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Case Report

Pericardial tuberculosis with an emphasis on empiric therapy in endemic areas for tuberculosis (a case report)

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ABSTRACT

Pericardial tuberculosis (TB) is rare, but has particularly severe complications and a high mortality rate when not treated. Prompt treatment of pericardial TB is important and can be life-saving. We report a 13-year-old girl with massive pericardial effusion and negative workup for TB, who was empirically treated with an excellent response.

Introduction

Pericardial tuberculosis is a rare form of mycobacterium tuberculosis infection. It is common in endemic areas for TB, like our region Asia. Many of the cases are old or may have a background of immunodeficiency (e.g. They may be human immunodeficiency virus-positive, or undergo immunosuppressive therapy) [1–3].

In many cases the workup for TB is positive and the patient may have a documented positive test (e.g. PCR, culture, biopsy, smear for acid fast bacilli, etc.) or a personal or family history of contact with TB or an infection with TB may be positive.

We introduce a young female, who was immunocompetent, with no history of TB and negative workup for mycobacterium tuberculosis. She responded rapidly to the empiric

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therapy and her condition was excellent at follow-up 2 months later.

Case Report

A 13-year-old female student presented to the urology clinic with fatigue, peripheral edema, dysuria, and hematuria, these symptoms having begun recently. She had a history of renal stone. The urologist ordered an abdominal and pelvic computed tomography scan with intravenous and oral contrast. In the computed tomography, moderate ascites, right-sided pleural effusion, and pericardial effusion were seen (Fig. 1).

She was admitted to the Emergency Department of the Shahid Rahimi Hospital of Lorestan, Iran. In the complete history, she reported dyspnea on exertion and weight loss in the past 1 month, but she had ignored them. She had no history of cough, chest pain, night sweats, orthopnea, tuberculosis (TB), diabetes mellitus, or immunosuppression. There was no personal or family history of contact with or an infection with *Mycobacterium tuberculosis*. She denied any recent chest infection.

On physical examination she seemed well and comfortable. She could talk well and answer questions completely. She did not have dyspnea at rest. On examination, at the time of admission, she was stable. Evaluation of the vital signs showed a temperature of 37 °C, heart rate 110 beats/min, blood pressure 110/80 mmHg, and respiratory rate 18 breaths/min.

Jugular venous pressure was raised. The respiratory sounds were normal in the left hemithorax but were decreased in the right hemithorax. The heart sounds were muffled. Also, she had paradoxical pulse and 1+pitting edema in the lower limbs. Electrocardiogram at the time of admission revealed sinus tachycardia and low voltage of the QRS complex. QRS is the second wave in a normal electrocardiogram and it shows the depolarization of the ventricles of a heart (Fig. 2).

On the initial echocardiogram, in apical four-chamber view, left ventricular ejection fraction was 65% and good, a 3.6-cm pericardial effusion was seen, and echocardiographic tamponade was detected. Additionally, the right ventricle was collapsed in diastole (Fig. 3).

Based on the massive pericardial effusion, emergency pericardiosynthesis and thoracoscopic pericardial window were recommended; 2.5 L of blood-stained fluid was drained. The result of the pericardial effusion analysis showed a white blood cell count of 5.34×10^6 cells/L including 5%

