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Case Report of Thyrotoxic Cardiomyopathy

[1]Julius Edeogu, [1]Rosemary Stanley [1]Chibuike Eze Nwafor, [1]Chioma Unachukwu
[1]Department of Internal Medicine, University of Port Harcourt Teaching Hospital, Rivers State, Nigeria.
Corresponding Author: Rosemary Stanley

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Abstract

Thyrotoxic cardiomyopathy is a rare but potentially fatal form of cardiomyopathy. It is a diagnosis of exclusion and prompt identification is crucial as it is a reversible cause of heart failure and cardiac function can recover after achieving euthyroid using anti-thyroid medications. The case record of a 40-year-old male who presented to the cardiology clinic of the University of Port Harcourt Teaching hospital was utilized.

Case Presentation: We present a case of 40-year-old business man who presented with progressive dyspnea of two weeks and bilateral leg swelling of one week on referral from a private center. He reported paroxysmal dyspnea, orthopnea and easy fatiguability. He volunteered further history of palpitation, heat intolerance, hyper-defecation and jaundice. He had noticed a neck swelling of two years. His initial evaluation is notable for a grade 4 finger clubbing, jaundice, bilateral pitting leg edema, tachypnea, tachycardia with diffused apex, S1, S2 and grade 3 pansystolic non radiating murmur, bi-basal rales, mildly tender hepatomegaly and ascites. Neck exam was remarkable for anterior neck mass of 14cm by 10cm moved with swallowing. Initial laboratory work-ups were notable for CXR evidence of cardiomegaly and unfolded aorta, moderately low TSH and mildly elevated T3, T4, and elevated total and unconjugated bilirubin, low albumin. Echo was consistent with heart failure with reduced EF. It was evident that our patient has thyrotoxic cardiomyopathy. Endocrine team was consulted early and patient was managed with combination of carbimazole and anti-failure regimen. After a hospital stay of about three weeks, he made a remarkable recovery and was discharged home fairly stable. Thyrotoxic cardiomyopathy is a rare but potentially lethal complication of thyrotoxicosis which is reversible if promptly identified and euthyroid status is achieved using anti-thyroid drugs.

Keywords Thyrotoxic cardiomyopathy, cardiology, cardiology, cardiomegaly

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1. Introduction

The common pathophysiologic state that perpetuates the progression of heart failure is extremely complex regardless of precipitating event. Compensatory mechanisms exist on every level from sub-cellular all the way through to organ-to-organ interactions. Only when this network of adaptations become overwhelmed does heart failure ensue. Though most cases of heart failure are caused by issues within the heart itself, rarely, heart failure can be caused by a non-cardiac pathology[1].

In this case report, we describe a male patient who was referred on account of breathlessness and bilateral leg swelling and preceding history of a neck swelling who was found to have CCF 2^0 thyrotoxic disease.

Thyroid disorders have female preponderance hence often missed in their male counterparts. This portends poor out-come in the setting of heart failure. The consequences of excess thyroid hormone in the myocardium have been well described. There is predictable increase in heart rate and contractility which leads to increased cardiac out-put[2] and ultimately resulting in undue strain on the heart. Thus, hyperthyroidism, if untreated significantly increases the risk of atrial fibrillation and heart failure. Restoration of euthyroid, slowing of ventricular rate leads to excellent out-come.[3,4]

Every physician needs to have high index of suspicion for thyrotoxic cardiomyopathy as it can mimic other common diagnosis such as acute coronary syndrome and tachycardia induced cardiomyopathy or cardiomyopathies of other etiologies, especially in males where thyroid disorder is rare compared to their female counterparts. This is pertinent as treatment options differ from that of heart failure of other etiologies.

ECG abnormalities are common even in asymptomatic thyroid patients hence any such abnormalities should be interpreted in the clinical context of patient.

2. Case Report

A 40-year-old business man with tertiary level of education referred from a private hospital on account of anterior neck swelling of one year, dyspnea of two weeks and bilateral leg swelling of one week.

