Original Research Paper

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General Medicine

REVERSIBLE DILATED CARDIOMYOPATHY IN THYROTOXICOSIS

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ABSTRACT Hyperthyroidism, though primarily recognized for its metabolic effects, can manifest with significant cardiovascular complications. Cardiomyopathy is observed in approximately 6% of hyperthyroid patients, yet progression to dilated cardiomyopathy with impaired left ventricular systolic function occurs in fewer than 1% of cases.(1) The cardiac effects are often reversible with appropriate management of the underlying thyrotoxicosis.(1)(2) We present the case of a 50-year-old male with no prior history of cardiovascular disease who developed worsening exertional dyspnea, edema, and fatigue. Initial evaluation revealed dilated cardiomyopathy with an ejection fraction of 20% and biochemical evidence of severe hyperthyroidism due to Graves' disease. After initiating antithyroid therapy and supportive cardiac medications, the patient demonstrated significant clinical and echocardiographic improvement, with left ventricular ejection fraction rising to 45% within a month. This case underscores the importance of considering thyrotoxicosis in patients presenting with unexplained heart failure.(2)(3) Early recognition and treatment are crucial, as the cardiac dysfunction in such cases is typically reversible(2)(4), emphasizing the need for thyroid function assessment in atypical cardiomyopathy presentations.

KEYWORDS : Graves' disease, thyrotoxicosis, cardiomyopathy, congestive heart failure

INTRODUCTION:

- The thyroid gland plays a crucial role in metabolic regulation, and its hormones significantly influence various organ systems, especially the heart. They impact both the rate and force of cardiac contractions.
- Research has shown that thyroid hormones directly stimulate cardiac function by upregulating beta-adrenergic receptors early on, and later altering myocardial electrophysiology.
- In animal studies, particularly with murine models, the hearts exposed to thyroid hormones demonstrated increased calcium influx and enhanced contractility compared to those in a hypothyroid state.
- Clinicians should be aware of the potential for hyperthyroidism to present as dilated cardiomyopathy, particularly in the absence of more common causes.
- Cardiomyopathies are primary myocardial disorders that are not attributable to conditions such as hypertension, congenital or valvular heart disease, coronary artery disease, or pericardial disorders.
- Certain forms of cardiomyopathy, including those linked to alcohol consumption, pregnancy, deficiencies in selenium or phosphate, thyroid dysfunction, chronic stimulant use, or sustained tachycardia, are known to be reversible.
- There is a scarcity of documented cases linking thyrotoxicosis to reversible dilated cardiomyopathy, underscoring the rarity and clinical significance of such presentations.

METHODOLOGY:

Prospective and observational case study.
TFT done by Vitrus 3600 machine.

CASE REPORT:

- A 50-year-old male with no history of alcohol use presented with a 10-day history of worsening shortness of breath on exertion (Grade III), reduced appetite, and low-grade fever. This was his initial visit to the clinic, although he had been consulting multiple primary care providers over the past two years for a persistent cardiac condition. His symptoms had notably deteriorated in the last week and a half, leading to feelings of hopelessness due to a lack of improvement despite ongoing treatment.

- The patient reported becoming breathless after walking approximately 300 feet. He also noted a significant loss of appetite but was unsure about recent weight changes. The fever was persistent but mild, and not associated with chills, night sweats, or rigors. There were no accompanying symptoms such as chest pain, cough, gastrointestinal disturbances, headache, or nausea.

External Investigations:

- Chest X-ray: Cardiomegal
- 2D Echocardiogram: Dilated cardiomyopathy with an ejection fraction (EF) of 20%, global hypokinesia , four-chamber dilation, and moderate pulmonary arterial hypertension (PAH)
- ECG: Sinus tachycardia with no ischemic changes

Past Medical History:
No known hypertension, ischemic heart disease, diabetes mellitus, or tuberculosis

Lifestyle History: Denied smoking, alcohol use, or recreational drug consumption

Family History: Unremarkable for cardiovascular or metabolic diseases

Current Medications: Torsid Plus (10/50) once daily

Clinical Examination:

- General Appearance: Mildly anxious with subtle exophthalmos
- Vital Signs: Pulse 132 bpm, BP 156/90 mmHg, RR 22/min, Temp 99.6°F, SpO_2 98% on room air, Weight 54 kg.
- Respiratory: Bilateral lower lobe crepitations and pedal edema
- Cardiovascular: Normal heart sounds, tachycardia with regular rhythm, JVP elevated to 10 cm, displaced apex beat, no murmurs or gallops
- CNS: Fine tremor with hands outstretched
- Abdomen: Mild hepatomegaly, positive hepatojugular reflux
- Neck: No visible or palpable thyroid enlargement
- Extremities: Bilateral pitting edema

Laboratory Investigations:

