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Research Article

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Thyroid storm induced congestive heart failure

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Abstract

Background: *Uncontrolled thyrotoxicosis is known to lead to a state of prolonged tachycardia that may then progress to the onset of reversible congestive heart failure.*

Case: *The patient is a 36-year-old female with a history of Asthma, bilateral ovarian benign cysts, chronic goiter presented due to persistent cough, wheezing, and shortness of breath for about two days duration, echocardiogram had been done for respiratory failure, showing reduced ejection fraction, diastolic dysfunction, and dilated cardiomyopathy. She was subsequently admitted to the MICU for treatment of respiratory failure due to asthma exacerbation. Then began feeling symptoms of anxiety, palpitations, tremulousness, facial flushing. Evaluation of her labs showed elevated T3 at >650, elevated T4 at 22.6, with diminished TSH level at 0.01. The patient was diagnosed with thyroid storm in consideration of lab values with clinical features. TSI was ordered, which returned at 674. Endocrinology was consulted; the patient added a history of tremors for three weeks and weight loss of about 5 pounds over several months, with symptoms of generalized anxiety and dysphagia; she also reported diarrhea in the past weeks. She refers to a family history of goiter in her aunt. The recommendation from an endocrinology perspective was to obtain TSI level and ESR, continue methimazole. The patient was clinically stabilized, discharged on medications for heart failure and methimazole. However, she developed a rash on methimazole, and this was stopped. The patient went for radioactive iodine ablation, and a repeat echo showed an improvement of her ejection fraction. The patient was asymptomatic at a one-year cardiology follow-up, and cardiology medications were discontinued.*

Conclusion: *Hyperthyroidism is a known cause of congestive heart failure secondary to tachycardia-induced cardiomyopathy if untreated. After the proper treatment, this heart failure can be reversed if tachycardia is controlled.*

Keywords: Heart Failure, Reversible Cardiomyopathy, Thyrotoxicosis, Thyroid Disease.

Introduction

Hyperthyroidism is a known and common endocrine disorder affecting up to 1.3% of the population [1,3]. Uncontrolled and prolonged hyperthyroidism is also known to directly affect the heart, affecting SVR, which subsequently causes changes in inotropy and the chronotropic function affecting the cardiac output. These changes lead to persistent tachycardia due to atrial fibrillation or sinus tachycardia and could result in congestive heart failure (CHF) [3].

However, this is typically irreversible if it is secondary to a prolonged hyperthyroid state with tachycardia, even if it causes reduced ejection fraction, is treatable, and reversion to euthyroid state can restore cardiac ejection fraction; this is why it is classified as reversible cardiomyopathy [4].

We present a case of a patient who presented with CHF due to thyroid disease that resolved after the euthyroid state was achieved.

Case

Patient is a 36-year-old African-American female, with a past medical history of Asthma (No prior intubations, not on chronic steroid therapy), bilateral ovarian benign cysts, chronic goiter presented to the emergency room initially for persistent cough,

wheezing and shortness of breath for about 2 days duration. Patient at this time recently had moved to New York City from South Carolina, and attributes these symptoms to the recent move. In the emergency room, temperature was 98.4F, Pulse was 136 bpm, Blood pressure 130/83 mmHg, SpO2 99% on room air. However throughout the emergency room course and on admission to the floor, the patient was in respiratory distress, complaining of a productive cough. She was also persistently tachycardic still, requiring oral metoprolol 25mg to be given, which brought HR down from 136 to 118. ICU was consulted, the evaluating physician noted the patient to be in significant distress, tachycardic to 130 bpm, tachypneic to 40 respirations per minute, with bilateral wheezing and accessory muscle use. Echocardiogram had been done, showing ejection fraction of 39%, concentric left ventricular hypertrophy, grade 2 diastolic dysfunction, dilated cardiomyopathy if left and right atrium, as well as right ventricle. She was subsequently admitted to the MICU for treatment of acute respiratory failure secondary to asthma exacerbation. In the ICU, the patient reported feeling respiratory symptoms for about 2 weeks, however after administration of albuterol, the patient began feeling symptoms of anxiety, palpitations, tremulousness, facial flushing, with these symptoms concerning her and her family enough for ED visit this admission. Evaluation of her labs showed elevated T3 at >650, elevated T4 at 22.6, with diminished TSH level at 0.01. Patient was diagnosed with thyroid storm in consideration of lab values with clinical features. TSI was ordered, which returned at 674. Propranolol had been started, however in light of acute asthma exacerbation, and after discussion with the cardiology team, the decision was made to start Diltiazem 30 mg q12 hours instead. Methimazole was started, 10mg q12 hours. Endocrinology was consulted, after discussion with patient, further history regarding thyroid symptoms was extracted; the patient had been having tremors for 3 weeks, as well as weight loss of about 5 pounds over several months, with generalized anxiety and dysphagia; she also noted diarrhea in the "past weeks". Reported a family history of goiter in her aunt. Recommendation from endocrinology perspective was obtain TSI level and ESR, methimazole was to be held due to risk of agranulocytosis and hepatotoxicity, but if TSI was elevated and ESR is low, start methimazole 20 mg twice daily after risk/benefit discussion with patient and family. As ESR was normal at 2.0, methimazole was started. Cardiology team noted JVD with respiratory distress, and recommended transfer to MICU to CCU for further monitoring and treatment of systolic heart failure, after giving furosemide IV doses. At this time, heart rate remained tachycardic at 115, temperature was 99, blood pressure 125/86, saturation 95% on 4L oxygen by nasal cannula. In the CCU, patient was cautiously transitioned from diltiazem to metoprolol to control heart rate, as well as low dose lisinopril for cardio-protection. Patient had persistent JVD with mild respiratory distress after transfer to CCU, so continuous IV furosemide regimen was started. Tachycardia also persisted, metoprolol was continued with close monitoring of respiratory status. Her clinical course improved over the next several days, furosemide was transitioned to 20mg oral daily, and she was transferred to the floor. Patient was discharged with core measures for heart failure as well as methimazole.

