

Biventricular Heart Failure, an early sign of thyrotoxicosis

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Abstract

The prevalence of congestive heart failure (CHF) is increasing. A rare cause of CHF is hyperthyroidism. It can affect the cardiovascular system manifesting from decreased systemic vascular resistance, increased left ventricular contractility, and sinus tachycardia to atrial fibrillation. Less than 0.5% is due to tachycardia-mediated mechanism. Heart failure in the absence of underlying cardiac disease or arrhythmia is thought to reflect a rate related cardiomyopathy that most likely resolves with treatment.

Our patient is a 56 year-old African American female with past medical history of subacute thyroiditis and medication noncompliance presented to ER with a two-week history of increasing exertional dyspnea and bilateral leg edema. She was hypertensive, tachycardic and tachypnic. Examination revealed bibasilar rales and 2+ pitting edema. Laboratory data revealed a TSH of 0.01 and free T4 of 3.3, consistent with hyperthyroidism. Two-dimensional echocardiogram revealed biventricular enlargement and ejection fraction of 25%. Thyroid uptake scan demonstrated significant homogenous uptake in both lobes consistent with Grave's disease. Six months ago her thyroid scan showed 5% uptake with hyperthyroid state, consistent with subacute thyroiditis. She was treated with beta-blockers and diuretics with profound symptomatic improvement. Definitive therapy consisted of maintaining euthyroid state.

This case illustrates an unusual presentation of Grave's disease with CHF without atrial fibrillation. Typically, thyrotoxicosis presents as high output failure. However, as time progresses it can cause low output failure. Dilated cardiomyopathy is an unusual manifestation of hyperthyroidism with unclear etiology. Early diagnosis is of utmost importance as some patients with hyperthyroidism may have a reversible form of dilated cardiomyopathy.

Keywords: Congestive heart failure, Grave's thyrotoxicosis, Tachycardiomyopathy.

Introduction

Roughly one out of 5 Americans above the age of 40 will develop congestive heart failure (CHF) at some point in their lives [1]. About half of these patients will not survive longer than 5 years after their hospitalization [2]. Therefore, it is important to determine what is the underlying cause of the heart failure, and if it is amenable to treatment. We present an unusual case of CHF due to hyperthyroidism. About half of CHF cases with hyperthyroidism actually occur in spite of increased cardiac output. Regardless of cardiac output, most CHF cases present with concurrent atrial fibrillation. Our patient had neither. She presented with systolic dysfunction, with a tachycardia induced cardiomyopathy.

Case Report

Our patient was a 56 years old African American female, who presented to the emergency room with a two week history of progressive exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea and bilateral lower extremity edema. The patient's past medical history was significant for subacute thyroiditis, and excessive alcohol abuse (about one pint of vodka per day). The patient admits to noncompliance with medication and noncompliance to follow up with her primary care provider.

Upon examination, she had a blood pressure of 203/121. Her heart rate was 136 beats per minutes, and regular. She had a respiratory rate of 32. She appeared to be in moderate distress. Chest auscultation revealed bibasilar rales. She was tachycardic, but had normal S1, and S2, with a regular rate, and rhythm. No murmurs, rubs, gallops were auscultated. There were 2+ pitting edema of both lower extremities, extending from ankles up to knees. The examination was otherwise unremarkable.

An electrocardiogram (EKG) was obtained in the emergency room. It showed sinus tachycardia (regular rhythm) without any ischemic changes (Figure 1). The patient was diagnosed with hypertensive emergency. A beta-blocker drip was started to titrate her blood pressure. Intravenous furosemide was also administered. An echocardiogram was performed

later. It showed biventricular enlargement, and systolic dysfunction with an ejection fraction (EF) of 25% (Figure 2).

Laboratory results demonstrated a normal complete blood count, blood urea nitrogen, creatinine, and electrolytes. Thyroid stimulating hormone (TSH) level was 0.01 (normal 0.4 - 4.0 mIU/L), and free thyroxine was (T4) 3.3 (normal 0.7 to 2 mcg/dL). Records from an outside institution showed that a thyroid scan had been done. The scan demonstrated 5% uptake, and she was diagnosed with subacute thyroiditis. However, due to the results of the recent thyroid panel, another scan was conducted at our institution. This scan showed significant homogeneous uptake in both lobes (Figure 3). This was consistent with Grave's disease.

As her medical history did not include an antecedent myocardial infarction or long standing angina, we initially believed her CHF to be either idiopathic in etiology or secondary to alcohol consumption. However, we did not catheterize her heart on this visit to definitively rule out coronary artery disease.

