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PERICARDIAL EFFUSION DUE TO *MYCOBACTERIUM TUBERCULOSIS* AND *STAPHYLOCOCCUS AUREUS* COINFECTION IN IMMUNOCOMPETENT PATIENT: A CASE REPORT

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ABSTRACT

Background: Pericardial effusion is a rare case, yet lethal if the treatment is not promptly given. *Mycobacterium tuberculosis* (MTB) is the leading cause of pericardial effusion in countries with high morbidity rate of tuberculosis, such as Indonesia. Generally, the infection happens in immunocompromised patients. **Case presentation:** This case report presented a 43-year-old female patient without comorbidities with worsening dyspnea exacerbated by dehydration due to acute diarrhea. The radiology imaging revealed the presence of cardiomegaly and pneumonia, meanwhile echocardiography revealed massive pericardial effusion. The pericardiocentesis result showed a negative Xpert TB result; however, elevated adenosine deaminase (ADA) and *Staphylococcus aureus* in the fluid culture were found. Antibiotic was given according to the culture sensitivity result as well as antituberculosis agents. **Conclusion:** From this case report, it is concluded that pericardial fluid culture is essential in improving diagnosis since coinfection can occur even to immunocompetent hosts. Immediate and appropriate management will reduce the fatal complication of cardiac tamponade.

BACKGROUND

Pericardial effusion is defined as the pathological accumulation of fluid inside pericardial cavity.[1] In normal condition, pericardial is the protective sac of the heart which consists of 2 layers, namely visceral and parietal pericardial, which is separated by small amount of plasma ultrafiltration volume (15 – 50 mL).[1] Several known etiologies of pericardial effusion are inflammation, microorganism infection, chest wall trauma, postsurgical complication, kidney failure, tumor, autoimmune diseases, and certain medications.[2] Pathophysiology of pericardial effusion is explained by three mechanisms, such as increased production of pericardial fluid, decreased absorption of pericardial fluid, and hemorrhage. Pericarditis, one of the etiologies of pericardial effusion, contributes toward the increase in pericardial fluid which is caused by increased vascular permeability due to inflammation.[3]

Epidemiologically, pericarditis caused by bacterial infection is a rare case, specifically in developed countries.[4,5] Generally, modes of infection in pericarditis are direct, hematogenous, and lymphatic spread.[4] The spreading from the focal infection around the heart due to pneumonia, empyema, and endocarditis, as well as chest trauma or surgery are the causes of bacterial pericarditis.[4] After the era of antibiotic discovery, pericarditis with bacterial etiology is found predominantly on immunocompromised, cancer, diabetic, chronic kidney disease, sepsis, and heart postoperative patients.[6] Intrapleural infection caused by *Streptococcus pneumoniae* develops intrathoracic foci for pericardial infection.[7] In contrast, purulent pericarditis caused by *Staphylococcus aureus* involves hematogenous route as the main mode of spreading.[7]

Mycobacterium tuberculosis is one of the notable etiologies of pericarditis, specifically in areas where tuberculosis (TB) incidence is considerably high.[4]

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According to World Health Organization (WHO) 2025, Indonesia has the second highest TB cases, contributing 10% of the total cases worldwide. The WHO also reported a high incidence rate of 1,080,000 cases and 118,000 mortality cases during 2024, which is further evidence of the population's susceptibility.[8] Extrapulmonary TB occurs more often in HIV patients, however about 10 – 20% of the cases are found in immunocompetent patients.[9] Tuberculous pericarditis happens in 1 – 2% of TB patients and it has high mortality rate, ranging from 17% to 40% after six months.[10] In developing country, pericardial effusion occurs in 50 – 70% of the tuberculous pericarditis cases.[11]

Coinfection is a phenomenon in which multiple pathogens are concurrently infecting the host, which often occurs in immunocompromised patients.[12] Coinfection might also occur in immunocompetent patients due to suppression of the immune system by multiple pathogens. The commonly reported coinfection with this mechanism is TB-HIV coinfection. However, TB-cryptococcus coinfection is also reported to have such immunosuppressive effect toward the immune system.[13]

This case report presents pericardial effusion originating from pericarditis which was caused by two bacteria, namely *Staphylococcus aureus* (*S. aureus*) and *Mycobacterium tuberculosis* (MTB), in immunocompetent patients. The case of pericardial effusion due to TB and *S. aureus* coinfection in patient with HIV was reported earlier by Lamas et. al.[14] Currently to the best of our knowledge, there are no reported cases of pericardial effusion with coinfection etiology in immunocompetent patients.

