J Nepal Health Res Counc 2020 Oct-Dec;18(49): 795-7

DOI: https://doi.org/10.33314/jnhrc.v18i4.2305

Tubercular Pericardial Effusion Presenting as Cardiac Tamponade

Alok Pradhan,[1] Ranjit Babu Jasaraj,[2] Bhesh Raj Karki,[3] Anish Joshi[4]

[1]Department of General Practice and Emergency Medicine, Dhulikhel Hospital, Dhulikhel, Nepal, [2]Department of Internal Medicine, Mount Sinai Hospital Medical Center of Chicago, Chicago, USA, [3]Department of Internal Medicine, SUNY Downstate Health Sciences University, New York, USA, [4]Department of Pediatrics, Dhulikhel Hospital, Dhulikhel, Nepal.

ABSTRACT

Pericardial effusion is an uncommon extra-pulmonary manifestation of tuberculosis, tamponade being even rarer. Here, a 14-year female presented with cough, chest pain and fever. She had raised jugular venous pressure, hypotension, and muffled heart sound, suggestive of cardiac tamponade, confirmed by echocardiogram. She underwent pericardiocentesis with continuous pericardial fluid drainage. Her jugular venous pressure normalized after the aspiration. The high adenosine deaminase level in pericardial fluid analysis was suggestive of tuberculosis for which she was treated with antitubercular therapy and steroid. This case highlights the importance of adenosine deaminase for diagnosing the etiology of a rare presentation.

Keywords: Adenosine deaminase; echocardiography; pericardial effusion; tamponade; tuberculosis

INTRODUCTION

Tuberculosis (TB) is a major public health problem affecting around 10 million people worldwide in 2018, with 1.1 million cases in children.[1] South Asian countries carry more than a third of the global TB burden. In Nepal, more than 30,000 cases were reported in 2017/2018 of which 71% were pulmonary TB.[2] Tuberculosis is a curable and preventable infectious disease caused by an acid fast bacillus Mycobacterium tuberculosis. Patients with pulmonary TB commonly present with cough, fever, weight loss and night sweat. Extra-pulmonary TB commonly involves lymph nodes and pleura while cardiovascular system rarely. Cardiac tamponade is even rarer and was reported only in 10 out of 53 patients having cardiac manifestations of TB.[3] Here, we present a case of a 14-year girl who developed tamponade secondary to tubercular pericardial effusion.

CASE REPORT

A 14-year fully-immunized girl presented to emergency room with complaints of chest pain and productive cough for two weeks associated with two episodes of blood-stained sputum, and fever for three days associated with sweating, chills and rigor. She had no history of TB contact.

She looked ill, weighed 40.8 kg (between 10th and 25th percentile) and measured 154 cm (between 10th and 25th percentile). She was tachycardic (130 beats/min), tachypneic (44/min), febrile (38.2^{0}C), hypotensive (80/50mmHg) and hypoxic (SpO_2-88%, improved to 92% with supplemental oxygen). She had prominent neck veins and muffled heart sounds. Her jugular venous pressure was elevated to 10 cm of water. There was bilateral decreased air entry with basal crepitations. She also had tender hepatomegaly and bilateral pitting edema.

Electrocardiogram showed sinus tachycardia, low voltage complexes and electrical alternans. Echocardiography revealed pericardial effusion with echogenic strands and diastolic collapse of right heart (Figure1).

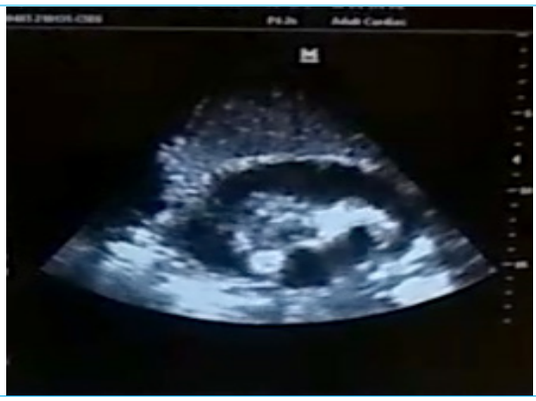

Figure 1. Pericardial effusion and right ventricular collapse in screening echocardiography.

Correspondence: Dr Alok Pradhan, Department of General Practice and Emergency Medicine, Dhulikhel Hospital, Dhulikhel, Nepal. Email: aalokpradhan@gmail.com, Phone: +9779842030577.