She followed up with endocrinology approximately 3 weeks later, recommended to keep the current medication regimen and repeat TFTs with close follow up. Repeat labs showed Free T4 now 2.25 (mild elevation), T3 down to 403, and TSH <0.01, still markedly reduced. Methimazole was increased to 20mg three times daily. One week later, labs were again repeated: Free T4 1.80, T3 316, TSH <0.01. After this change in dosage, the patient developed a facial rash. Methimazole was stopped, and the patient was then scheduled for radioactive iodine ablation. Repeated echocardiogram was done 5 months later, it showed the resolution of the reduced ejection fraction to 61.5%, normal size and thickness of Left ventricle, normal right ventricle size, moderate dilation of left atrium, all of which are significant improvements to the prior echocardiogram; her heart rate also was noted to have normalized after adjustments of beta blocker dose.

After receiving radioactive iodine ablation, the patient reported becoming asymptomatic on subsequent follow ups. However during this phase, repeat labs still were in the hyperthyroid range, and the patient was still having tachycardia on metoprolol. At this point the patient stopped taking all medications, including metoprolol.

At 6 month follow up with the endocrinologist, the patient reported not taking her medications, as well as chest pain. Thyroid function testing continued to remain in the hyperthyroid range despite ablation months prior. Patient did not follow up until 1 year later, when she saw cardiology. Patient's tachycardia was improved, core measures were no longer indicated and were thus discontinued. However, she did begin to complain of mild exophthalmos despite having completed treatment with radioactive iodine approximately 1 year prior.

Discussion

Thyroid hormone causes specific changes in the cardiovascular system, affecting the systemic vascular resistance (Decreased), leading to a compensatory increase in blood volume due to renal sodium reabsorption, causing increased inotropy and chronotropy with the resultant increase in cardiac output [2,7,13]. Cardiac contractility is also increased directly and indirectly due to thyroid hormone stimulation, increasing contractility in the form of increased oxygen consumption [3,13].

The predominant molecule mediating these changes is Triiodothyronine (T3), as this hormone is present in the heart muscle [1,8]. Once in the heart muscle (through presumed transmembrane entrance proteins), T3 activates nuclear transcription of mRNA that synthesizes proteins that stimulate G-protein receptors resulting in physiologic changes, including a cAMP-mediated increase in inotropy and Beta-adrenergic receptor-mediated chronotropic [7]. In layman's terms, the direct effects of T3 on the heart are

increased muscle contraction in the heart and increased heart rate. Triiodothyronine also is noted to create these changes through actions on the calcium, sodium, and potassium receptors that can influence levels of these ions in the cells and thus alter inotropy and chronotropy [1].

A hyperthyroid state will pathologically alter the cardiac myocytes due to the excessive stimulation produced by the T3 in favor of a tachycardic and hypercontractile state. Thyroid hormone is known to cause the new onset or trigger Atrial Fibrillation, which in and of itself is a cause of tachycardia [3]; however, many patients, as in the case presented, can be in persistent sinus tachycardia. It is also widely known that treating hyperthyroidism alone will also successfully treat tachycardia [3].

Congestive heart failure is a known complication of uninhibited continued hyperthyroidism in the setting of prolonged tachycardia, being sinus or atrial fibrillation. The patient should be in this state for a protracted period of time, and present hypertrophy of cardiac muscle, arrhythmia, and effect of elevated cardiac preload will all happen, to result in CHF [5]. Interestingly, resolution of hyperthyroid state led to consequent resolution of heart failure and symptoms, with one study reporting 85% of patients (50% of which had systolic CHF) improving after being euthyroid for three months [4].

