

Case of Massive Pericardial Effusion in Primary Hypothyroidism

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Running Title: *Massive Pericardial Effusion In Hypothyroidism*

Abstract: *Hypothyroidism is a common endocrine disorder. Pericardial effusion is frequently found in patients with hypothyroidism, but it is rarely associated with cardiac tamponade. Hypothyroidism complicated by cardiac tamponade is rarely reported in the medical literature. Here we present a case of hypothyroidism presenting with a cardiac tamponade. A 27 year old lady presented with symptoms and signs suggestive of hypothyroidism. In addition, ECG and chest X-ray indicated and echocardiogram confirmed that the patient had a massive pericardial effusion. Under the guidance of echocardiography about 3 litres of pericardial fluid was aspirated through apical approach. The aspirated fluid was exudative and sterile. The patient improved symptomatically on pericardial aspiration, started on high dose thyroxine and is on regular follow-up. Considering high prevalence of thyroid disease, high degree of suspicion is mandatory while dealing with cardiac manifestations of thyroid disease. Since cardiac changes are completely reversible at least in initial stages of the disease, it's imperative to have a high suspicion of thyroid disorders in cardiac cases. A diagnosis of hypothyroidism should be considered in the differential diagnosis of patients presenting with unexplained pericardial effusion, even in the absence of accompanying signs and symptoms of hypothyroidism.*

Keywords: Cardiac tamponade, hypothyroidism, pericardial effusion

1. Introduction

Pericardial effusion is a known cardiac manifestation of hypothyroidism, but the diagnosis is often difficult and delayed due to silent nature of the disease and low index of clinical suspicion. Frequent rarity of hemodynamic premonitory signs, even in the presence of large effusions is due to the slow accumulation of fluid in the pericardial space. Though massive pericardial effusions as a presenting feature are extremely uncommon, we report a case of a lady who presented with massive pericardial effusion with tamponade physiology secondary to primary hypothyroidism.

2. Case Report

A 27 year old married lady presented to the physician with symptoms of easy fatigability, generalized body swelling, increased hair loss, hoarseness of voice, weight gain despite decreased appetite, exertional breathlessness and palpitations and sensation of heaviness over the central chest of one year duration. Also she had primary infertility (was unable to conceive in the past 2 years of her marriage) and irregular menses (heavy bleeding lasting 7-8 days every 2-3 months) for the last 2 years. She had no history of chest pain, cough, haemoptysis, orthopnoea, paroxysmal nocturnal dyspnoea, fainting spells, wheezing, fever, abdominal pain or distension, jaundice, hematemesis, melaena or any urinary disturbances. She did not have any features of cold intolerance, constipation, difficulty concentrating, poor memory or, impaired hearing. Examination revealed mild facial (especially peri-orbitally) puffiness and oedematous (non-pitting) hands and feet. Her skin was coarse and dry. No obvious thyroid swelling was evident. Her pulse was 62/min, regular rhythm with pulsus paradoxus. Her blood pressure was 110/90mmHg and respiratory rate was 16

breaths/minute. Neurological examination revealed delayed relaxation of bilateral ankle jerks. Cardiovascular examination revealed distant and muffled heart sounds and elevated jugular venous pressure. Gastrointestinal and respiratory systems were within normal limit.

Hemogram revealed a haemoglobin of 6gram%, peripheral blood smear and iron profile were suggestive of severe iron deficiency. Blood sugar, liver and kidney function tests, including prothrombin time, serum electrolytes had no significant abnormalities. Lipid profile was normal except for raised triglycerides (238mg/dL). HIV and hepatitis B surface antigen were negative. Standard twelve lead electrocardiogram revealed low voltage complexes.

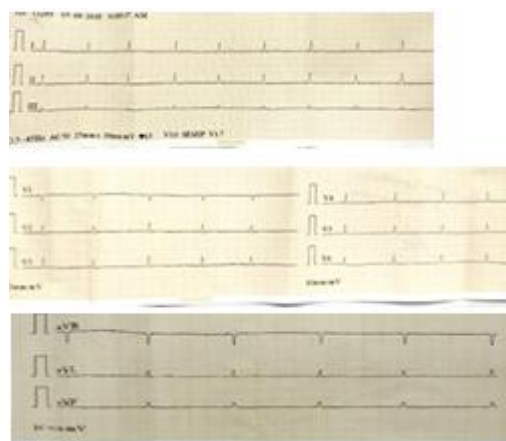


Figure 1: 12-lead electrocardiogram showing low voltage complexes and sinus bradycardia

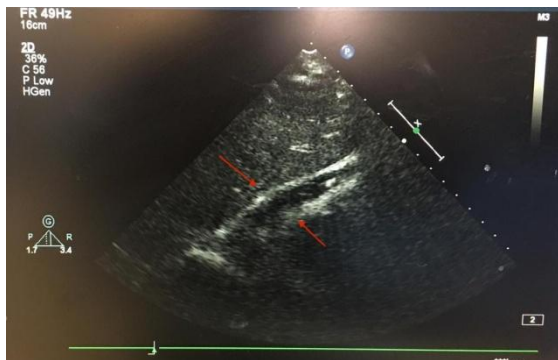
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Figure 2: Chest X-ray showing enlarged cardiac silhouette giving a money-bag appearance



