Case Series

Case series of unusual cases of hypothyroidism-induced pericardial effusion

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Abstract

Hypothyroidism is an endocrine disorder with a high worldwide prevalence and diverse clinical presentation and can affect multiple organ systems. It can be asymptomatic and subclinical or overtly symptomatic and can prove to be fatal if left untreated. It is an established cause of pericardial effusion, which can rarely lead to cardiac tamponade and severe haemodynamic instability. Herein, we present a few unusual case reports of patients presenting with hypothyroidism with varied causes who presented with tamponade.

Keywords: Cardiac tamponade, central hypothyroidism, hypothyroid, myxoedema coma, pericardial effusion, pericardiocentesis

Introduction

Thyroid dysfunction can affect various organs of the body, including the heart. It increases the permeability of albumin across the pericardial membrane, which causes exudative effusion. Patients with hypothyroidism-related cardiac tamponade usually remain asymptomatic or present with atypical symptoms such as bradycardia and a regular heart rate or high blood pressure; therefore, they can be left undiagnosed until the cardiac issue complicates, causing haemodynamic instability. We believe hypothyroidism-related cardiac tamponade is a preventable condition if detected and treated in outpatient settings by family physicians. Hence, it prevents the occurrence of various complications arising from hypothyroidism, including pericardial effusion, and leads to a better quality of life among patients with the added benefit of reduced healthcare burden due to reduced frequency of hospital admissions of acutely ill patients.

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Clinical Case Series

Case 1

A 62-year-old female presented with a history of progressive shortness of breath, associated with orthopnoea, a history of chest pain over precordium since four days non-radiating associated with diaphoresis; there is a history of constipation, cold intolerance, generalized fatigue since one month, no h/o palpitation, pedal oedema, decreased urine output, cough, fever, weight change, diabetes mellitus (DM)/hypertension (HTN)/coronary artery disease (CAD)/tuberculosis (TB). On examination, she was conscious and oriented with a pulse of 94/min, blood pressure of 90/70 mmHg, oxygen saturation (spo2) was 95 percent on room air, tachypnoeic, skin was dry, the temperature was 98.2°F and there was pallor. Respiratory examination showed b/l end-inspiratory crepitations, cardiovascular system (CVS) examination showed that heart sounds were muffled, but no murmurs were heard and electrocardiogram (ECG) showed low voltage complexes; per abdomen examination was normal. Trop I was negative, and arterial blood gas (ABG) showed respiratory alkalosis. Cardiac tamponade was suspected, and the patient was taken for immediate pericardiocentesis. Pericardial fluid analysis suggested

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acellular exudative fluid with biochemical analysis of protein 6 mg/dl and albumin 4.1 mg/dl, ADA 9.1 glucose 64, Cartridge Based Nucleic Acid Amplification Test (CBNAAT) was negative, acid-fast bacilli (AFB) stain was negative, malignant cytology was negative and culture was sterile and autoimmune workup was negative. Routine examination showed iron deficiency anaemia with haemoglobin (Hb) 10.8 g/dl, total leucocyte count (TLC) 6500 (70/28/0/2) and total platelet count (TPC) 2.8 lakh. With normal kidney function, liver function and electrolytes, lipid profile and cardiac markers were within normal range; thyroid profile showed thyroid-stimulating hormone (TSH) 162 mIU/L, free tri-iodothyronine (t3)-1.4 pg/ml and free T_4 (fT4)- 0.25 ng/dl; ultrasonography (USG) of the neck showed normal thyroid gland; and anti-thyroid peroxidase (TPO) was negative. The thyroid hormone was replaced, and gradually, the patient improved; the pigtail was removed, and the patient was discharged. The final diagnosis of the patient was cardiac tamponade (recovered) secondary to primary hypothyroidism.