Treatment of Grave's Disease through radioactive iodine ablation is effective and safe, as the hyperthyroid state will bring the iodine to the thyroid, creating necrosis and loss of function over 6-18 weeks [2,7,14]. As seen in this case, thyroid function tests persistently elevated but improved and led to symptom resolution.

Toft et al. [9] refer a similar case reported in 1992, in which a young female patient presented with symptoms of hyperthyroidism with concurrent heart failure without any prior cardiac comorbidities that successfully resolved after adequate treatment with oral medication, suggesting a thyroid cause of heart failure and a need to consider it in the differential diagnosis. However, Froeschl et al. [11] reported a case in which a 26-year-old male presented with severe thyrotoxicosis, leading to heart failure that resulted in pancreatitis and mortality due to a history of significant alcohol dependence, multiorgan system failure, and protracted ICU level of care. On autopsy, hypertrophic changes in the heart consistent with non-ischemic cardiomyopathy of hyperthyroidism were confirmed.

In their case report and discussion, Choudhary et al. [10] incur the importance of managing heart failure directly with loop diuretics and beta-blockers for both heart failure management and control of the excess adrenergic activity seen in hyperthyroidism. Schrier et al. [13] stress the importance of ACE inhibition in the setting of heart failure with hyperthyroidism, suggesting that while treating the root cause of the heart failure results in resolution of such, *treating the heart failure itself is equally important.*

Our case showed a patient who was noted to have sinus tachycardia with altered thyroid function in the setting of congestive heart failure with reduced ejection fraction; besides her changes in history, she was started on appropriate therapy. However, she developed a rash on methimazole, for which this medication was discontinued, iodine ablation was done and despite her limited adherence to the medications, on her follow up she was largely asymptomatic (the main complaint at this time was mild exophthalmos) with regular heart rate, and the medications for her heart condition were discontinued.

Exophthalmos is seen almost exclusively in Grave's disease [12], in which hyperthyroidism is mediated from the Thyroid-Stimulating Immunoglobulin, which was positive in this patient. The presence of exophthalmos after treatment was noted in a small subset of patients and is an especially higher risk if the treatment is radioactive iodine therapy [15] (Table1 1-4).

Table 1: Pertinent laboratory findings on admission.

Laboratory variable	Lab result	Reference range
T3	>650 ng/dL	60-181 ng/dL
T4	22.6 ug/dL	4.8-10.4 ug/dL
TSH	<0.01 mIU/dL	0.40-4.50 mIU/dL
TSI	674	<140
Hgb	13.4 g/dL	12.0-16.0 g/dL
Hct	40.6%	42.0-51.0%
WBC Count	7.3 k/uL	4.8-10.8 k/uL
Platelet Count	155 k/uL	150-400 k/uL
Sodium	145 mEq/L	135-145 mEq/L
Potassium	4.4 mEq/L	3.5-5.0 mEq/L
Chloride	104 mEq/L	98-108 mEq/L
BUN	14 mg/dL	6-20 mg/dL
Creatinine	0.5 mg/dL	0.5-1.5 mg/dL
AST	96 u/L	9-36 u/L
ALT	117 u/L	5-40 u/L
ALP	106 u/L	42-98 u/L
Bilirubin, Total	0.5 mg/dL	0.2-1.2 mg/dL
Bilirubin, Direct	0.3 mg/dL	0.0-0.3 mg/dL

Table 2: Thyroid Function Tests on Outpatient Follow-up Three Weeks After Discharge.

Laboratory variable	Lab result	Reference range
T3	403 ng/dL	60-181 ng/dL
Free T4	2.25 ug/dL	0.8-2.20 ng/dL
TSH	<0.01 mIU/dL	0.40-4.50 mIU/dL

Table 3: Thyroid Function Tests Five Weeks After Initial Outpatient Follow-up.

Laboratory variable	Lab result	Reference range
T3	316 ng/dL	60-181 ng/dL
Free T4	1.80 ug/dL	0.8-2.20 ng/dL
TSH	<0.01 mIU/dL	0.40-4.50 mIU/dL

Table 4: Most Recent Thyroid Function Tests One Year After Initial Outpatient Follow-up

Laboratory variable	Lab result	Reference range
T3	224 ng/dL	60-181 ng/dL
T4	13.1 ug/dL	4.8-10.4 ug/dL
TSH	0.01 mIU/dL	0.40-4.50 mIU/dL

Conclusion

Hyperthyroidism is a known cause of cardiomyopathy that could lead or trigger congestive heart failure secondary to tachycardia-induced cardiomyopathy. A proper assessment and treatment are crucial to reverse this condition and avoid potential complications that could directly impact the patient's outcome with reversible cardiomyopathy.

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