CASE PRESENTATION

A 43-year-old female patient, a housewife, was admitted to ER with the chief complain of dyspnea in the last 2 weeks. The dyspnea wasn't affected by activity nor position, as well as without wheezing, chest pain, nor palpitation. The accompanying complaints were fatigue, night sweat, and anorexia. The dyspnea was worsening in the last 2 days when the patient had diarrhea with a frequency of 15x without mucus; however, a small amount of fresh blood was present. The patient denied nausea, vomiting, fever, cough, rhinorrhea, dysphagia, leg edema, nor weight loss. No symptoms during urination were reported by the patient.

The patient had no history of any illness. She is married, has 1 child, and is currently not using any contraceptive method. There was no history of consuming any routine medications or nonprescribed medications. There were no histories of illness in the family. She denied smoking and drinking alcohol, but she rarely exercised.

The physical examination in the ER revealed the patient looked heavily ill, anxious, compos mentis, with the blood pressure of 130/70mmHg, heart rate of 104 beats per minute, respiration rate of 24 times per minute, temperature of 36^0C, and oxygen saturation of 97% with O_2 given at 2 liters per minute (LPM) with nasal cannula. The remarkable physical examination results, including anemic conjunctiva, dry lips mucosa, tachypnea, tachycardia, cardiomegaly with muffled heart sound, increased bowel sound, and lower abdominal pain upon palpation.

Initial hematological laboratory test revealed hemoglobin 9.7 g/dL (normal 11.7 – 15.5 g/dL), hematocrit 29% (normal 35–47%), *mean corpuscular volume* (MCV) 78fL (normal 80 – 100 fL), leukocyte of 36.810/mm^3 (normal 4,000 – 10,000/mm^3) with segmental neutrophil 90% (normal 50 – 70%) , and platelet of 647,000/mm^3 (normal 150,000 – 300,000/mm^3), creatinine 0.82 mg/dL (normal 0.51-0.95 mg/dL), urea at 62.8 mg/dL (normal 20 – 40 mg/dL), random glucose level 115 mg/dL (normal < 140 mg/dL), hyponatremia of 101 mEq/L (normal 135 – 145 mEq/L), and hyperkalemia 5.6 mEq/L (normal 3.5 – 5.1 mEq/L). Electrocardiogram showed sinus rhythm with the heart rate of 95x/minute, low voltage, and elevated ST segment in inferior leads **(Fig. 1)**. Because of this finding, troponin T test was ordered, and the result came back normal (< 40 ng/L) which ruled out myocardial infarction.

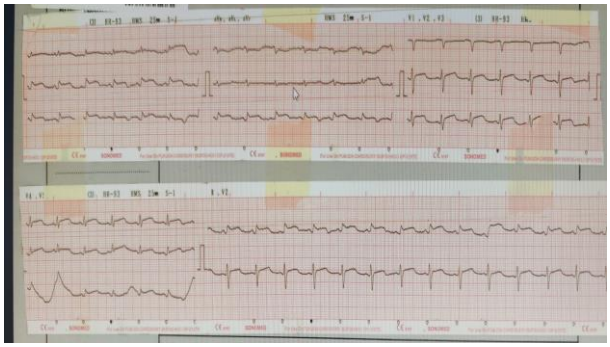

Figure 1. The patient's ECG revealed low voltage in limb leads and ST segment elevation in inferior leads

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The patient's chest X-ray result revealed cardiomegaly with the early sign of lung congestion, right pneumonia, and the minor fissure thickening on the right lung **(Fig. 2**).