He had noted a neck swelling of one year. Swelling had increased in size over the time with associated heat intolerance, unintentional weight loss despite voracious appetite. He also noted episodes of frequent stooling. He reported visual impairment although no protrusion of the eyes.
Progressive dyspnea had lasted two weeks prior to presentation, initially exertional and progressed to dyspnea at rest. He reported associated PND and orthopnea, palpitations, cough and easy fatigability. He also noticed jaundice though did not report pruritus, pale stool or

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abdominal pain. Bilateral leg swelling was noticed one week following on-set of dyspnea and progressively worsened. He did not report facial swelling or reduction in urine output.
He was previously diagnosed of hyperthyroidism in a private center one year prior to presentation and has been on carbimazole though inconsistently. He was also previously diagnosed hypertensive one year prior to presentation. His family history is notable for hypertension in his mother.

His general examination was notable for moderate respiratory distress, jaundice and grade IV finger clubbing. The systemic examination carried out was remarkable for tachypnea, tachycardia with irregular rhythm and small volume, with pulse deficit of 25b/min, diffused apex, S_1, S_2 with ectopic beats and bi-basal crackles.

Neck exam was notable for an anterior neck mass measuring 14 x 10cm, non-tender and firm in consistency. Eye exam was unremarkable. PNS/CNS was notable for sweaty palms and fine tremors. A preliminary diagnosis of thyrotoxic congestive heart failure was considered to r/o hypertensive CCF and ischemic cardiomyopathy.

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Investigation:

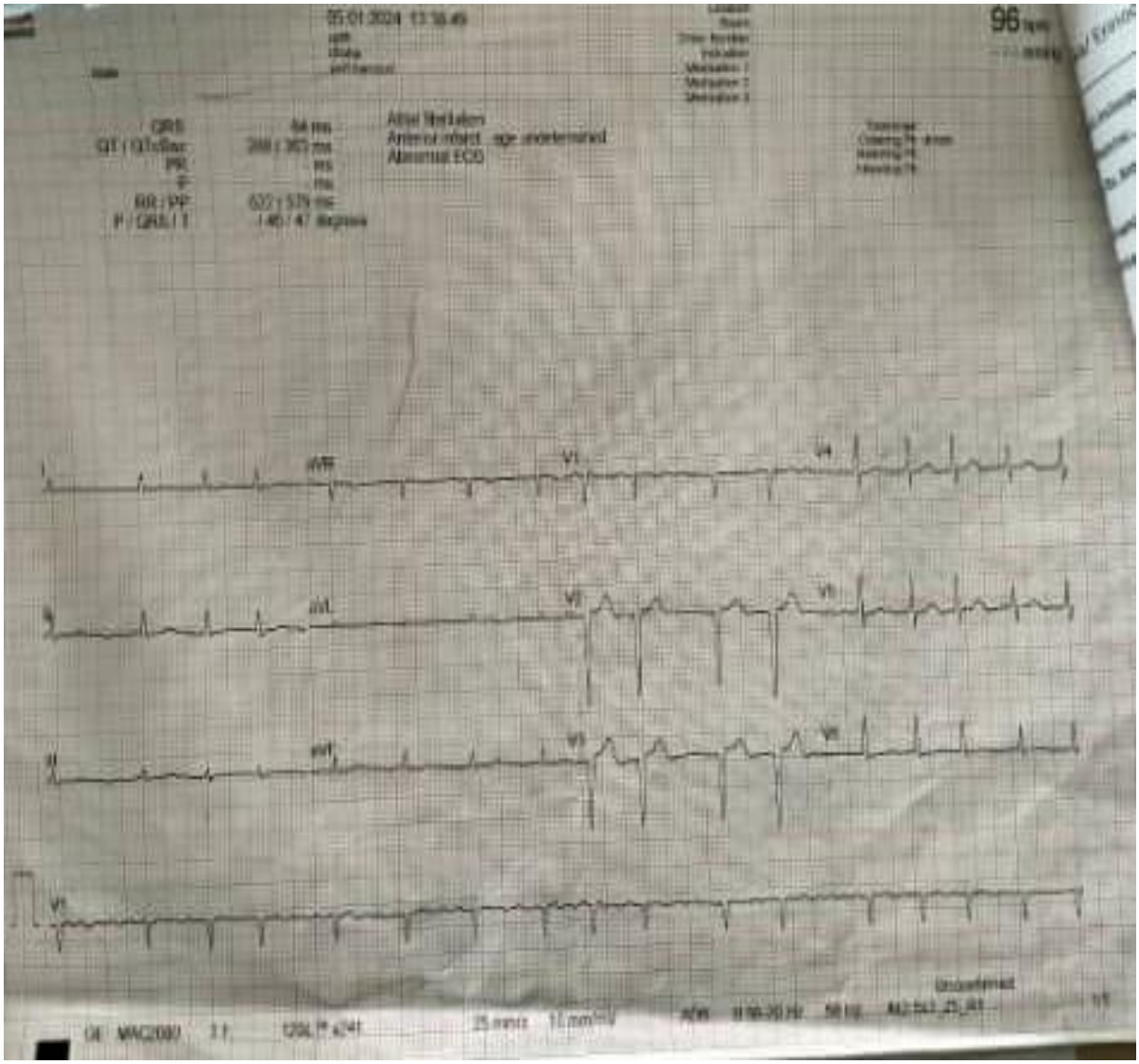

Plate 1: ECG- Atrial fibrillation with high normal ventricular rate and old anterior- septal old infarct.

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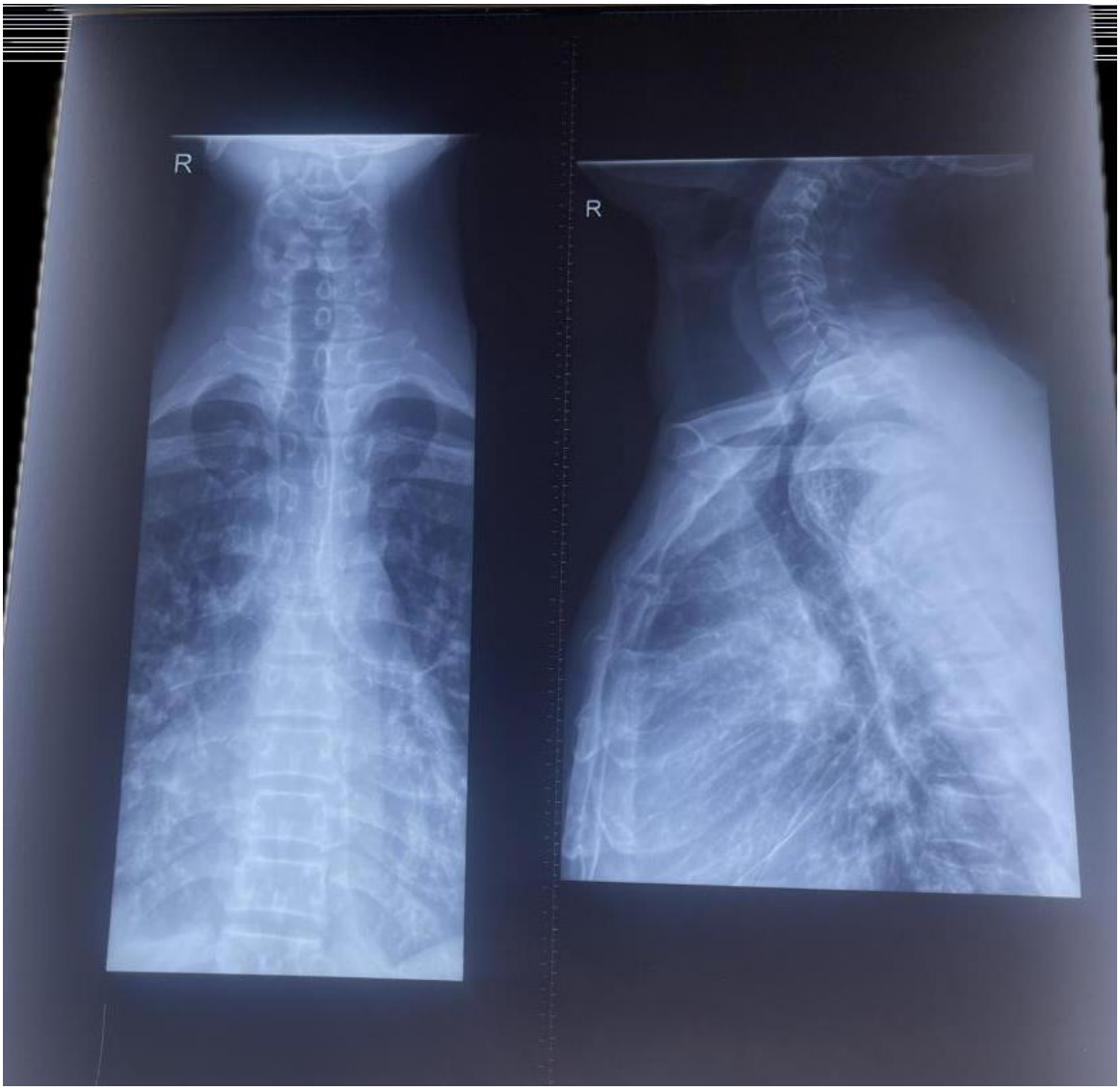

