Case Report

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The Importance of Hyperthyroid Screening in Acute Decompensated Heart Failure with Persistent Tachycardia Despite Optimal Decongestion: A Case Report

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Abstract

Background: Hyperthyroid has various effects on the cardiovascular system. If hyperthyroidism is recognized and treated early, the cardiac dysfunction could be resolved by restoring thyroid hormone to normal levels. Here, we highlight the importance of thyroid function screening in acute decompensated heart failure with persistent tachycardia.

Case Illustration and Discussion: A man was admitted to the emergency department with signs and symptoms of acute decompensated heart failure. After optimal decongestion, his heart rate remained tachycardic. Thus, we ordered a thyroid function test, although no clinical presentation of hyperthyroidism was found. Tachycardia was resolved after we administered anti-thyroid medication and he was discharged on the fifth day of hospitalization with euvolemic and stable hemodynamic condition.

Conclusion: Regardless of the hyperthyroidism clinical presentation, we suggest to performed thyroid function tests in suspected cases of acute decompensated heart failure with persistent tachycardia due to the variety of symptoms in hyperthyroidism. Thus, hyperthyroidism could be recognized and treated early to optimize patient outcomes.

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Keywords: Hyperthyroid, Acute Decompensated Heart Failure, Persistent Tachycardia.

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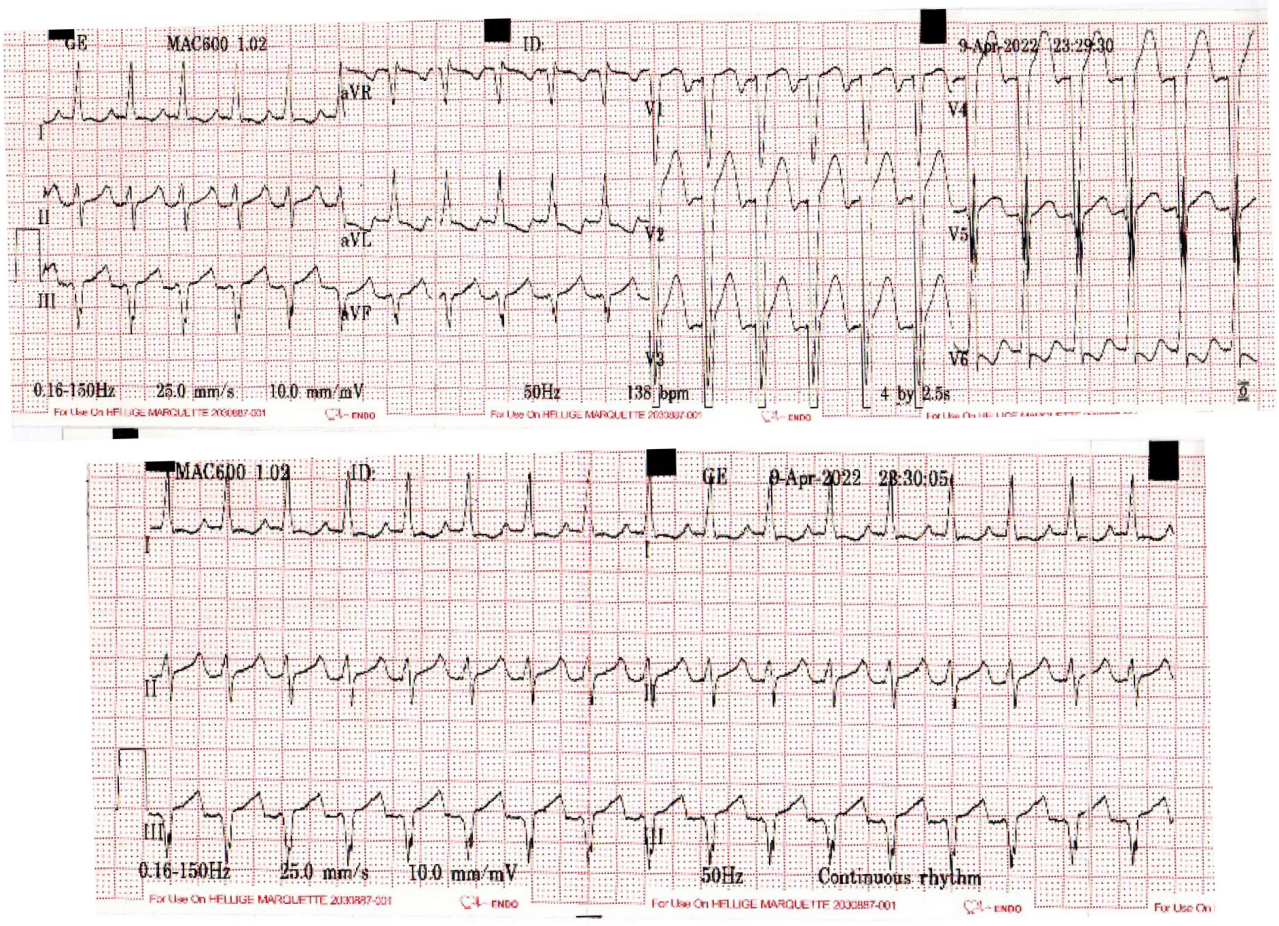
Introduction