Case 2

A 22-year-old male presented with the following complaints. Shortness of breath for one week, which was insidious onset gradually progressive associated with orthopnoea with a significant history of weight loss (12 kg in 6 months) and loss of appetite. There was no history of fever, cough, chest pain, palpitation, congenital heart disease, decreased urine output or lower limb swelling. The patient did not give any history of use of recreational drugs—no family history of cardiac disease. On examination, the patient had a temperature of 98.2°F, pulse of 78/min and blood pressure of 90/60 mm hg with SPO2 of 97% on room air. The patient had a thin build and multiple palpable nodules in the bilateral lobes of the thyroid gland and cervical lymph nodes. In systemic examination, patients had muffled heart sounds with no murmur and no pericardial rub; other systems were normal on examination. The ECG on admission showed low voltage complexes with electrical alternans. Chest X-ray shows the increased diameter of cardiac impression (money bag appearance). Two-dimensional echocardiography (2D Echo) confirmed our finding and a pericardial catheter was placed. Cardiac troponin and creatine kinase-myocardial band (Ck-mb) were negative; other parameters on admission were Hb-12, TLC-7300, platelet (Plt)-4.9 lakh, with normal liver function test (LFT), kidney function test (KFT), serum electrolytes and iron profile. The pericardial fluid study was sent, showing a golden yellow colour fluid with a total protein of 1.6 g/dl, glucose-100 mg/dl, albumin-0.8 g/dl, with 15 cells/mm^3, all mononuclear, ADA was 50, CBNAAT and Gram stain and culture were negative for tuberculosis.

Malignant cytology of the fluid was also negative. The patient's autoimmune profile was also negative, with the retroviral test non-reactive. The thyroid profile of the patient showed TSH-98.7 mIU/L, fT3-2.5 pg/ml, fT4-0.7 ng/dl, anti-TPO-0.88IU/ml. Ultrasonography of the neck was performed, showing multiple variable-size solid lesions of the thyroid gland with lymph nodes. FNAC of lymph nodes suggested positive for AFB stain. However, the FNAC of the thyroid gland nodule did not suggest malignancy or tuberculosis. Our patient was treated with thyroid replacement and antitubercular drugs with steroids. The final diagnosis was kept as pericardial effusion secondary to primary hypothyroidism and tuberculosis (cervical + pericardial).

Case 3

A 47-year-old female presented with complaints of progressive shortness of breath for 7–8 days, not associated with the change of posture. She also complained of facial swelling for five days, not involving the lower limb or another part of the body. The patient was also in altered sensorium for three days in the form of increased sleeping and irrelevant talk. The patient also did not pass stool for two days. There was no h/o fever, decreased urine output, loss of consciousness or abnormal body movement. No h/o tuberculosis, diabetes and hypertension were present. There is a history of massive blood loss during her last childbirth 15 years back (P3L3A0). The patient was drowsy on examination and not oriented to place and time. Bp-80 systolic, extremities cold, respiratory rate 10/min, pulse rate-45/min, spo2-91% on room air, random blood sugar was 100 mg/dl, pallor-present, oedema-facial swelling present, with mild pedal oedema, central nervous system (CNS) examination hypotonia in muscles, cranial nerves were normal, reflexes were diminished, plantar b/l were flexor, myxoedema score more than 60. ABG showed type 2 respiratory acidosis. On systemic examination, heart sounds were muffled, mild tenderness over the right hypochondrium and bilateral crepitations were present. ECG showed low voltage complexes, and chest X-ray showed massive cardiomegaly. The patient was immediately taken for screening echo, which showed pericardial effusion. A pericardial catheter was placed with a drain of around 550 ml. The patient was evaluated on the lines of myxoedema coma (myxoedema coma score of 60). On admission, biochemical parameters were Hb-10.4, tlc-13000, platelet-2.3 lakh, Na+/k-123/3.9. The LFT, KFT, cardiac profile and iron profile were normal. Cardiac troponin was negative. Pericardial fluid analysis showed cytology-250 cells/mm^3 (polymorph 60%, mononuclear 40%). Glucose-138 mg/dl, protein-5.8 g/dl, lactate dehydrogenase (LDH)-791 and adenosine deaminase (ADA)-6.27 (normal). CBNAAT and malignant cytology were negative. Autoimmune workup was negative. Thyroid function test showed free T3-1.1 pg/ml, free T4-0.2 ng/dl, TSH-0.36 uIU/mL and anti-TPO-0.7 IU/ml. USG of the neck was normal. The cerebrospinal fluid (CSF) study showed no nucleated cells, 61 mg/dl glucose and protein-74.5 mg/dl. Other pituitary hormonal profiles showed serum insulin-like growth factor-1 (IGF1)-8 ng/ml (68–220), follicle-stimulating hormone (FSH)-0.9 mIU/ml normal range (1.3–23.4), luteinizing hormone (LH)-0.1 mIU/ml (0.8–15), prolactin-0.73 ng/ml (normal range 3–18 ng/ml), serum cortisol-34.7 nmol/l (normal range 123–626 nmol/l). Contrast-enhanced magnetic resonance imaging (CEMRI) brain was done, which showed empty sella. The patient was diagnosed as empty sella with panhypopituitarism, presenting with myxoedema. The pericardial catheter was removed, and