Plate 2: Thoracic inlet X-R: soft tissue bulge at the anterior neck with no retro-sternal extension.

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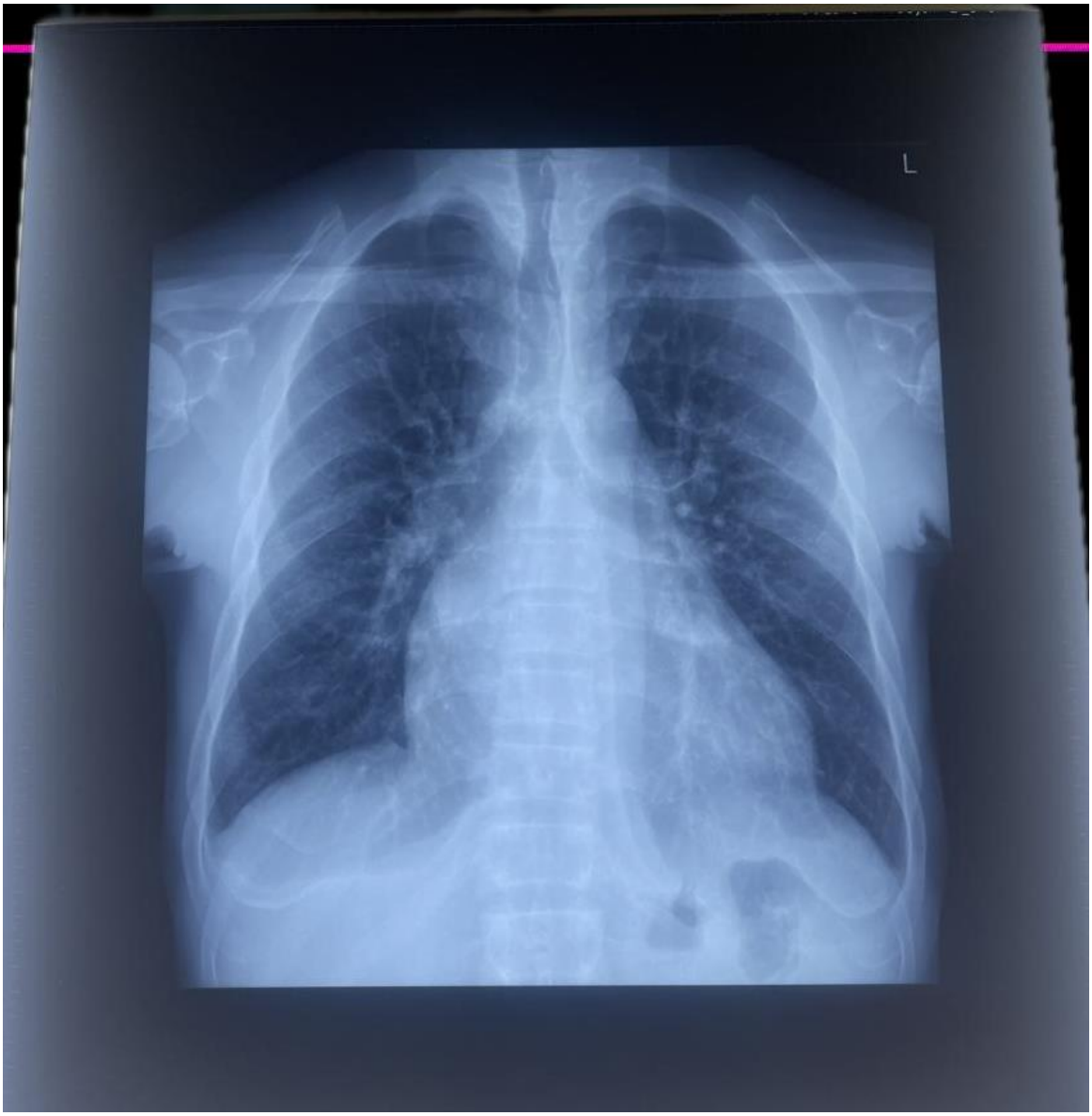