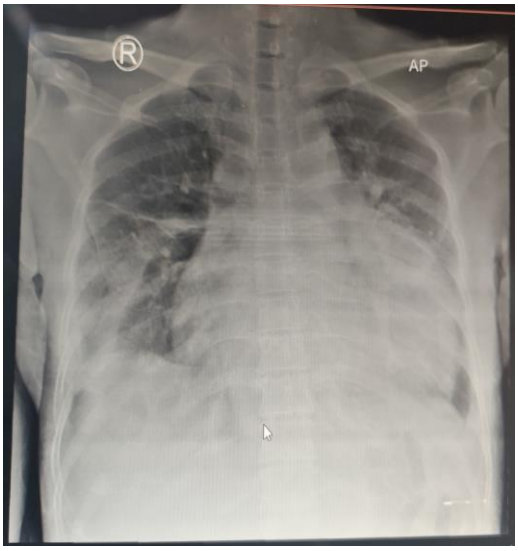

Figure 2. Chest X-ray before treatment

Based on the history taking, physical examinations, and additional tests done, she initially had the diagnosis of bacterial dysentery, with the complication of moderate dehydration, electrolytes imbalance (hyponatremia and hypokalemia), right bronchopneumonia, and cardiomegaly. The treatment given in ER is rehydration with 1500 mL of isotonic saline, followed by 500 mL of NaCl 3% each day, as well as serial Na^+ measurement every 24 hours. The patient also received Ciprofloxacin 400 mg IV every 12 hours, Metronidazole 500 mg IV every 8 hours, and Attapulgite 2 tablets after each diarrheal defecation. The fecal examination was also planned to be done.

The patient's diarrhea had stopped and hyponatremia was getting better (120 mEq/L) on the next day of follow up. However, she still complained of dyspnea and chest pain on the left side, hence she was consulted for an echocardiography examination. The imaging revealed normal heart chambers, valves' anatomy and physiology, systolic contraction, also ejection fraction of 75%. However, the echocardiography also revealed massive pericardial effusion with a tendency toward cardiac tamponade **(Fig. 3).** Thus, the pericardiocentesis was planned to be done on the third day.

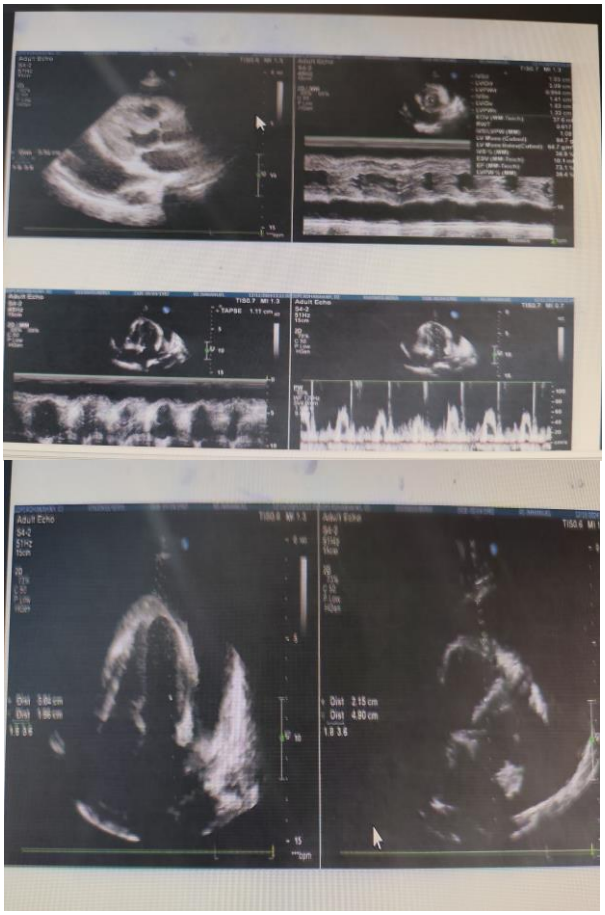

Figure 3. The patient's echocardiography revealed pericardial effusion

Pericardiocentesis was done on the third day and 520 mL of yellowish exudate fluids were pulled out. A catheter was put into the pericardial cavity for subsequent aspirations. The pericardial fluid specimen was sent for culture, adenosine deaminase (ADA), and Mycobacterium TB Gene Expert (MTB Xpert PCR). Since the patient cannot tolerate Ibuprofen 600 mg per day, Prednisone was started with a low dose (10 mg thrice daily) to reduce complication of constrictive pericarditis. The vital signs of the patient after pericardiocentesis were unremarkable.

