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Thyrotoxic Cardiomyopathy: A Case Report

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Abstract INTRODUCTION: Thyrotoxic cardiomyopathy defines a myocardial damage caused by the toxic effects abundant thyroid hormones that result in altered energy production by myocytes, intracellular metabolism and myofibril contractile function. CASE REPORT: A 29 year old female presented with recurrent leg swelling of three months, abdominal swelling of one week and breathlessness of a day's duration. There was paroxysmal nocturnal dyspnea, orthopnea and non-productive cough. She also complained of persistent symptoms of palpitations, heat intolerance, weight loss, excessive sweating, weight loss, bulging eyes and progressive neck swelling over a three year period for which she was diagnosed with hyperthyroidism. Significant examination findings include tachycardia, elevated JVP, S3 gallop, bibasal fine crepitations, hepatomegaly and ascites. DISCUSSION: ECG showed atrial flutter with fixed 2:1 block. Echocardiography showed dilated cardiac chambers with severe left ventricular systolic dysfunction and restrictive diastolic dysfunction, impaired right ventricular function and severe mitral and tricuspid regurgitation. Thyroid function (Serum TSH=0.005IU/L, free T4=39.8pmol/l, free T3=10.1 pmol/l). She responded to anti-failure medication but repeatedly developed adverse reactions to antithyroid medication. CONCLUSION: Thyrotoxic cardiomyopathy in adults has been reported in very few publications, but immediate antithyroid therapy has been shown to provide good long-term outcome in these patients. The challenge with our patient was getting her to tolerate her antithyroid medication.

Keywords Thyrotoxic, Cardiomyopathy, Hyperthyroidism, Heart failure

1. Introduction

Cardiomyopathy is an uncommon presentation in hyperthyroid patients and as an initial presentation, has been reported in 6% of hyperthyroid patients while less than 1% of them developed dilated cardiomyopathy with severe left ventricular dysfunction. [1] The cardiovascular manifestations of thyrotoxicosis may be due to direct effects of thyroid hormones at the cellular level, their interactions with the sympathetic nervous system, or alterations of peripheral circulation and metabolism. [2] Here we present the challenging case of a lady who presented with dilated cardiomyopathy with florid features of hyperthyroidism.

2. Case Report

A 29 year old Nigerian female presented with recurrent leg swelling of 3 months, abdominal swelling of one week and breathlessness of a day's duration. There was paroxysmal nocturnal dyspnea, orthopnea and non-productive cough. She also complained of persistent symptoms of palpitations, heat intolerance, weight loss, excessive sweating, weight loss, bulging eyes and progressive neck swelling over a 3 year period for which she was diagnosed with hyperthyroidism. She was commenced on carbimazole on two occasions over the past 3 years but she stopped the medication on account of side effects which she attributed to the medication. Significant examination findings include tachycardia, elevated JVP, S3 gallop, bibasal fine crepitations, hepatomegaly and ascites.

Electrocardiogram done showed atrial flutter with fixed 2:1 block (Figure 1). Echocardiography showed dilated cardiac chambers with severe left ventricular systolic dysfunction (EF= 25%) and restrictive diastolic dysfunction. Other features found during echocardiography include impaired right ventricular function and severe mitral and tricuspid regurgitation (Figure 2). Thyroid function (Serum TSH=0.005IU/L, free T4=39.8pmol/l, free T3=10.1 pmol/l).

She was commenced on frusemide, lisinopril, spironolactone, atenolol, and subcutaneous heparin with initial clinical improvement. On the third day on admission carbimazole was commenced but three hours after taking it she was noticed to be very restless and examination showed absent pulses and very low blood pressure. An assessment of cardiogenic shock was made and she was commenced on dopamine infusion. Lisinopril and carbimazole were stopped and digoxin was increased to 0.25 mg daily. She responded to this treatment and after a week on admission, was weaned off the dopamine. She was also switched to propylthiouracil which she tolerated and was discharged home after two weeks later. She however presented again a month later with

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similar symptoms after stopping her medications for two weeks on account of feeling restless and refused taking any antithyroid medication. She was stabilized on anti-failure medication and referred for radioiodine therapy. She tolerated the radioiodine therapy well and is currently stable on her heart failure medications.