Plate 3: Chest x-ray showed aortic unfolding with cardiomegaly, prominent pulmonary vasculature with clear lung field.

Abdominal USS: mildly enlarged liver with normal out-line and echo texture. Enlarged spleen regular in out-line and parenchymal echogenicity. Other visceral organs were essentially normal.
Thyroid scan: multiple hypo echoic nodules bilaterally. Blood works are as represented in the tables below.

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Fine needle aspiration cytology: smear shows few clusters of cells with bland nuclear features. The background consists of colloid and haemorrhage. There are few clusters of benign looking thyroid cells.

Echo was notable for mildly reduced EF, dilated LV, mild PHTN, moderate MR and TR.

Table 1: E/U/Cr

	Result	Normal range
Sodium	138 mmol/L	135-145 mmol/L
Potassium	3.8 mmol/L	3.5 – 5.5 mmol/L
Bicarbonate	26 mmol/L	24-30 mmol/l
Urea	9.9 mmol/L ⇧	2.4-6.2 mmol/l
Creatinine	55 umol/L ⇩	60-130 umol/L

Table 2: Thyroid function test

	Result	Normal range
TSH	0.05 m/u/ml ⇩	0.4-4.3 m/u/ml
Free T3	4.9ng/ml ⇧	1.4- 4.2 ng/ml
Free T4	2.4 ng/ dL ⇧	0.8- 2.0 ng/dl

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Table 3: LIVER FUNCTION TEST

	Result	Normal range
Total Bilirubin	45umol/l ↑	<21 umol/L
Conjugated Bilirubin	22umol/l ↑	<5 umol/ L
AST	44 IU/L ↑	<31 IU/L
ALT	20 IU/L ↓	<35 IU/L
ALK Phosphatase	242 IU/L ↑	30-120 IU/L
Gamma GT	97 IU/L ↑	<32 IU/L
Total Protein	59 g/L ↓	66-83 g/l
Albumin	29 g/l ↓	35-52 g/l

Table 4: SEROLOGY

	Result
RVS	Sero-negative
HBsAg	negative
HCVab	negative
VDRL	non-reactive

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Table 5: FBC +ESR RESULT

	Result	Normal Range
Hb	6.9g/dL ⇩	13-18 g/dl
PCV	22% ⇩	40-45 %
RBC	2.5 x 10^{12}/L ⇩	4.5-5.5 x 10^{12}/L
MCV	79 fl × 10^{15}/L	76-93 fl × 10^{15}/L
MCH	28(PG)× 10	27-31 PG)× 10
WBC	3.3× 10^{9}/L	4-10.8 × 10^{9}/L
Platelet	98 × 10^{9}/L	90-300 x 10^{9}/L
ESR	42 mm/hr	

Table 6: THYROID ANTIBODIES

	Result	Normal range
Thyroglobulin Ab	144.34 ⇧	< 4.11 IU/ml
Thyroid peroxidase Ab	>2000.00 ⇧	<5.61 IU/ml

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Table 7: CLOTING PROFILE

	RESULT
PT	13 seconds
Control	13 seconds
Ratio	1.0
INR	1.0
APTT	36 seconds
Control	35 seconds

On the basis of physical examination and laboratory findings, a diagnosis of CCF 2^0 thyrotoxic cardiomyopathy was sustained.

3. Treatment:

Patient was initially managed with diuretics and other anti- failure regimen and transferred to cardiology unit in the medical ward.