The blood investigations showed leukocytosis (32,000/mm^3) with neutrophilia (89%) and raised erythrocyte sedimentation rate (40mm/hour). Chest radiography showed cardiomegaly with blunting of bilateral costophrenic angles (Figure2).

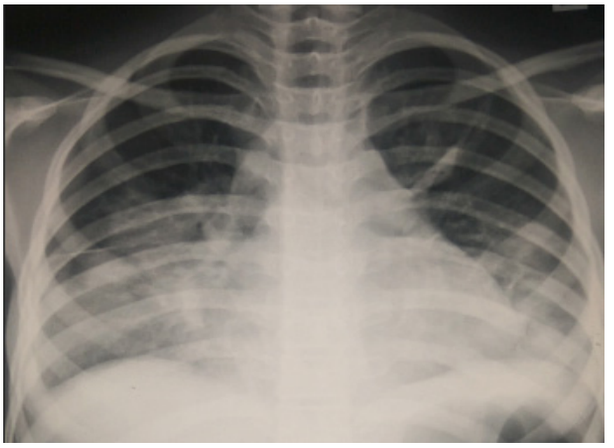

Figure 2. Chest x-ray showing cardiomegaly with blunting of bilateral costophrenic angles.

The child was shifted to pediatric intensive care unit; pericardiocentesis was performed, aspirating 240 ml of turbid purulent fluid (Figure3).

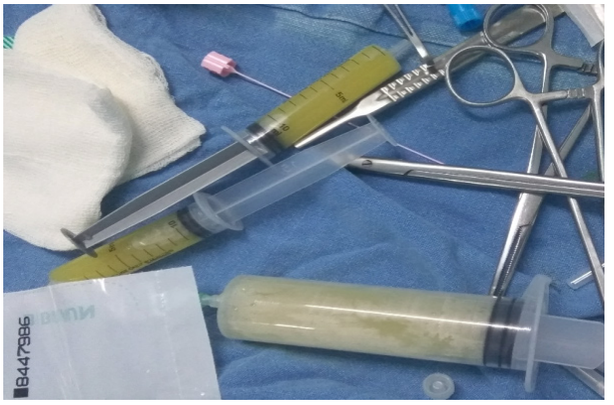

Figure 3.Turbid to purulent pericardial fluid aspirate during pericardiocentesis.

Ultrasound guided pericardial catheter was inserted through subxiphoid approach and kept in situ for further drainage of the fluid. Intravenous ceftriaxone two grams twice a day and amikacin 300 milligrams once a day were given for seven days. She was started on dopamine infusion for hypotension at 12 mcg/kg/min, tapered slowly and stopped on the fourth day. Pericardial fluid analysis showed leukocytosis (38,000/mm^3) with predominant neutrophils (60%) and lymphocytes (40%), high protein (4.8mg/dl), high lactate dehydrogenase (14,330U/L) and low glucose (<10mg/dl). Adenosine deaminase (ADA) was elevated (114U/L). The pericardial fluid culture, however, did not show any growth.

In view of tubercular pericardial effusion, antitubercular therapy (ATT) was started on the third day with daily three fixed dose combination tablets [Isoniazid (75 mg), Rifampicin (150 mg), Pyrazinamide (400 mg) and Ethambutol (275 mg)]. Oral prednisolone (60 mg) was also started on the same day. The child became afebrile on 4th day of admission and repeat fluid culture from the same day showed no growth. Pericardial fluid drainage was continued till the seventh day, when the catheter was removed (total aspirate-1480 ml).

Repeat echocardiography before discharge showed minimal pericardial effusion and thickened pericardium. So, we consulted cardiothoracic vascular surgery, who recommended pericardiotomy and this plan was discussed with the parents. The patient was discharged on ATT and prednisolone (60 mg), and scheduled for follow up after two weeks.

DISCUSSION

Our patient had hypotension, raised jugular venous pressure and muffled heart sounds (Beck's triad) on presentation. Thus, the echocardiography was performed in view of cardiac tamponade, which was confirmed. It was challenging to establish the etiology of the tamponade. A systemic review showed that the etiology of pericardial diseases could not be established in 15-20% of cases.[4]

Although tuberculosis is a major public health problem in Nepal, the girl had fever for only three days, and did not have any weight loss and contact history. Antinuclear antibody and thyroid function test were normal. Echocardiographic finding of fibrinous strands was the initial imaging clue for tuberculosis as the etiology. Presence of fibrinous strands on the visceral pericardium is typical but not specific to tuberculous pericardial effusion.[5]

The diagnosis of tubercular pericarditis is done either by detection of tubercle bacilli in pericardial fluid analysis and/or on histological examination of the pericardium or by presence of TB elsewhere in a patient with otherwise unexplained pericarditis, a lymphocytic pericardial exudate with elevated ADA level and/or appropriate response to ATT.[6] We could not find focus of TB elsewhere. The pericardial fluid was exudative with predominance of neutrophils rather than the lymphocytes. However, tubercular pericardial fluid is predominantly lymphocytic. The AFB smear of the pericardial fluid did not show any bacilli; neither did the

culture reveal any growth. We were not able to perform the histological examination of the pericardium.