Hyperthyroidism has various effects on the cardiovascular system. Cardiac arrhythmias ranging from sinus tachycardia to atrial fibrillation and low/high cardiac output state to congestive heart failure are observed in patients with hyperthyroidism.[1] Patient with hyperthyroid who did not receive proper medication had a higher risk of cardiovascular death (CVD).[2] If hyperthyroidism is recognized and treated early, the cardiac dysfunction could be resolved by restoring thyroid hormone to normal levels.[3] This case highlights the importance of thyroid function screening in acute decompensated heart failure with persistent tachycardia.

Case illustration

A 67-year-old man was admitted to the emergency department with chief complaint of breathlessness for three days. There was dyspnea on exertion and orthopnea. He also complained about cough four days prior to admission and fatigue. He had a history of two-vessel coronary artery disease With prior history of percutaneous coronary intervention. The patient had a lousy adherence. His routine medication was only furosemide 40 mg per oral once daily (od).

During admission, his vital signs were: blood pressure 140/119 mmHg, heart rate 138 bpm, respiratory rate 24 times per minute, oxygen saturation 97%, and temperature 36.20^0. On physical examination, jugular venous pressure was slightly elevated up to 3 cmH_2O. Thyroid gland was not palpable. Cardiac examination revealed normal first and second heart sounds, no murmur, and no gallop. Vesicular pulmonary sounds with bilateral rales were found on lung examination. The abdomen revealed no abnormalities. Slight pitting ankle edema was observed bilaterally. Electrocardiography



