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Case Report

Impending Cardiac Tamponade caused by Tuberculous Pericarditis

Authors
Dian Daniella[1*], Candra Wiguna[2], Leonardo Paskah Suciadi[3]
[1]General Practitioner, Siloam Hospitals KebonJeruk, Jakarta, Indonesia
[2]Internist, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia
[3]Cardiologist, Siloam Hospitals Kebon Jeruk, Jakarta, Indonesia
[*]Corresponding Author
Dian Daniella
Siloam Hospitals Kebon Jeruk
Jl. Pejuangan Raya number 8, Kebon Jeruk, West Jakarta

Abstract
Tuberculosis is responsible for 70% cases of large pericardial effusion and 2% of cardiac tamponade. A-21-year-old female sought medical care due to progressive dyspnea and chest discomfort. Tuberculous pericarditis with pericardial effusion was diagnosed based on muffled heart sound along with regular tachycardia and jugular vein distention on cardiovascular examination, high CRP with positive IGRA, ECG showed low voltage criteria, right pericardial effusion on chest x-ray, and echocardiography showed massive pericardial effusion with fibrous (echogenic) profile surrounding the heart which was swinging inside with signs of early tamponade. The patient underwent an urgent pericardiocentesis and analysis of pericardial fluid was suggestive of TB. Patient was given oral anti-tuberculosis therapy, corticosteroid and colchicine and showed remarkable improvement. Pericardial effusion contribute to higher morbidity and mortality of the patients with TB. Early identification and diagnosis confirmation are essential to start the appropriate medication and intervention to improve the outcomes of the patients.
Keywords: *Tuberculosis, Pericarditis, Pericardial Effusion.*

Introduction

Extrapulmonary tuberculosis (TB) occurs in 20% of patients with TB,[(1)] with tuberculous pericarditis is rare and only found in approximately 1% of all autopsied cases of TB and in 1% to 2% of instances of pulmonary TB.[(2)] The incidence of tuberculous pericarditis is increasing with the advent of the Acquired Immunodeficiency Syndrome (AIDS) pandemic.[(1)] Tuberculosis is responsible for 70% cases of large pericardial effusion in developing countries,[(3)] and 2% of cardiac tamponade.[(4)] Among the potential reasons for pericardial effusion, tuberculous pericarditis should be considered as the reason, especially in developing countries such as African and Asian countries, including Indonesia.[(2,5,6)]

The presence of tuberculous pericarditis increases mortality rate of the patients to 14-40%,[(7)] therefore timely and accurate identification of tuberculous pericarditis is crucial, because emergent procedures are needed in cases with

massive pericardial effusion.[5] Tuberculosis is a relative rare cause of pericardial diseases in Human Immunodeficiency Virus (HIV) negative and immunocompotent persons.[4] We reported a case of massive pericardial effusion caused by tuberculous pericarditis in an immunocompetentyoung patient.

Case Report

A-21-year-old female, domiciled in Wamena, Papua, East Indonesia, sought medical care due to progressive dyspnea and chest discomfort especially while in supine position since two months ago, with the worsening of the symptoms in the last five days. She also complained of fever in the last five days. She denied coughing, weight loss and night sweats. She denied history of other disease or previous use of medications. Most people in her neighborhood had been diagnosed with TB. At physical examination, the patient was lucid and oriented, normal body weight, non cyanotic, normal blood pressure,persistent tachycardia (heart rate 110-120 bpm) and slight tachypnea (respiratory rate 22x/minute), with fever (temperature 38.4 C at admission). A muffled heart sound along with regular tachycardia and jugular vein distention were noted by cardiovascular examination. Respiratory apparatus with clear lung by auscultation and dullness in lower right hemithorax. Abdomen examination revealed within normal findings.

Her initial laboratory examination showed high C-reactive protein (CRP) (table 1). The aadditional laboratory testindicated positive Interferon Gamma Release Assay (IGRA) and negative HIV.

