

## **Massive Pericardial Effusion as a sole Manifestation of Hypothyroidism – A Case Report**

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**Massive pericardial effusion (PE) was diagnosed in a 50 years old Bahraini lady which turned to be secondary to severe hypothyroidism. PE is well known in hypothyroidism, but it is rare for it to be the sole presenting sign. The case history, the incidence, pathogenesis, diagnosis tool, treatment and prognosis of hypothyroid related PE together with the pathological and haemodynamical alteration in the hypothyroid heart are discussed.**

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Cardiovascular syndromes are common targets for thyroid disorders. In this article we are reporting the clinical story of a middle-aged lady who presented massive pericardial effusion, the latter turned to be secondary to severe hypothyroid state. This patient responded to simple thyroxin replacement with almost complete recovery of her pericardial effusion on future follow up.

### **THE CASE**

A 50 year old Bahraini lady presented on 25<sup>th</sup> of April 1999 with history of progressive abdominal distention of 3 months duration. The problem started insidiously when she noticed gradual painless increase in her abdominal girth causing tightness of her clothes. Swelling of both lower limbs was also progressive becoming clearly evident since two weeks prior to her hospital admission.

On further questioning, she denied history of chest pain, shortness of breath, paroxysmal nocturnal dyspnea, jaundice, abdominal pain, haematemesis, urinary symptoms, fever, anorexia or weight loss. She had no history of recent travel, or blood transfusion, or close contact with tuberculosis patient.

Examination revealed a pale, fully conscious oriented, apathetic middle-aged lady, with mild peri-orbital puffiness. Her skin was dry and her ankle jerks were slow in the relaxation phase.

The patient was afebrile with a heart rate of 75/min, blood pressure 131/84 and her jugular venous pressure was elevated (11 cm above the sternal angle) with normal pericardial examination apart from muffled heart sounds. The lungs were clear.

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The abdomen was massively distended with non-tender ascites and no evidence of organomegaly or clinical signs of portal hypertension were detected. Patient had bilateral pitting ankle edema with preserved normal peripheral pulses.

The initial laboratory investigations of full blood count, ESR, random blood sugar, urea electrolytes, creatinine and mid stream urine examination for both routine and microscopy were normal.

The result of diagnostic paracentesis was exudative ascites with normal leucocytic count and differential. Cytological examination of the peritoneal fluid was normal apart from reactive, esothelial cells. Tuberculosis screening was negative.

Chest radiograph showed a huge global cardiac enlargement with no evidence of pulmonary venous congestion or parenchymal pulmonary pathology (Fig 1).

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*Figure 1. Chest x-ray showing global cardiac enlargement with no evidence of pulmonary congestion*

Computerized tomography of the chest and abdomen were negative apart from massive pericardial effusion and ascites respectively (Fig 2, 3).

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*Figure 2. Computerized tomogram of the chest showing features of massive pericardial effusion*

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*Figure 3. Computerized tomogram of the abdomen showing massive ascites with normal appearance of intra-abdominal organs*

ECG showed sinus rhythm of eighty beats per minute, low voltage QRS complexes, diffuse non progressive ST-T changes and prolonged Q-T interval of 0.42 seconds.

The echocardiogram showed a large circumferential echo-free space consistent with massive pericardial effusion (Fig 4).

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*Figure 4. Four chamber view echocardiogram. A “v” shaped  
Collapsed right atrium donating massive pericardial effusion  
(RA-right atrium, RV-right ventricle, LV-left ventricle,  
PE – pericardial effusion)*

Thyroid function test subsequently confirmed severe hypothyroidism; TSH 392 MU/ml (normal range 0.25-4) and free T4 of 1.9 (normal range 5-12µg/dl). In consultation with endocrinologist she was started on low dose thyroxin which was increased gradually. To hasten the recovery of pericardial effusion and to rule out any other underlying pathology, pericardial drainage with pericardiectomy was performed. A total of 1200 milliliters of straw colored fluid was aspirated. Cytological examination of the pericardial fluid demonstrated features of cholesterol effusion and the pericardial histopathology was nonspecific. Culture of the pericardial fluid was negative for bacteria and acid-fast bacilli.

Subsequent hospital course was non-complicated; she was discharged with TSH of 138 MU/ml and a T4 of 6.5 µg/dl. On a follow up visit to medical clinic on 21<sup>st</sup> June 1999, the patient was asymptomatic and the ascites had totally subsided. Her repeated echo showed trivial pericardial effusion with normal cardiac function.

## **DISCUSSION**

Cardiovascular syndromes are common presentations of thyroid disorders with the terms apathetic thyrotoxicosis and myxoedema heart commonly mentioned in the medical textbooks. Hypothyroidism can have extremely rare modes of presentations like pre-eclampsia in pregnancy<sup>1</sup> and acute massive macroglossia<sup>2</sup>. Pericardial effusion (PE) is a common finding in myxoedema but is seldom the only presenting feature of this disorder<sup>3</sup>.