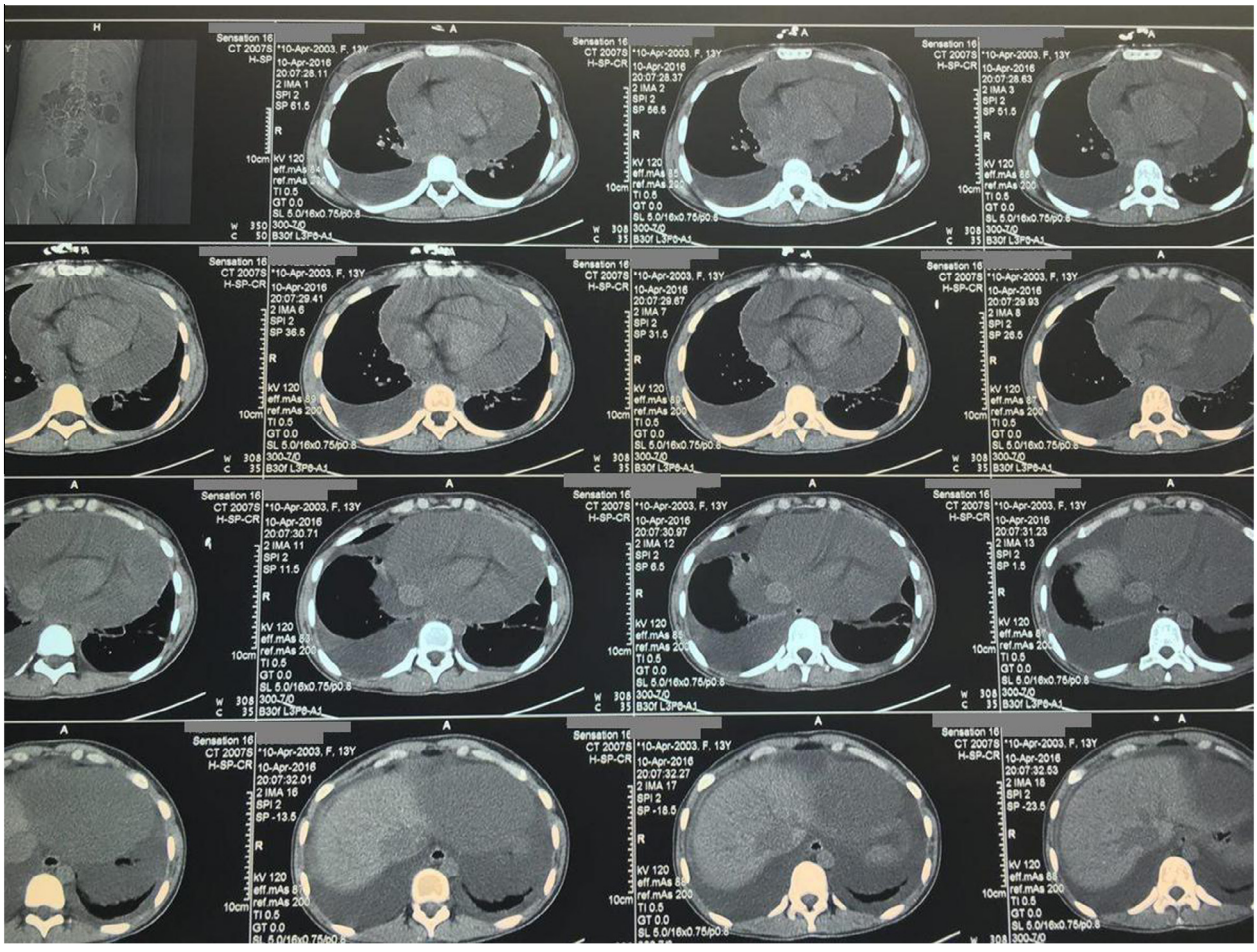

Fig. 1 – Abdominal and pelvic computed tomography scan with intravenous and oral contrast showing moderate ascities, right-sided pleural effusion, and massive pericardial effusion.

