

CASE REPORT

INTERMEDIATE

CLINICAL CASE

# Concomitant Purulent Pericarditis and Pleuritis Due to *Actinomyces odontolyticus* Following Endobronchial Biopsy for Pulmonary Sarcoidosis



Abdulaziz Joury, MD,<sup>a,b</sup> William West, MD,<sup>c</sup> Nadia Abelhad, MD,<sup>a,d</sup> Jeremy Teruel, MD,<sup>c</sup> Joseph A.R. Englert III, MD<sup>a,d</sup>

## ABSTRACT

The authors present a very rare case of bacterial purulent pericarditis due to *Actinomyces odontolyticus* 2 weeks following an endobronchial ultrasound bronchoscopy. On his presentation, he was in cardiac tamponade, for which he underwent an emergent pericardiocentesis with purulent drainage. Similar organisms grew in his left pleural effusion. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:1026-1031) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## HISTORY OF PRESENTATION

A 44-year-old man, recently diagnosed with pulmonary sarcoidosis, presented to a community emergency department with pleuritic chest pain. He reported fever, chills, night sweats, and persistent chest pain following an endobronchial ultrasound (EBUS) bronchoscopy. His chest pain was continuous, sharp, and relieved with leaning forward. His vital signs were significant for tachycardia to 110 beats/min and oxygen saturation of 92% on room air. His physical findings were notable for mild distress and distant heart sounds with no pericardial rubs. His oral temperature was 97.9°F (36.6°C), and blood pressure was 110/71 mm Hg; pulsus paradoxus was not present.

## LEARNING OBJECTIVES

- To be able to create a differential diagnosis for pleuritic chest pain in the setting following noncardiac procedure (ie, endobronchial ultrasound).
- To understand the occurrence of bacterial pericarditis as a secondary infection by contiguous spread following invasive thoracic procedures, and among these, direct extension from lung or pleural cavity.
- To be aware of the morbidity and mortality associated with bacterial purulent pericarditis and the importance of early implantation of therapeutic intervention.

From the <sup>a</sup>Department of Cardiology, Ochsner Medical Center, New Orleans, Louisiana, USA; <sup>b</sup>King Salman Heart Center, King Fahd Medical City, Riyadh, Saudi Arabia; <sup>c</sup>Department of Internal Medicine, Ochsner Medical Center, New Orleans, Louisiana, USA; and the <sup>d</sup>University of Queensland School of Medicine, Ochsner Clinical School, New Orleans, Louisiana, USA.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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## PAST MEDICAL HISTORY

The patient's past medical history is notable for well-controlled essential hypertension. Two weeks before this presentation, he underwent EBUS bronchoscopy at an outside hospital as part of further investigation for mediastinal and hilar lymphadenopathy on his chest computed tomography (CT). The pathology result of his mediastinal lymph node biopsy revealed noncaseating granulomas, suggestive of pulmonary sarcoidosis. He denied smoking, alcohol use, or illicit drug use. He denied prior international travel, and he did not own any pets. Six years before this presentation, he underwent a chest x-ray for routine pre-employment screening, which showed scattered pulmonary nodules, prompting a comprehensive chest CT. His chest CT showed small scattered pulmonary nodules (Figure 1A); however, no further investigation was pursued at that time.

## DIFFERENTIAL DIAGNOSIS

Given his presentation of chest pain, he had wide differential diagnoses including evolving COVID-19 pneumonia, acute pericarditis, pulmonary embolism, myocarditis, and acute coronary syndrome.

## INVESTIGATION

His electrocardiogram showed diffuse ST-segment elevations and PR depression (Figure 2). His laboratory investigation revealed serial negative troponin (ie, <0.006 ng/mL) and D-dimer >250 ng/mL [normal <0.50 mg/L]. The inflammatory markers C-reactive protein (CRP), and sedimentation rate were 204.5 mg/L (normal 0.0-8.2 mg/L) and 65 mm/h (normal 0-23 mm/h), respectively. He eventually underwent a chest CT with pulmonary angiography, which was negative for pulmonary embolism but revealed a significant circumferential pericardial effusion and worsening of multiple diffuse pulmonary nodules compared with prior imaging (Figure 1B). Two sets of blood cultures were negative for growth.

