# A Rare Case of Massive Pericardial effusion as the first presentation of Hypothyroidism

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***Abstract*—** One of the multiple clinical manifestations associated with hypothyroidism is pericardial effusion. Pericardial effusions are commonly seen in cases of severe hypothyroidism, however large to massive pericardial effusions with cardiac tamponade are exceptionally rare. We report an atypical case that was diagnosed to have massive pericardial effusion and early echocardiographic features of cardiac tamponade secondary to primary hypothyroidism. Following repeated pericardiocentesis and thyroid replacement therapy, eventually the patient's symptoms improved with no recurrence of pericardial effusion and is on regular follow-up.

***Index Terms* — HYPOTHYROIDISM, PERICARDIAL EFFUSION, CARDIAC TAMPONADE, PERICARDIOCENTESIS, THYROXINE, ECHOCARDIOGRAPHY.**

## Introduction

Hypothyroidism is an endocrine disorder with multiorgan involvement and various complications. One of the significant but less often seen complications is pericardial effusion [1,2] . Since it can progress to life-threatening conditions like cardiac tamponade and hemodynamic instability, early diagnosis, and management of the pericardial effusion in hypothyroidism is a must [3,4,5]. Echocardiography is the gold standard diagnostic test with a very high sensitivity and specificity in the diagnosis of pericardial effusion [6].

## Case Report

A 13-year-old girl had a history of gradually increasing breathlessness, difficulty in swallowing, abdominal fullness, generalized weakness, and decreased appetite for 2 months. She had no history of fever, chest pain, vomiting, arthralgia or recent travel. Her psychomotor development was normal. Vital signs on admission were temperature 99.4 $^{O}$F; blood pressure 96/60 mmHg; heart rate 110/min; respiratory rate 30/min. Physical examination found coarse hair, dry skin, pallor, muffled heart sounds. Chest X-ray showed globular enlargement of the cardiac silhouette. (Figure 1). An electrocardiogram revealed low P wave and QRS complexes (Figure 3). An echocardiogram demonstrated normal heart size but with massive pericardial effusion and signs of early systolic right atrium (RA) and early diastolic right ventricle (RV) collapse (Figure 2).

The following blood investigations were conducted on the patient. The complete hemogram showed Hb: 10.9 gm% (N: 12-14 gm%); TLC: 8,100 cells/cu.mm (N: 4,000-11,000); DLC: P: 64% (N: 60-70%), L: 30% (N: 20-30%), E: 02% (N: 0-5%); ESR : 14 mm at 60 mins (N: 0-20 at 60 mins); platelets: 2.60 lakhs/cu.mm (N: 1.5-4.5 lakhs/cu.mm); bleeding time: 3 mins (N: 2- 4 mins); clotting time : 4.30 min (N: 5-7 mins); urea: 20 mg/dL (N: 20-40 mg/dL); serum creatinine: 0.6 mg/dL (N: 0.7-1.2 mg/dL); serum cholesterol: 112 mg/dL (N: 150-200 mg/dL); triglycerides: 78 mg/dL (N: <150 mg/dL); HDL: 38 mg/dL (N: 30-45 mg/dL); LDL: 102 mg/dL (<100 mg/dL); liver function tests : normal study. The thyroid profile of the patient showed - T3: 0.19 ng/dL (N: 0.9 -1.690 ng/dL); T4: 0.12 µg/dL (N: 5-12 µg/dL); TSH: 142 mIU/mL (N: 0.35-5.5 mIU/mL).

Figure (1)

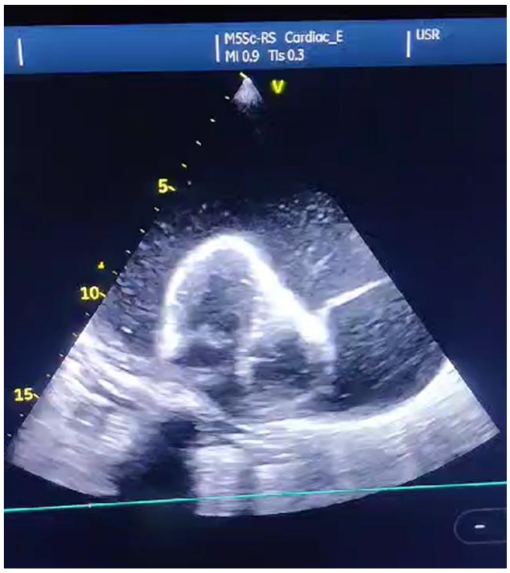


Figure (2)

