ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
Index Copernicus Value: 49. 23

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 3, Issue –3, June - 2018, Page No. : 67 - 72

Thyrotoxicosis: Unmasking the Underlying Cardiac Disease - A Case Series

Dr. Praphull Deepankar, Assistant Professor (General Medicine), IGIMS, Patna
Dr. Ved Prakash, Assistant Professor (Department Of Endocrinology), IGIMS, Patna
Dr. Bhim Ram, Associate Professor (General Medicine), IGIMS, Patna
Dr. Manoj Kumar Choudhary, Assistant Professor (General Medicine), IGIMS, Patna

Correspondance Author: Dr. Bhim Ram, Associate Professor (General Medicine), IGIMS, Patna

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

The prevalence of congestive heart failure (CHF) is increasing and in the majority of these cases the etiology is underlying cardiac disease. Common causes of CHF include coronary artery disease, valvular heart disease, hypertension, alcoholic cardiomyopathy, and dilated cardiomyopathy. Thyroid hormone homeostasis is vital to the optimal functioning of the cardiovascular system. Hyperthyroidism can affect the cardiovascular system in a variety of ways. The cardiovascular manifestations range from sinus tachycardia to atrial fibrillation and from a high cardiac output state to CHF due to systolic left ventricular dysfunction. We present a case series of five patients of CHF due to systolic left ventricular dysfunction secondary to pre-existing cardiac illness. Here CHF was precipitated by simultaneous occurrence of thyrotoxicosis and showed considerable improvement in the left ventricular function once the thyrotoxicosis was treated along with CHF.

Key words: thyrotoxicosis, congestive heart failure, CHF, hyperthyroidism, TSH, pulmonary edema.

Introduction

The association of thyrotoxicosis and heart disease is well known. Thyrotoxicosis can aggravate pre-existing cardiac disease and can also itself lead to atrial fibrillation and congestive heart failure (CHF) or worsening of angina pectoris. [1] Several studies have documented enhanced myocardial performance in the hyperthyroid state. Hyperthyroidism causes an increase in resting heart rate, systolic blood pressure, blood volume, left ventricular mass, stroke volume, left ventricular ejection fraction, and cardiac output and a reduction in systemic vascular resistance with a widened pulse pressure [2]. Hyperthyroidism may cause sinus tachycardia, atrial fibrillation, atrial flutter, paroxysmal atrial tachycardia, premature ventricular beats, premature atrial beats, atrioventricular block, myocardial ischemia, angina pectoris, and congestive heart failure. [2, 3] Congestive heart failure is common in patients with thyrotoxicosis and heart disease in whom the heart is unable to increase cardiac output appropriately to match the increase in myocardial oxygen demand. [3] The Heart Failure Society of America guideline recommends that for patients suspected of having CHF, evaluation of thyroid disorder should be part of the standard laboratory tests, in recognition that both hyperthyroidism and hypothyroidism can be primary or contributory causes of HF. [4]

Here we present five cases of our institute where patient's underlying cardiac disease is unmasked by occurrence of thyrotoxicosis.

CASE 1

A 58-year-old man presented to the emergency room of the hospital with a one week history of upper abdominal pain, mostly localized to the left upper quadrant and epigastric regions, plus palpitation, shortness of breath on exertion, abdominal swelling, and ankle edema for 3 days. He denied any chest pain, syncope or past history of diabetes mellitus, hypertension and coronary artery disease. He had smoked two packs of cigarettes per day for 25 years but denied any history of alcohol or drug abuse. On arrival, the patient was comfortable and in no acute distress. His vital signs were: pulse 146 beats per minute, irregularly irregular, blood pressure 130/70 mm Hg, and respiratory rate 18/min. He was afebrile. Jugular venous pulsation was elevated up. A symmetrically enlarged, nontender thyroid gland was palpable. Tremor of the outstretched hands is present. Chest examination revealed bilateral basal rales. Cardiovascular examination revealed normal first and second heart sounds. A third heart sound and a grade 3/6 holosystolic murmur were heard at the apex, with radiation to the axilla were present. Mild tender hepatomegaly and bilateral pitting ankle edema was noted bilaterally. Patient was treated symptomatically for congestive cardiac failure.