Table 1 Laboratory Results

Parameter	Value	Unit	Normal value	Interpretation
Hemoglobin	13.2	g/dL	13.0-18.0	
Leucocyte	8.8	103/uL	4.0-10.0	
Basofil	0	%	0-1	
Eosinophil	0	%	0-4	
Banded neutrophil	0	%	2-6	
Segmented neutrophil	77	%	50-70	High
Lymphocyte	15	%	20-40	
Monocyte	8	%	2-8	
ESR	5	mm	0-15	
Hematocryte	42	%	40-54	
Thrombocyte	215	103/uL	150-400	
Reticulocyte				
Albumin	3.2	g/dL	3.5-5.5	Low
SGOT	37	U/L	5-34	High
SGPT	86	U/L	< 55	High
Blood glucose	91	mg/dL	60-180	
Ureum	39	mg/dL	10-50	
Creatinin	0.7	mg/dL	0.1-1.3	
Natrium	140	mmol/L	135-145	
Kalium	3.7	mmol/L	3.5-5.1	
Chloride	103	mmol/L	97-111	
CRP	16.1	mg/L	<5	High
IGRA	(+)		(-)	
Anti HIV	(-)		(-)	

ESR: erythrocyte sedimentation rate; SGOT: serum glutamic oxaloacetic transaminase ;
SGPT: serum glutamic pyruvic transaminse ;CRP: C-reactive protein; IGRA: Interferon Gamma Release Assay; Anti HIV: Anti Human Immunodeficiency Virus

Electrocardiogram (ECG) at admission showed sinus tachycardia, low voltage criteria, and nonspecific T wave changes at precordial leads (figure 1).

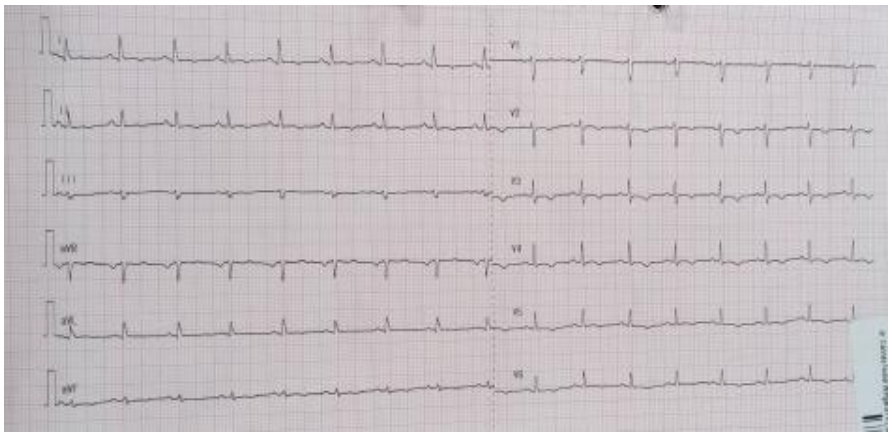

Figure 1 Electrocardiogram

Her chest x-ray showed remarkably cardiomegaly, right pericardial effusion, with slight opacity in right paracardial (figure 2).

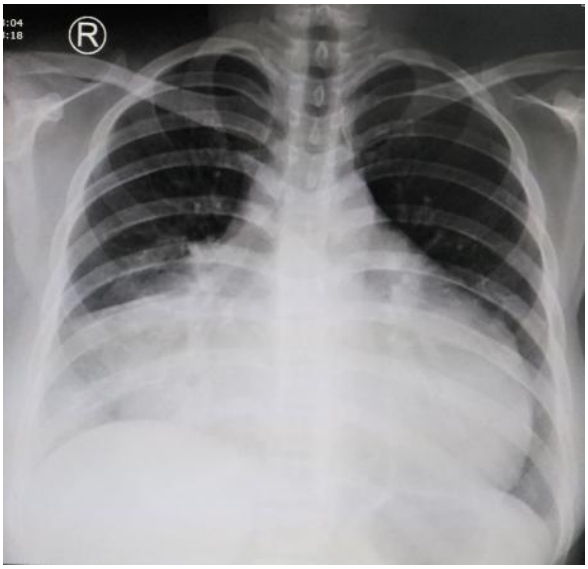

Figure 2 Chest X-Ray

The patient was managed as inpatient by an Internist with some medication. As there was a clinical suspicion of pericardial effusion, consultation to cardiologist and echocardiography evaluation were performed then.