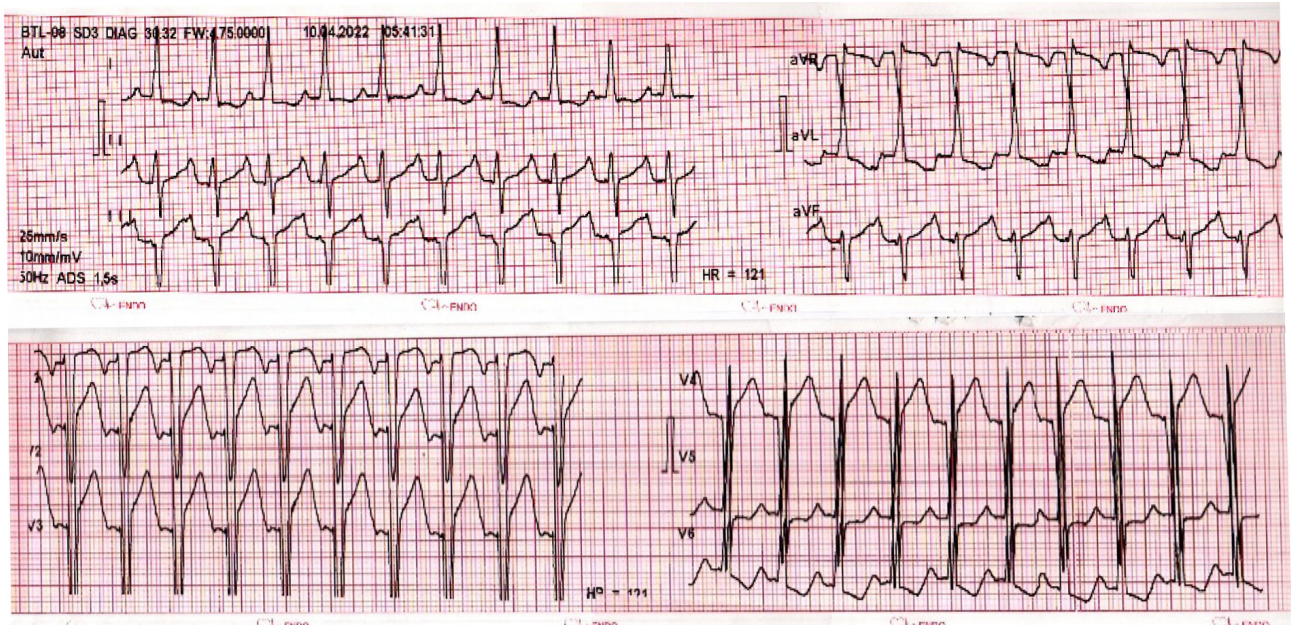


Figure 2. ECG in the ward.

(ECG) in the emergency department showed sinus tachycardia with a heart rate of 138 bpm, left axis deviation, complete LBBB, and left ventricular hypertrophy **(Figure 1.)**. Thus, we administered 100 mg of intravenous furosemide. On evaluation, target urine output was achieved and dyspnea was relieved. Furosemide 10 mg continuous pump was given afterward. However, his heart rate remained tachycardia at 121-125 bpm until the following day **(Figure 2.)**.

Initial laboratory examination showed mild anemia with a hemoglobin level of 12.7 g/dL and slightly low red blood cells, 4.37x106/µl. Cells count revealed neutrophilia and lymphopenia. Renal function was slightly decreased with creatinine serum level of 1.28 mg/dL (eGFR 56.1 mL/min/1.73m^2). The potassium level was mildly reduced to 3.10 mmol/L, and the calcium ion was also reduced to 0.85 mmol/L.

Due to tachycardia still persisted after target urine output was achieved and dyspnea was relieved on furosemide, we ordered thyroid function tests and blood gas analyses, although there was no clinical presentation of hyperthyroidism. FT4 was found elevated, 29.25 ρmol/L, and TSH serum level decreased to 0.44 µIU/ml, confirming the diagnosis of hyperthyroidism. Blood gas analysis revealed uncompensated respiratory alkalosis.

Chest X-ray **(Figure 3.)** discovered cardiomegaly with a disappeared cardiac waist and grounded cardiac apex. It also showed perihilar haze and increased pulmonary vascularity. We found all chamber dilatation with a severely reduced left ventricular ejection fraction (LVEF) of 16.9% in echocardiography examination. Grade 2 diastolic dysfunction with elevated left atrial pressure (LAP) was revealed. Moderate mitral regurgitation and mild aortic, pulmonary, and tricuspid valve regurgitation were also found. No thyroid abnormality was found on neck ultrasonography.

After initial treatment was given, the patient was treated with aspirin 80 mg o.d., atorvastatin 20 mg o.d., ramipril 5 mg b.i.d., spironolactone 25 mg o.d., and 10 mg of continuous pump furosemide. Methimazole 20 mg b.i.d. was administered on the second day of hospitalization. Within a few days, tachycardia was resolved and after the patient was in stable condition, we administered propranolol 10 mg t.i.d. During hospitalization, heart failure medication was up titrated to the target dose.

On the fifth day, he was discharged with furosemide 40 mg o.d., spironolactone 50 mg o.d., ramipril 5 mg b.i.d., propranolol 40 mg t.i.d., aspirin 80 mg o.d., atorvastatin 40 mg o.d., and methimazole 20 mg b.i.d. in euvolemic and stable hemodynamic condition.

