

Case Report

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Massive Pericardial Effusion – Rare and Only Presentation of Hypothyroidism – Myxedematous Heart

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Abstract

Cardiovascular symptoms are often predominant features in patients with hyperthyroidism. But cardiovascular findings in hypothyroidism are more subtle. Hypothyroidism mainly presents as lethargy, cold intolerance, constipation, proximal muscle weakness, weight gain, decreased appetite, coarse dry skin, hair loss and non-pitting edema. Cardiovascular findings in hypothyroidism are mild degree of bradycardia, diastolic hypertension and narrow pulse pressure. Mild degree of pericardial effusion is seen in up to 30% of overtly hypothyroid patients. There are several case reports on myxedema ascites, but only few case reports of hypothyroidism presenting as massive pericardial effusion. We report a case of 54-year-old female presenting with breathlessness and found to have massive pericardial effusion without tamponade. After extensive workup, she was found to have primary hypothyroidism. In contrast, she had no other symptoms and clinical signs suggestive of hypothyroidism. She was improved after treatment with levothyroxine.

Keywords: Hyperthyroidism; Pericardial effusion; Levothyroxine

Case Presentation

A 54-year-old female (weight 49 kg; BMI 18 kg/m²) presented to our department with class 2 breathlessness for last 6 months which had progressed to class 3 for last 1 month. She had no previous history of diabetes, hypertension and coronary artery disease. Vitals showed, blood pressure of 110/70 mmHg, heart rate of 112/min, respiratory rate of 20/min, SpO₂ of 94% and she was afebrile. Her general physical examination was normal, JVP was not raised and there was no pedal edema. Cardiovascular examination showed only muffled heart sounds, respiratory system was normal and on neurological examination her tendon reflexes were not delayed.

On investigations, electrocardiogram showed low voltage complexes, chest X-ray showed water bottle heart (Figure 1) and on echocardiography there was massive pericardial effusion without evidence of diastolic collapse of right atrium and right ventricle. Thickness of pericardial effusion in apical 4 chamber view was 33 mm (Figure 2), in parasternal long axis view was 31.8 mm (Figure 3) and in parasternal short axis view was 21.5 mm (Figure 4). Hb was 13 gm%, ESR 21 mm 1st hr, urea 31 mg/dl, creatinine 0.9 mg/dl, serum proteins (total 6.5 gm/dl, albumin 3.5 gm/dl), SGOT 31 IU, SGPT 35 IU, ANA was negative and urine examination was also normal.

She underwent pericardiocentesis through subxiphoid approach. Around 500 ml of light golden colour fluid tapped and sent for analysis. Cytology showed 120 cells with lymphocytic predominance without

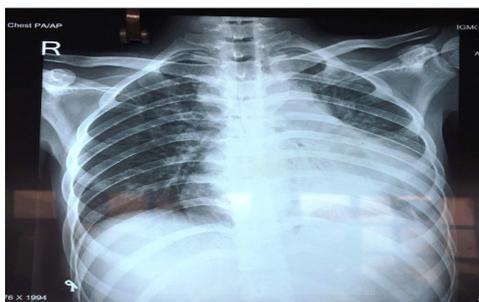


Figure 1. X-ray chest showing water bottle heart suggestive of massive pericardial effusion.



Figure 2. Echocardiogram apical 4 chamber views showing pericardial effusion of thickness 33 mm.



Figure 3. Echocardiogram PLAX view showing pericardial effusion of thickness 31.8 mm and there was no evidence of right ventricle diastolic collapse.

any malignant cells, proteins 1.1 gm/dl (transudative), ADA was 10 IU/L, genexpert for tuberculosis was negative and PCR for tuberculosis

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Figure 4. Echocardiogram PSAX view showing pericardial effusion of thickness 21.5 mm.

was also negative. Contrast enhanced CT did not reveal any pathology. Finally, TFT were sent and it showed T3 0.31 ng/ml, T4 3.58 ng/ml and TSH 136 μ IU/ml. So final diagnosis of primary hypothyroidism was made and she was started on Levothyroxine 100 microgram/day. There was gradual improvement in patient symptoms. After one month during follow up, on repeat echocardiography there was no evidence of pericardial effusion.

Discussion

Thyroid disorders are highly prevalent, occurring most frequently in aging women. The most common signs of hypothyroidism are bradycardia, diastolic hypertension, a narrowed pulse pressure, and attenuated activity on the precordial examination. Other characteristic but nonspecific findings are high serum concentrations of cholesterol and creatine kinase [1,2]. Pericardial effusions and non-pitting edema (myxedema) can occur in patients with severe, long-standing hypothyroidism. Hypothyroidism prolongs the cardiac action potential and the QT interval [3].

