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Case Report

Echocardiographic Findings of Tuberculous Pericardial Effusion in Early Stage of Chronic Kidney Disease

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ABSTRACT:

Pericardial Effusion (PE) is a common clinical entity that is routinely diagnosed by echocardiography. The presenting syndrome depends on the volume, rate of accumulation and characteristics of fluid. Tuberculous Pericardial Effusion (TPE) is an accumulation of fluid in the pericardial cavity with Mycobacterium tuberculosis (MTB) in the immunocompromised patients and it is uncommon form of extra-pulmonary tuberculosis being increasingly found with common symptoms of chest pain, dyspnea, weight loss, general weakness. In this case report patient is in early stage CKD, 2D-Echocardiogram revealed a large pericardial effusion with fibrin strands. Usually in many cases pericardial effusion occur in end-stage renal failure but in this case pericardial effusion was seen in early stage. In endemic areas mostly MTB may cause PE in early stage of chronic renal failure. The patient was treated and responded well with ATT and other supportive therapy.

KEYWORDS: Pericardial Effusion, Mycobacterium tuberculosis, Chronic Kidney Disease, 2D-Echocardiogram.

INTRODUCTION:

Pericardial Effusion is a common clinical entity that is routinely diagnosed by echocardiography. The presenting syndrome depends on the volume, rate of accumulation and characteristics of fluid.[1] Tuberculous Pericardial Effusion (TPE) is an accumulation of fluid in the pericardial cavity with Mycobacterium tuberculosis (MTB) in the immunocompromised patients and it is uncommon form of extra-pulmonary tuberculosis being increasingly found [2] with common symptoms of chest pain, dyspnea, weight loss, general weakness. The pericardium is a relatively avascular fibrous sac that surrounds the heart. It consists of 2 layers: the visceral and parietal pericardium. The 2 layers of the pericardium are separated by a potential space that can normally contain 15 to 35 mL of serous fluid distributed mostly over the atrial-ventricular and interventricular grooves. Because of the limited amount of space in the pericardial cavity, fluid accumulation leads to an increased intrapericardial pressure which can negatively affect heart function. The pericardium may also function as a barrier to infection.[3-4]The most common causes of moderate to large pericardial effusions are iatrogenic, infections, malignancy, chronic idiopathic effusion, post-acute myocardial effusion, autoimmune diseases, radiation, renal failure with uremia and hypothyroidism. [5-6] The most frequent causes of hemorrhagic pericardial effusion are malignancy, post-procedural (transcatheter interventions and pacemaker insertion), post-pericardiotomy syndrome, aortic dissection and trauma. In addition, tuberculosis is frequent cause of hemorrhagic effusion in endemic areas. [7-11] Pericardial involvement usually develops by retrograde lymphatic spread of *MTB* from peritracheal, peribronchial, or mediastinal lymph nodes or by hematogenous spread from primary tuberculous infection. [12-13]

Severities of Symptoms (dyspnea) vary according to the amount of pericardial fluid which may vary from 150ml to 1000 ml. [14] Echocardiographyis the most useful and confirmatory as one sees echo-free space between two pericardial layers and patchy deposits 4-8mm in thickness with fibrinous strands criss-crossing the pericardial space. [15]

The diagnosis is confirmed by history of low-grade fever, malaise, loss of appetite, loss of weight, cough, dyspnea and

clinical signs of pericardial effusion. Raised ESR, elevated absolute lymphocyte count, lymphocytic exudative nature of pericardial fluid culture of MTB, pericardial fluid PCR, estimation ADA and IFN-ɣ levels in pericardial fluid help in confirming the TB as etiology of pericardial effusion. [16]

Pericardiocentesis is indicated if malignancy, bacterial, mycobacterial, or fungal pericardial effusion is suspected. It is indicated in the setting of large pericardial effusion with associated pericardial tamponade with large effusions of recent onset, close clinical and echocardiographic follow-up is warranted. [17]

Anti-tuberculous treatment (ATT) with primary four drugs (rifampicin, isoniazid, pyrizinamide and ethambutol) is given for first two months. Later pyrizinamide isomitted and the remaining three drugs are given. Total 9-12 months of treatment is recommended with periodic follow-up of symptoms and signs of pericardial effusion, ESR, X-ray chest, ECG and 2D-echo till the complete disappearance of fluid and other clinical features. [18] High doses of steroids decrease the incidence of pericardial constriction and mortality following TPE. [19]

CASE REPORT

A 58years old male patient presented with a 4 days history of shortness breath, mild chest pain, fever and having past medical history of chronic kidney disease (CKD) associated with anemia and Chronic obstructive pulmonary disease (COPD) from past one year and he was on calcitriol OD, calcium carbonate 500mg BID, ferrous fumerate BD for CKD and for hypertension metoprolol 50mg BD, prazosin 5mg OD, for COPD salbutamol theophylline, prednisolone 80mg.

On physical examination he was thin, cachetic and febrile with respiratory rate of 30 breaths/minute, blood pressure 180/90 mmHg and pulse rate 85 beats/minute.