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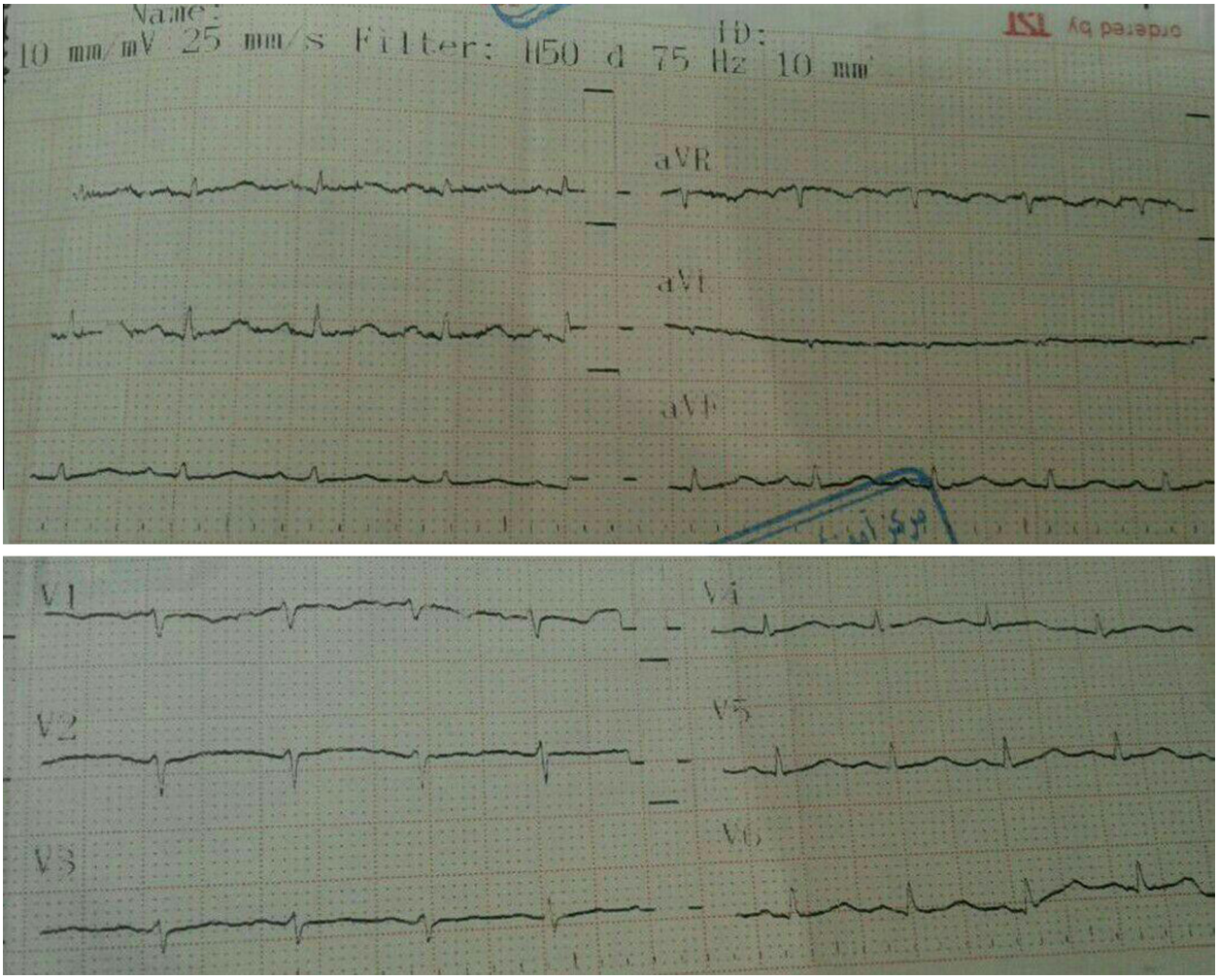

Fig. 2 – Electrocardiogram at the time of admission showing sinus tachycardia and low voltage QRS complex.

polymorphonuclear leukocytes, 94% lymphocytes, and 1% monocytes, and the red blood cell count was 1.9×10^9 cells/L. Adenosine deaminase (ADA) in pericardial fluid was 80 U/L.

Because of the presentation of pleural effusion, thoracosynthesis was recommended. Pleural fluid was exudative and the results of the analysis indicated that the white blood cell count was 4.93×10^6 cells/L including 7% polymorphonuclear leukocytes, 92% lymphocytes, and 1% monocyte. The red blood cell count was 1.398×10^8 cells/L.