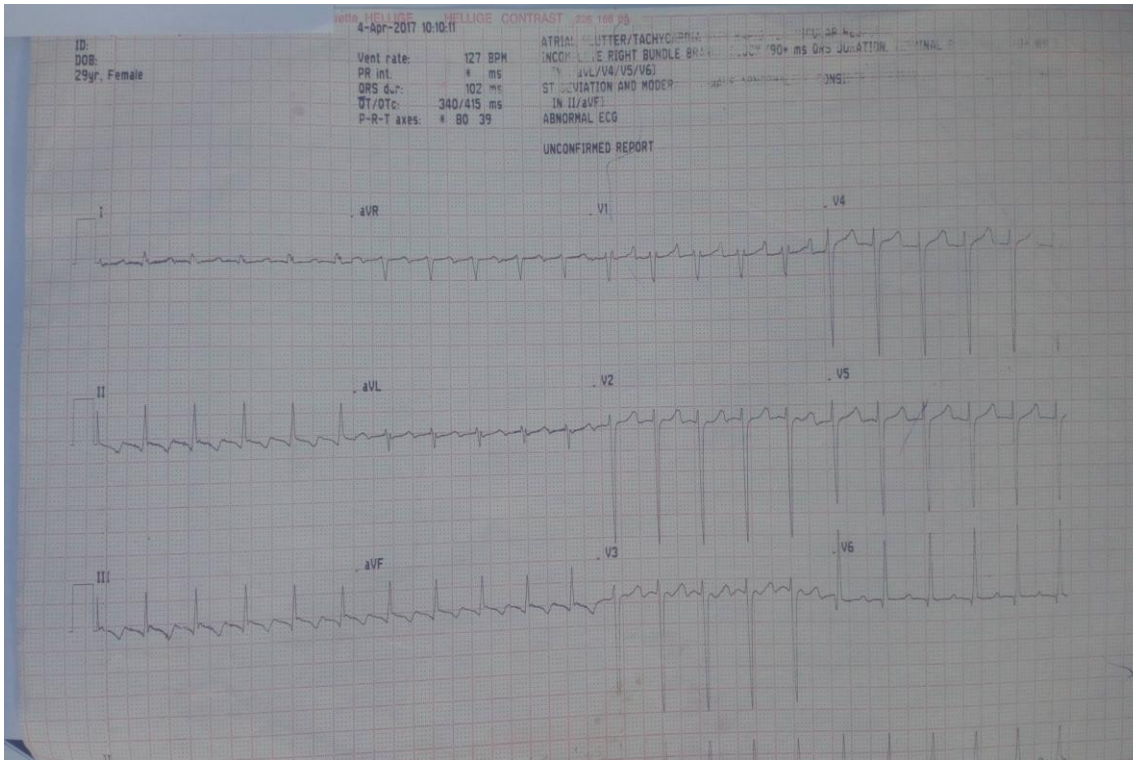

Figure 1. Electrocardiogram showing atrial flutter with fixed block

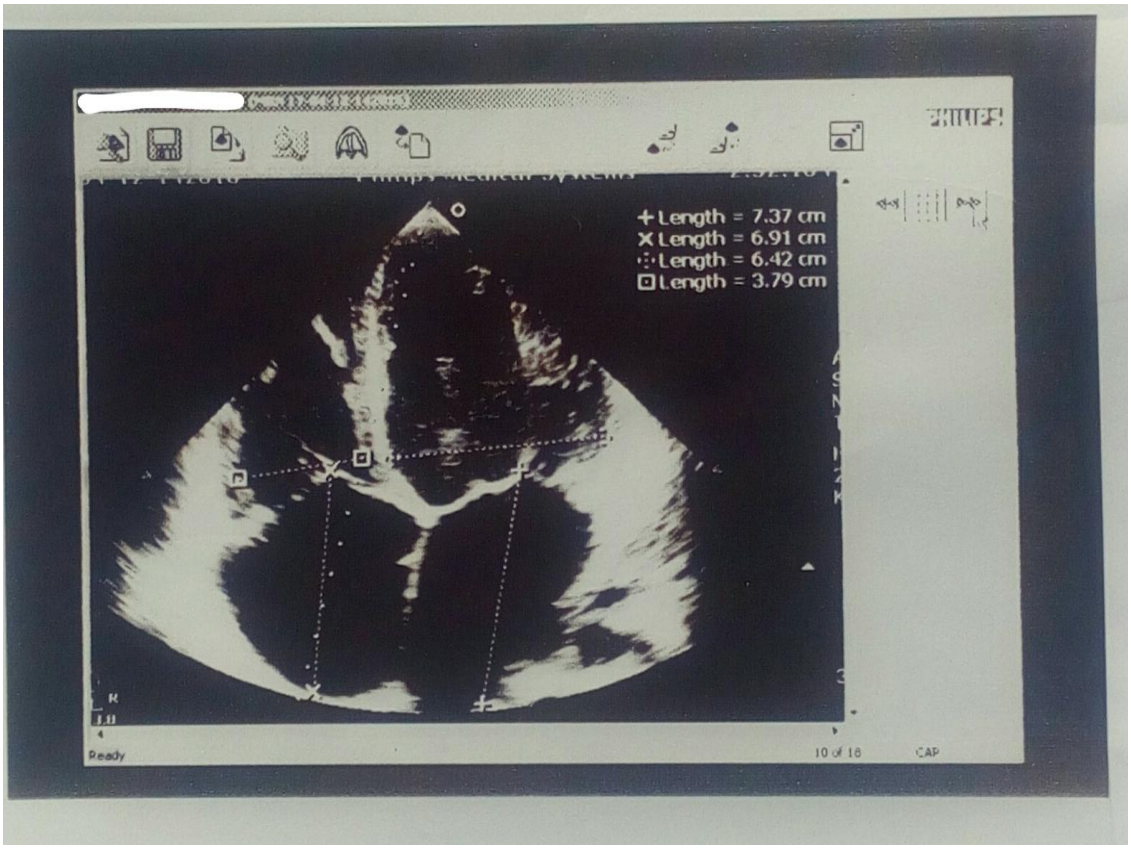

Figure 2. Apical 4 chamber view on echocardiography

3. Discussion

Perry and Graves first described the effects of thyrotoxicosis on the cardiovascular system nearly 200 years ago. Excess thyroid hormone has a direct inotropic and chronotropic action on the heart. It also increases the total blood volume and decreases the total systemic vascular resistance. All these factors contribute to the increase in cardiac output. [3] Hyperthyroidism that initially presents as heart failure is an uncommon event, as only 6% of hyperthyroid patients have heart failure. 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) [4] Reduced left ventricular contractile reserve may impair the ability to raise cardiac output to match the increase of peripheral metabolic demand. Left ventricular hypertrophy may result in impaired left ventricular filling, in particular when associated with accelerated heart rate. Atrial fibrillation may further compromise left ventricular filling because of loss of the atrial contribution and a rapid ventricular response rate. In addition, increased myocardial oxygen demand may result in myocardial ischemia, particularly in the presence of coronary artery disease or spasm, and may contribute to the occurrence of heart failure. [5]

However, our patient had an unusual presentation. She had dilated cardiomyopathy, with an ejection fraction of 25%. Moreover, electrocardiogram showed atrial flutter rather than atrial fibrillation which is more common. From the literature, it was found that some mechanisms which include genomic, nongenomic and direct action of thyroid hormone on the cardiac muscle may cause cardiomyopathy. [1] Tachycardia has been shown to cause cardiomyopathy in the long term. [6] A possible mechanism may be that chronic tachycardia increases the concentration of calcium within the cytosol during diastole, with reduced ventricular contractility. 