Initial laboratory data analysis showed: hemoglobin- 12.6 g/dL; hematocrit- 38%; white cell count- 8,600/mm^3; platelets- 250,000/mm^3; blood urea nitrogen- 15 mg/dL; and creatinine- 0.8 mg/dL. Electrolytes and cardiac enzymes were normal. Chest x-ray revealed cardiomegaly and pulmonary edema. Twelve-lead ECG revealed atrial fibrillation with a fast ventricular rate. The transthoracic echocardiogram (TTE) revealed a left ventricular (LV) hypokinesia (RWMA) with ejection fraction (EF) of <20%, with moderate to severe mitral regurgitation without evidence of thrombus.

An initial diagnosis of atrial fibrillation and coronary artery disease with CHF was made. The patient was started on intravenous furosemide, orally with digoxin and angiotensin-converting enzyme (ACE) inhibitors, along with intravenous low molecular weight heparin.

Thyroid function tests ordered on admission revealed a thyroid-stimulating hormone (TSH) level- 0.056 μU/mL (normal- 0.5–5.0 μU/mL) and a free thyroxine level of 4.6 ng/dL (normal- 0.8–1.8 ng/dL), confirming the diagnosis of thyrotoxicosis. And treatment for thyrotoxicosis was started. Over the next 7 days, the patient's condition improved, with complete resolution of hepatomegaly and fluid overload. On follow-up 2D Echo confirmed a marked improvement in EF (45%–50%) and the patient continued to do well.

CASE-2

A 34-year-old male was referred to the outpatient clinic of our institute with complaints of ankle edema and dyspnea on exertion, for last one week plus history of orthopnea and paroxysmal nocturnal dyspnea for few days. There was no history of chest pain, or syncope. The medical history and family history were unremarkable. He was a smoker but no history of alcohol abuse. He was afebrile. Vital signs were: pulse- 144/min, regular; blood pressure- 114/68 mm Hg; respiratory rate-16/min. JVP – raised. On chest auscultation bilateral basal crepts were noted. Cardiovascular examination revealed a non-displaced apical impulse. First and second heart sounds were normal; no third or fourth heart sounds were heard, and no murmurs could be appreciated due to tachycardia. No hepatomegaly appreciated.

Initial laboratory data were: hemoglobin- 13.6 g/dL; hematocrit- 36%; white cell count- 6000/mm^3; platelet count- 260,000/mm^3; blood urea nitrogen- 16 mg/dL; and creatinine-1.0 mg/dL. Electrolytes were normal. Chest x-ray revealed mild cardiomegaly and pulmonary edema. Twelve-lead ECG revealed sinus tachycardia with a ventricular rate of 152/min. 2D echocardiography showed

features of rheumatic heart disease with mild mitral stenosis.

Patient was diagnosed as Rheumatic heart disease (RHD) and mitral stenosis (MS) with sinus tachycardia with pulmonary edema and was managed accordingly. Symptoms of pulmonary edema were improved with treatment but tachycardia persisted. In view uncontrolled heart rate, treatment was revised with cardiologist consultation and thyroid function tests were ordered. TSH level- 0.06 μU/mL (normal, 0.5–5.0 μU/mL) and free thyroxine 4.80 ng/dL (normal, 0.8–1.8 ng/dL) confirmed the suspicion of thyrotoxicosis. The patient was treated with methimazole and propranolol and thyroid scan was planned. Gradually the rhythm was controlled and patient was discharged without symptoms.