Considering her thyroid function, we were highly suspicious that her CHF was an atypical presentation of hyperthyroidism. She did not have atrial fibrillation (as most patients do), but she did have sinus tachycardia. Administration of a beta-blocker with furosemide substantially improved the symptoms. She was scheduled for thyroid ablation with radio active iodine and methimazole was administered in the interim period. To follow up, we planned to monitor her TSH, and maintain her on replacement thyroid hormones once she becomes hypothyroid. We also planned to monitor her cardiac function. However, she was lost to follow up.

Discussion

Some of the common underlying etiologies for CHF include hypertension, diabetes mellitus, coronary artery disease, and antecedent myocardial infarction [1]. She did not report long standing hypertension, or any of these other conditions. Her current episode of hypertensive emergency was thought to be related her thyrotoxicosis.

Hyperthyroidism that initially presents as heart failure is an uncommon event, as only 6% of hyperthyroid patients have heart failure [3]. About half of these cases have normal ventricular ejection fractions (>50%), and fall under the category of high output heart failure. These cases are also highly associated with coexisting atrial fibrillation (odds ratio of 37.4) [3]. There have been several case reports of CHF with atrial fibrillation in thyrotoxicosis, which improved upon restoration of a euthyroid state [5-8].

However, our patient had an unusual presentation. She had dilated cardiomyopathy, with an ejection fraction of 25%. Moreover, telemetry did not show atrial fibrillation. Her cardiomyopathy was thus attributed to sinus tachycardia.

Tachycardia has been shown to cause cardiomyopathy in the long term [4]. A possible mechanism may be that chronic tachycardia increases the concentration of calcium within the cytosol during diastole, with reduced ventricular contractility. In our case, thyrotoxicosis may well have been the cause of her sinus tachycardia. Tachycardia induced cardiomyopathy, or simply tachycardiomyopathy, is a term that most frequently refers to cardiac dysfunction caused by faster heart rate, usually in the range 130's. The array of etiologies involved can range from sinus tachycardia to atrial fibrillation.

Limited literature on this subject indicates that the heart failure is often reversible. It is unclear as to how much time recovery takes for an average patient. A case report by Walker et al. described improvement of heart function in their patient "rapidly" after restoration of a normal heart rate [10]. Another report by Singh et al. reported improvement after 3 months [11]. Another study documents 6 patients with tachycardia, caused by an accessory pathway were, who were treated with surgical ablation. On average, the patients had an improvement of 22.3% in their ejection fraction after a mean of almost 22 months [12]. Iyer suggests that the prognosis is affected by two main factors: the rate and duration of the arrhythmia [13].

To summarize differential diagnosis for our patient's the heart failure, we were mainly concerned about three principle etiologies. First was the possibility of ischemic heart disease.

We could not definitively rule out CAD with a cardiac catheterization, as it would entail administering iodinated contrast media to a patient in thyrotoxicosis. This diagnosis was not high on differential. She did not give a history of long standing angina, or have a prior myocardial infarction. Our remaining differential included thyroid cardiomyopathy, and tachycardiomyopathy.

The recommended treatment for such patients includes beta blockers. Beta blockers help control the tachycardia as well other symptoms of hyperthyroidism. ACE-inhibitors and diuretics should be used as well. The underlying cause must be addressed with antithyroid medications (such as propylthiouracil or methimazole) or ablation of the thyroid. If atrial fibrillation is present, the patient must be cardioverted after euthyroidism is restored.

We ablated our patient's thyroid, and planned to monitor her for improvement thereafter. If she fails to show improvement after a few months of becoming euthyroid on replacement thyroxine, we planned to do the cardiac catheterization (as the iodinated media would not pose a risk after the patient becomes euthyroid) to check for CAD. However, the patient was lost to follow up.

Conclusion

It is important to recognize cases of heart failure that may have treatable etiologies. Heart failure due to hyperthyroidism (whether high output heart failure, or congestive heart failure with systolic dysfunction) is one such example. Early treatment may reverse the cardiomyopathy. Lowering the threshold of suspicion to check for thyroid function in newly diagnosed patients with heart failure, or previously diagnosed patients presenting with exacerbation may help to identify cases that are treatable. Vice versa, there may be a role for monitoring cardiac function in patients with thyroid dysfunction as well.

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Figure1: 12 lead Electrocardiogram showing Sinus Tachycardia.