[i]



ii [i] (a)



ii (b)

Figure 3 Trans-thoracic two-dimensional echocardiography showing [i] subcostal view showing 3 cm peri-cardial effusion anterior to right ventricle with diastolic RV collapse. **After 2 litres pericardial tap and one month of thyroxine replacement therapy**, [ii (a)] subcostal view showing a collection in the pericardium of 1.35cm anterior to right ventricle [ii (b)] long parasternal view showing a collection of 1.4cm posterior to left atrium.

Routine chest X-ray revealed enlarged cardiac silhouette with money-bag appearance. Transthoracic two-dimensional echocardiography revealed presence of massive pericardial effusion with signs of tamponade (early diastolic right ventricular collapse, with preserved left ventricular systolic function; ejection fraction of 60%). Pericardiocentesis was performed and 2 litres of straw-coloured fluid was removed. The analysed fluid had normal adenosine deaminase (ADA) and sugar levels, high protein, and was negative for malignant cells. Culture was negative for bacteria and acid-fast bacilli. Mantoux test was non-reactive. Autoimmune profile including anti-nuclear antibody (ANA) and rheumatoid factor were negative. Thyroid stimulating hormone (TSH), free T₃ (fT₃), free T₄ (fT₄) levels were found to be >500 μ IU/mL (0.34-5.6), 0.709 pg/mL (2.5-3.9) and 0.064 ng/dL (0.6-1.12) respectively. Ultrasonography showed atrophic changes in both thyroid lobes. Anti-thyroid peroxidase antibody (TPO) was positive.

She was started on 100 micrograms levothyroxine once a day (later stepped up to 150 micrograms), oral iron supplements and transfused 1 packed red cells. Further hospital course was uneventful and she was discharged. On 4 weeks follow-up she was in good health and had resolution of tissue oedema and minimal pericardial effusion was remaining, her TSH was 49 and she continued on the same therapy with regular follow up.

3. Discussion

Overt hypothyroidism has been associated with several cardiovascular abnormalities like sinus bradycardia, non-pitting or pitting peripheral oedema, reduced stroke volume due to impaired cardiac contractility, diastolic hypertension resulting from increased peripheral resistance, the latter two contributing to a narrow pulse pressure, pericardial effusions (muffled heart sounds and cardiomegaly), rarely cardiac tamponade and possibly increased risk of atherosclerosis and coronary artery disease. Also reduced cutaneous circulation is responsible for sensitivity to cold.

Normally, there is 10-50 ml of fluid present between visceral and parietal layers of pericardium, which is produced by pericardium through ultrafiltration of plasma. Pericardial effusion in hypothyroidism has been reported in older literature with variable incidence ranging from 30% to 80%. The recent studies, however, conclude that it is extremely infrequent in hypothyroidism, with an incidence of 3% to 6%.

The accepted mechanisms of serous cavity effusions in hypothyroidism are as follows (a) As a part of the generalized polyserositis. (b) Increased leak of plasma proteins because of abnormal capillary permeability and the

lack of a compensatory increase in lymph flow and protein return rate. (c) Exudative polyserositis due to alteration in albumin metabolism. (d) Deposition of mucopolysaccharides and protein in pericardial space. (e) Hyaluronic acid accumulation in the skin which causes oedema by a direct hygroscopic effect, and interaction with albumin to form complexes that prevent the lymphatic drainage of extravasated albumin. Autoimmunity does not seem to play a major role in the pathophysiology of pericardial effusions.

A majority of such cases are asymptomatic due to slow fluid accumulation. The diagnosis is generally made when the pericardial disease is associated with an elevated TSH level, and other causes are excluded.

The mainstay of treatment for this is simple and gratifying. Majority need just thyroxine replacement, except in those patients with pericardial tamponade or, impending tamponade, where urgent pericardiocentesis is mandatory. An important thing to note is that a rapid increasing of replacement dose could induce a heart failure or, acute forms of coronary heart disease in a patient with underlying coronary artery disease.

4. Conclusion

Because of insidious onset, gradual progression and non-specificity of signs and symptoms of hypothyroidism along with rare occurrence of moderate to massive pericardial effusion in these patients, hypothyroidism induced pericardial diseases are underdiagnosed. As hypothyroidism is treatable cause of pericardial effusion the diagnosis must be established and treatment promptly started. In patients with unexplained pericardial effusion, thyroid function tests and two-dimensional echocardiography should always be performed. Moderate to massive pericardial effusion can be completely cured only with thyroid hormone supplementation without pericardiocentesis. On obtaining euthyroid status, pericardial effusion slowly disappears (may take as long as months or years).

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