CASE-3

A 51-year-old woman was admitted to the hospital with history of progressive dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, ankle edema, and weight gain for last weeks. Shortness of breath and fatigue had increased over the previous two days. She denied any history of chest pain, palpitations, weight loss, diabetes, hypertension or any cardiac illness. The patient denied any tobacco, alcohol, or drug abuse. 2D echocardiography done outside had shown an EF of 20%, with global hypokinesia, and was advised coronary angiography. She had been on diuretics, digoxin, an ACE inhibitor, and spironolactone for CHF, with some improvement in symptoms. She had also been on long-term, low dose prednisone for reactive airway disease.

On examination, the patient was in respiratory distress. Vital signs were pulse-126 beats/min and regular; blood pressure- 108/70 mm Hg; respiratory rate- 22/min. She was afebrile. JVP was raised. Ankle edema was appreciated. Mild fullness was palpated in the neck, and no thyroid bruits were heard. Examination of the chest revealed basilar rales bilaterally. Examination of the heart revealed apical impulse in the 5th intercostal space, 3 cm lateral to the mid-clavicular line. First and second heart sounds were normal. Third and fourth heart sounds were absent. A soft grade 3/6 holosystolic murmur was heard at the left parasternal area and mitral area.

Initial laboratory data included: hemoglobin- 15 g/dL; hematocrit- 46%; white cell count- 11,000/mm^3; platelet count- 170,000/mm^3; blood urea nitrogen- 27 mg/dL; and creatinine- 1.0 mg/dL. Cardiac enzymes & electrolytes were normal. Chest x-ray showed cardiomegaly with pulmonary edema. Twelve-lead ECG showed sinus tachycardia of 130/min. The patient was admitted to the HDU with a diagnosis of worsening heart failure and was started on intravenous diuretics. Over the next 24 hours, the patient's condition deteriorated, with increasing tachypnea, tachycardia, and hypotension despite good diuresis initially. The patient was transferred to the coronary care unit and intravenous dopamine infusion. Bedside echocardiography showed finding consistent of CAD and coronary angiography was planned. Although her blood pressure improved, she became more tachycardia, requiring discontinuation of dopamine. Thyroid function tests, sent as a part of the "routine work-up" became available and revealed a TSH 0.025 μU/mL (normal, 0.5–5.0 μU/mL) and free thyroxine 6.80 ng/dL (normal, 0.8–1.8 ng/dL). Patient was started on methimazole, and the prednisone dose was increased. In addition, intravenous normal saline was infused to keep the systolic blood pressure >90 mm Hg. Over the next 24–48 hours, the patient's condition gradually improved. When her blood pressure was stable, she was started on propranolol.

The patient was discharged after 5 days on methimazole, with plan of thyroid scan and other advice for coronary artery disease. Six months later, the patient is clinically doing well. A repeat 2D echocardiography showed an improvement in LV function, with an EF of 35%–40%.

CASE- 4

A 22-years-old married female presented with of abdominal pain with for last 7 days and developed acute onset dyspnea, orthopnea, cough and sore throat one day back. There was no history of fever, chest pain, pedal edema or addiction of alcohol and smoking. There was no history of chronic medical and surgical illness. On examination she was anemic, and vitals are pulse- 128/min, regular, blood pressure- 108/76 mm Hg and respiratory rate of 32/min. Raised JVP and tenderness over neck without thyromegaly were noted. On chest examination bilateral basal rales were present. Cardiovascular examination revealed tachycardia, loud S_1 and no murmur appreciated. Other systemic examinations were normal.

Initial investigation showed Hemoglobin- 9.2gm/dl, total leucocyte count- 12600/mm^3, platelet count- 1.68 lacs /mm^3, blood urea nitrogen -18mg/dl, creatinine -0.8, total bilirubin -1.8 mg/dl and normal serum lipase level. Arterial blood gas analysis showed hypoxemia. ECG showed sinus tachycardia. Chest X-ray showed pulmonary edema. Patient shifted to HDU and intravenous diuretic started in view of acute pulmonary edema. 2D echocardiography showed rheumatic heart disease, severe mitral stenosis, with normal ejection fraction. Patient's condition improved with treatment. She vomited round worm out during hospital stay.

