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Think outside the "box" Revisiting Heart Failure and Hypothyroidism

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Abstract

Heart failure is one of the most common health problems requiring hospitalization. Usual causes exacerbating HF include cardiomyopathies, coronary heart disease and uncontrolled hypertension. When presented with such a case, while it is important to rule out underlying cardiac causes, one must also think of reasons outside the heart. Hypothyroidism is a common non-cardiac etiology for heart failure, which may be missed especially given its subtle clinical features. We present a 64 year old woman, who presented with heart failure secondary to longstanding hypothyroidism. She developed dilated cardiomyopathy which was not reversible even with thyroxin supplementation and eventually needed an ICD placement. This case highlights the importance of thyroid disorder on heart disease and how on occasion, it can even be permanent.

Keywords: Hypothyroidism; Cardiomyopathy

Introduction

Heart failure is the second most common reasons for hospitalizations and health care expenses. 5% of the cases are secondary to non-cardiac etiologies including hypothyroidism [1]. While it is important to rule out underlying coronary artery disease, one must also be aware of thyroid hormones and its effects on the heart. Usually, these changes are reversible with thyroxin supplementation but we present a case of non-ischemic cardiomyopathy with permanent damage from long-standing hypothyroidism.

Case Report

This is a 64-year-old woman with history significant for hypertension, who had a total thyroidectomy for multi-nodular goiter about four years ago and has been on replacement therapy with levothyroxine since then. About a year ago, she lost her insurance and wasn't able to afford health care, including medications. For the past few months, she started to note difficulty breathing which eventually progressed to occurring even on mild activity. In addition, she also noted swelling in her feet for about a month, spreading upwards to her legs with minimal improvement with leg elevation. Given her difficulty breathing along with feeling tired and fatigue, she decided to come to the ER.

On examination, she appeared ill and hypervolemic with BP of 157/124. She had a raspy voice and dry, flaky skin. Respiratory examination was benign except for decreased breath sounds in the bases. Cardiovascular examination revealed normal S1, S2 with 2/6 ejection systolic murmur along with 1+ to 2+ pitting pedal edema. Laboratory studies revealed microcytic anemia with hemoglobin of 11.3 g/dl, creatinine of 1.19 mg/dL and elevated BNP of 1434 pg/mL. A 12-lead EKG showed sinus rhythm, left ventricular hypertrophy with occasional paroxysmal ventricular complexes (PVCs) and the chest X-ray showed an enlarged cardiomediastinal silhouette with minimal trace right pleural effusion. Treatment with intravenous furosemide was initiated for possible acute exacerbation of right heart failure and cardiac consult was obtained.

Given the history of hypothyroidism, thyroid panel was obtained, which revealed elevated TSH at 66.5 mIU/L and a low free T4 of 0.33 ng/dL. A 2D Echo cardiogram was showed moderate to severely dilated left-sided chambers with severely depressed function. The estimated ejection fraction was about 10-15% and there was also mild-to-moderate pericardial effusion but without any evidence of tamponade. Cardiac catheterization showed very mild coronary artery disease ruling out ischemia and her symptoms were attributed to dilated non-ischemic cardiomyopathy likely from severe hypothyroidism and uncontrolled hypertension. Medical management was optimized with 50 mcg of levothyroxine in addition to aspirin, carvedilol, lisinopril, digoxin, spironolactone and furosemide and she was sent home on a life-vest with a plan to re-evaluate in 90 days for a possible intracardiac defibrillator. Repeat TSH in a few weeks showed improvement to 2.76 mIU/L but follow up echo in 90 days showed persistent cardiomyopathy with an estimated ejection fraction persisting at 15% requiring a defibrillator implant. Although rare, her cardiac function had not improved even with levothyroxine supplementation, but she had been following up at the out-patient clinic and has been doing well.