Echocardiography showed massive pericardial effusion with fibrous (echogenic) profile surrounding the heartwhich was swinging inside (figure 4.A). Signs of early tamponade by some echocardiography parameters were also recognized.

The patient underwent an urgent pericardiocentesis by ultrasound guided apical approach puncture at ICCU. Using a dwelled pigtail, 300cc of serosanguinous pericardial fluid was drained initially, and followed by periodic aspiration to collect a total drainage of 485 mL within a week.

Table 2 Pericardial Fluid Analysis

Parameter	Value	Unit	Normal value	Interpretation
pH	8			
Albumin				
Albumin	2.5	g/dL		
Albumin serum	3.2	g/dL	3.5-5.3	low
Albumin ratio	0.78			
Fluid analysis				
Sample	Pericardial fluid			
Macroscopic				
Color	red			
Clarity	not clear			
Clot	negative			
Rivalta	positive			
Microscopic				
Cells	4060	/uL		
Differential count				
PMN	5	%		
MN	95	%		
Protein				
Protein	6300	mg/dL		
Protein serum	7.8	g/dL	6.2-8.5	
Protein ratio	0.81			
LDH				
LDH	5036	U/L		
LDH serum	552	U/L	<480	high
LDH ratio	9.12			
Glucose	19	mg/dL		
CEA	1.78	ng/mL	0-5	
Adenosine deaminase	127	U/L	TB	
	Exudate			

pH: power of hydrogen; PMN: polymorphonuclear; MN: mononuclear; LDH: lactate dehydrogenase; CEA: carcinoembryonic antigen

Analysis of pericardial fluid was suggestive of TB (table 2), supported by histology examination that showed no signs of malignancy. Oral anti-tuberculosis therapy and corticosteroid (Methylprednisolon 2x125 mg IV) were instituted then, and colchicine 1x0.5 mg was addedlater to suppress the inflammation process on the pericardium and prevent constrictive pericarditis as a common complication later. Follow up chest X-ray showed better findings (figure 3) along with clinical improvement of the patients. As the consequence, pericardial drain was removed at the following day.

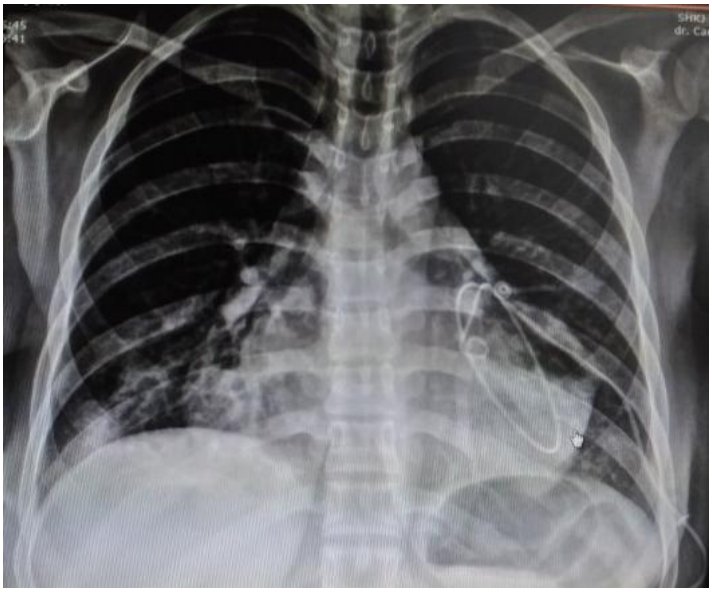

Figure 3 Chest X-ray after Pericardiocentesis

Follow up echocardiography after removal of pericardial drain indicatedminimal pericardial effusion located at posterior, with normal cardiac structure and function (figure 4.B).