Early consultation with the Endocrine team was made. Patient was started on antithyroid medication (Carbimazole 10mg daily) alongside anti failure drugs including propranolol 20mg daily, IV Lasix 40mg daily, Tab Digoxin 0.125mg daily, Tab Dapaglifozin 10mg daily, Tab Aldactone 25mg daily, Tab Rivaroxaban 20mg daily. He was covered for chest infection with IV Rocephine 1g daily and was weighed daily. He was nursed in cardiac position with close vital signs monitoring. Dietary measures were instituted. Propranolol was changed to B1-selective blocker with good response. Haematology team was consulted in view of moderate anaemia and mild leukopenia. Gastroenterology team consultation in view of deranged liver enzymes and hypo-albuminemia was sought. He was commenced on Livolin forte.

Outcome and follow up:

Patient was discharged home after three weeks of care in fairly stable state. He was seen in the MOPC two weeks post discharge. His review showed remarkable clinical improvement. Repeat echocardiography revealed interval improvement with EF appreciating to 50% with improvement in MR and TR. Repeat TFT also improved remarkably. His Carbimazole, antifailure regimen and life-style modification were sustained.

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4. Discussion

Thyrotoxic cardiomyopathy is a rare complication of thyrotoxicosis with tendency for a bleak out-come. Cardiomyopathy has been reported as initial presentation in 6% of patients[3] though <1% developed severe LV dysfunction.[5] It is very difficult to establish the incidence of HF related to thyroid diseases because of other etiologies such as ischemic, hypertensive and valvular heart disease that are usually not well excluded.

Cardiovascular manifestations of thyrotoxicosis may be due to both direct effects of thyroid hormones on cardiac myocytes and on the systemic vasculature that alter hemodynamics.[4] Thyroid hormones have both positive inotropic and chronotropic effects on the heart along with increased adrenergic sensitivity accounting for the tachycardia and increased contractility of the heart in hyperthyroidism.[6]

In thyrotoxic cardiomyopathy, myocardial injury is caused by over production of thyroid hormones particularly triiodothyronine. There are various ways in which excessive circulating T3 affects the cardiovascular system. This include increased myocyte metabolic rate leading to increased contractility and hence increased cardiac out-put as well as increased heart rate.[6] There is also an increase in blood volume. T3 acts on the vascular smooth muscle to cause vasodilation[2] which in turn lowers systemic vascular resistance. This in turn activates the renin-angiotensin-aldosterone system which causes fluid and salt retention. T3 induces erythropoiesis which results in a net increase in total blood volume and stroke volume. These changes lead to high out-put state leading to signs and symptoms of heart failure.[7]

Atrial fibrillation has been the most prevalent clinically significant arrhythmias associated with thyrotoxicosis which presents in 20% of the patients.[8] Overt and sub-clinical hyperthyroidism are well known risk factors for AF.[9] Other risk factors out-lined in some studies include male sex, advancing age, ischemic heart disease, congestive heart failure and valvular heart disease.[9] Sinus tachycardia is reported in 56.52% of patients with mild to moderate hyperthyroidism and 76.47% of patients with severe hyperthyroidism.[10] Hyperthyroidism causes sympatho-vagal imbalance with enhanced sympathetic and reduced vagal regulation of the heart rhythm. The first goal of treatment of thyrotoxic heart is to reduce heart rate by adrenergic blockade. In addition, in patients with HF, the use of digoxin and diuretics are appropriate.[11]However the definitive treatment of hyperthyroidism and AF is controversial. The risk of systemic or cerebral embolism must be weighed against the potential for bleeding and other complications.[12]

The case presented by us is unique in the following ways when compared with other previously published case reports. First, our patient is a young man. Thyroid disease is rare in males compared to their female counterparts.

Secondly, our patient presented with unusual symptoms and signs including jaundice, moderate anaemia and neutropenia with laboratory evidence of chronic liver disease. There were no obvious ophthalmic signs.

Thirdly, our patient fared better with B1 selective than B2 blocker. This raises question about suitability of the use of B2 blocker as 1ST line option in thyrotoxic heart disease rather than B1 blocker.

Learning points:

1. Cardiac dysfunction can occur in hyperthyroidism in the absence of obvious thyrotoxic eye signs and symptoms.
2. There can be marked hematologic and hepatic involvement in hyperthyroidism.
3. Further study of the use of B1 selective blocker for rate control in thyrotoxic heart disease is warranted.

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Patient consent for publication was obtained.

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