In initial blood tests, white blood cell count was 1.11×10^7 cells/L (differential: neutrophils, 77%; lymphocytes, 15%; monocytes, 1%; and band cells, 7%), hemoglobin was 10.9 g/dL and the erythrocyte sedimentation rate was 51 mm/h. Urinary analysis and creatinine were normal.

The workup for TB was negative, as were the skin test, acid-fast bacilli smear, and fluid polymerase chain reaction. The histological examination was consistent with the chronic reaction and no granuloma was observed. Pericardial fluid culture was not available. Other causes of pericardial effusion were ruled out.

Later in the hospital, the temperature increased to 38 °C. Pericardial TB was considered as a clinical diagnosis because of the pleural effusion, ascites, hemorrhagic pericardial effusion, and the systemic symptoms (including weight loss and peripheral edema) and ADA of 80 U/L, and lymphocyte dominancy in the pericardial fluid. Standard TB treatment was initiated with isoniazid, rifampicin, pyrazinamide, and ethambutol. Additionally, prednisolone was added to the therapy.

Her clinical condition improved rapidly during the hospitalization. The temperature became normal and pericardial and pleural effusion decreased. The clinical response to the therapy was excellent and she was discharged after 10 days. Electrocardiogram at the time of discharge revealed normal QRS complex (Fig. 4).

The patient's response to the treatment was excellent. Follow-up echocardiography after 8 weeks showed only mild pericardial effusion (0.6 cm) with no evidence of constructive pericarditis (Fig. 5). Chest radiography was normal. Also the erythrocyte sedimentation rate decreased to the normal range (18 mm/h). During 12 weeks of follow-up, we did not detect any recurrence of symptoms or signs of deterioration. There were no adverse reactions to the drugs observed and she had gained weight.

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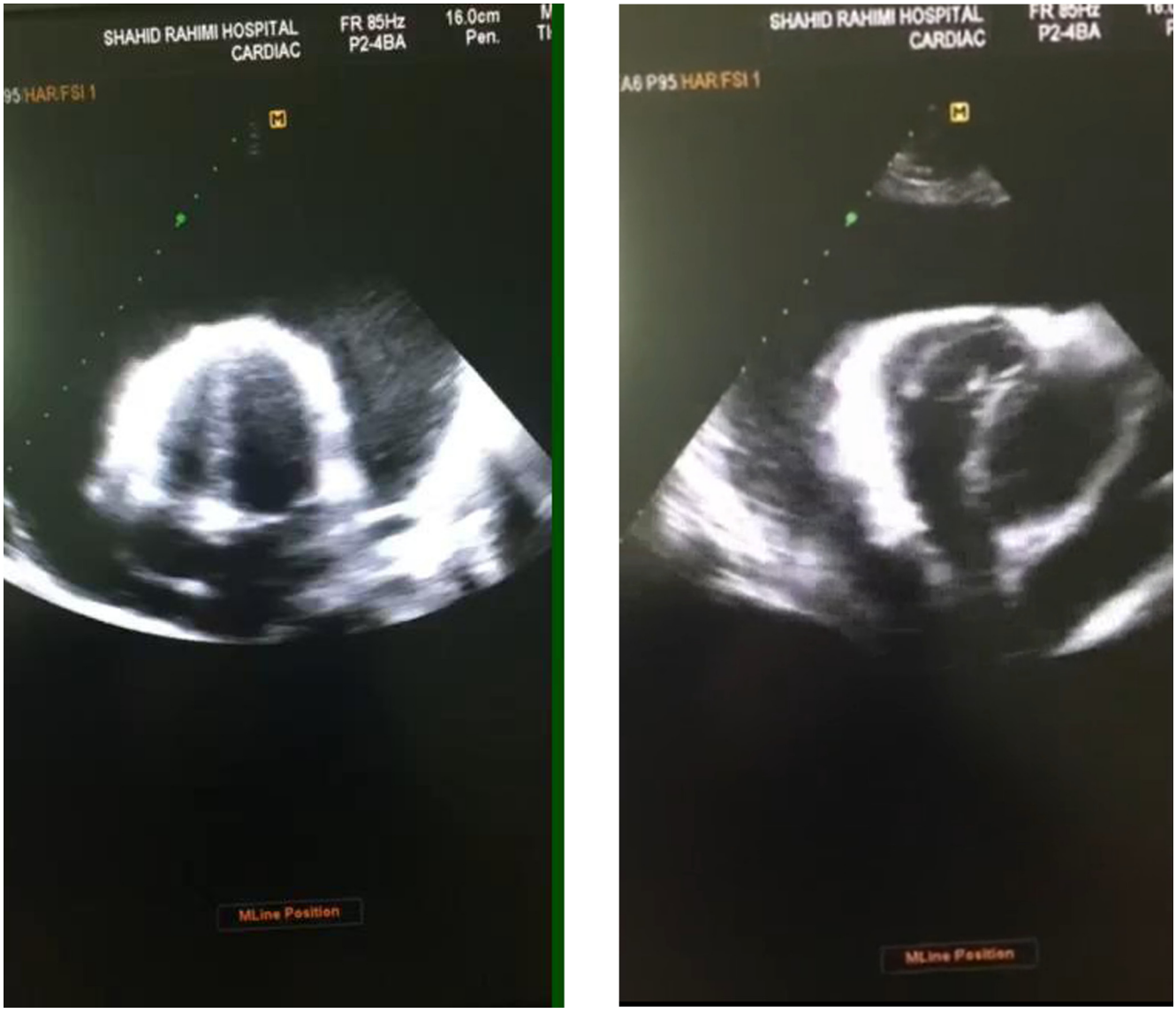