Although there were no clinical signs of tamponade, an urgent transthoracic echocardiogram showed preserved left ventricular ejection fraction of 55% and the presence of a moderate-size circumferential pericardial effusion with maximum thickness at 1.8 cm in subcostal window. There were echocardiographic signs suggestive of cardiac tamponade, including early diastolic right ventricular collapse,

significant variation in mitral inflow E velocity with inspiration, and plethoric inferior vena cava (Figure 3).

## MANAGEMENT

Supplemental oxygen was provided, and oral analgesia subsided his pain. He underwent an echocardiography-guided pericardiocentesis within hours of presentation via subxiphoid access with drainage of 250 mL of purulent material. Following this procedure, he was admitted to the intensive care unit. He was started on broad spectrum antibiotics with intravenous piperacillin/tazobactam 4.0 g/0.5 g every 12 hours and intravenous vancomycin with initial dose of 20 mg/kg. A pericardial drain remained and was removed 6 days later. Although his chest pain improved, on day 4 of admission, his chest CT showed worsening of left pleural effusion. On day 4, he underwent a left thoracentesis and left chest tube placement with intrapleural thrombolysis for loculated pleural effusion. His pericardial fluid, as well as pleural fluid, analysis showed an exudative process. Fluid cultures grew *Actinomyces odontolyticus*. His antibiotics were de-escalated to intravenous ampicillin-sulbactam 3 g every 6 hours, with an anticipation to complete 6 weeks of therapy. The chest tube was removed 7 days after its placement.

His pericardial effusion recurred after removal of the drain (hospital day 10); however, he was not in clinical or echocardiographic tamponade. After multidisciplinary discussion with cardiothoracic surgery, he eventually underwent placement of a pericardial window via a left anterior thoracotomy approach on hospital day 13. Over his hospital course, his inflammatory markers showed improvement toward the normal range, and he was discharged on hospital day 15 with a plan to finish intravenous antibiotics at home for 6 weeks.