the patient was discharged on thyroxine. The final diagnosis was kept as empty sella with panhypopituitarism with central hypothyroidism with myxoedema coma (recovered) with cardiac tamponade (recovered) with hypocortisolism.

Discussion

Hypothyroidism is a common endocrine disorder involving various systems with diverse clinical symptoms. If left untreated or with a lack of prompt diagnosis, it can lead to fatal complications. Hypothyroidism can cause a myriad of symptoms, including weight gain, fatigue, cold intolerance, constipation, dry skin, oedema, muscular weakness and diminished deep tendon reflexes. It is associated with cardiovascular findings, such as increased systemic vascular resistance, diminished cardiac contractility, low cardiac output, atherosclerosis, CAD, bradycardia and conduction abnormalities. However, diagnosing hypothyroidism requires a simple laboratory test, which is cheap, cost-effective and readily available. Although there are various other aetiologies of pericardial effusion like, including malignancy, infection, heart failure, radiation, trauma, connective tissue disorder, iatrogenic cause and metabolic causes, idiopathic pericarditis, usually of viral origin, remains the common cause of pericardial effusion.

In contrast, a metabolic disorder like hypothyroidism is considered unusual.[1,2] According to the literature, pericardial effusion occurs in only 3 percent of mild forms of the disease and is uncommon as a first presentation.[3,4] It is challenging to diagnose it early due to the discordance between the fluid's volume and clinical symptoms presented by the patient.[5] The effusion volume is directly proportional to the duration and severity of the hypothyroid state. However, some authors point out that there is no relation to the duration of the disease.[6] The mechanism by which exudative effusions are caused is by the extravasation of hygroscopic mucopolysaccharides within the pericardial space, along with an increase in capillary permeability, decreased lymph drainage and increased salt and water retention, along with increased albumin volume distribution.[6-8] As in our first case, a defect of the hypothalamus-hypophysis-thyroid axis can result in the development of hypothyroidism.

Currently, the incidence of pericardial effusion in early hypothyroidism is 3% to 6%.[8,9] Even though most cases have been described in clinical hypothyroidism, Dattilo *et al.* reported a single case of pericardial effusion in subclinical hypothyroidism, describing a 41-year-old patient with increased TSH levels and normal hormone thyroid levels.[10] Pericardial effusion can be present in hypothyroidism rarely causing cardiac tamponade in both primary and secondary disease.[5,11,12]

Our third case presented with acute full-blown complicated overt hypothyroidism, leading to pericardial effusion that required urgent pericardiocentesis and intensive care unit (ICU) admission because of myxoedema coma with delayed diagnosis. *Sheehan syndrome* is a rare disorder that leads to 6% of all cases of hypopituitarism.[13,14] In postpartum haemorrhage (PPH), Sheehan syndrome occurs due to spasms in smaller vessels, apoplexy and pituitary necrosis.[15] Due to its long latency period, it remains undiagnosed for a long period. The severity and type of hormone insufficiency influences all clinical manifestations,[14] such as adrenal crisis, circulatory collapse, myxoedema coma and hyponatremia, which are fatal.[15] A very uncommon symptom of central hypothyroidism is pericardial effusion.

Thyroid function should be routinely tested and emphasis should also be made to educate the patients about the nature of their disease and the complications that may arise if left untreated. This way, a family physician can play a huge role in significantly reducing hypothyroidism-related cardiovascular complications.

Conclusions

Patients with pericardial effusion should be examined in detail. Before starting antitubercular therapy, other causes of pericardial effusion, like hypothyroidism should be ruled out. Women with PPH should be followed for at least five years for early detection of panhypopituitarism.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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