Discussion

Heart failure (HF) is a complex clinical condition that can result from any structural or functional cardiovascular disorder causing inadequate systemic perfusion. It is characterized by symptoms such as dyspnea, weight gain and fatigue, and signs, such as fluid retention, elevated jugular venous pulse. More than 20 million people suffer from HF worldwide. In 2011, HF was the most common reason for hospitalization in patients older than 85 years of age and the second most common for those aged 65–85 years [2].

The most common causes of HF are coronary (ischemic) heart disease, hypertension, and valvular disease. In one review, coronary disease and hypertension accounted for 62 and 10 percent of cases, respectively [1]. About 5% of the cases of HF were secondary to other causes, like thyroid disorders, alcohol, sarcoidosis, and amyloidosis.

Maintenance of thyroid hormone homeostasis is required for proper cardiovascular function. Thyroid hormones affect the cardiovascular system either directly by modifying cardiac contractile force and frequency,

or indirectly by affecting the tissue metabolism, fluid volume status and by altering the sympathetic and vagal modulation [3,4]. Therefore, both deficiency and excess of thyroid hormone can be associated with cardiovascular injury and heart failure.

Cardiac abnormalities have been studied extensively in patients with primary hypothyroidism. Various types of cardiomyopathies, dilated or hypertrophic, increased peripheral vascular resistance, arrhythmias, and conduction defects have been reported. Almost one-third of patients also are found to have pericardial effusion that resolves when euthyroidism is achieved [5].

The exact mechanism of the effect of thyroid hormones on the heart is still unknown. Many theories have been postulated including a study conducted on adult rats, which showed that chronic hypothyroidism affected the coronary arterioles thereby impairing the blood flow. This caused changed in the size, shape and structure of the myocytes leading to HF [6]. Usually these changes in the cardiac structure and function are dependent on the severity of the thyroid hormone deficiency and generally regress with T4 replacement.

Thyroid hormones, mainly triiodothyronine (T3) has also shown to play a role at the genetic level by regulating the expression of myosin heavy chains. These chains encode the contractile protein of the cardiac myocyte resulting in decreased contractility, slower heart rate, and decreased conduction. It also has non-genomic action on specific membrane proteins (like ion transporters ($Ca^{2+,}$ Na^{+}) and glucose transporters), organelles (including mitochondria) and cytoskeletal proteins. The cardiac myocyte is very sensitive to changes in serum T3, and this is evidenced by rapid changes in the expression of T3 mediated genes [7]. Ultimately, it affects the myocyte calcium cycling leading to improved contraction and relaxation and overall enhances the cardiac function. Another possible mechanism is related to increased oxidative stress from reduced glutathione levels in the myocardial tissue causing direct myocardial damage [8].

Hypothyroidism has also been associated with prolongation of QT and increased QT dispersion. The sympathovagal imbalance and increased ventricular deregulation can predispose to potentially life-threatening arrhythmias [4].

In addition to cardiomyopathies, hypothyroidism has also been associated with pericardial effusions. Typically, these are hemodynamically inconsequential with an incidence reported to be as low as 3% in an early mild stage to 80% when myxedema is present [9]. Echocardiography demonstrates small to moderate effusions in as many as 30% of overtly hypothyroid [7]. This effusion usually has a "gold paint" appearance and is exudative in character with increased cell count, increased cholesterol and plenty of cholesterol crystal with mononuclear infiltrates [10]. The likely mechanism for the effusions appears to be from increased capillary albumin leak, extravasation of hygroscopic mucopolysaccharides into the pericardial space, decreased lymphatic drainage, as well as increased retention of salt and water. On rare instances, these can get quite large but cardiac tamponade is typically rare because of the slow accumulation of fluid, and the pericardial distensability allowing for a large amount of fluid collection without hemodynamic compromise [9,10]. Given the reversal of the effusion with therapy, pericadiocentesis is rarely required, unless in tamponade [11,12].

Typically, these changes are reversible by thyroid hormone replacement, but it has been found in rare cases that long-term hypothyroid state may cause irreparable myocardial damage. Shuvya et al. [8] reported a case of significant, irreversible and biopsy-proven myocardial injury attributed to a long standing hypothyroid state.