On the subsequent day, pericardiocentesis was repeated; 230 mL of exudate fluids were pulled out. Hematologic profiles were improved after three days of antibiotics administration, with decreased leukocyte number ($18,000/mm^3$ from $36,810/mm^3$) and segmental neutrophil (74% from 90%). Ciprofloxacin and metronidazole were shifted to the oral regimen.

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On the fifth day of the treatment, the pericardial fluid culture showed *Staphylococcus aureus,* which is sensitive to Ciprofloxacin. The MTB PCR's result was negative, meanwhile the level of ADA was 140.1 U/L (normal < 40 U/L). Based on these results, the patient's diagnosis was confirmed as pericarditis caused by coinfection of TB and *S. aureus.* Screening for HIV then was done and turned out to be negative. Anti-TB regiment was given based on WHO guidelines, 2 months of intensive phase and 4 months of continuation phase. Ciprofloxacin was continued until 10 days duration and Prednisone's dose was tapered off. She was discharged after one week of hospitalization in good condition.

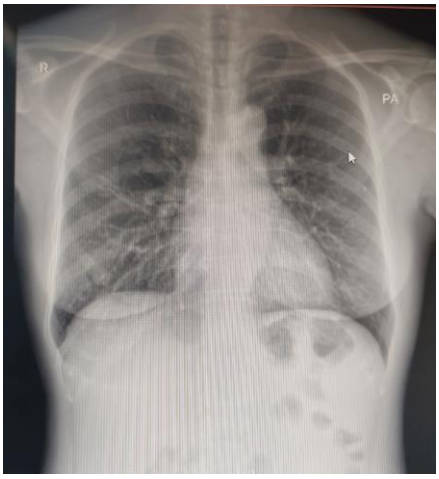

Figure 4. Chest X-ray posttreatme

Another follow up was done on the sixth month of TB treatment, with her chest X-ray showed an unremarkable result. Therefore, the anti-TB regiment was stopped and the patient was categorized as having completed the TB treatment course **(Fig. 4).**

DISCUSSION

Tuberculous pericarditis manifests as acute pericarditis (4%), cardiac tamponade (7%), and constrictive pericarditis (6%).[15] MTB usually spreads to pericardium retrogradely from peritracheal, peribronchial, or mediastinum lymph nodes. MTB also spreads through hematogenous route from the primary infection focus. The direct spreading from TB lesion in the lungs or hematogenous spreading from the far-located lesions are rarely found.[16]

Tuberculous pericarditis doesn't show sudden clinical symptoms in a similar fashion as acute pericarditis caused by *S. aureus*. Generally, tuberculous pericarditis is accompanied by nonspecific systemic signs, such as fever, night sweats, fatigue, and weight loss.[17] Chest pain is rarely found in slow-progressing pericarditis caused by tuberculosis infection, tumor, uremia, and postradiation therapy. If the pericarditis effusion exists, the heart sound will be muffled, the apex of the heart is not visible upon inspection, and pericardial friction rub is not heard.[2]

The patient in this case had dyspnea for two weeks, accompanied by systemic signs such as fatigue and night sweats, with the symptoms worsened since the patient had dehydration caused by acute gastroenteritis. The dyspnea was accompanied by cardiomegaly and muffled heart sound, which led to the pericardial effusion diagnosis.