PE in hypothyroidism has been reported in the literature with extremely variable incidence ranging from 30% to 80%<sup>3,4</sup>. The recent studies, however, conclude that PE is extremely infrequent in hypothyroidism, with an incidence of 3% to 6% and state that the old figures ie, 30% to 80% are exaggerated and require reassessment. This is because the subjects studied in the old literature were severely hypothyroid at the time of the study, reflecting delayed diagnosis of hypothyroidism. Thus PE may be a frequent manifestation in myxoedema (advanced severe stage) but a rare

manifestation in hypothyroidism (early mild stage), as the latter condition nowadays is detected in the very early stage<sup>4</sup>.

The accepted pathogenesis of PE in hypothyroidism is that it is part of the generalized polyserousopathy in hypothyroidism. The increased permeability of capillaries to protein accounts for the exudative PE in this disorder. In addition, a greater than normal proportion and quantity of exchangeable albumin is localized to the extra vascular space, aggravated by the greater decrease in albumin degradation than in albumin synthesis with resultant exudative polyserousopathy<sup>5</sup>. Echocardiography remains the most reliable diagnostic modality with exceptionally high specificity and sensitivity<sup>6,7</sup>.

There are certain pathological and haemodynamical changes, both reversible and irreversible reported in the literature that describe the hypothyroid heart. The classical pathological examination reveals a dilated heart with pale and floppy myocardium. Coronary atherosclerosis is common. Histopathological examination shows interstitial edema and swelling of the muscle fibers with loss of striations. The pericardial sac contains fluid rich in protein and mucopolysaccharides<sup>5</sup>. Vacuolated degeneration has been reported by transvenous right ventricular endomyocardial biopsy in myxoedema heart, which improved after thyroxin therapy, despite persistence of slight degree of fibrosis<sup>8</sup>.

Alteration in the haemodynamics has been well documented in the hypothyroid heart, which can even lead to diagnostic confusion. The haemodynamic alterations at rest resemble those of congestive cardiac failure, but cardiac output increases normally and peripheral vascular resistance decreases normally in response to exercise<sup>5</sup>. The Q-T interval, pre-ejection period (PEP) and pre-ejection period / left ventricular ejection time (PEP/LVET) were significantly greater in the hypothyroid heart (both congenital and acquired) before initiation of thyroxin therapy. Left ventricular fractional shortening on the other hand was significantly lower before thyroxin replacement<sup>5,9</sup>.

Reversible echocardiographic abnormalities are well known in the hypothyroid heart. Heart dilatation, reduced myocardial contractility, enlarged thickness of the interventricular septum and posterior wall of the left ventricle with deviation in the mitral valvular movement, retarded rate parameters of movements of the valvular structures and increases in the left ventricular volume have been described<sup>10</sup>. Echocardiographically the hypothyroid heart can mimic features of both hypertrophic and dilated cardiomyopathies with reversal of these abnormalities upon normalization of the hypothyroid state<sup>11</sup>. It has been well documented in the literature that the hypothyroidism per se can lead to concentric left ventricular thickening that responds to gradual thyroxin therapy<sup>12</sup>.

The main stay treatment for the hypothyroid PE is simple thyroxin replacement. Exceptional are those patients with pericardial tamponade or impending tamponade as the patient might develop paradoxical tachycardia as a warning sign for tamponading<sup>13</sup>, the condition that mandates urgent pericardiocentesis.

Satisfactory clinical resolution of PE in hypothyroidism is the usual outcome with thyroxin replacement, although its very slow<sup>14</sup>, which can take as long as months or

years after the euthyroid state has been reached<sup>15,16</sup>. Most of the reported cases in the literature conclude that thyroxin has been adequate for symptomatic treatment and prevention of recurrences of hypothyroid related PE<sup>16</sup>. Rarely, recurrent PE and even pericardial tamponade has been reported despite maintaining the euthyroid state and adequate replacement therapy. The postulated mechanism for this rare complication is cholesterol pericarditis, the condition that mandates pericardial drainage and pericardiectomy. Cholesterol pericarditis should be suspected in hypothyroid related large PE.

Patients with myxoedema related PE, should be carefully followed up for an extended period, even when the patient has been successfully treated with thyroxin and rendered euthyroid<sup>16</sup>.

## CONCLUSION

**Hypothyroidism can have rare modes of presentation and on many occasions it is silent. Hypothyroidism should be ruled out as a hidden underlying cause of pericardial effusion especially in the middle or old age people. The treatment is simple and gratifying with almost very rare recurrence rates upon maintenance of euthyroid state.**

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