



Case Report

Cardiac tamponade as initial presentation of primary hypothyroidism: A rare 21 case report

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ABSTRACT

Patients with primary hypothyroidism are often complicated with pericardial effusion (3- 6%) but its association with cardiac tamponade is rare in medical literature. Here we report an unusual case of 55 year old female who presented with breathlessness and bilateral lower limb edema was found to have primary hypothyroidism with massive peri cardiac effusion with cardiac tamponade. Patient was managed with emergency pericardiocentesis and thyroid hormone replacement.

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1. Introduction

Pericardial effusion is one of the most important cardiovascular complications of hypothyroidism. According to recent studies, the overall incidence of pericardial effusion in hypothyroid patients is estimated to be around 3-6% but cardiac tamponade is very rare.¹ One of the distinguishing feature of pericardial effusion caused from hypothyroidism and other causes is absence of sinus tachycardia which should prompt towards diagnosis.² The occurrence of pericardial effusion has been related to severity of disease. Early intervention in the form of emergency pericardiocentesis is unnecessary unless there is development of cardiac tamponade. 2d echo remains the gold standard for diagnosis of pericardial effusion. Hypothyroidism has been documented to cause concentric left ventricular thickening which is responsive to thyroxine therapy.³⁻⁶ Here we present a case of primary hypothyroidism presenting with cardiac tamponade as initial presentation.

2. Case Report

A 55 year old female presented to our emergency department with complaints of gradual onset breathlessness since 6 months. Patient was apparently alright 6 months back, when she gradually starting having malaise, lethargy, slow speech, edema of the face and extremities, and progressive weight gain. There was no significant past medical and drug history. Vital signs were recorded at the time of admission (temperature-36.2 c: heart rate regular at 78 beats/min, blood pressure -94/60 mm hg) body mass index was 27.1 kg/m². Physical examination showed facial edema, coarse hair, dry skin, mild pallor and non pitting edema of the extremities, raised JVP Oxygen saturation was 92%.

On cardiovascular examination apical impulse was not visualized. Apex beat was not felt. Heart sounds were not heard. Respiratory system showed bilateral rales in both lung fields. Per abdomen examination was within normal limits. CNS examination showed delayed relaxation (hung up) of deep tendon reflexes. Chest x- ray showed cardiomegaly with globular enlargement of cardiac silhouette with “water bottle” configuration (Figure 1).

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Fig. 1: Chest X ray suggestive of pericardial effusion

ECG showed heart rate of 78/min with low voltage complexes with electrical alternans. 2d echocardiography was done showed small sized heart with massive pericardial effusion and signs of early diastolic right ventricular collapse which is echocardiographic hallmark of cardiac tamponade (Figure 2). CECT thorax was done to rule out local malignancy presenting as pericardial effusion which showed pericardial effusion with drainage catheter in situ with sub centrimetric mediastinal lymphadenopathy with bilateral pleural effusion with sub segmental collapse. Thus a provisional diagnosis of massive pericardial effusion with cardiac tamponade was kept. Immediately the patient was taken for pericardiocentesis. Around 1.1 litre of pericardial fluid through xiphisternal route was removed under aseptic precautions under cardiac monitoring and pigtail catheter was kept in situ for further draining of fluid. Pericardial fluid was golden yellow in appearance (“Gold Paint” effusion). Immediately after fluid removal patient cardiopulmonary status improved significantly. Follow up 2d echo showed minimal pericardial effusion with no evidence of cardiac tamponade.

INVESTIGATIONS Haemoglobin(hb)-9.4gm%, erythrocyte sedimentation rate(ESR) was 85 at the end of 1 hour; TLC10500 mm³; platelet count-2.19 lac; serum urea-46 serum creatinine-1.10; liver function test was within normal limits. Thyroid function test :serum TSH,>150 mU/ml(normal 0.35-5.5 mU /ml) serum T3 and serum T4 was undetectable. Lipid profile HDL-32 mg/dl, total cholesterol-274 mg/dl, LDL-121 mg/dl, VLDL-21 mg/dl, triglyceride-194 mg/dl, serum sodium was 142 meq /l and serum potassium was 4.3 meq /l. HIV and HBsAG were negative.

