Case Report

**Massive Amount of Pericardial Effusion Secondary to Hashimoto’s Thyroiditis: A Case Report**

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**Abstract**

Hypothyroidism remains a rare but significant cause of pericardial effusion. Pericardial effusion secondary to hypothyroidism is a diagnostic challenge for physicians because of its characteristics and chronology. Pericardiocentesis is unnecessary in most of the patients with hypothyroidism-associated pericardial effusion. We reported a 57-year-old lady with exertional dyspnea for several weeks. After detailed history taking, physical examination, echocardiography and thyroid function survey, pericardial effusion secondary to Hashimoto’s thyroiditis was impressed and it completely disappeared after 6-month thyroid hormone replacement. We recommend that hypothyroidism should be included in the differential diagnosis of unexplained pericardial effusion.

(Formos J Endocrin Metab 2009; 1: 29-32)

**Key words: Pericardial effusion, Hashimoto’s thyroiditis, Hypothyroidism.**

**Introduction**

A wide variety of pathologic process can lead to pericardial effusion1. Early recognition of etiology is very important due to a great diversity of management. Pericardial effusion secondary to hypothyroidism is a diagnostic challenge for physicians because of discrepancy between clinical symptoms and amount of pericardial effusion2-4. Individual with massive amount of pericardial effusion secondary to hypothyroidism can be asymptomatic or of few symptoms. It tends to regress slowly and disappear several months after patients were reverted to euthyroid status. Pericardiocentesis was usually unnecessary until significant cardiac tamponade developed4. In order to avoid unnecessary pericardiocentesis and improving prognosis, early recognition of hypothyroidism as the etiology of pericardial effusion is important.

**Case Report**

A 57-year-old housewife visited our Cardiology outpatient department due to progressive shortness of breath, especially while quick walking for several weeks in November, 2005. No orthopnea or paroxysmal nocturnal dyspnea was complained. There was no history of chest pain, febrile diseases, airway symptoms, body weight loss or other clinically important comorbidities. She had been treated with an Iron preparation (Ferrum Hausmann 1# bid po) for iron deficiency anemia for about five years in Hematology OPD.

Vital signs included a temperature of 37.2 °C, and a regular pulse rate of 70 bpm, a respiratory rate of 20 breaths per minute, and a blood pressure of 110/70 mmHg. Physical examination revealed engorged jugular vein, regular and distant heart sounds,

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and clear breath sounds. There was no ventricular gallop rhythm, audible cardiac murmur, pitting edema or sign of pulse paradoxus. The maximal cardiac apical impulse was palpated at 5th intercostals space; left mid- clavicular line. Electrocardiography showed sinus rhythm with low voltage over all of the 12 leads; this finding suggested the presence of the pericardial effusion (Figure 1).

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 2).

After the diagnosis of pericardial effusion was established, series surveys of etiologies were started.

The patient had no virus infection or fever history. No malignancy, rheumatologic disorders or trauma was noted. Hemogram, liver function, renal function and electrolyte were all within normal limits. C-reactive protein (<0.100 mg/dl) and anti-nuclear antibody (160X, coarse speckled pattern) revealed no significant findings. TSH was 604 uIU/ml (normal range 0.27-4.20 uIU/ml), T3 was 0.20 ng/ml (normal range 0.80-2.00 ng/ml), and T4 was 0.5 ug/dl (normal range 5.10-14.10 ug/dl); thyroid function survey was compatible with hypothyroidism. Thyroid sonography showed heterogenous echogenicity over bilateral lobes. Thyroglobulin antibody was 1600X+ and anti-microsomal antibody was 6400X+; thus, Hashimoto’s thyroiditis was impressed. Tracing back the history, the patient denied fatigue, myalgias, weight gain, constipation, dry skin or coarse brittle hair. Lipid abnormalities including total cholesterol 352 mg/dl, triglyceride 243 mg/dl, HDL-C 75 mg/dl and LDL-C 273 mg/dl were noted.

Echocardiography revealed mild diastolic compression of right atrium, and clinical condition of this patient was relatively acceptable. Thus, we did not recommend pericardiocentesis for pericardial effusion. She received thyroid hormone replacement with Eltroxin (0.1mg per day). Six months later, thyroid function and lipid profiles normalized gradually and follow-up echocardiography showed disappearance of previous pericardial effusion (Figure 3). Patient is well with OPD follow-up.
Discussion

Previous studies have shown the comorbidity of the hypothyroidism and pericardial effusion\(^1\). Hypothyroidism-associated pericardial effusion is sometimes massive but it rarely causes cardiac tamponade. 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\(^5,6\).

The diagnosis of myxedematous pericardial effusion is usually overlooked because of the unremarkable clinical symptoms and signs\(^7\). Early recognition of myxedematous pericardial effusion is very important since it responds dramatically to simple thyroid hormone replacement and pericardiocentesis can be avoided. Patients should be followed up closely at an extended period for clinical response because myxedematous pericardial effusion usually regressed slowly after patient was reverted to euthyroid status\(^8\).

A relationship between hypothyroidism and lipid abnormalities was described in the previous studies\(^9,10\). Among the 248 patients referred to a lipid disorders clinic, 2.8 % had overt hypothyroidism and 4.4 % had subclinical hypothyroidism; thus, all patients with hypercholesterolemia should be cautiously screened for hypothyroidism\(^9,10\). The mechanisms of the hypercholesterolemia secondary to hypothyroidism are the reduction in hepatic LDL receptor function and delayed clearance of LDL. Simple thyroid hormone replacement usually ameliorates hypercholesterolemia\(^9,10\).

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.

References