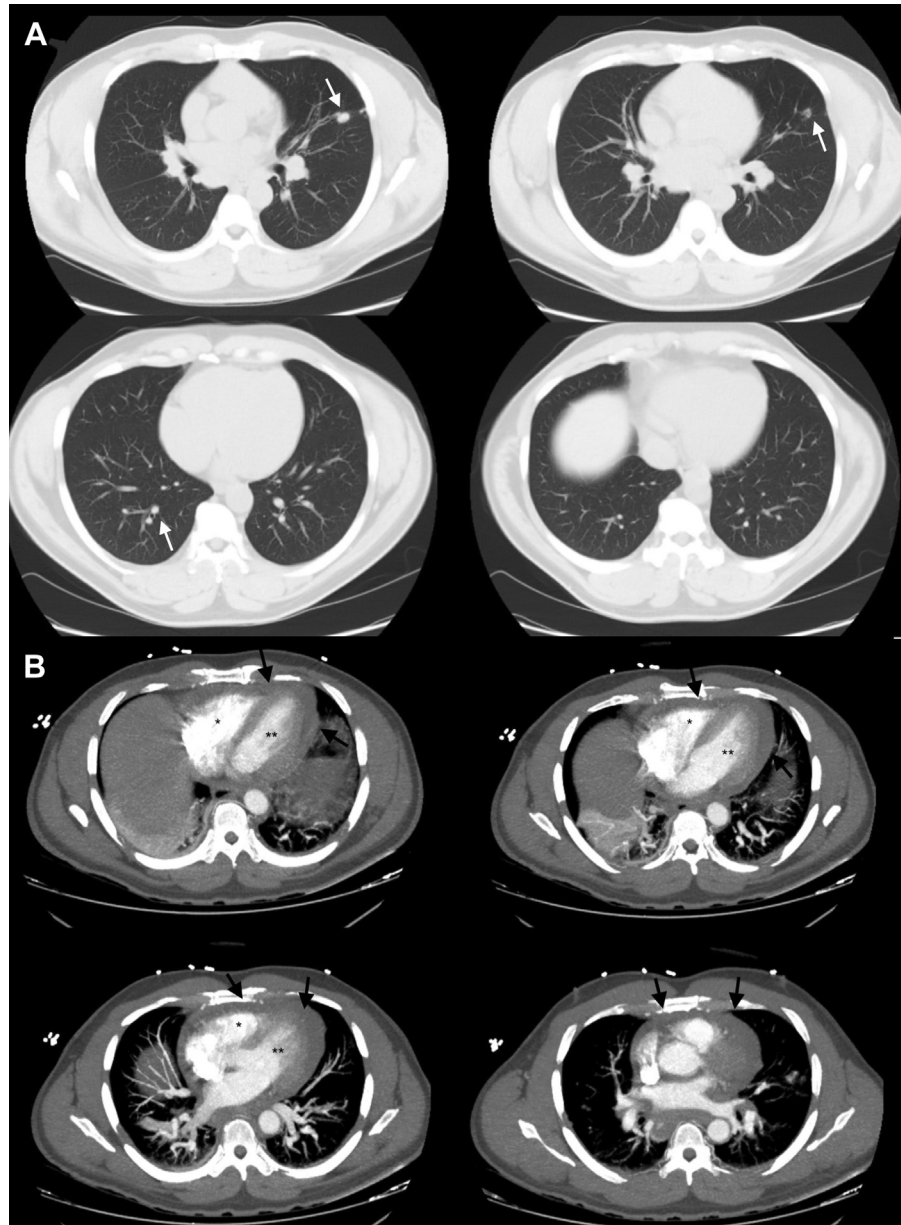
## DISCUSSION

Purulent pericarditis is a localized inflammation of the pericardium due to an infectious process, resulting in a fibrinous hypercellular exudate. Although rare, around 1% of total pericarditis, purulent pericarditis is associated with a higher mortality rate.<sup>1</sup> In Western countries, the common organisms are *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Str. viridans*; however, in immunocompromised patients, fungal pericarditis has been previously reported.<sup>2</sup> Contiguous spread following invasive thoracic procedures involving

## ABBREVIATIONS AND ACRONYMS

CT = computed tomography

EBUS = endobronchial ultrasound

**FIGURE 1** Baseline and Current Chest Computed Tomography Scan

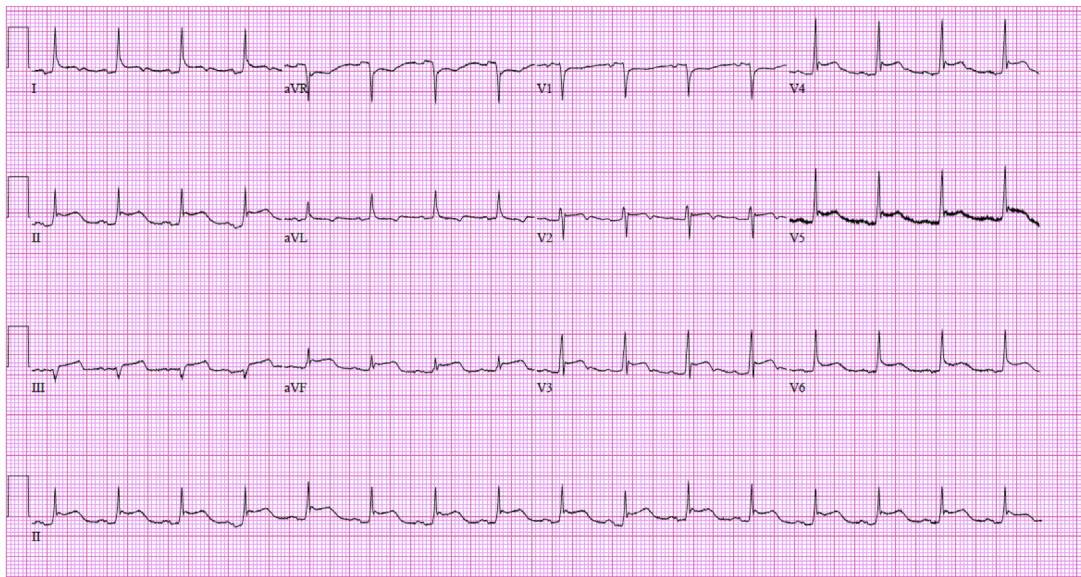
**(A)** Baseline chest computed tomography 6 years before the current presentation. This study was performed to further evaluate incidentally found pulmonary nodules on routine pre-employment screening chest radiographs. **Arrows** = pulmonary nodules. **(B)** Current presentation chest computed tomography with contrast showing no signs of pulmonary embolism; however, there are multiple pulmonary nodules and a new pericardial effusion. **Asterisk** = right ventricle; **double asterisks** = left ventricle; and **arrows** = pericardial effusion.

