# Massive Pericardial Effusion: A Rare Cardiac Manifestation in Hypothyroidism

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## Abstract

Pericardial effusion is a common finding in hypothyroidism patients. We had three cases of hypothyroidism with cardiac manifestations of massive pericardial effusion accompanied by echocardiographic evidence of tamponade. After diagnosis, pericardiocentesis and treatment with hormonal drugs were commenced, as a result of which the symptoms were alleviated. Tamponade is a serious, albeit rare, clinical manifestation of hypothyroidism. It is, therefore, advisable that hypothyroidism be considered in all patients with pericardial effusion ***(Iranian Heart Journal 2010; 11 (3):43-46).***

**Key words:** pericardial effusion ■ hypothyroidism ■ cardiac tamponade

Hypothyroidism is the most common clinical disorder of thyroid function; and worldwide, iodine deficiency is the most common cause of hypothyroidism. In iodine-sufficient areas, the most common cause is autoimmune thyroiditis.[1] The initiation of symptoms is very gradual, occasionally with durations of several months or years.[2] The cardiac manifestations of hypothyroidism are almost always occult,[3] with bradycardia, diastolic dysfunction, narrow pulse pressure, and motionless pericardium being some of the most common presentations.[4] The incidence of pericardial effusion varies, ranging from 3% in the initial stages of hypothyroidism to 80% in myxedema; and improvement is normally seen after weeks or months of treatment.[5] The protein content of pericardial fluid could be exudative or transudative. In one study on 11 patients with tamponade,[6] the amount of pericardial fluid protein was variable (2.2 - 7.6 mg/dl). Except for prolonged hypothyroidism, pericardial effusion is minor and the cardiac symptoms are alleviated with hormonal replacement therapy.

Occasionally, pericardial effusion is substantial and cardiomegaly is evident in chest X-ray. We had three cases of primary hypothyroidism with clinical cardiac symptoms of progressive dyspnea on exertion. Further evaluations revealed massive pericardial effusion, which was subsequently improved with interventional and hormonal treatment.

### Case 1

A 45-year-old woman with progressive dyspnea on exertion and weakness was admitted to the emergency ward. She had no history of cigarette smoking, alcohol consumption, or cardiopulmonary disorder. Body mass index (BMI) was 23.5 (weight=60kg, height, 160cm) with puffy face

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and extremities. She was sluggish and had some degrees of hearing loss.

Vital signs were: BP=130/70mmHg with 30mmHg paradoxical pulse, PR=110/min, and RR=18/min. The thyroid gland was normal in size and firm on palpation. There was an elevated jugular venous pressure, and pulmonary examination showed positive Ewart sign on percussion and heart sounds were muffled.

An ECG showed low voltage QRS with sinus tachycardia and no significant ST-T change. Chest X-ray revealed cardiomegaly, and there was massive pericardial effusion with diastolic right atrial and right ventricular collapse and significant respiratory variations of the mitral and tricuspid valves inflow on echocardiography. The patient underwent pericardiocentesis, during which approximately 300mL of serous fluid was drained (Table I). Lab data are presented in Table II.

**Table I. Characteristics of Pericardial Effusion**

| Patient | Volume ($cm^3$) | Protein (g/dL) | LDH[1] (IU/L) | Glucose (mg/dL) | AlK[2] (IU/L) | TG[3] (mg/dL) | Cholesterol (mg/dL) |
|---|---|---|---|---|---|---|---|
| **Case 1** | 300 | 6 | 823 | 88 | 10 | 25 | 43 |
| **Case 2** | 1200 | 7.3 | 762 | 69 | 4 | 20 | 52 |
| **Case 3** | 1000 | 7.1 | 792 | 70 | 4.5 | 22 | 50 |

LDH: lactic dehydrogenase, AlK: alkalin phosphatase,
TG: triglycerides

**Table II. Biochemical Characteristics**

| Patient | Hb[1] (g/dL) | Hct[2] (%) | WBC[3] cels/UL) | MCV[4] (FL) | ESR[5] (mm/h) | LDH[6] (IU/L) | CPK-MB[7] (IU/L) |
|---|---|---|---|---|---|---|---|
| Case 1 | 11.8 | 36 | 7100 | 99 | 32 | 766 | 24 |
| Case 2 | 12 | 36 | 7300 | 103 | 15 | 556 | 55 |
| Case 3 | 10 | 30 | 5200 | 103 | 44 | 460 | 97 |

1 - hemoglobin, 2 - hematocrit, 3 - white blood count, 4 - mean corpuscular volume , 5 - erythrocyte sedimentation rate, 6 - lactine dehydrogenase, 7 - creatine phosphokinase-MB

Thyroid function tests showed primary hypothyroidism: TSH > 30 mlu/l (0.4 - 4), thyroxine (T4) level 0.3 ng/ml (5 - 11.7), triiodothyronine (T3) 0.1 ng/ml (0.8 - 2.1), T3RU 22% (25 - 35), FTI 0.7 (1.5- 3.6). Cytology study revealed no evidence of malignant cells, and AFB (acid-fast bacilli) study was negative. Pericardial biopsy show mild scattered chronic inflammation. No malignancy or granulomatous inflammation was seen. Hormonal replacement with levothyroxine was commenced, and the patient's clinical and cardiac symptoms of hypothyroidism were subsequently improved. Two months later, the patient was euthyroid with no pericardial effusion on echocardiography.