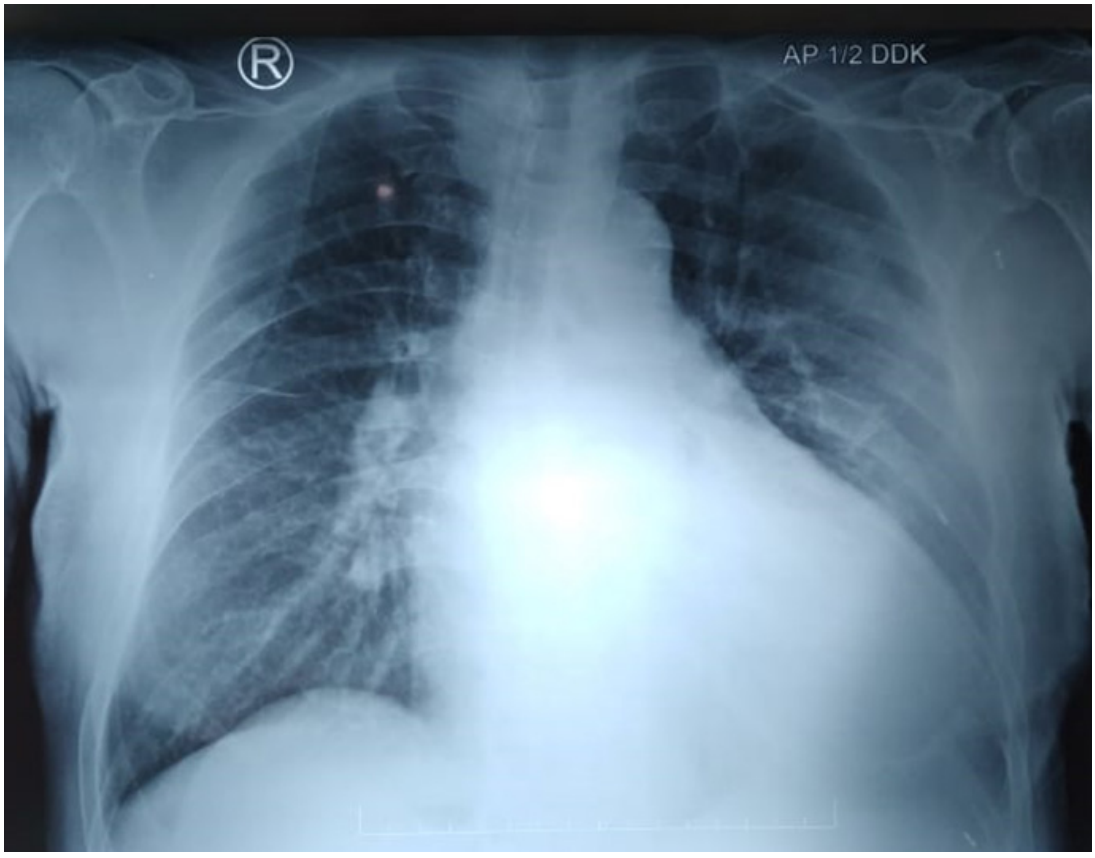


Figure 3. Chest X-Ray of the patient.

