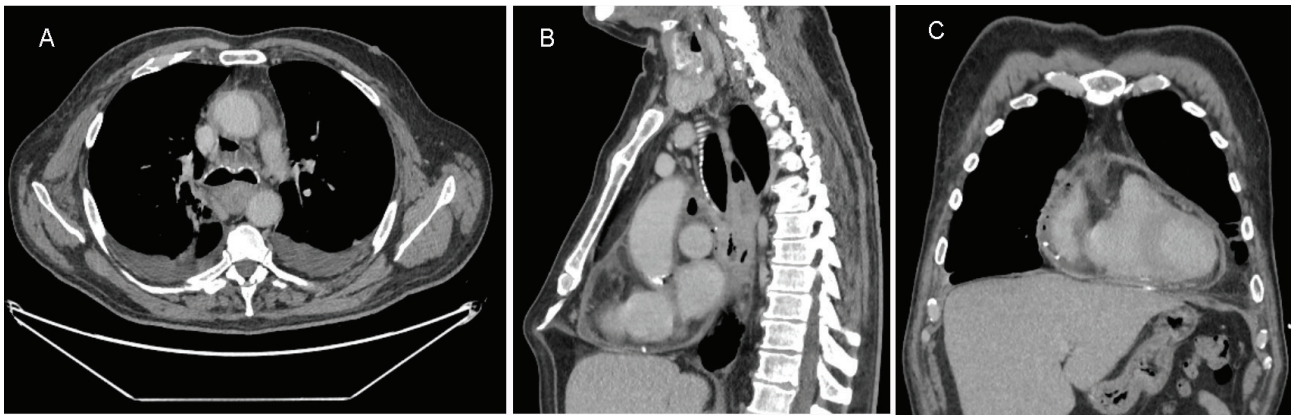


Figure 3

Computed tomography images in the transverse (A), sagittal (B) and coronal (C) planes. Wall thickening of the esophagus as well as periesophageal inflammatory infiltrate and gas-filled spaces in the mediastinum and pericardial cavity are observed.

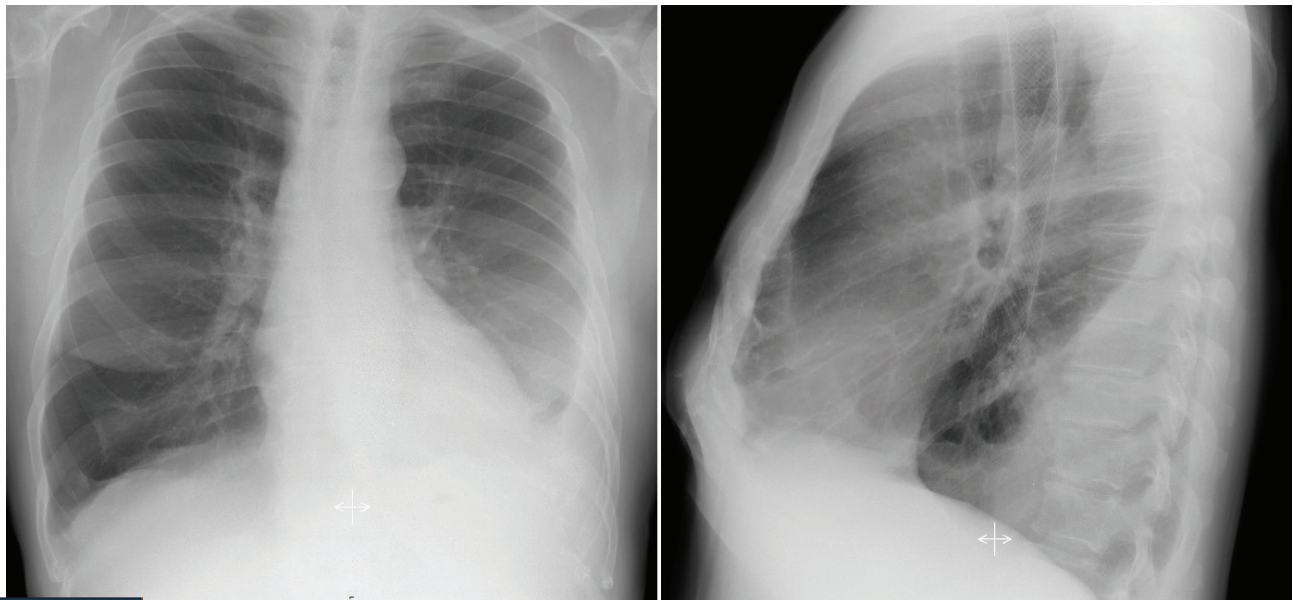


Figure 4

Plain chest x-ray after esophageal stent placement

show leukocytosis with neutrophilia and elevated C-reactive protein⁴⁻⁶ while the 12-lead electrocardiogram typically shows decreased amplitude of the QRS complex and concave elevation of the ST segment in anterior and inferior leads³.

Occasionally, chest x-ray may show hydro-pneumopericardium which is highly suggestive of this condition². Findings on CT scan of the chest may include pericardial effusion, the presence of gas outside the esophageal lumen, and occasionally, a frank communication between the esophagus and the pericardial cavity³⁻⁶.

Cardiac tamponade is usually confirmed by echocardiography where pericardial effusion is observed,

generally moderate in volume, with significant compression of the right cardiac chambers and characteristic echo Doppler parameters³⁻⁶. The fluid drained in pericardiocentesis is usually purulent in nature with a predominance of neutrophils without neoplastic cells³⁻⁷. The microorganisms observed are usually typical of the oral flora such as *Streptococcus anginosus*³, coagulase-negative *Staphylococci*⁸, *Streptococcus constellatus*⁹, and *Candida* species¹⁰. However, more than one species can frequently be isolated⁷.

The prognosis of patients with esophago-pericardial fistula secondary to malignancy is generally poor and a significant proportion of these patients may die within days or

weeks^{2,3,8}. Treatment is aimed primarily at relieving symptoms, preventing recurrence, improving quality of life and, in selected cases, treating the neoplastic process³.

Purulent pericarditis and cardiac tamponade require urgent management by pericardiocentesis, subxyphoid drainage, or thoracotomy^{3,4,5,8}. The pericardial sac can be washed with saline solution, or alternatively, with antibiotics^{3,7}.

The implantation of an esophageal stent allows the exclusion of the fistula and ensures the patency of the esophageal lumen, although it is not always possible due to the fragility of the malignant tissues^{3,4}. Despite some cases of long-term survival described after combined surgery, chemotherapy, and radiotherapy^{7,6}, most patients with poor prognosis may only receive palliative interventions^{3,8}.

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