In view of persistent tachycardia and anxiety thyroid profile of the patient ordered. It showed TSH – 0.025 (0.35 – 5.50), FT4 – 4.08 (normal value- 0.89 – 1.76) and higher anti TPO antibody titre. Thyroid scan (Tc99) performed which showed overall reduced thyroid uptake reduced (0.2%) feature suggesting of sub-acute thyroiditis. Patient was treated with methimazole and propranolol and her tachycardia and other symptoms improved gradually over 2-3 days.

CASE- 5

56 years old gentleman came to the emergency department with the symptoms of dyspnea on exertion, palpitation and chest heaviness for last 5-7 days. He was hypertensive and on amlodipine 5mg for last 15 yrs. There was no history of chest pain, abdominal discomfort, cough and fever. There was also no history of diabetes, coronary artery disease, or other medical illness. On examination patient was dyspneic and his vitals were; pulse rate- 164/min, & irregularly irregular, blood pressure- 156/90 mm Hg, respiratory rate 28/min and 90% sO_2. Bilateral basal crepts were heard on chest auscultation. On cardiovascular auscultation muffled heart sound heard.

Initial blood investigations showed hemoglobin- 11.8 gm. /dl, total leucocyte count – 5400/ mm^3, platelet count–1.86 lac. /mm^3, normal kidney and liver function test. Cardiac enzymes and serum electrolyte were normal. ECG showed atrial fibrillation with fast ventricular rate and poor progression of R wave. Chest X-ray demonstrated pulmonary edema with cardiomegaly. 2D echocardiography was done which revealed features of coronary artery disease and mild pericardial effusion.

Patient was treated with intravenous diuretics, digoxin, anticoagulant and other conservative management. Chemical cardioversion was done with amiodarone after transfer to coronary care unit. Patient was improved symptomatically from pulmonary edema and sinus rhythm restored. Patient was transferred to the medicine department for further observation and management. After 3days hospital stay, patient again became symptomatic with pulmonary edema and atrial fibrillation recurred. At this time thyroid function ordered which revealed thyrotoxicosis [TSH – 0.08 (0.35 – 5.50), FT4 – 3.06 (normal value- 0.89 – 1.76)]. Patient was started methimazole and propranolol. Patient heart rate and rhythm were controlled gradually and pulmonary edema didn't recur.

TABLE.1 SUMMARY OF PATIENT CHARACTERISTICS:

CASE NO.	AGE/SEX	SIGN & SYMPTOMS OF THYROTOXICOSIS AT PRESENTATION	DURATION OF CARDIAC SYMPTOMS	NYHA ON ADMISSION / FOLLOWUP	HEART RHYTHM ON PRESENTATION	CARDIAC ILLNESS DIAGNOSED
1.	58/M	++	3 DAYS	III/I	ATRIAL FIBRILLATION	CAD
2.	34/M	-	7 DAYS	IV/II	SINUS TACHYCARDIA	RHD/MS
3.	51/M	+/-	2-7 DAYS	IV/II	SINUS TACHYCARDIA	CAD
4.	22/F	++	7 DAYS	III/I	SINUS TACHYCARDIA	RHD/MS
5.	56/M	+/-	5 DAYS	III/I	ATRIAL FIBRILLATION	CAD

Discussion

We represented five cases of cardiac disease presented with new onset congestive heart failure (CHF) precipitated by thyrotoxicosis. These five patients were from different age group, indicating that development of overt CHF due to thyrotoxicosis is not limited to the elderly population, but can precipitate in younger patients if thyrotoxicosis is there. (TABLE-1) All patients showed improvement of EF with usual CHF treatment and therapy aimed for thyrotoxicosis. Typically, thyrotoxicosis presents with the features of heat intolerance, weight loss, sweating, palpitation, and tremors. Occasionally, it may precipitate heart failure in the absence of any classic symptoms of hyperthyroidism, as is seen in our case no.2. [5]

