Pericardial Effusion with Cardiac Tamponade as a form of presentation of Primary Hypothyroidism

Arun Agarwal1, Nikhil Chowdhury2, Ankit Mathur3, Samiksha Sharma4, Aakanksha Agarwal5

Abstract
Hypothyroidism is a rare cause of pericardial effusion (PE). Pericardial effusion secondary to hypothyroidism remains a diagnostic challenge for clinicians because of its inconsistency between symptoms and amount of pericardial effusion. We report an atypical case that presented with ascites and was diagnosed to have cardiac tamponade secondary to primary hypothyroidism. Besides repeated pericardiocentesis she eventually required surgical management and optimization of medical therapy to manage the massive pericardial effusion.

Introduction
Hypothyroidism can have atypical modes of presentations like pre-eclampsia in pregnancy, acute massive macrogllosis and pericardial effusion (PE) as a sole manifestation. Patients with massive amount of pericardial effusion secondary to hypothyroidism may be asymptomatic or have few symptoms. Cardiac tamponade as a complication of hypothyroidism is very rare. This is probably due to the slow accumulation of fluid and to cardiac distensibility. Pericardial effusion has a high concentration of protein and, like other serous effusions of hypothyroidism, its pathogenesis is not fully understood. Controversy exists regarding the form of drainage of cardiac tamponade which shall be discussed here.

Case Report
A 40 year female, known case of depression since 2003 and not a known case of hypothyroidism had a history of feverishness, gradually increasing abdominal distention, weakness, and decreased appetite for 2 months. She was initially admitted at a government hospital from 10.09.2015 to 16.09.2015. She was discharged on thyroxine 100 ug, diuretics, oleanzapine and supportive treatment with a diagnosis of anemia, hypothyroidism and high gradient ascites. She was later referred to our institution for further work-up in view of persistent abdominal symptoms. She had no history of shortness of breath, chest pain, vomiting, arthralgia or recent travel. Vital signs on admission were temperature 99.4°F; blood pressure 100/64 mmHg; heart rate regular at 110 beats/min; respiratory rate 20/min. There was no pulsus paradoxus. Physical examination found coarse hair, dry skin, elevated jugular venous pressure, muffled heart sounds, bilaterally decreased basal air entry in lungs, non tender hepatomegaly (2 cms below costal margin), moderate ascites, mild pitting edema of the legs bilaterally, and a non-palpable thyroid gland. Chest roentgenogram showed globular enlargement of the cardiac silhouette (Figure 1A). An electrocardiogram revealed low P wave and QRS complexes (Figure 1B). 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 1C). Cardiac tamponade was diagnosed and pericardiocentesis was done on 29.09.2015 via sub costal approach using 6F pigtail catheter which was left in situ. 700 ml of golden brown color fluid was drained. After the procedure, her heart rate decreased in the range of 50-60 beats/min and blood pressure remained around 100/60 mm Hg. Her hematology, biochemistry and other reports are mentioned in tables 1 and 2 respectively. The test for Dengue IgM antibody was also positive but she had no symptoms suggestive of Dengue fever. There were no features of capillary leak syndrome. The pericardial fluid bacterial culture and tuberculosis/non-tuberculosis (TB/NTB) real time PCR were negative. Blood, urine and pericardial fluid cultures, malarial antigen, scrub typhus IgM, serology and MT were negative. All other causes of cardiac tamponade except hypothyroidism were ruled out. Thyroxine dose was optimized. However, there was a persistent collection of pericardial fluid and daily 200-350 ml fluid was being aspirated. A repeat echocardiography on 08.10.2015 showed persistent moderate size PE, RA and RV collapse and thickened, shaggy visceral pericardium. NCCT and CECT chest were done (Figure 2A). Cardio thoracic and vascular surgery (CTVS) consult was taken and it was decided to do a surgical procedure with a diagnostic and therapeutic intent in view of recurrent PE. Pericardiectomy and pericardial pleural window formation was done on 12.10.2015. Intra-operatively pericardium was found to be thickened and adherent to the heart. Bilateral pleural effusions were drained, mediastinal and bilateral pleural drains were placed and biopsy was sent. She evolved favorably during the post operative period. She was discharged on 21.10.2015. A chest x-ray taken at discharge showed that the cardiac silhouette had returned to normal size with minimal left pleural effusion (Figure 2C). The pericardium biopsy showed chronic non specific inflammation, fibrosis and congested vessels.(Figure 3). The patient is currently receiving follow-up care through the outpatient department. When last seen in February 2016, she was asymptomatic, X ray chest and inflammatory markers were normal, and thyroxine dosage were adjusted. She is off antidepressants now.