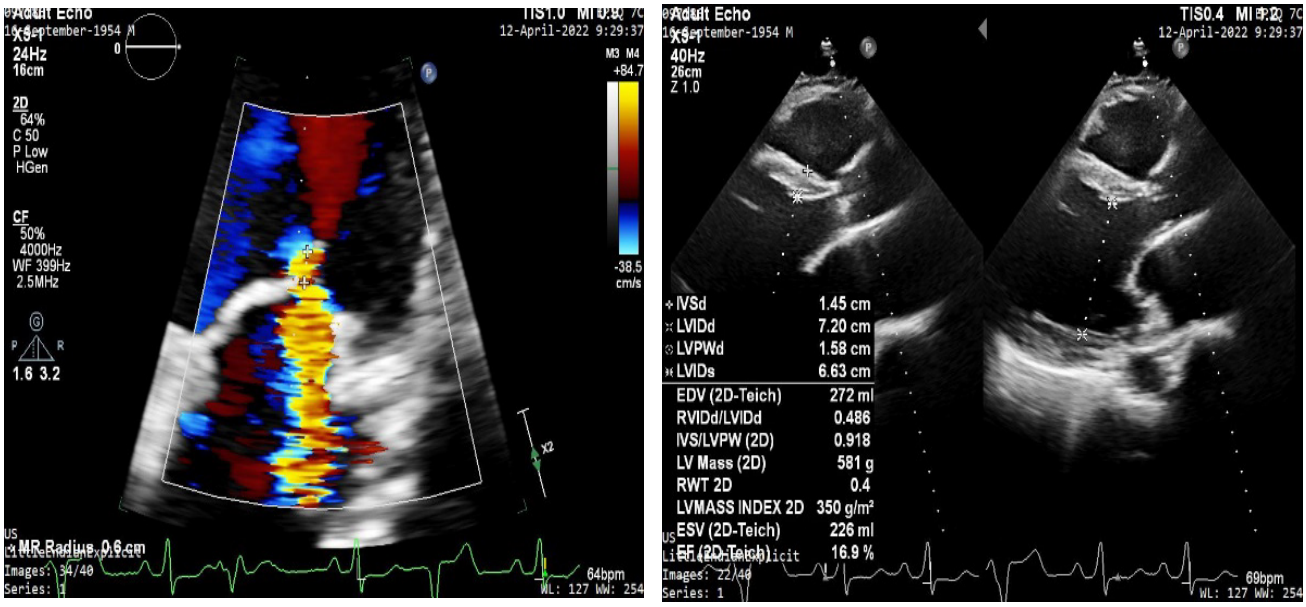


Figure 4. Transthoracic echocardiography of the patient.

Discussion

The thyroid gland secreted two iodinated hormones, T3 (triiodothyronine) and T4 (tetraiodothyronine). T3 and T4 exert biological activity in responsive tissues through binding to thyroid hormone receptors (TRs). T3 is a biologically active hormone, while T4 has a few non-genomic effects but is mainly thought of as a prohormone.[1,4] In the liver, kidneys, and skeletal muscle, most T4 is deiodinated to T3. Hyperthyroidism might present with various symptoms, including cardiac and hemodynamic symptoms, such as palpitations, expanded pulse pressure, exertional dyspnea, tachycardia, exercise intolerance, and atrial fibrilation.[1] Here, our patient had persistent tachycardia despite optimal decongestion. Thus, this raised our suspicion of hyperthyroidism although no clinical presentation of hyperthyroidism was found. If left untreated, hyperthyroidism had a higher risk of cardiovascular death (CVD).[2]

Thyroid hormone has cardiac effects directly on the heart and indirectly through its impact on vasculature and body metabolism.[5] In addition, thyroid hormones affect cardiac intracellular pathways by genomic and non-genomic mechanisms.[6] Thyroid hormones upregulate myocardial contractility, resting heart rate, and blood volume compared to expected.[1] T3 enters myocytes by particular transport proteins, resulting in increased contractility and relaxation of cardiac cells via transcription and non-transcription mediated actions. Increased contractility is caused by transcriptional effects on sarcoplasmic reticular calcium (Ca++) release and uptake and phosphorylation of phospholamban. The thyroid hormone's influence on various ion channels mediates the non-transcriptional effects.[5] Thyroid hormone can affect the sinus node directly and increase the heart rate.[7] T3 has been found to reduce systemic vascular resistance (SVR) via inducing vasodilation on the periphery. The direct effect of T3 on vascular smooth muscle mediates this activity. The renin-angiotensin-aldosterone system is activated as a result of the decrease in SVR, resulting in sodium (Na+) and fluid retention.[5,7]