Fig. 3 – Transthoracic echocardiography at the time of admission, showing good left ventricular ejection fraction (65%) and a large pericardial effusion (3.6 cm) and a complete collapse of the right ventricle.

Discussion

Although there has been a consequential decrease in TB in wealthy industrialized countries over the past decades, Africa, Asia, and Latin America with 86% of the world's population, have 95% of all cases of active TB and 98% of nearly 2 million deaths from this disease each year [1]. Pericardial TB is often seen in old patients in countries with low TB prevalence. It is also diagnosed regularly in human immunodeficiency virus-infected patients [2]. Pericardial TB has also been diagnosed with immunosuppressive therapy, such as patients who undergo renal transplants [3]. The reported patient was young, human immunodeficiency virus-negative, immunocompetent, and without any history of pulmonary infection or a family history of exposure to *M. tuberculosis*.

The clinical signs and symptoms of pericardial TB are variable and nonspecific. Symptoms may include fever, weight loss, fatigue, and night sweats and more common symptoms are cough, shortness of breath, and chest pain [4]. Patients may present with cardiac tamponade [5], but can also present with heart failure as a result of chronic cardiac compression [4]. The patient was afebrile when admitted to the hospital and her fever increased during hospitalization. She had weight loss (5 kg) in the past month, but had ignored it during this time.

Cardiac tamponade happens when the fluid in pericardial space collects more quickly in comparison to the stretching of the pericardial sac, it causes high pressure and may compress the heart. This high pressure can prevent the heart from expanding completely. When cardiac trauma occurs, blood can fill the space immediately, and a small amount of fluid