The laboratory studies revealed hemoglobin level 8g/dl, and ESR 140mm/1sthour, the other findings of blood were normal.

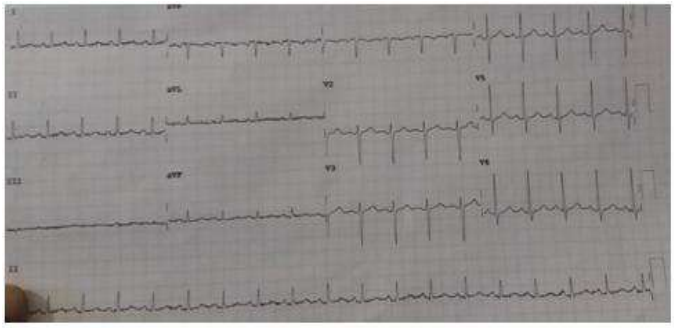
Liver function tests revealed Total bilirubin-1.4mg/dl, direct bilirubin-0.4mg/dl, AST-75U/L, ALP-59U/L. Total protein, albumin and globulin level were normal.

Renal profile revealed that blood urea was 69mg/dl (slightly elevated) and serum creatinine found to be 3.1mg/dl (abnormal).

ECG:

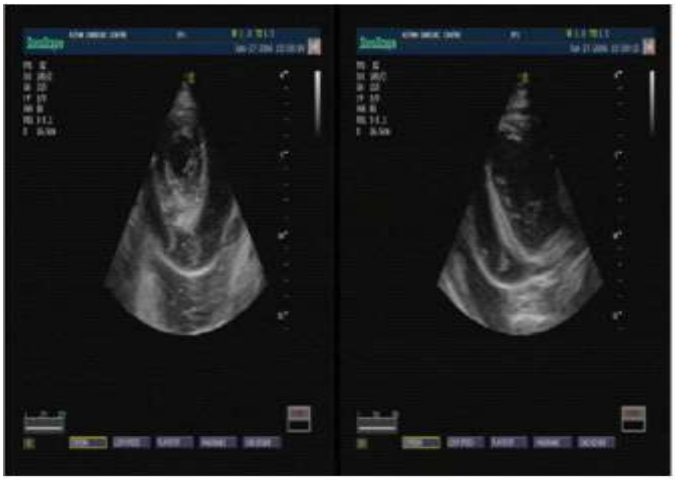
Figure-1: Sinus tachycardia with low voltage complexes

Chest X-ray showed cardiomegaly.



Echocardiogram revealed a large pericardial effusion with fibrin strands. Valves and chambers were normal.

Figure-2: Large pericardial effusion with fibrin strands



Treatment advised during hospital stay was furosemide 20 mg stat, infusion of nitroglycerin1 ampoule in 50ml NS at 0.3ml/hr, ceftriaxone 1g BD, and paracetamol 650mg TID, ATT, prednisolone 10mg OD, pyridoxime 40mg OD were started immediately.

Patient underwent pericardiocentesis and about 800ml of pericardial fluid tapered by pericardiocentesis process. Pericardial fluid color was hemorrhagic and turbid.

On the next day ADA levels in pericardial fluid was 23U/L. Pericardial fluid contains plenty of RBC, cell count was 100cells/cmm, N-10% L: 90% sugar-121mg/dl, and no microorganisms were found.

The other diagnostic tests like Mantoux test, hepatitis-C and B, HIV I and II, Anti-nuclear antibody (ANA) test were detected to be negative.

From day 1 to day 4, vital signs were monitored and patient relieved from SOB.

On day-5, pericardiocentesis sheath was removed and patient was stable.

On day-6, patient had complaints of palpitations and ECG shows paroxysmal atrial fibrillation which is controlled with bolus dose of amiodarone 300mg stat at 4ml/hr.

On day 8, patient discharged in stable conditions.

DISCUSSION:

Tuberculous pericardial effusion though uncommon is being

increasingly found, especially in immunosuppressed persons. Thickened pericardium and fibrinous strands on echocardiography are suggestive of infective etiology. Tuberculous pericarditis can present without any previous history of exposure to tuberculosis. [2] It is usually seen in predialysis patients rather than in patients who are undergoing maintenance renal replacement therapy. [20] This study has shown that, pericardial disease was common in patients with chronic kidney disease (CKD) at first presentation to a nephrology unit before the commencement of dialysis. This was evident as early as stage 3 CKD. Pericardial thickening was more prevalent than pericardial effusion. Echocardiography to detect pericardial disease should be part of routine investigation of patients with CKD. [21]

CONCLUSION:

In this case report patient is in early stage CKD with normal urine output, blood urea was 69mg/dl (slightly elevated), and serum creatinine was found to be 3.1mg/dl (abnormal), and also all other diagnostic tests were found to be normal, 2D-Echocardiogram revealed a large pericardial effusion with fibrin strands. Usually in many cases pericardial effusion occur in end-stage renal failure but in this case pericardial effusion was seen in early stage. So in endemic areas mostly mycobacterium tuberculosis may cause PE in early stage of chronic renal failure. The patient was treated and responded well with ATT and other supportive therapy.

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