Pericardial fluid analysis: appearance golden yellow in colour (“gold paint “ effusion) TLC-21 cells/ cumm DLC-70% lymphocytes 30% neutrophils. Biochemical analysis of fluid showed glucose-78, protein-7.7 albumin-3.9 LDH-262. Culture of fluid showed no growth of organism. ADA of pericardial fluid was negative-20u/l(normal >40iu/ml)

Keeping a suspicion of hypothyroidism based on history and clinical findings, a thyroid profile was done which was suggestive of primary hypothyroidism Patient was started on thyroid replacement therapy in form of thyroxine 50ug/day which was gradually increased to 125 ug/day. Patient’s general condition improved and she was discharged after 10 days.

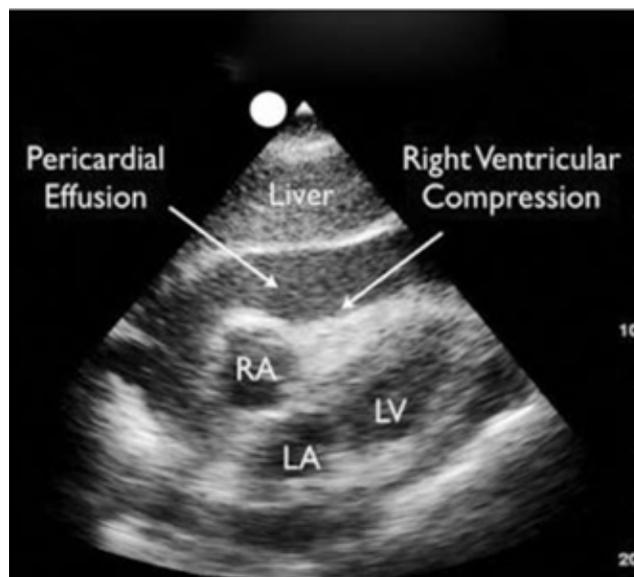


Fig. 2: Suggestive of Pericardial Effusion

3. Discussion

Increased systemic capillary permeability and disturbances of electrolyte metabolism has been found responsible for fluid collection in serous cavities of hypothyroid patients.⁷ The term “Gold Paint” effusion was coined by Alexander to describe the golden brown appearance of pericardial fluid due to shimmering satin cholesterol crystals. Disturbances in lipid metabolism has been attributed to high cholesterol content in the fluid⁷⁻⁹ Cardiac tamponade as a complication of hypothyroidism is very rare; Jiménez- Náchter et al cite that until 1992 less than 24 cases had been described in the world literature. Such a low incidence has been attributed to the slow accumulation of liquid and also cardiac distensibility.⁶ Factors described as provoking cardiac tamponade include infection, spontaneous pericardial hemorrhage, thyroid therapy, and abdominal paracentesis.¹⁰ Identification of cardiac tamponade in hypothyroidism is therefore difficult and commonly mistaken for cardiac

failure due to its symptoms of tachycardia, rise in venous pressure, lower limb edema, and increased cardiac silhouette on radiography. The primary and the most important phenomenon in cardiac tamponade is the compression of all the four cardiac chambers after the pericardial content reaches the limit of pericardial reserve volume. With smaller cardiac chambers, the myocardial diastolic compliance is reduced and cardiac inflow becomes limited, ultimately equalizing mean diastolic pericardial and chamber pressures. This equalization of pressures is the hallmark of cardiac tamponade¹¹

Hypothyroidism as the cause of the pericardial effusion and tamponade was diagnosed by an exclusion criterion, because other afflictions (neoplasm, tuberculosis) are the most frequent causes of nontraumatic pericardial effusion. Treatment of hypothyroidism is always mandatory following tamponade drainage, because, generally, a residue of the effusion following pericardiocentesis which has a high potential for recurrence disappears following appropriate therapy over a period which may vary between 1 month and 1 year, ranging up to 15 months. The most recommended therapeutic scheme is L-thyroxin, which is usually started at an initial low dose (25mcg/day), increased very progressively, because initiating treatment with high doses may precipitate new effusions or decompensation towards tamponade. In the present case, the dose used was of 50 mcg/day, which was gradually increased to 125mcg/day within 6 days, with indefinite continuation.

4. Conclusion

Hypothyroidism is common problem in India. Though pericardial effusion and tamponade secondary to thyroid dysfunction are rare in western scenarios, we do get cases in India because of high burden of primary disease. As diagnostic and therapeutic modalities are sophisticated in nature, including primary hypothyroidism as a differential diagnosis of pericardial effusion and cardiac tamponade looks essential in India

5. Source of Funding

None.

6. Conflict of Interest

None.

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