## Case 2

51-year-old woman with dyspnea on exertion and atypical chest pain was admitted to the emergency ward. The patient had mental retardation since childhood (without clinical evidence of Down syndrome), with coarse skin, and a mildly enlarged and firm thyroid on palpation. Her weight was 64 kg and height was 150 cm (BMI 28.2). She had BP=120/80mmHg with 25mmHg paradoxical pulse, HR=100/min, and RR=16/min. There was an elevated jugular venous pressure with prominent X descent and muffled heart sounds and positive Ewart sign with clear lung fields. In ECG, there was sinus rhythm and low-voltage QRS and QRS alternans. Transthoracic echocardiography showed normal LV size and systolic function with no valvular abnormality, but there was massive pericardial effusion with diastolic RA and RV collapse. Pericardiocentesis was performed and 1200ml liquid was drained. Thyroid function tests were ordered and showed severe primary hypothyroidism: TSH > 25 mlu/l (0.3 - 7.12), thyroxine level (T4) 2.8 ng/ml (5 - 11.7), T3 level 0.6 ng/ml (0.8 - 2.1), T3RU 26% (25 - 35), FTI 0.7(1.5 - 3.6). Pericardial biopsy showed mild chronic inflammation and focal hemorrhage. No

malignancy or granulomatous inflammation was seen. Cytological study was negative for malignancy and AFB. Laboratory studies indicated primary hypothyroidism. With hormonal treatment, the patient became euthyroid.

**Case 3**

A 78-year-old woman with severe progressive dyspnea commencing five days ealier, together with fatigue and malaise, referred to the emergency ward. The patient was obese (BMI 34) (weight=95 kg, height=167 cm), sluggish, puffy-faced, and had severe hearing loss. On physical examination, the thyroid was enlarged and soft, BP=110/60 mmHg, HR=46/min, and RR=18/min. There was an elevated jugular venous pressure with muffled heart sounds and positive Ewart sign with clear lung fields. ECG showed low voltage QRS with normal sinus rhythm, and echocardiography revealed diastolic RA and RV collapse as well as significant respiratory variation in the mitral and tricuspid valve inflow. Thyroid function tests showed primary hypothyroidism with TSH more than 30 mlu/l (0.4 - 4), thyroxine level (T4) 3.4 ng/ml (5 - 11.7), T3 level 0.3 ng/ml (0.8 - 2.1), T3RU 24% (25 - 35), and FTI 0.8 (1.5 - 3.6). Pericardiocentesis withdrew 1000mL serous fluid, which was negative for malignancy and AFB. Pericardial biopsy showed mild chronic inflammation and focal hemorrhage. No malignancy or granulomatous inflammation was seen. Given the clinical manifestations of hypothyroidism, hormonal replacement was initiated before the results of the lab tests. Three months afterwards, the patient was euthyroid and had no evidence of recurrent pericardial effusion.

## Discussion

Massive pericardial effusion as a presentation of hypothyroidism is very rare.[7,8] Common causes of cardiac tamponade are dissection of the aorta, malignancy, uremia, pericarditis, tuberculosis, hemorrhage, radiation, and cardiac trauma; nonetheless, hypothyroidism is a rare etiology of cardiac tamponade. One of the culprits is over-time fluid accumulation.[3,9] Pericardial effusion after medical therapy improves gradually; occasionally after hormonal treatment. Be that as it may, there remain small amounts of residual pericardial fluid.[10] The diagnosis of hypothyroidism with cardiac symptoms in the emergency ward is difficult. Classic symptoms of tamponade are chest pain, dyspnea, hypotension, and tachycardia. Multiple studies have shown that thorough history, physical examination, and non-invasive laboratory studies can diagnose the etiology in 55% of cases with pericardial effusion.[11] In cases of tamponade with hypotension, bradycardia with other symptoms of hypothyroidism should arouse a suspicion of hypothyroidism. Due to the increase in the incidence of hypothyroidism, in all elderly patients with a suspicion of hypothyroidism and in patients with unknown pericardial effusion, hypothyroidism should be ruled out.[12] Although hypothyroidism a rare disease, it remains one of the most important differential diagnoses of moderate to large pericardial effusion.

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