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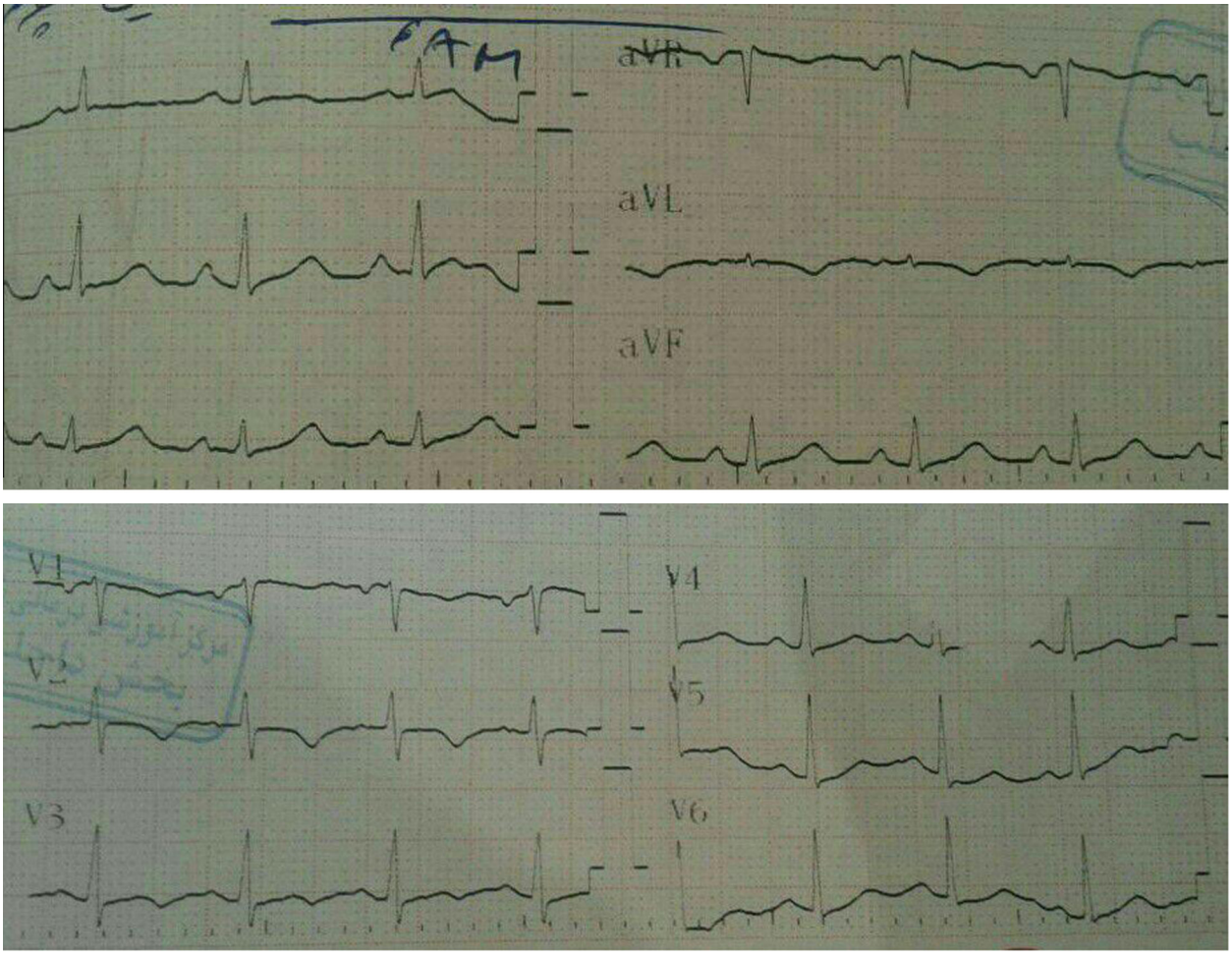

Fig. 4 – Electrocardiogram at the time of discharge showing normal QRS complex.