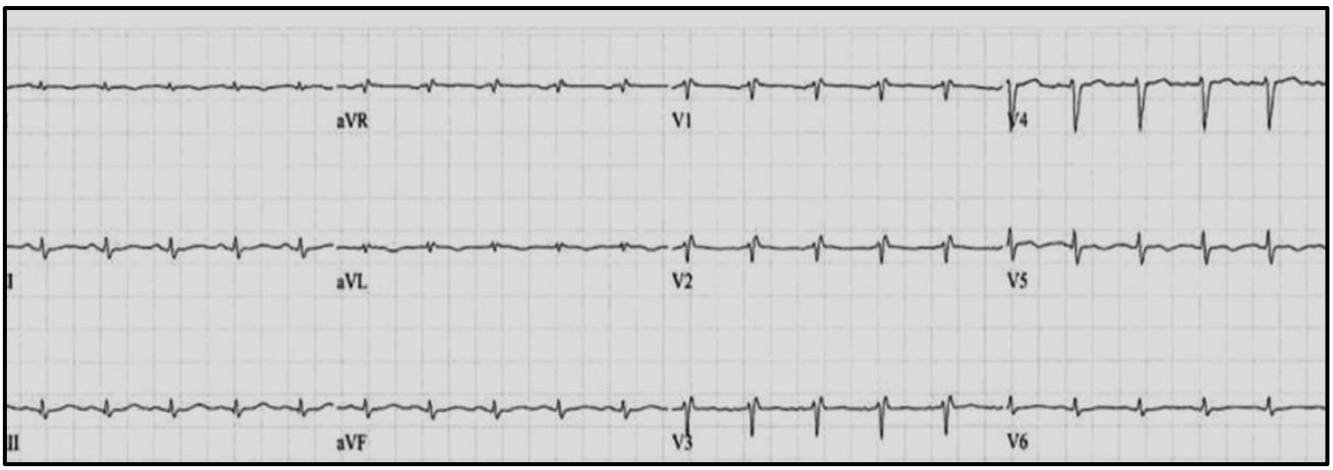


Figure (3)

**Figure (1) Chest X-ray PA view showing massive cardiomegaly with normal lung fields**
**Figure (2) Echocardiography showing large pericardial effusion**
**Figure (3) ECG showing low voltage complexes**

Cardiac tamponade was diagnosed and pericardiocentesis was done via sub-costal approach using 6F pigtail catheter which was left in situ. Examination of sputum and Mantoux test were negative. Cytological, microbiological and biochemical examination of the aspirated fluid were inconclusive other than increased protein content and a predominance of lymphocytes in the differential count. Cytological examination were total cells of 1250 cells ( 40% PMNs & 60% Lymphocytes). Biochemical examination were glucose 59 mg/dL, protein 4.8 gm/dL, LDH 637 U/L. Smear examination of pericardial fluid by Gram stain and Ziehl–Neelsen stain failed to demonstrate the presence of bacteria or tubercle bacilli. Cultures of the pericardial fluid were negative for *Mycobacterium tuberculosis*. The activity of adenosine deaminase (ADA) was not elevated in pericardial effusion. All other causes of cardiac tamponade except hypothyroidism were ruled out.

After the fluid aspiration the patient symptoms improved, dyspnea was reduced. A repeat X-ray had showed the size of the heart had decreased. Patient was started on Tab. Levothyroxine 100 mcg initially. However, there was a persistent collection of pericardial fluid and daily 200-350 ml fluid was being aspirated. CECT Thorax with Abdomen-Pelvis were done, suggetive of Pericardial effusion with mild ascites and mesenteric lymphadenopathy. Whole body PET-CT scan was done to rule out neoplastic etiology. No evidence of any active neoplastic etiology was detected on whole body PET-CT scan. Ultrasonography of the thyroid with a high frequency probe featured normal thyroid gland with normal size, echotexture, and normal vasculature.