CHF was related to tachycardia-induced systolic LV dysfunction. Peripherally, triiodothyronine (T3) has been shown to decrease systemic vascular resistance (SVR) by promoting vasodilatation. The resulting decrease in SVR activates the renin-angiotensin-aldosterone system, leading to retention of sodium (Na+) and fluid. Thyroid hormone also increases erythropoiesis. [6] The net effect is a resultant increase in the total blood volume and stroke volume. These cardiac effects, coupled with a generalized increase in tissue metabolism, low SVR, and increase in total blood volume, lead to a high cardiac output state in hyperthyroidism. Clinically, the effects of excess thyroid hormone on the cardiovascular system translate into a wide variety of signs and symptoms, ranging from sinus tachycardia to the development of severe LV dysfunction and heart failure. Resting sinus tachycardia is the most common finding in hyperthyroidism, second only to goiter. [7]

Thyrotoxicosis is not an uncommon cause of atrial fibrillation; it is found in 5%–22% of hyperthyroid patients and is probably the most common cardiovascular problem that brings this disease to medical attention. In addition to overt thyrotoxicosis, the risk of atrial fibrillation is increased even with subclinical hyperthyroidism. [8] In our case series two patients had atrial fibrillation while other three had sinus tachycardia at presentation.

Clinically significant CHF due to hyperthyroidism per se is considered not a common occurrence. This is more commonly seen in patients with pre-existing heart disease, such as ischemic, hypertensive, rheumatic heart disease or alcoholic cardiomyopathy. [9] In this case series thyrotoxicosis precipitated CHF in patient all having pre-existing heart disease. LV function tends to improve within a few weeks of initiation of treatment of thyrotoxicosis and heart failure. In particular, LV function improves when the rapid ventricular rate, due to either sinus tachycardia or atrial fibrillation, is brought under control. [10]

Conclusion

Thyroid hormone influences every cell, tissue, and organ in the body and its homeostasis is essential to the optimal functioning of the heart. Hyperthyroidism can precipitate severe LV dysfunction in patients with pre-existing cardiac illness. This disease can be diagnosed earlier by a detailed history and physical examination, and TSH level should be checked as a part of the initial laboratory work-up of every patient with new-onset CHF.

References

1. Woeber KA. Thyrotoxicosis and heart failure. NEngl J Med 1992; 327: 94-8.

2. Dillmann WH (2001) Thyroid hormone influences on the cardiovascular system: molecular and clinical studies. Thyroid Today 24: 1-13.
3. Aronow WS (2013) Cardiovascular Manifestations of Hyperthyroidism. J Clin Case Rep 3: e120.
4. Heart Failure Society of America. Executive summary: HFSA 2010 comprehensive heart failure practice guideline. J Card Fail 2010; 16: 475–539.
5. Polikar R, Burger AG, Scherrer U, et al. The thyroid and the heart. Circulation. 1993;87:1435–1441.
6. Ojamaa K, Klemperer JD, Klein I. Acute effects of thyroid hormone on vascular smooth muscle. Thyroid. 1996;6:505–512.
7. Klein I, Ojamaa K. Thyrotoxicosis and the heart. Endocrinol Metab Clin North Am. 1998;27:51–62.
8. Krahn AD, Klen GR, Kerr CR, et al. How useful is thyroid function testing in patients with recent-onset atrial fibrillation? Arch Intern Med. 1996;156:2221–2224.
9. Nordyke RA, Gilbert FI Jr, Harada ASM. Graves' disease: influence of age on clinical findings. Arch Intern Med. 1988;148: 626–631.
10. Choudhury RP, Macdermot J. Heart failure in thyrotoxicosis, an approach to management.Br J Clin Pharmacol. 1998; 49:421-424.