The patient's chest radiograph in this case revealed cardiomegaly and pneumonia on the right lung. *Staphylococcus aureus* as the cause of pericarditis in this case was presumed to originate from the pneumonia, since there was no other infection found in the common locations of *S. aureus*, such as skin infection. Afşin et al reported a case of pneumonia in immunocompetent host due to coinfection of *Staphylococcus aureus* and *Mycobacterium tuberculosis*. Whether or not coinfection develops is determined by the host's immune response and the virulence of the infecting organisms. Comorbidities such as impaired cough reflex and mucociliary clearance, impaired local and humoral immunity, alcohol use and smoking, chronic obstructive pulmonary disease (COPD), congestive heart failure, chronic kidney disease, liver disease, and immunodeficiency conditions constitute a predisposition for the development of community-acquired pneumonia.[18]

S. aureus can weaken T cell immunity and innate immune responses to secondary bacterial infections. Thus, it can also pave the way for the reactivation of latent tuberculosis and spread to the pericardium, causing pericarditis and effusion. The infection caused by *Mycobacterium tuberculosis* alone may suppress the immune system. Furthermore, the inflammation process which is usually chronic also contributes toward the further suppression of the immune system. Therefore, opportunistic pathogens might proliferate in the weakened state of TB patient's body.[19]

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Diagnosis of tuberculous pericarditis can be determined through several methods: analysis of pericardial fluid in which the early phase shows dominant neutrophil, meanwhile the late phase is dominated by mononuclear cells. Biomarkers, such as adenosine deaminase (ADA) enzymes with sensitivity of 88% and specificity of 83% as well as gamma interferon (IFN-γ) with the sensitivity of 92% and specificity of 100% can be used. Although IFN-γ offers advantages in specificity, its high cost, complexity, and infrastructure requirements make it less practical as a frontline diagnostic tool. Current evidence suggests that biomarkers like ADA, an enzyme pivotal in purine metabolism, offer a more accessible and economically viable alternative for differential diagnosis. ADA is found predominantly in lymphocytes, monocytes, and macrophages, rendering it a potentially sensitive indicator of TB-associated inflammation.[20]

The nucleic acid amplification test, such as Xpert MTB/RIF, also can be used to diagnose, even though the sensitivity is only 63,8%. Histopathological examination on the more specific tissue can only diagnose 10 – 64% of tuberculous pericarditis cases. In this case, the patient had a high level of ADA enzyme, and yet Xpert MTB showed negative results. The meta-analysis study by Andrianto et al. showed that Xpert MTB had a sensitivity of 0.676 and specificity of 0.994 on diagnosing tuberculosis. Therefore, the negative result of Xpert MTB cannot rule out tuberculous pericarditis.[21]

Pericardial fluid culture is a gold standard to determine non tuberculosis etiology of infection, since culture of TB results after several months. The presence of concurrent infections in tuberculosis patients creates great clinical and public health complications, changing the disease progression and management. A wide range of microbial pathogens, from viruses to bacteria, frequently accompany *Mycobacterium tuberculosis* infections, thus worsening health outcomes and mortality risks. A survey study by Abdulkadir et al in 2020 showed coinfection with bacteria in TB patients. *Klebsiella pneumoniae, Staphylococcus aureus* and *Pseudomonas aeruginosa* were the most prevalent organisms isolated with 21.90%, 19.66%, and 19.10% respectively. [22]

The presence of bacterial coinfections in tuberculosis patients causes a lot of therapeutic challenges, often resulting in prolonged treatment regimen and poor clinical outcomes. Coinfections frequently contribute to the development of antimicrobial resistance and create complex medication protocols that complicate standard TB therapy.[23]

The patient in this case received six months of anti-TB regiment in accordance with WHO guidelines, antibiotic based on the culture result, and one month of corticosteroid to prevent the inflammation and fibrosis effect on the pericardium. During outpatient follow up on the sixth month, the chest X-ray result was unremarkable, and the patient no longer had any complaints. Precise treatment of pericardial effusion caused by infection mainly has three goals: eliminating the bacteria causing infection, preventing short term cardiovascular effect caused by pericardial inflammation, and preventing the long-term cardiac sequelae caused by the imperfect healing process, such as constrictive fibrosis.[24]

CONCLUSION

Pericardial fluid culture is essential in improving diagnosis since coinfection can occur even to immunocompetent hosts. Immediate and appropriate management will reduce the fatal complication of cardiac tamponade.

ETHICAL APPROVAL

Written and verbal informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Conceptualization, LK; methodology, LK; formal analysis, LK; investigation, LK; resources, LK and AC; data curation, LK; writing—original draft preparation, LK; writing—review and editing, LK and AC; visualization, LK; supervision, LK; project administration, LK.

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