*Consultant and Head, Department of Internal Medicine, 1Senior Consultant, Department of Cardiology, 2Senior Consultant, Department CTVS, 3Consultant, Department of Pathology, Narayana Multispeciality Hospital, Jaipur, Rajasthan; 4Intern, BJ Medical College, Ahmedabad, Gujarat
Received: 27.11.2015; Revised: 05.05.2016; Re-revised: 26.07.2016; Accepted: 08.08.2016
### Discussion

Pericardial effusion (PE) as an initial clinical presentation of primary hypothyroidism has been reported in literature.\(^1\)\(^2\) Our patient had severe hypothyroidism with undetectable T3, T4 and markedly raised TSH. As discussed she had massive PE with features of cardiac tamponade and the fluid had a high concentration of protein and was an exudate (Table 3). Though the Light’s criteria had been described for pleural effusions it has been applied for other serous effusions too to classify them as exudates or transudates.\(^3\) She had been treated for depression for a long period but it appears that her symptoms had been treated but her clinical picture had never been investigated.

PE in hypothyroidism is a part of the generalized polyserositis and the pathophysiological changes responsible are capillary dysfunction, albumin leak into interstitial and extra cellular space, reduced lymph clearance probably due to poor lymphatic tone, and disturbances in electrolyte metabolism.\(^2\)\(^4\) The term “Gold Paint Effusion” has been used to describe the golden brown appearance of the pericardial fluid due to the shimmering satin cholesterol crystals. Changes in lipid metabolism are probably responsible for the high cholesterol content of the fluid. The case discussed had high total cholesterol (47 mg/dl) in pericardial fluid. A churning action of the heart may play a role in the precipitation of cholesterol from the pericardial fluid or the poor absorptive capacity of the pericardium may be a major factor.\(^1\)\(^4\) Mercedes Camprubi et al\(^5\) reported a case of cholesterol pericarditis with hypothyroidism. However no such crystals could be demonstrated in the pericardial biopsy of our patient, though the fluid did had golden brown appearance. Having ruled out an infectious process a tumor or an underlying immunologic disorder and because of the persistence of the effusion a surgical approach was
considered and a pericardial biopsy was performed. 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 data from pathologic study showed enlarged pericardium with chronic non specific inflammation and fibrosis.

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 pericardiocentesis or creation of a pericardial window. The advantages of pericardial window are possible tissue biopsy and prevention from recurrences. On literature search we could find only one case report of a case of cardiac tamponade secondary to hypothyroidism being managed with surgical intervention.

The bilateral pleural effusion and ascites were due to congestive heart failure and not a part of capillary leak syndrome (CLS). In CLS in dengue fever, the fluid clears rapidly within a week’s time, is associated with some degree of thrombocytopenia in all cases, bleeding manifestations, and other clinical and sonography features such as edematous gall bladder wall which were not present in the case discussed. Further, Pericardial effusion, though reported in dengue fever, is not massive and the pericardial biopsy features of chronic inflammation also rules out acute febrile tropical illness as a likely etiology in the present case. We did not find any evidence in favor of dengue fever which could have caused or complicated the patient illness and positive serology for dengue fever could be the result of a subclinical infection that may have occurred in the preceding 2 to 3 months period or a false positive test due to cross reactivity with other flaviviruses.

### Conclusion

Hypothyroidism presenting with massive pericardial effusion and cardiac tamponade is an uncommon and rare presentation as in the present case report. Mild pericardial effusion respond to thyroxine supplementation over a long period. Pericardiocentesis is necessary only when it is massive and tamponade develops. Very rarely in recurrent PE surgical management such as pericardiectomy and/or pericardial pleural window is needed as in the present case reported.

### References