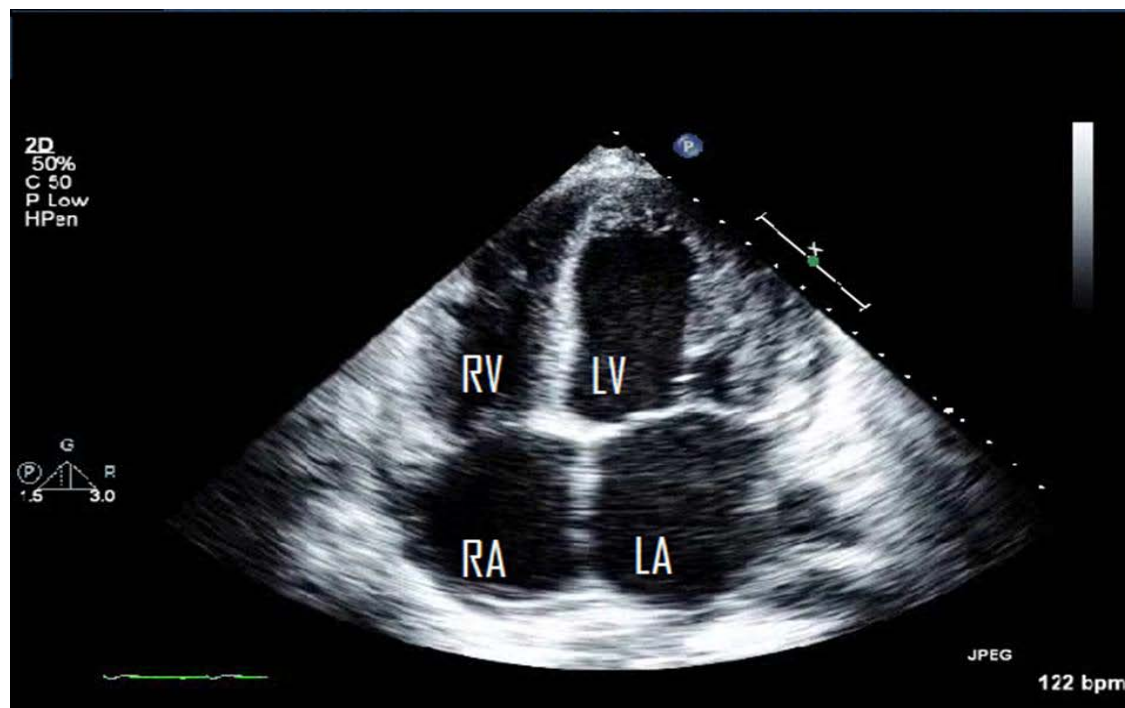


Figure 2: Two dimensional Echocardiogram showing enlarged heart chambers; consistent with Biventricular failure. LV – Left ventricle. LA – Left atrium. RV – Right ventricle. RA – Right atrium.

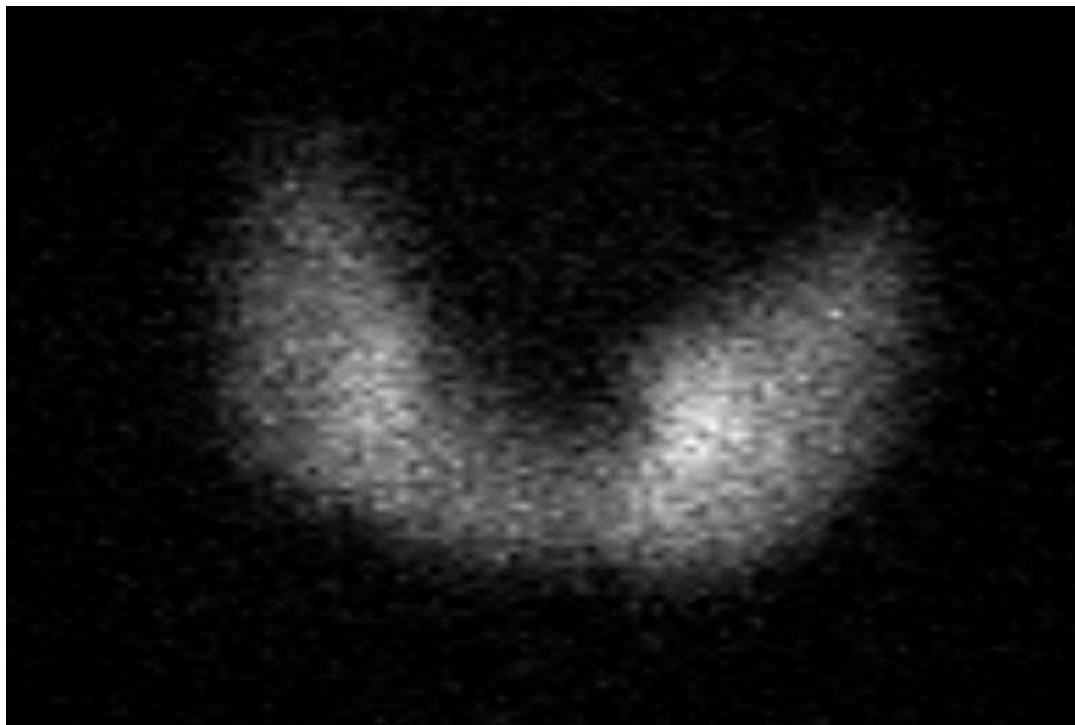


Figure 3: Thyroid uptake scan showing diffuse uptake in both lobes; consistent with Graves's thyroid disease.