

## A Case of Actinomycosis Presenting as Purulent Pericarditis with Cardiac Tamponade

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### INTRODUCTION

Purulent pericarditis (PP), a neutrophilic effusion in the pericardial space, is relatively uncommon and frequently identified only post-mortem.<sup>1</sup> When presented, prompt diagnosis and intervention is a necessity, as the mortality rate ranges from 20 - 30% and can be as high as 80% if left untreated.<sup>1,2</sup> It also may result in cardiac tamponade which is a rare yet life-threatening complication reported in patients of all ages and levels of immunocompetency.<sup>3</sup> The antibiotic era has changed the landscape of PP incidence, with pneumonia being the most common risk factor, along with cardiothoracic procedures, and immunocompromised states.<sup>4</sup> The most common organisms isolated from such infections are usually gram positive bacteria including *Staphylococcus* and *Streptococcus*. While other organisms are uncommon due to effective antibiotics and antifungals in the post-antibiotic era, their incidence may be increasing, likely from increased use of percutaneous procedures.<sup>5</sup> The following case highlighted *Actinomyces* as a cause of PP and cardiac tamponade in a patient initially misdiagnosed with sarcoidosis.

### CASE REPORT

A 22-year-old male presented to the emergency department (ED) with intermittent, non-specific shortness of breath. In the ED, his vitals and physical exam were unremarkable and overall, he appeared well. Laboratory studies, including basic chemistry and blood counts, were also unremarkable. No cultures were done. Initial x-ray showed enlarged hila and lymphadenopathy (Figure 1). Computed tomography (CT) of the chest revealed mediastinal lymphadenopathy, multiple solid and subsolid nodules, most notably in the right upper and middle lobes.

The patient was discharged from the ED with a referral to pulmonology for suspected lymphoma versus sarcoidosis given imaging findings. He underwent an endobronchial ultrasound with the biopsy of station 4R and 7 lymph nodes. Pathology revealed necrotizing granulomas with a negative stain for fungal or mycobacterial organisms. Further workup for *Histoplasma capsulatum* was negative. Given the unremarkable findings on physical exam and in the laboratory studies, the patient tentatively was diagnosed with sarcoidosis and started on prednisone.

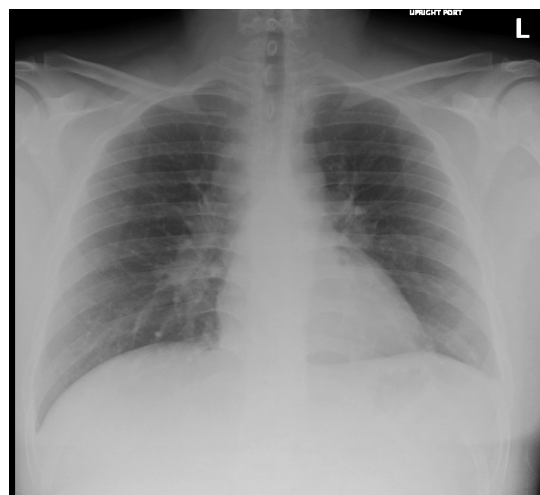


Figure 1. Chest x-ray showed enlarged hila and lymphadenopathy.

Approximately three weeks later, the patient presented to the ED with progressive, non-specific chest pain of two to three weeks duration. Vitals and physical exam were pertinent for tachycardia, and the patient appeared generally anxious. His electrocardiogram (EKG) showed diffuse ST segment elevation (Figure 2). Repeat imaging was unable to rule out pulmonary embolism definitively, but chest x-ray and CT were unchanged. The patient was diagnosed with acute pericarditis and his two-day hospital course was unremarkable. At this time, 2-D echocardiogram (echo), blood cultures, basic chemistries, and troponin levels were unremarkable. The patient's pain significantly improved with limited intervention, and he was given a prescription for colchicine 0.6 mg twice a day upon discharge.

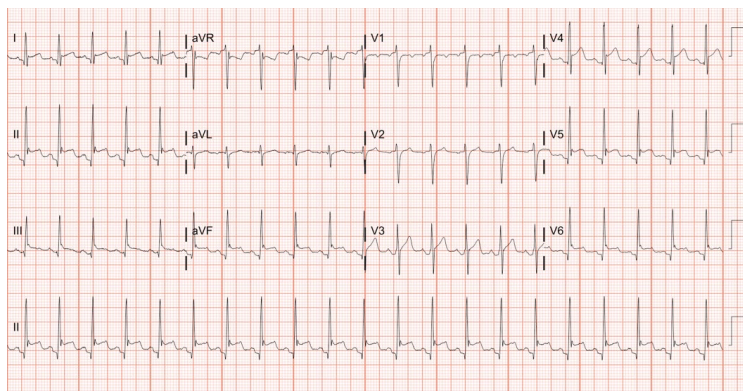


Figure 2. Electrocardiogram showed diffuse ST segment elevation.