A repeat echocardiography showed decreased in pericardial effusion. A chest x-ray taken at discharge (Figure 6) showed that the cardiac silhouette had returned to normal size with minimal pericardial effusion. The patient is currently receiving follow-up care through the outpatient department. When last seen recently, she was asymptomatic, X ray chest and 2D Echo were normal, and thyroxine dosage was adjusted.

**Figure (4)**

**Figure (5)**

**Figure (6)**

**Figure (4,5,6) Chest X-ray PA view showing subsequent resolving pericardial effusion with normal cardiac silhouette at discharge (Figure 6)**

## Discussion

Accumulation of transudate, exudate or blood in the pericardial sac is a complication of pericardial heart disease which results from several conditions; hypothyroidism is one of them [7,8]. Small Pericardial effusion is frequently found in patients with hypothyroidism whereas moderate to large PE is relatively rare. Massive Pericardial effusion (PE) with tamponade as an initial clinical presentation of primary hypothyroidism has been reported in literature. This low incidence is probably due to the pericardial distensibility and the slow accumulation of fluid, allowing significant fluid accumulation without hemodynamic compromise. Though heart failure is unusual in hypothyroidism but should be distinguished from tamponade because of similar presentations [9].

Tuberculous pericardial effusion should be considered as an important etiology in the evaluation of all cases of pericardial effusion without a rapidly self-limiting course. Its presentation may vary as purulent pericardial effusion with predominance of polymorphonuclear leukocytes and relatively abundant mycobacterial; serosanguinous effusion with predominantly lymphocytic exudates; absorption of effusion with pericardial thickening and ultimately fibrosis and constrictive pericarditis. Definite tubercular pericarditis can be diagnosed by one or more of the following criteria: (a) Isolation of *M. tuberculosis* from pericardial fluid or pericardial biopsy; (b) Demonstration of granulomatous inflammation on histological examination of pericardial biopsy; (c) Isolation of *M. tuberculosis* from sputum or non pericardial effusion exudates in the presence of clinical and/or radiological evidence of tuberculosis, associated with positive response to antitubercular therapy (ATT) and in the absence of any other obvious cause of pericardial effusion. In the present case we could isolate *M. tuberculosis* neither from smear examination nor from culture of the pericardial fluid. ADA activity was also not elevated.

Pericardial effusion in hypothyroidism is considered to be a part of the generalized polyserousopathy and the accepted pathogenic mechanism being a combination of extravasation of albumin and inadequate lymphatic drainage, accounts for the exudative nature of the accumulated fluid in this disorder [10,11]. The pericardial fluid in hypothyroidism is characteristically straw color due to high content of alpha and beta globulins with predominance of lymphocytes.[8] In our case, the fluid obtained in initial pericardiocentesis was serosanguinous with predominantly lymphocytic exudates and we consider trauma as a possible cause, but in second aspiration it was serous in nature. In our case diagnosis was based on clinical and echocardiography findings and hypothyroidism as a cause for pericardial effusion and tamponade was diagnosed by an exclusion criteria. The management varies depending on size of effusion and hemodynamic stability of the patient. Management strategy ranges from conservative management with close monitoring and thyroxine replacement to urgent pericardiocentesis or creation of a pericardial window.

The clinical resolution of pericardial effusion is satisfactory in majority of the hypothyroid patients with thyroxine replacement therapy, but it takes as long as months or years after the patient reach the euthyroid

state [12]. In our patient, at follow up her thyroid status was well improved and she had no symptoms. Although essentially treatment remains similar to other kinds of pericardial effusion with tamponade, identifying the primary disorder in a patient where other causes cannot be found out is very difficult. So, high degree of suspicion is necessary in which hypothyroidism is endemic, so as to give long lasting cure for the patient from this reversible but uncommon entity.

## Conclusion

Hypothyroidism is common problem in India. Though pericardial effusion and tamponade secondary to thyroid dysfunction are rare, in India we do get cases because of high burden of primary disease. Mild pericardial effusion respond to thyroxine supplementation over a long period. Pericardiocentesis is necessary only when it is massive pericardiocentesis and tamponade develops. Very rarely in recurrent pericardial effusion required surgical management such as pericardiectomy and/or pericardial pleural window is needed as in the present case reported.

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