lungs or pleural cavity is a rare procedural complication.<sup>3</sup>

*A. odontolyticus* is a gram-positive anaerobic facultative nonsporulating bacteria seen in the oral cavity, colon, and urogenital cavity.<sup>4</sup> It has a lower

potential for virulence compared with other gram-positive bacteria; however, when the natural barrier (ie, mucosa) is disturbed, invasive disease with higher morbidity and mortality can occur.<sup>5</sup> In our patient, the most likely source of his *Actinomyces* pericarditis

**FIGURE 2** Electrocardiographic Signs of Pericarditis



Electrocardiographic images showing diffuse ST-segment elevation and PR-segment depression resembling electrocardiographic signs of pericarditis.

was from contiguous spread from the oropharynx during EBUS bronchoscopy.

To our knowledge, the first case report describing the presence of *A. odontolyticus* in pericardial fluid was described by Litwin et al<sup>6</sup> in 1999. They described a 68-year-old man who underwent gastric endoscopic biopsy followed by surgical resection of gastric adenocarcinoma. Two weeks postoperatively, he presented with fever and chills, and was found to have *A. odontolyticus* in his exudative pericardial effusion.<sup>6</sup> Our patient was healthy and in an immunocompetent state. Although he was incarcerated for a short period of time, his tuberculosis workup showed negative results. Two weeks before this presentation, he underwent an EBUS biopsy. We assume this invasive procedure was responsible for the introduction of *A. odontolyticus* to his pericardium and pleural cavity. The timing between the EBUS until his presentation (ie, 2 weeks) is slightly shorter than previously reported for EBUS bronchoscopy complicated by purulent pericardial tamponade.<sup>7</sup>

Aggressive management of purulent pericardial effusion with urgent pericardiocentesis to identify

the causative organism and implement antibiotics is imperative. Despite the medical progress in managing purulent pericarditis, the mortality exceeds 20% to 30%, and death usually occurs due to untreated cardiac tamponade, constrictive pericarditis, and sepsis.<sup>8</sup> Our patient developed an early recurrence of pericardial effusion without tamponade physiology, and a pericardial window was performed for chronic management.