Triiodothyronine causes increased tissue thermogenesis, decreased systemic vascular resistance, increased RAAS activity leading to increased blood volume, increased cardiac ionotropy and cardiac output. So, hyperthyroidism causes systolic hypertension and hypothyroidism causes diastolic hypertension because of change in SVR and RAAS activity [4]. Thyroid hormone causes activation of β 1-adrenergic receptors and causes increased cardiac contraction. Thyroid hormone also causes phosphorylation and inactivation of phospholamban protein [5], the protein which normally inhibits SERCA channel on sarcoplasmic reticulum. This results in activation of SERCA and increased uptake of calcium in sarcoplasmic reticulum which causes relaxation of heart [5].

Hypothyroidism can cause mild pericardial effusion in upto 30% cases as demonstrated by Hardisty et al. [6]. There were many case reports regarding hypothyroidism as a cause for ascites. But hypothyroidism causing massive pericardial effusion have rarely been reported. The patients in available case reports are overtly hypothyroid and clinically symptomatic. Our patient had no other symptoms and clinical signs suggestive of hypothyroidism and massive pericardial effusion is exclusive presentation.

Mechanism of Effusions in Hypothyroidism

Serous cavity effusion is seen less frequently in hypothyroidism.

Following mechanism are described:

1. Low level of thyroxine hormone causes increase in capillary permeability and leakage of albumin and lack in compensatory increase in lymph flow [7].
2. Hyaluronic acid accumulates in body cavities and form complex with albumin that prevent the lymphatic drainage of extravasated albumin [7].
3. Recent studies have shown that a decrease in nitric oxide levels causes an increase in capillary permeability, increased plasma protein filtration out of the microvasculature and endothelial dysfunction [8].

So, effusions occurring because of hypothyroidism are mainly exudative. But in our case effusion is transudative. Gottehrar et al. reported that pleural effusions because of hypothyroidism were borderline between exudates and transudates and showed little evidence of inflammation [9].

This is rare case of hypothyroidism primarily presenting as massive pericardial effusion. Interestingly is this case there were no other clinical findings suggestive of hypothyroidism.

Conclusion

In conclusion, hypothyroidism presenting as massive pericardial effusion is rare but easy to treat. Treatment with thyroid hormone replacement therapy leads to complete regression of the pericardial effusion. On routine evaluation of massive pericardial effusion after excluding common causes such as tuberculosis, malignancies and infections and connective tissue disorders, thyroid function tests should be performed even if there are no other signs and symptoms suggestive of hypothyroidism.

References

1. Klein I (1990) Thyroid hormone and the cardiovascular system. *Am J Med* 88: 631-637.
2. Klein I, Ojamaa K (2000) The cardiovascular system in hypothyroidism. In: Braverman LE, Utiger RD, eds. *Werner & Ingbar's the thyroid: A fundamental and clinical text*. (8th edn). Philadelphia: Lippincott Williams & Wilkins; pp. 777-782.
3. Ojamaa K, Sabet A, Kenessey A, Shenoy R, Klein I (1999) Regulation of rat cardiac Kv1.5 gene expression by thyroid hormone is rapid and chamber specific. *Endocrinology* 140: 3170-3176.
4. Dillmann WH (1990) Biochemical basis of thyroid hormone action in the heart. *Am J Med* 88: 626-630.
5. Kiss E, Jakab G, Kranias EG, Edes I (1994) Thyroid hormone-induced alterations in phospholamban protein expression: Regulatory effects on sarcoplasmic reticulum Ca^{2+} transport and myocardial relaxation. *Circ Res* 75: 245-251.
6. Hardisty CA, Naik DR, Munro DS (1980) Pericardial effusion in hypothyroidism. *Clin Endocrinol* 13: 349-354.
7. Parving HH, Hansen JM, Nielsen SL, Rossing N, Munck O, et al. (1979) Mechanisms of edema formation in myxedema increased protein extravasation and relatively slow lymphatic drainage. *N Engl J Med* 301: 460-465.
8. Kubes P, Granger DN (1992) Nitric oxide modulates microvascular permeability. *Am J Physiol* 262: H611-H615.
9. Gottehrar A, Stanford GG, Sahn SA (1990) Hypothyroidism and pleural effusions. *Chest* 98: 1130-1132.