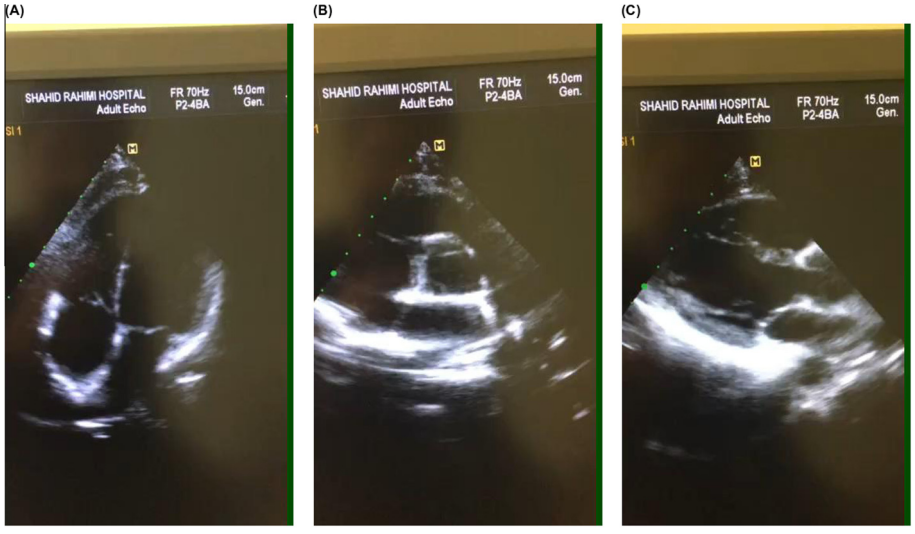

Fig. 5 – (A–C) Transthoracic echocardiography after 8 weeks' follow-up, showing mild pericardial effusion (0.6 cm) without respiratory variation and no evidence of constrictive pericarditis (septal bounce or hemodynamic effect).

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as 100 mL can cause cardiac tamponade. However, in the slow collection of the fluid in the pericardial space, as we observe in pericardial TB, the pericardial sac can expand to hold >1 L of fluid before serious compression arises [6]. We should definitely treat these patients with a pericardial window [7].

Mayosi et al. [4] stated: "The diagnosis is established by detection of tubercle bacilli in smear or culture of pericardial fluid and/or by detection of tubercle bacilli or caseating granulomata on histological examination of the pericardium." Stout [8] said: "Tuberculous pericarditis is considered likely in the setting of pericarditis with TB demonstrated elsewhere in the body, lymphocytic pericardial exudate with elevated ADA level, and/or clinical response to antituberculous therapy."

Acid-fast bacilli may be present in the pericardial fluid smear of 40–60% of the pericardial TB cases [9–11]. Histology findings are frequently nonspecific; in one prospective series including 78 patients with tuberculous pericarditis, characteristic granulomatous changes on histopathology were observed in 53% of cases [10].

Elevation of ADA activity $\geqslant$40 U/L is diagnostic with 87% sensitivity and 89% specificity [11]. The positive results from the cultures of pericardial effusion are seen in only 55–93% of the patients with pericardial TB [12,13].

For a diagnosis of pericardial TB, the sensitivity of pericardial biopsy ranges from 10% to 64% [14,15]. Therefore, pericardial TB cannot be ruled out with a normal pericardial biopsy specimen; in some cases, the examination of the full pericardium is required to establish the diagnosis [7]. For patients in areas where TB is endemic and who are highly suspicious of pericardial TB, pericardial biopsy is not required before the initiation of empiric anti-TB therapy [4].

Although the initial workup for TB was negative, according to the patient's symptoms, living in an endemic area for TB, and the high level of ADA, the patient was started on empirical anti-TB treatment irrespective of test results. The response to treatment was a valuable diagnostic argument in this case.

Conclusion

Pericardial TB is a rare presentation of TB. It may be life-threatening, so early diagnosis is very important. In areas endemic with TB, we recommend initiating empirical therapy as soon as possible even with negative workup of TB or even if reliable laboratory results are not available.

Conflicts of interest

All authors have no conflicts of interest to declare.

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