Two days following this discharge, the patient presented to the ED with severe dyspnea, diaphoresis, and chest pain. Pertinent physical exam findings included distant heart sounds, shallow breathing, and obvious distress. Labs were pertinent for white blood cell count of  $45 \times 10^3/\mu\text{L}$ , troponin of 0.62 ng/ml, and overall metabolic acidosis. EKG again showed diffuse ST elevations, and bedside echo showed large pericardial effusion with suspected tamponade. He subsequently underwent subxiphoid pericardiectomy (pericardial window) with drainage of grossly purulent fluid with cultures collected and sent to the lab. Empiric antibiotics, vancomycin and piperacillin-tazobactam, were started.

Although blood cultures were not recollected on this admission, specimen cultures grew *Parvimonas micra*, *Prevotella* (undifferentiated), *Actinomyces odontolyticus*, and *Peptostreptococcus* (undifferentiated). Targeted therapy with ampicillin-sulbactam was started. Echo obtained four days following admission showed near complete resolution of pericardial effusion, but had residual fluid with fibrous appearance. Given organisms cultured, further investigation into the oral cavity was done. Panorex imaging of the mandible showed one dental caries, and physical exam of the oral cavity was unremarkable. He was discharged home with plan to receive four to six weeks of intravenous antibiotics before changing to oral maintenance for several months, pending clinical picture.

## DISCUSSION

The most common organism isolated in PP infections is *Staphylococcus aureus*, accounting for approximately 31% of cases, followed by *Streptococcus pneumoniae*.<sup>1</sup> Fungal infections have increased in incidence recently. Anaerobic causes of PP are uncommon, although increasing;<sup>5</sup> multi-organism anaerobic infections are rarer.<sup>6</sup> *Actinomyces* isolated from PP is rare, fastidious, and multi-factorial in nature. Patients can have subclinical presentations that delay diagnosis. *Actinomyces* as a cause of PP dates as far back as 1950.<sup>6</sup> A majority of cases are related to either significant cardiothoracic surgery, periodontal infection, alcohol abuse, or a previous diagnosis of pneumonia or disseminated *Actinomyces* infection. The four organisms isolated in this case were far higher than the average of 1.4 per specimen.<sup>7</sup> While it is suspected that *Actinomyces* is the predominate infectious organism present, the other organisms may increase virulence or provide other unknown contributions.<sup>8</sup> This requires further investigation.

The pathogenesis of PP is well-documented and includes hematogenous spread, intrathoracic trauma, subdiaphragmatic spread, or from an intrathoracic site such as pneumonia or suppurative mediastinal lymphadenitis.<sup>6</sup> *Actinomyces* causing PP is usually secondary to intrathoracic spread.<sup>9</sup> In this patient, it was suspected that his initial symptoms were from pulmonary actinomycosis and the diagnosis of sarcoidosis was inaccurate.

*Actinomyces* is usually spread from the oropharynx or upper gastrointestinal track via aspiration or inhalation and is related to poor oral and dental hygiene.<sup>8</sup> Unfortunately, the biopsy tissue was sent for pathology and not for culture, thus *Actinomyces* could not be identified. It is likely that the transbronchial lymph node biopsies of station 4R and 7 may have allowed the infection to spread from the respiratory track into the pericardium given the anatomical proximity.

Antimicrobial agents, proper source control, and drainage are the mainstay of treatment in patients with PP.<sup>9</sup> Prior to culture results, the patient was started on empiric antibiotics, vancomycin and piperacillin-tazobactam. *Actinomyces* is classically sensitive to penicillin, but given the presence of other anaerobic organisms on culture, the treatment was broadened to ampicillin-sulbactam. Most patients require at least four to six weeks of intravenous antibiotics followed by oral suppression to clear the infection.<sup>10</sup>

## CONCLUSIONS

Sarcoidosis is a diagnosis of exclusion and should be made only after infection has been ruled out by tissue culture. Purulent pericarditis

caused by *Actinomyces* and other anaerobic organisms is uncommon. Both the indolent and acute presentations of PP should be recognized, as well as how the clinical course impacts the management, treatment, and outcome of the infection.

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