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. [7] In our case, thyrotoxicosis may well have been the cause of her atrial flutter. Our patient was intolerant of her antithyroid medication even though there are reports in the literature of significant improvement in ejection fraction in patients with thyrotoxic cardiomyopathy who attain euthyroid state. [8] In a study of a series of seven patients with hyperthyroidism and congestive heart failure, the mean left ventricular systolic ejection fraction increased from 28% to 55% after treatment for thyrotoxicosis. The ejection fraction normalized in 5 patients, and an improvement from severe to mild systolic dysfunction was noted in the other two patients. [9] Fewer studies have shown irreversible cardiomyopathy in patients presenting with thyrotoxicosis despite achieving euthyroid state with antithyroid medication and optimal treatment with anti-failure medications. [2, 10] A severe hypotensive episode occurring after commencement of beta adrenergic blockade has been reported [8] though our patient believes that her episode was a side effect of the antithyroid medication.

4. Conclusions

Thyrotoxic cardiomyopathy, though uncommon, is an important cause of cardiomyopathy because it is reversible in most patients if euthyroid state is achieved with antithyroid medication. Prompt diagnosis and attainment of euthyroid state has also been shown to improve clinical outcomes. Patients with dilated cardiomyopathy should routinely be screened for hyperthyroidism. More studies need to be done to correctly define the role of thyroid hormones in the pathophysiology of dilated cardiomyopathy.

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