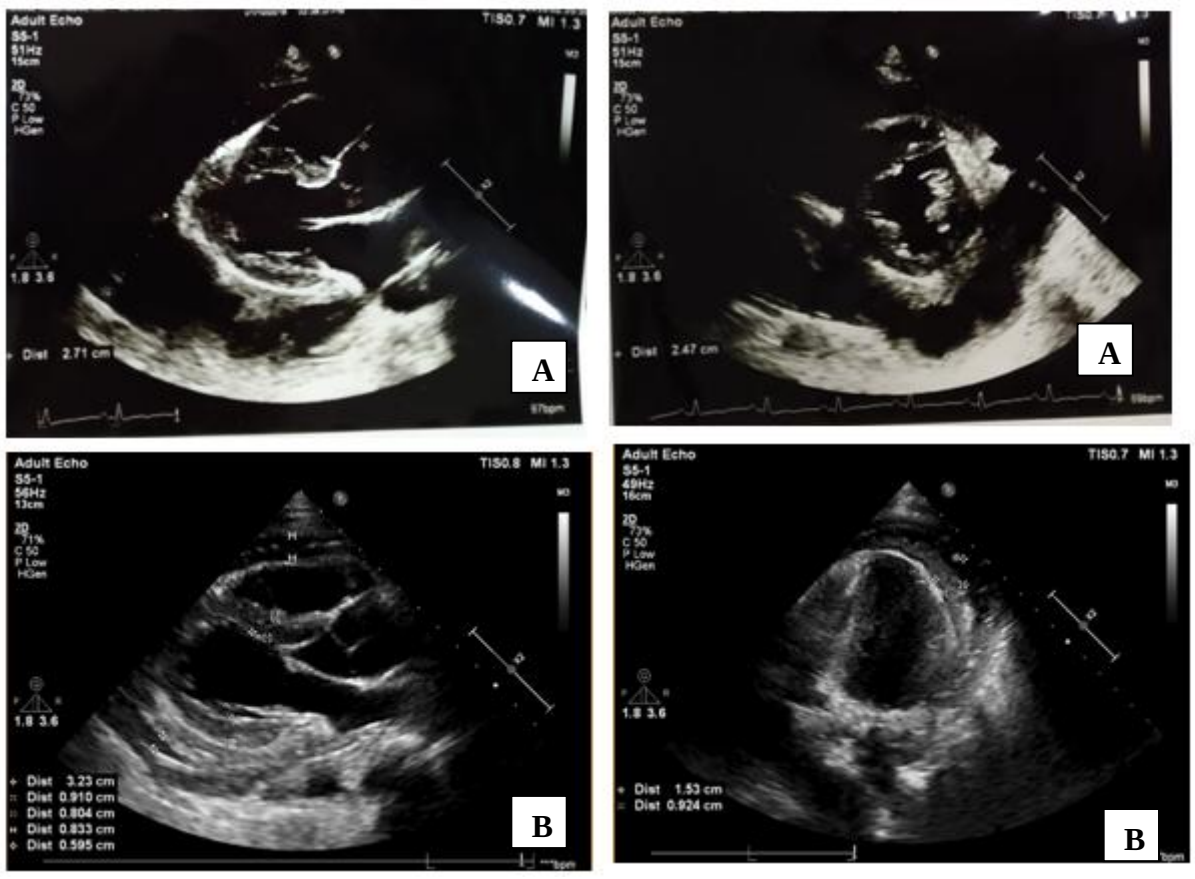

Figure 4 Echocardiography A. Before Pericardiocentesis B. After Pericardiocentesis

Patient was discharged after completing 2 weeks of hospitalization, with continuing medication of oral anti-tuberculosis therapy, corticosteroid, and colchicine. Follow up at outpatient clinic at 2 weeks later indicated that the patient was free of symptoms and nearly no remained pericardial effusion by echocardiography examination.