Because of its influence on glucose transporter and mitochondrial metabolism, severe hypothyroid states can reduce muscle energy production, which can lead to extensive muscle injury and elevated serum creatinine kinase (CK) anywhere from 50% to 10-fold times. Rhadbomyolysis (elevated CK) is seen in up to 30% of the patients with hypothyroidism. In addition, hypothyroidism also produces increase in total and LDL cholesterol as well as apolipoprotein B leading to dyslipidemia. As a result of an increase in risk factors, including hypercholesterolemia, hypertension, and elevated levels of homocysteine, patients with hypothyroidism may by themselves have increased risk for atherosclerosis and heart disease [7,13].

Conclusion

While coronary artery disease and hypertension continue to be the most common causes for heart failure, it is important to consider other causes, especially thyroid disorders. The clinical features of hypothyroidism, such as weight gain, fatigue, cold intolerance, constipation, dry skin, edema, and slow mentation are usually subtle and the diagnosis may be missed when they present with heart failure. Patients with sub-clinical hypothyroidism may not even have any features of hypothyroidism, but have been found to have cardiac manifestations, which are reversible with replacement therapy when caught in time. Thus it is very important to keep in mind, that it's not always coronary heart disease of hypertension or a cardiac disease which can cause heart failure, but also rule out hypothyroidism before it causes permanent damage.

References

1. He J, Ogden LG, Bazzano LA, Vupputuri S, Loria C, et al. (2001) Risk factors for congestive heart failure in US men and women: NHANES I epidemiologic follow-up study. Arch Intern Med 161: 996-1002.
2. Tognini S, Pasqualetti G, Calsolaro V, Polini A, Caraccio N, et al. (2014) Cardiovascular risk and quality of life in elderly people with mild thyroid hormone deficiency. Front Endocrinol (Lausanne) 5: 153.
3. Ladenson PW (1990) Recognition and management of cardiovascular disease related to thyroid dysfunction. Am J Med 88: 638-641.
4. Muthu V, Bhatta L (2013) Sudden Cardiac Death Due to Untreated Hypothyroidism. The Journal of Innovations in Cardiac Rhythm Management 4: 1097–1099.
5. Kissell N, Mudd JO, Gelow JM, Chong LE, Yuen KC (2014) Cardiogenic Shock Due to Non-Ischemic Cardiomyopathy Induced by Severe Anterior Hypopituitarism. Endocr Pract 4: 1-17.
6. Gerdes AM, Iervasi G (2010) Thyroid replacement therapy and heart failure. Circulation 122: 385-393.
7. Danzi S, Klein I (2014) Thyroid disease and the cardiovascular system. Endocrinol Metab Clin North Am 43: 517-528.
8. Shuvy M, Shifman OE, Nusair S, Pappo O, Lotan C (2009) Hypothyroidism-induced myocardial damage and heart failure: an overlooked entity. Cardiovasc Pathol 18: 183-186.
9. Chou SL, Chern CH, How CK, Wang LM, Huang CI, et al. (2005) A rare case of massive pericardial effusion secondary to hypothyroidism. J Emerg Med 28: 293-296.
10. Zimmerman J, Yahalom J, Bar-On H (1983) Clinical spectrum of pericardial effusion as the presenting feature of hypothyroidism. Am Heart J 106: 770-771.
11. Setty NS, Sadananda KS, Nanjappa MC, Patra S, Basappa H, et al. (2014) Massive pericardial effusion and cardiac tamponade due to cholesterol pericarditis in a case of subclinical hypothyroidism: a rare event. J Am Coll Cardiol 63: 1451.
12. Ekka M, Ali I, Aggarwal P, Jamshed N (2014) Cardiac tamponade as initial presenting feature of primary hypothyroidism in the ED. Am J Emerg Med 32: 683.
13. Zare-Khormizi MR, Rahmanian M, Pourrajab F, Akbarnia S (2014) Massive pericardial effusion and rhabdomyolysis secondary to untreated severe hypothyroidism: the first report. Acta Clin Belg 69: 375-378.