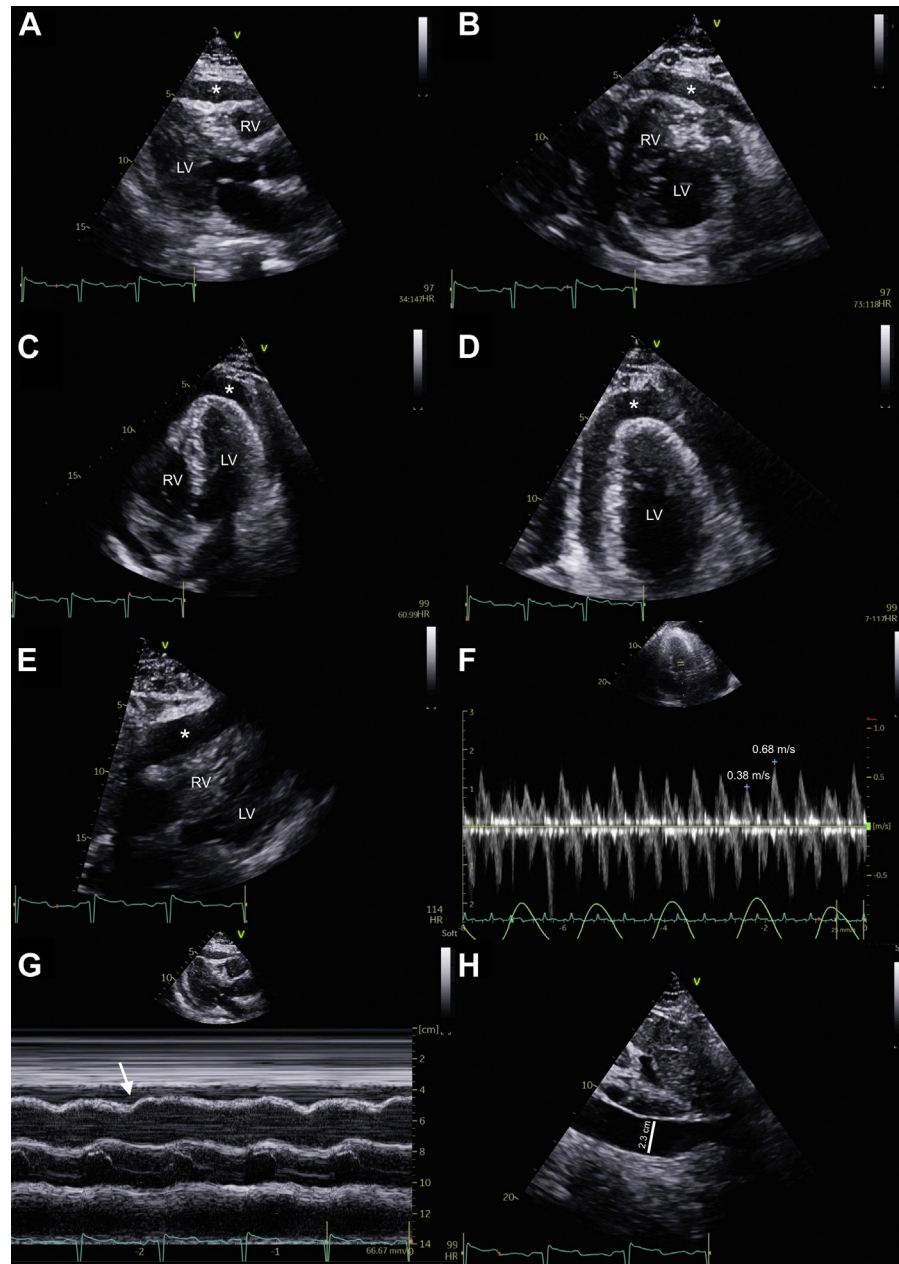
#### FOLLOW-UP

His inflammatory markers showed improvement in his CRP to 19 mg/L and sedimentation rate to 40 mm/h. To date, there is no recurrence of his acute pericarditis.

#### CONCLUSIONS

Bacterial purulent pericarditis is a rare entity but carries significant morbidity and mortality. Early diagnosis and aggressive intervention of purulent pericarditis should be implemented to avoid adverse outcomes.



**FIGURE 3** Echocardiographic Signs of Cardiac Tamponade

**(A)** Parasternal long-axis (PLAX) view revealing circumferential pericardial effusion; **(B)** short-axis; **(C)** apical 4-chamber; **(D)** apical 2-chamber; **(E)** subcostal view; **(F)** pulse-wave Doppler of transmittal diastolic flow reveals >40% respiratory variation; **(G)** M-mode in the parasternal long-axis showing early right ventricle (RV) diastolic collapse (**arrow**); **(H)** plethoric inferior vena cava measuring 2.3 cm without collapse during respiration. The **asterisk** indicates pericardial effusion.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

**ADDRESS FOR CORRESPONDENCE:** Dr Joseph A.R. Englert III, Ochsner Medical Center, 1514 Jefferson Highway, New Orleans, Louisiana 70121, USA. E-mail: [Joseph.englert@ochsner.org](mailto:Joseph.englert@ochsner.org).

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**KEY WORDS** *Actinomyces odontolyticus*, bacterial pericarditis, infectious pericarditis