Discussion

Progressive dyspnea and chest discomfort which is alleviated while taking leaning forward position, along with tachycardia, muffled heart sound, jugular vein distension, relatively clear lung by auscultation, and low voltage criteria on ECGfound in patients with TB should raise a clinical suspicion of significant pericardial effusion or cardiac tamponade if hypotension occurs.

Pericardial involvementis usually developed by retrograde lymphatic spread of *Mycobacterium tuberculosis* from peritracheal, peribronchial, or mediastinal lymph nodes, by hematogenous spread from primary tuberculous infection or direct spread from lung such as from military TB.[2] Four pathological stages of tuberculous pericarditis are recognized: (1) fibrinous exudation with initial polymorphonuclear leukocytosis, relatively abundant mycobacteria, and early granuloma formation with loose organization of macrophages and T cells; (2) serosanguineous effusion with a predominantly lymphocytic exudate with monocytes and foam cells; (3) absorption of effusion with organization of granulomatous caseation and pericardial thickening caused by fibrin, collagenosis, and ultimately, fibrosis; and (4) constrictive scarring: the fibrosing visceral and parietal pericardium contracts on the cardiac chambers and may become calcified, encasing the heart in a fibrocalcific skin that impedes diastolic filling and causes the classic syndrome of constrictive pericarditis.[2]

Aside than physical examination, laboratory examination is useful in confirming diagnosis of tuberculous pericarditis, such as Adenosine Deaminase (ADA). Adenosine Deaminase is an enzyme produced by leucocyte in pericardial fluid.[7] Recent studies have demonstrated that elevated ADA activity is suggestive of tuberculous pericarditis.[2] The usefulness of ADA as a diagnostic tool applies both to HIV-positive and HIV-negative patients, although lower ADA levels are observed in HIV-positive patients with severe CD4 lymphocyte depletion.[2] Pericardial fluid high in ADA activity (>40 U/L) is of great value in early diagnosis of tuberculous pericarditis, with a sensitivity of 94% and a specificity of 97% for tuberculous pericarditis.[4] Analysis from pericardial fluid of our patient indicated a high ADA level of 127 U/L, which suggestive of tuberculous pericarditis.

In addition to ADA test, collecting IGRA is also recommended and useful to support the diagnosis. Interferon-gamma is a type of cytokine produced by T helper cells in response to *Mycobacterium tuberculosis*.[5] The diagnostic accuracy of tests for IGRA in pericardial effusion were extremely high, with an overall sensitivity and specificity of 97% and 99%, respectively.[5] Positive IGRA result found in our patient gave additional diagnostic confirmation for tuberculosis in this case.

Tuberculous pericardial effusions are typically exudative and characterized by a high protein content and increased leukocyte count, with a predominance of lymphocytes and monocytes.[2] The pericardial fluid is usually a yellow-citrus-like exudate with low glucose.[9] Light's criteria (whereby an exudate is defined as having 1 or more of the following: pleural fluid protein divided by serum protein $\geq$ 0.5; pleural fluid lactate dehydrogenase [LDH] divided by serum LDH $\geq$ 0.6; and/or pleural fluid LDH level $\geq$ 66% of the upper limit of normal for serum LDH) are the most reliable diagnostic tool for identifying pericardial exudates.[2] In this patient, pericardial fluid showed red, with positive rivalta, mostly mononuclear, protein ratio 0.81, LDH ratio 9.12, with normal CEA and high ADA level 127 U/L. This pericardial fluid analysis was reasonably considered as exudate caused by TB. Specimen culture obtained from pericardial fluid is considered as a gold standard in making diagnosis of tuberculous pericarditis, however, this workup is time-consuming, costly and not sensitive.[5]

The ECG is abnormal in virtually all cases of tuberculous pericardial effusion, usually in the form of nonspecific ST-T–wave changes.[2]

Diffuse PR-segment depression and ST-segment elevation as characteristic of acute pericarditis are found in only 9% to 11% of cases.[2] The presence of low voltage criteria (complexes < 5 mm in limb leads and < 10 mm in precordial leads) suggests a large pericardial effusion, and cardiac tamponade is unlikely in the absence of this ECG microvoltage.[2] In extreme case of tamponade, QRS voltage alternans can be viewed on ECG indicating the heart swinging inside the fluid accumulation.