The major lab investigation supporting TB as the etiology was the pericardial fluid ADA level which was measured to be 114 U/L. An ADA level greater than 40 U/L has a sensitivity of 90% and a specificity of 86%.[7] When the threshold is decreased to 35 U/L, the sensitivity increases to 95.7% and specificity decreases to 84%.[8] Although both the sensitivity and specificity for Interferon Gamma Release Assay (IGRA) has been demonstrated to be 94%[9] this modality of investigation was not available in our centre. There is also a positive correlation between high ADA value and development of constrictive pericarditis suggesting its prognostic value.[10]

ADA is a ubiquitous enzyme present in all cells that catalyses the deamination of adenosine and deoxyadenosine into inosine and deoxyinosine respectively. We want to highlight the role of ADA in diagnosis of tubercular pericardial effusion in resource limited setting like ours, where evaluation of IGRA level in pericardial fluid, its culture and histological examination of the pericardium may not be possible in every hospital. Thus, a strong clinical suspicion along with high ADA can be a strong supportive evidence for tuberculous pericarditis; and prompt initiation of ATT can prevent further complications like cardiac tamponade and constrictive pericarditis.

CONCLUSIONS

Pericardial tamponade is a rare presentation of tuberculosis even in developing country like ours where tuberculosis is a common illness. Our case emphasizes the role of ADA in establishing TB as the etiology of pericardial effusion and, hence allowing timely intervention.

REFERENCES

1. World Health Organization. Global tuberculosis report. 2019. https://apps.who.int/iris/bitstream/handle/10665/329368/9789241565714-eng.pdf?ua=1
2. National Tuberculosis Program Nepal. Annual Report. 2018.[Download PDF]
3. Shakya S, Jha SC. Cardiac manifestations of tuberculosis in a tertiary care center of Nepal. Nepal Heart J. 2018 May 8;15(1):35–8.[Article]
4. Noubiap JJ, Agbor VN, Ndoadoumgue AL, Nkeck JR, Kamguia A, Nyaga UF, et al. Epidemiology of pericardial diseases in Africa: a systematic scoping review. Heart. 2019 Feb 1;105(3):180–8.[Article]
5. Liu PY, Li YH, Tsai WC, Tsai LM, Chao TH, Yung YJ, et al. Usefulness of echocardiographic intrapericardial abnormalities in the diagnosis of tuberculous pericardial effusion. Am J Cardiol. 2001 May 1;87(9):1133–5. [Article]
6. Mayosi Bongani M., Burgess Lesley J., Doubell Anton F. Tuberculous Pericarditis. Circulation. 2005 Dec 6;112(23):3608–16.[Article]
7. Xie DL, Cheng B, Sheng Y, Jin J. Diagnostic accuracy of adenosine deaminase for tuberculous pericarditis: a meta-analysis. Eur Rev Med Pharmacol Sci. 2015 Nov;19(22):4411–8.[Download PDF]
8. Pandie S, Peter JG, Kerbelker ZS, Meldau R, Theron G, Govender U, et al. Diagnostic accuracy of quantitative PCR (Xpert MTB/RIF) for tuberculous pericarditis compared to adenosine deaminase and unstimulated interferon-γ in a high burden setting: a prospective study. BMC Med. 2014 Jun 18;12:101.[Article]
9. Seo HT, Kim YS, Ock HS, Kang LH, Byun KS, Jeon DS, et al. Diagnostic performance of interferon-gamma release assay for diagnosis of tuberculous pericarditis: A meta-analysis. Int J Clin Pract. 2020 May;74(5):e13479. [Article]
10. Komsuoglu B, Göldeli Ö, Kulan K, Komsuoglu SS. The diagnostic and prognostic value of adenosine deaminase in tuberculous pericarditis. Eur Heart J. 1995 Aug 1;16(8):1126–30.[Article]