Received: 16 February 2020 | Revised: 26 June 2020 | Accepted: 6 July 2020

DOI: 10.1002/emp2.12202

JACEP OPEN
JOURNAL OF THE AMERICAN COLLEGE OF EMERGENCY PHYSICIANS OPEN
A GLOBAL JOURNAL OF EMERGENCY MEDICINE
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CASE REPORT

Cardiology

Idiopathic hemodynamically unstable polymicrobial purulent pericarditis: a rare case presentation

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Funding and support: By *JACEP Open* policy, all authors are required to disclose any and all commercial, financial, and other relationships in any way related to the subject of this article as per ICMJE conflict of interest guidelines (see www.icmje.org). The authors have stated that no such relationships exist.

Abstract

Purulent pericarditis is an extremely rare disease accounting for <1% of pericarditis cases. Purulent pericarditis with hemodyamically unstable tamponade if untreated is fatal. Furthermore, although idiopathic polymicrobial disease is documented, a combination *Haemophilus parainfluenzae*, *Prevotella buccae*, and *Citrobacter freundii* have not been found in the literature by the authors. What follows is a case of a 58-year-old male who presented to the emergency department (ED) with these features and underwent emergent bedside pericardiocentesis and a brief review of current pericardiocentesis techniques in the emergency department.

KEYWORDS
emergency medicine, emergency department, pericardiocentesis, tamponade, sepsis, pericarditis, pericardial effusion

1 | INTRODUCTION

Pericarditis has many causes, primarily extension from other thoracic structures, oral infections, hematogenous spread, in the case of Dressler's syndrome – postischemic heart disease, and viruses.[1-3,10] Often the origin of infection is unknown. Current mortality is unclear. However, mortality is extremely high; 40% in an 86-year series done in 1977 and 77% in a 15-year series done in 1974.[3,4] Acute hemodynamically unstable cardiac tamponade secondary to purulent pericarditis warrants emergent pericardiocentesis or cardiac window along with broad-spectrum antibiotics, fluid analysis, and cultures. In this case report, a 58–year-old male, transferred from an outside hospital, presented with idiopathic hemodynamically unstable cardiac tamponade, requiring emergent bedside pericardiocentesis.

2 | CASE REPORT

This is a 58-year-old male with a history of hypertension, smoking, and alcoholism who was transferred from an outside hospital by rotary wing for cardiac catheterization due to acute ST-segment elevation myocardial infarction. The outside-hospital clinician evaluated the patient for an acute ST-segment elevation myocardial infarction. There, the patient complained of abdominal pain, which led to obtaining a computed tomography (CT) scan of the abdomen and pelvis. The images included the inferior portion of the chest, and the report indicated that there was a pericardial effusion. Upon arrival, he stated that he had been feeling fatigued for 2 weeks and went to the ED because he wasn't feeling "right." He denied any vomiting, foreign travel, or history of tuberculosis exposure.

At the outlying facility, the patient's blood pressure was 63/46. He was started on intravenous fluids and dopamine. Laboratory workup was remarkable for a troponin of 0.170. We were unable to obtain a record of anticoagulation or antiplatelet administration. Upon arrival after transfer, the physical examination showed that he was tachycardic with a blood pressure of 111/61 on dopamine. The cardiovascular examination did not show jugular venous distension, muffled heart sounds, or pulsus paradoxus. Electrocardiogram did not show low voltages or electrical alternans. Upon further evaluation of the transferring facilities records, it was unclear why an emergent pericardiocentesis was not conducted at the outlying facility. However, we conducted a point-of-care cardiac ultrasound, which in addition to confirming the large pericardial effusion, kinetics consistent with tamponade

Supervising Editor: Nicholas Caputo, MD, MSc.

FIGURE 1 Purulent drainage from emergency department pericardiocentesis

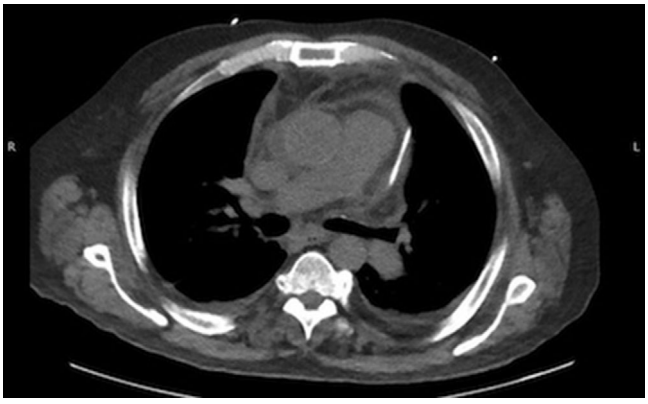

FIGURE 2 Computed tomography of the chest confirming drainage catheter placed in the pericardial space

physiology were also revealed. Subsequently, emergent pericardiocentsis was performed at the bedside by the emergency physician. The procedure was conducted with a classic subxiphoid approach. Purulent, non-bloody fluid came from the pericardial sac with a total of 1100 cc of output in the ED [Figure 1]. Bedside ultrasound and a CT scan of the chest were obtained immediately after the procedure in order to verify that the catheter was in the pericardial space [Figure 2]. The patient was started on vancomycin and piperacillin/tazobactam. The fluid was sent for analysis, which was positive for *Haemophilus parainfluenzae*, *Prevotella buccae*, and *Citrobacter freundii*.