- Thyroid Profile:
- TSH: 0.0018 µIU/mL (↓)
- Free T4: 21.99 ng/dL (↑)
- Free T3: 54.7 pg/mL (↑)
- Autoimmune Markers:
- TSH receptor antibodies: 35 IU/L (↑)
- Anti-TPO: >2000 IU/mL (↑)
- Anti-thyroglobulin: >5000 IU/mL (↑)
- CBC, LFTs, electrolytes, and cardiac enzymes were within normal limits
- Technetium Thyroid Scan: Showed diffuse uptake consistent with toxic goiter due to Graves' disease

Diagnosis: Dilated cardiomyopathy attributed to Graves' thyrotoxicosis

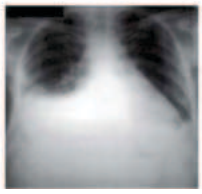
Treatment Initiated:

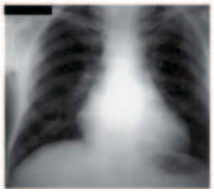
- Neomercazole 10 mg three times daily
- Continued Torsid Plus 10/50 mg once daily
- Advised salt-restricted diet

Follow-Up (1 Month):

Marked clinical improvement was observed. The patient no longer experienced breathlessness or edema and was now able to walk 3 kilometers without discomfort. Weight increased by 3.5 kg. Echocardiogram showed EF improved to 45%. Torsid Plus was discontinued, and metoprolol XL 25 mg daily was added. Neomercazole was continued, and the patient was advised to undergo total thyroidectomy. This case underscores a reversible cardiomyopathy secondary to Graves' disease, without accompanying arrhythmia.

BEFORE TREATMENT: AFTER TREATMENT:





DISCUSSION:

Unmanaged thyrotoxicosis can lead to structural and functional changes in the heart, including ventricular dilation, sustained tachycardia, and chronic heart failure[2][4] Thyroid hormones play a central role in stimulating the sympathetic nervous system, largely through upregulation of β1-adrenergic receptors. Additionally, these hormones enhance the activity of sarcoplasmic reticulum Ca^{2+} ATPase, a key player in myocardial excitation-contraction coupling and calcium handling.[2]

- The resulting increase in intracellular calcium release promotes stronger cardiac muscle contractions. Free T3 and T4 also stimulate expression of fast-contracting alpha-myosin heavy chain isoforms and accelerate SA node activity, both of which contribute to increased cardiac output and heart rate.[5]
- These physiological changes result in heightened chronotropic and inotropic effects. Thyroid hormone also increases erythropoietin production, expanding circulating blood volume. The cumulative hemodynamic effects—elevated cardiac output and blood volume—can eventually overwhelm the heart, resulting in high-output heart failure.[4][5]
- Although uncommon, less than 1% of individuals with thyrotoxicosis develop dilated cardiomyopathy with left ventricular dysfunction[1]. The precise pathophysiology is not entirely understood but likely involves a mix of prolonged sympathetic stimulation, sustained tachycardia, and direct metabolic impacts of excessive thyroid hormone on cardiac myocytes.[1][3]
- Risk factors such as advanced age and preexisting cardiac conditions (e.g., valvular disease, hypertension, or ischemia) increase susceptibility to heart failure in the setting of hyperthyroidism. In this case, the patient had no known prior cardiovascular illness, yet long-standing sinus tachycardia likely played a central role in the development of heart failure. While atrial fibrillation was not documented, silent paroxysmal episodes cannot be ruled out.
- Clinicians should maintain a high index of suspicion for hyperthyroidism in patients presenting with unexplained dilated cardiomyopathy. Early diagnosis is crucial, as treatment of the underlying thyrotoxicosis often results in rapid and significant cardiac improvement.
- Hyperthyroid patients typically experience high-output heart failure, sometimes accompanied by right-sided dysfunction, tricuspid regurgitation, or pulmonary hypertension. While these manifestations are rare, their recognition is critical. Studies have demonstrated that heart failure related to hyperthyroidism is largely reversible. For instance, one study involving seven patients reported improvement in EF from 28% to 55% following treatment. In another report, two middle-aged patients with EFs near 35% showed near-complete recovery post-treatment of hyperthyroidism.
- This case clearly demonstrates the importance of evaluating thyroid function in patients with new-onset heart failure, particularly in the absence of conventional cardiovascular risk factors. Reversal of cardiac dysfunction following treatment supports the causative role of thyrotoxicosis.

CONCLUSION:

Thyrotoxicosis, though an uncommon cause of dilated cardiomyopathy, should be considered in patients presenting with unexplained heart failure, especially when accompanied by signs of hyperthyroidism. Timely recognition and management can lead to complete reversal of cardiac dysfunction, highlighting the need for routine thyroid function testing in such clinical scenarios.[2][3][5]

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