Chest X-Ray can raise initial clinical suspicion of massive pericardial effusion in patients with suspected TB. An enlarged cardiac shadow resembling a bottle pattern, along with characteristic distribution of infiltrate in lung is an important clue to send the patient for additional imaging test such as echocardiography.[2]

Echocardiography plays a pivotal role in diagnosis making of TB pericarditis. This method can identifyand confirm pericardial effusion, evaluate the distribution of the collected fluid (circumferential or loculated), estimate the volume of the fluid, and detect early signs of cardiac tamponade ("echocardiographic tamponade"). Furthermore, this tool is also important to guide pericardiocentesis procedure as a definitive therapy to evacuate the fluid. The pericardial exudate of TB is thick and fibrinous (echogenic) with a tendency to form adhesions and in some instances constriction.[6] On echocardiography there are patchy deposits 4–8 mm in thickness with ''fibrinous'' strands criss crossing the pericardial space.[6] The appearance is quite characteristic.[6]

Some of proposed diagnostic criterias of tuberculous pericarditis can be seen on Table 3.

Table 3 Diagnostic criteria of Tuberculous Pericarditis

Cherian et al. [6]	**Wanjari et al.**[1]	**Mayosi et al.**[2]
Any of the following: Invasive 1. Culture of *M. tuberculosis* from pericardial fluid or tissue 2. Pericardial tuberculous granuloma with acid fast bacilli 3. Pericardial tuberculous granuloma + positive tuberculin skin test 4. Pleural tuberculous granuloma with acid fast bacilli 5. Pleural tuberculous granuloma + positive tuberculin skin test 6. Tuberculous granuloma in scalene node or peripheral lymph node Non invasive: 1. Active TB elsewhere in the body 2. Mediastinal (non-hilar) lymph nodes on chest ct scan with hypodensecentre and matting + positive tuberculin skin test) 3. Response to specific anti-tuberculosis therapy	Definite tuberculous pericarditis can be diagnosed by one or more of the following criteria: 1. Isolation of *M. tuberculosis* from pericardial effusion fluid or pericardial biopsy. 2. Demonstration of granulomatous inflammation on histologic examination of pericardial biopsy sample. 3. Isolation of *M. tuberculosis* from sputum or non pericardial effusion exudates in the presence of clinical and/or radiological evidence of TB, associated with a positive response to antitubercular therapy and in the absence of any other obvious cause for pericarditis.	Proposed Diagnostic Criteria for Tuberculous Pericarditis for Countries and Communities in Which TB Is Endemic Definite tuberculous pericarditis 1. Tubercle bacilli are found in stained smear or culture of pericardial fluid; 2. Tubercle bacilli or caseating granulomata are found on histological examination of pericardium Probable tuberculous pericarditis 1. Evidence of pericarditis in a patient with TB demonstrated elsewhere in the body; and/or 2. Lymphocytic pericardial exudate with elevated ADA activity; and/or 3. Good response to anti-tuberculosis therapy

Management of tuberculous pericarditis consists of medication and pericardiocentesis. Medication such as anti-tuberculosis therapy should be given. A regimen consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol for at least two months followed by isoniazid and rifampicin

(total six months of therapy) have been shown to be highly effective.[1] Treatment for 9 months or longer gives no better results and has the disadvantages of increased cost and poor compliance.[2] The available trial data show effects supporting the use of steroids in the treatment of pericarditis in both HIV-negative and positive patients.[11] The recommended prednisolone dose for tuberculous pericarditis is 1 mg/kg/day in the acute phase tapered off during the next 3 months.[10] Patient was given colchicinesto suppress the inflammation process on the pericardium and prevent constrictive pericarditis as a common complication later. Colchicine concentrates in leucocytes and inhibits the process of microtubule self-assembly by binding b-tubulin, thus interfering with chemotaxis, degranulation and phagocytosis.[13] Low fractionated doses improve tolerability: 0.5–0.6 mg twice daily in general or 0.5–0.6 mg once daily for patients <70 kg or intolerant to higher doses.[13]