The patient was subsequently admitted to the intensive care unit. Because of the patient's polymicrobial infection, it was suspected that the source of the infection was extension from the gastrointestinal system. Upper endoscopy did not find a fistulous tract; however, a small hiatal hernia was discovered. Blood cultures and nasopharyngeal swab for viral illnesses were negative. Antibiotics were continued from the ED and were tailored to culture sensitivity. The catheter was removed and the patient was discharged in good condition with outpatient ertapenem for 2 weeks. Follow-up with the patient in the infectious disease clinic showed that he had no complaints and final cultures were negative.

3 | DISCUSSION

The most common causes of purulent pericarditis are because of extension from other structures or from an underlying disease state where either the origin is unknown at the time of diagnosis or is not found.[2-4] In most extant literature, the majority of cases were caused by gram-positive infection due to secondary spread from pulmonary disease in children and young adults.[3,4] However, Rubin and Guze over 40 years ago identified a shift from gram-positive infection to disease caused by fungi and gram-negative rods in the elderly and chronically debilitated.[3] This is reflected in culture results from live tissue and autopsy literature showing positive growth for *Escherichia coli* and *Pseudomonas aeruginosa*.[2-4] A 2009 case report documented an instance of idiopathic cardiac tamponade caused by *Proteus mirabilis* and *Citrobacter diversus*.[5] The cause of pericardial effusions overall is usually associated with an underlying condition or chronic disease state or is idiopathic without any acute precipitating factor.[1,2,10] In our case, *Haemophilus influenzae*, *Prevotella buccae*, and *Citrobacter freundii*, anerobic gram negative bacteria, were isolated. These are commonly found in polymicrobial infections. The patient did not have any acute precipitating factors. He did, however, have an underlying risk factor in the form of alcoholism.

The emergency department management for acute hemodynamically unstable cardiac tamponade is pericardiocentesis. Common methods include landmark and ultrasound-guided approaches. A landmark or blind approach should be avoided unless the effusion is an immediate risk to life. [8] Ultrasound has become a standard of care adjunct when performing pericardiocentesis if available in the emergent setting. For decades it has been used to identify pericardial effusion with tamponade physiology and which approach may be the easiest and safest for the patient. There are 3 approaches that are conducted emergently at bedside: parasternal, apical, and subxiphoid. One can best identify the safest approach (without lung interference, closest to largest pocket of effusion, etc) utilizing bedside ultrasound. Several key features identified on bedside ultrasound include right atrial collapse, right ventricular collapse, plethoric inferior vena cava, and swinging heart.[8] The European Society of Cardiology has indicated guidance either with ultrasound or fluoroscopy is a must, although

they do not specify that ultrasound guidance must be continuous throughout the procedure.[8] Furthermore, there has been a trend toward real-time continuous guidance both with phased array and high-frequency linear transducers.[11,12] Ultrasound-guided pericardiocentesis has an overall complication rate of 4.7%.[6]Morbidity and mortality from the blind approach are 50% and 6%, respectively.[6,9] Fluoroscopy-guided pericardiocentesis is more commonly found in catheterization laboratories and operating rooms and may not be feasible in the emergent setting. At this time, there appear to be no explicit approach recommendations by US societies. In resource-poor settings, the blind approach is recommended over medical management, which includes fluids to increase preload and dobutamine to improve contractility.[7] Vasopressor support is temporary and controversial and has shown to improve hemodynamics only in animal models.[7]

After the acute intervention, it is important to continue treatment in the emergency department. This includes obtaining cultures for speciation and antibiotic sensitivity, starting broad-spectrum antibiotics, and beginning the initial testing for the underlying cause. In this case, because of the polymicrobial, gram-negative anaerobic isolates, it was initially thought that the source was from a gastrointestinal-pericardial sac fistula. The patient underwent esophagogastroduodenoscopy and no communication was found. Although the etiology is unknown, one reason, albeit unsatisfactory, could be bacteremia secondary to the patient's history of alcoholism. Current recommendations by the European Society of Cardiology include broad-spectrum antibiotics, and narrowing treatment once cultures result, a class 1 level C recommendation.[8,13]

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How to cite this article: Ased S, Payne AN, Yang C. Idiopathic hemodynamically unstable polymicrobial purulent pericarditis: a rare case presentation. *JACEP Open*. 2020;1:744–746. https://doi.org/10.1002/emp2.12202