UNUSUAL PRESENTATION OF PERICARDIAL EFFUSION: A CASE REPORT

*Neki N.S.
Department of Medicine, Govt. Medical College/ Guru Nanak Dev Hospital, Amritsar, India- 143001
*Author for Correspondence

ABSTRACT
Hypothyroidism is a rare but significant cause of pericardial effusion. Pericardial effusion secondary to hypothyroidism is a diagnostic challenge for physicians because of its characteristics. Pericardiocentesis is not necessary in most of the patients with hypothyroidism associated pericardial effusion since it is reversible and can be treated with the management of underlying hypothyroidism. We report a 61-year-old lady with exertional dyspnea for several weeks. After detailed history taking, physical examination, echocardiography and thyroid function survey, a diagnosis of pericardial effusion secondary to Hashimoto’s thyroiditis was made and it completely disappeared after 6-month thyroid hormone replacement. Hence hypothyroidism should be included in the differential diagnosis of unexplained pericardial effusion.

Keywords: Hypothyroidism; Pericardiocentesis; Hashimoto's Thyroiditis; Pericardial Effusion

INTRODUCTION
Numerous pathologic processes can lead to pericardial effusion (Levy et al., 2003). Pericardial effusion secondary to hypothyroidism is a diagnostic challenge for physicians because there is discrepancy between clinical symptoms and amount of pericardial effusion (Lin et al., 2003). Worldwide, iodine deficiency is the usual cause of hypothyroidism, but in iodine-sufficient areas, the most common cause is autoimmune thyroiditis. The development of symptoms is very gradual, with the duration of several months, occasionally even one or two years (Petel et al., 1991). Individuals with massive amount of pericardial effusion secondary to hypothyroidism can be asymptomatic or with few symptoms. It tends to regress slowly and disappears several months after patients revert to euthyroid status. Pericardiocentesis is usually not necessary. In order to avoid unnecessary pericardiocentesis and improving prognosis, early recognition of hypothyroidism as the etiology of pericardial effusion is important. Pericardial effusion is a common finding in patients with hypothyroidism which represents the most common clinical disorder of thyroid function. The incidence of pericardial effusion in patients of mild hypothyroidism ranges from 3% to 6%, but in those with severe deficiency, the incidence ranges from 30% to 80%. However, pericardial effusion can also be associated with subclinical hypothyroidism (Meares et al., 1993).

CASES
A 61-year-old female farmer visited our medical outpatient department with complaints of progressive shortness of breath which started gradually while on routine activity in her field, then gradually progressed to rest also. There was no history of orthopnea or paroxysmal nocturnal dyspnea, chest pain, febrile diseases and airway symptoms. But there was history of body weight gain and decreased appetite. There was no other history regarding comorbid conditions like hypertension, diabetes mellitus, and tuberculosis in past. Vital signs included temperature of 37.4 °C, and a regular pulse rate of 62 beats per minute, respiratory rate of 24 breaths per minute and a blood pressure of 116/72 mmHg measured in right arm and in sitting position. Physical examination revealed engorged jugular veins, bilateral non-pitting edema, regular and muffled heart sounds but no audible murmur and clear breath sounds. The maximal cardiac apical impulse was palpated at 5th intercostals space in the left mid-clavicular line. Electrocardiography showed sinus rhythm with low voltage in all 12 leads (Figure 1). Chest X-ray showed picture suggestive of massive pericardial effusion (Figure 2). Echocardiography was arranged and it showed large amount of pericardial effusion with swing heart motion, mild diastolic compression of
right atrium, normal cardiac chamber size and fair left ventricular contractility (Figure 3). After the diagnosis of pericardial effusion was established, search for etiologies was started. The patient had no virus infection or fever history. Laboratory investigations including hemoglobin, total leucocyte count, blood urea, serum creatinine and urine complete examination were within the normal limits. Thyroid profile showed TSH 304uIU/ml (normal range 0.27-4.20 uIU/ml), T3 0.15 ng/ml (normal range 0.85-2.10 ng/ml), and T4 0.60ug/ dl (normal range 5.00-14.00 ug/dl); thyroid function survey was compatible with hypothyroidism. Thyroid sonography showed heterogenous echogenicity over bilateral lobes. FNAC cytology showed thyroid follicular cells in cluster with background of pleomorphic population of lymphocyte multinucleate giant cells and fibrosis suggestive of Hashimoto’s thyroiditis. Anti-thyroglobulin antibody was 22.34 IU/ml and anti-TPO antibody was 332.3 IU/ml; thus diagnosis of Hashimoto’s thyroiditis was made. Lipid abnormalities including total cholesterol 300 mg/dl, triglyceride 203 mg/dl, HDL-C 65 mg/dl and LDL-C 243 mg/dl were noted. Thus pericardiocentesis was not recommended for her pericardial effusion. She was started on thyroid hormone replacement with eltroxin 100 micro gram per day empty stomach. Six months later, thyroid function and lipid profiles normalized gradually and follow-up echocardiography showed disappearance of previous pericardial effusion. Patient is now asymptomatic and is on OPD follow-up.

Figure 1: Electrocardiography of patient showing low voltage complexes with sinus rhythm

Figure 2: Chest X-ray of patient showing massive pericardial effusion
DISCUSSION
Various studies have shown the comorbidity of the hypothyroidism and pericardial effusion (Omura et al., 2007). Hypothyroidism-associated pericardial effusion sometimes can be massive. The mechanisms of myxedematous pericardial effusion are the increased permeability of capillaries with subsequent leakage of fluid rich in protein into the interstitial space, impaired lymphatic drainage and salt and water retention. The diagnosis of myxedematous pericardial effusion is usually undiagnosed because of the absent clinical symptoms and signs. Early recognition of myxedematous pericardial effusion is very important since it responds dramatically to simple thyroid hormone replacement and hence pericardiocentesis can be avoided. The etiology of hypothyroidism is autoimmune in most of the cases. The incidence and amount of pericardial effusion in myxedema correlates with the severity and duration of disease. Pericardial effusion due to hypothyroidism regresses slowly under hormonal replacement therapy (Tudoran et al., 2011). In our case this massive pericardial effusion was associated with Hashimoto’s thyroiditis which was proven biochemically and histologically. Patients should be followed up closely at an extended period for clinical response because myxedematous pericardial effusion usually regresses slowly after patient reverts to euthyroid status (Zimmerman et al., 1983). Hypothyroidism can be associated with lipid abnormality because of the reduction in hepatic LDL receptor function and delayed clearance of LDL (Friis and Pedersen, 1987). Severe hypothyroidism can be associated with cardiac tamponade but it is also rare (Virendra et al., 2011). Thus, all patients with hypercholesterolemia should be cautiously screened for hypothyroidism (Muls et al., 1984). Therefore, hypothyroidism should be included in the differential diagnosis of unexplained pericardial effusion, especially combined with lipid abnormalities. Early tests for thyroid function are essential for the diagnosis of Hashimoto’s thyroiditis in the form of T3, T4, TSH and anti-thyroglobulin antibody and anti TPO antibody.

Conclusion
In a patient with pericardial effusion and lipid abnormality, thyroid functions should be routinely done in the line of diagnosis and should not be missed as it is a reversible condition and thus unnecessary pericardiocentesis (which is a risky procedure and should be done in expert hands) can be prevented and patient can be managed conservatively.

REFERENCES

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