Percutaneous pericardiocentesis using needle should be considered in large pericardial effusion, especially if the fluid collection is circumferential or not loculated in the posterior region. This minimally invasive procedure can be performed safely under local anesthesia and ultrasound guided. Depending on area with the largest fluid accumulation and operator preference, puncture site can be approached from subcostal, apex, lateral, or precordial. We performed puncture from apical approach in this patient based on the aforementioned considerations. Drain catheter, usually pigtail, is usually inserted to pericardial space and kept for several days to assure periodic aspiration of the residual or additional fluid later.

In other situations when transcutaneous pericardiocentesis is not feasible, ec. posteriorly loculated effusion or very thick and lots of debris fluid, surgical pericardial window is a considerable option. Cardiac thoracic surgeon conducts this minor surgery in operating theatre under sedation. This larger-bore catheter placed surgically to pericardium space allows evacuating more densed fluid especially if it is located in posterior.

Conflicts of Interest

Authors declare no conflict of interest.

Acknowledgment

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Conclusion

Tuberculous pericarditis with or without significant pericardial effusion is a potential clinical manifestation of TB, even in immunocompentent patients.This complication contribute to higher morbidity and mortality of the patients with TB. Early identification and diagnosis confirmation are essential tostart the appropriate medication and intervention to improve the outcomes of the patients.

References

1. Wanjari K, Baradkar V, Mathur M, Kumar S. A Case of Tuberculous Pericardial Effusion. Indian J Med Microbiol. 2009;27(1):75–7.
2. Mayosi BM, Burgess LJ, Doubell AF. TuberculousPericarditis.Circulation. 2005 Dec 6;112(23):3608–16.
3. Syed FF, Mayosi BM. A Modern Approach to Tuberculous Pericarditis. Prog Cardiovasc Dis. 2007 Nov;50(3): 218–36.
4. Rai DK, Kumar A. A rare case of cardiac tamponade due to tuberculosis.Int J Adv Med. 2017 Jan 23;4(1):296.
5. Liu C, Cui Y-L, Ding C-M, Wu Y-H, Li H-L, Liu X-F, et al. Diagnostic accuracy of interferon-gamma in pericardial effusions for tuberculous pericarditis: a meta-analysis. J Thorac Dis. 2018 Feb;10(2):854–60.
6. Cherian G. Diagnosis of tuberculous-aetiology in pericardial effusions. Postgrad Med J. 2004 May 1;80(943):262–6.

7. Shaheen, Rajyashri S, Anjum P, Jayshree P. An Unusual Presentation of Tuberculous Pericardial Effusion. South Asian Fed Obstet Gynecol. 2009;1(3):71–2.
8. Trautner BW, Darouiche RO. Tuberculous Pericarditis: Optimal Diagnosis and Management. Clin Infect Dis. 2001;33:954–61.
9. Farnetano B dos S, Farias ML, Mota GSM, Leao CN, Machareth MMV. Pericardial Tuberculosis-A Case Report. J Tuberc Ther. 2007;1(1).
10. Singh S, Tiwari K. Use of corticosteroids in tuberculosis. J Assoc Chest Physicians. 2017;5(2):70.
11. Evans DJ. The use of adjunctive corticosteroids in the treatment of pericardial, pleural and meningeal tuberculosis: Do they improve outcome? Respir Med. 2008 Jun;102(6):793–800.
12. Shah SR, Alweis R, Shah SA, Arshad MH, Manji AA-K, Arfeen AA, et al. Effects of colchicine on pericardial diseases: a review of the literature and current evidence. J Community Hosp Intern Med Perspect. 2016 Jan;6(3):31957.
13. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, et al. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC) Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2015 Nov 7;36(42):2921–64.