About 6% of people with hyperthyroidism have heart failure (HF) as their initial clinical presentation, with half having left ventricular (LV) dysfunction.[8] Due to several mechanisms, including arrhythmias, cardiac enlargement, and higher blood volumes, hyperthyroidism may cause HF.[9] Patients with hyperthyroidism might develop "high-output HF" or “low-output HF”. High-output HF is congestive HF with rising cardiac output without underlying heart disease.[9] High-output HF may result from “tachycardia-induced cardiomyopathy” due to compensating for the increased stress level, the heart develops a remodeling process.[1,9,10] Breathlessness at rest, fatigue, fluid accumulation with peripheral edema, pleural effusion, pulmonary hypertension, and hepatic congestion are all characteristics of high-output HF.[1] In elderly patients with hyperthyroidism, HF may develop accompanied by a low ejection fraction (EF) known as low-output HF.[9,11] Low-output HF is characterized by patients with underlying heart disease such as ischemic heart disease, hypertensive or valvular disease, and/or atrial fibrillation. In this condition, patients had low cardiac output, high systemic vascular resistance, reduced left ventricular contractility, and impaired left ventricular filling, while blood volume was increased.[9,12]

In this case, congestive heart failure could be worsened due to untreated hyperthyroidism, besides his lousy compliance and history of coronary artery disease. Coronary artery disease generates direct ischemia injury to the myocardium, which results in remodeling and scarring, reducing contractility and cardiac output.[13] In hyperthyroidism, sinus tachycardia is the most common rhythm disturbance.[10] Most cases of sinus tachycardia are physiological and triggered by catecholamines. Tachycardia affects the myocardial oxygen supply and demand by reducing the duration of diastole. This condition leads to insufficient blood perfusion and worsening the left ventricular dysfunction.[14]

Tachycardia is a common compensatory mechanism for patients with acute heart failure to fulfill hemodynamic demands.[15] This condition is often resolved by optimal decongestion. In our patient, after optimal decongestion, tachycardia still persisted, leading to clinical suspicion of other conditions that generated persistent tachycardia. Identifying the specific cause of heart failure is pivotal because conditions that generate heart failure may necessitate disease-specific therapies.[16]

Hyperthyroidism with heart failure is challenging for clinicians because it is a clinical entity with variety of symptoms. The majority of patients are over the age of 60, with a history of cardiovascular disease and tachyarrhythmias.[17] Due to potential symptom reversal and normalization of cardiac structure and function after achieving euthyroid status, the primary goal of

hyperthyroidism with heart failure treatment is to treat underlying hyperthyroidism.[16–19] Radioiodine therapy (RAI), anti-thyroid medications (ATD), and thyroidectomy are the three treatment choices for hyperthyroidism.[20] Although normalization of thyroid hormones level is the primary therapeutic goal, the treatment takes roughly 4 to 6 weeks, necessitating the clinical requirement for symptom management while on anti-hyperthyroid medication.[17] All patients with symptomatic hyperthyroidism with heart failure and other thyrotoxic patients with resting heart rates > 90 bpm or accompanying cardiovascular disease should be considered beta-blockers as a safe and effective symptom management therapy.[17,18,21] Treatment with propranolol, atenolol, metoprolol, or another beta-blocker is recommended.[17] However, beta-blockers should be avoided in patients with reversible airway illness, severe hypotension, and severe bradycardia, especially for patients with second or third-degree atrioventricular block.[5] Calcium channel blockers (verapamil and diltiazem) is recommended for rate control in patient contraindicated to beta-blockers. Nevertheless, the use of non-dihydropyridine CCB is limited in the presence of LV dysfunction.[17] Diuretics should be used in patients with heart failure and pulmonary congestion.[18]

Conclusion

Here we present a case of hyperthyroidism and acute decompensated heart failure with persistent tachycardia after optimal decongestion. Due to the variety of symptoms in hyperthyroidism, we suggest to performed thyroid function tests in suspected cases of acute decompensated heart failure with persistent tachycardia, regardless of the hyperthyroidism clinical presentation. Therefore, hyperthyroidism could be recognized and treated early to optimize patient's outcome.

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