CLINICAL VIGNETTE

Severe Hypothyroid-Induced Cardiomyopathy

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A 61-year-old female was admitted with severe weakness. Her history includes mitral valve prolapse, and thyroidectomy and radioactive iodine ablation (RAIA) for a malignant thyroid nodules. Admission las noted severe hyponatremia, as well as elevated thyroid stimulating hormone (TSH) of 130mIU/mL and a free thyroxine (T4) of 0.2ng/dL. Patient reported diarrhea, poor appetite and decreased oral intake. In orthostatic position she felt lightheaded and fell, causing a head injury. Telemetry was reviewed and noted sinus bradycardia around 60 beats per minute. There were no other cardiac arrhythmias. Blood pressure was 94/62mmHg but ranged as high as 154/87mmHg. ECG revealed sinus rhythm at 64 BPM, left axis deviation, incomplete right bundle branch block, and diffuse t-wave inversions (TWI) consistent with inferior ischemia and a QTc of 532ms. When compared to her outpatient ECG, the TWI were new. Serial troponin enzymes and BNP were all within normal range. Given ECG changes and her unwitnessed fall, an echocardiogram was obtained. It revealed a mildly reduced ejection fraction of 50% with global hypokinesis and normal left ventricle size. Her known mitral valve prolapse without significant regurgitation, was unchanged. She denied alcohol abuse, autoimmune disease, or family history of cardiomyopathy. She had no signs or symptoms of congestive heart failure. Her fall was felt to be vasovagal based on orthostatic vital signs and absence of arrhythmia on telemetry.

Additional record review noted normal ECG, myocardial perfusion study and coronary artery calcium (CAC) score of 0 making coronary artery disease (CAD) an unlikely etiology of her mildly depressed ejection fraction. ECG discharge showed persistent inferior TWI. Her QT had shortened and lateral TWI had improved. Given she was asymptomatic, with lower blood pressures, no new medical therapy was initiated for her mildly reduced ejection fraction. Her marked hypothyroidism was aggressively treated, and her ECG and echocardiogram were repeated. The ECG showed resolution of TWI and normalization of QTc interval, her echocardiogram showed normalization of ejection fraction back to baseline of 60-65%. Her blood pressure also normalized to 130/72mmHg.

Dilated cardiomyopathy (DCM) is characterized by dilation and impaired contraction of one or both ventricles. Patients usually have signs and symptoms of heart failure or present with arrhythmia symptoms. Some patients may not develop overt heart failure. The most common cause of dilated cardiomyopathy is ischemic heart disease. Our patient had undergone recent stress test and had a CAC score of 0 suggesting a low risk of coronary arterial disease. While uncalcified three vessel or left main disease remains possible, it was unlikely with absence of chest pain and normal troponin, and BNP. The patient had ECG abnormalities and since 7% of patients with DCM can have ischemic disease, outpatient CT coronary angiogram was ordered to confirm no CAD. [1] This was not obtained during hospitalization due to hypothyroidism concerns of iodine toxicity.

Other causes of DCM include stress-induced cardiomyopathy and viral cardiomyopathy. While hospitalization and severe hypothyroidism represented a major stress on patient, she did not have classic echocardiographic findings of stress cardiomyopathy. These include apical hypokinesis with basal wall hyperkinesis. Patient also had no recent viral symptoms to explain her cardiomyopathy. Her diarrhea was non-infectious and improved on a lactose restricted diet.

Thyroid dysfunction is one of a few endocrine abnormalities that can cause cardiac dysfunction, with pheochromocytoma and Cushing's syndrome being two others. The exact mechanism by which hypo or hyperthyroidism can lead to DCM are not known. Typically, patients are asymptomatic and do not present with signs or symptoms of congestive heart failure.[2] Patients who develop overt heart failure tend to have pre-existing risk factors, marked elevations in BNP, and worse overall prognosis.[3] Rhythm disturbances are common and include prolonged QT interval and increased risk of ventricular tachycardia.[2] Despite the cardiovascular effects of hypothyroidism, the all-cause mortality is not increased.[4] Our patient had QT interval prolongation, without increased ectopy on telemetry monitoring. Her QT interval normalized with her thyroid replacement.

Our patient has American Heart Association (AHA) class B heart failure. This is a "pre-heart failure" category and describes people who have evidence of structural heart disease without any signs or symptoms of heart failure.[5] The structural change is a mild reduction in ejection fraction. The identified risk factor is metabolic, severe hypothyroidism; and treatment is focused on the risk factor.[5] Our patient received intravenous levothyroxine with serial monitoring of free T4 levels. There was no concern for adrenal insufficiency given normal cortisol of 21.7ug/dL and steroids were not given. The manifestations of her cardiovascular disease resolved as her TSH and free T4 normalized.

If the patient had congestive heart failure (CHF) symptoms she would be AHA class C. The New York Heart Association (NYHA) classification characterizes the severity of a patients' symptoms. For patients with AHA class C and D, treatment is geared towards reducing symptoms, morbidity and mortality.[5] Treatment is based on ejection fraction and includes beta blockers, angiotensin converting enzyme inhibitors/angiotensin receptor blockers/angiotensin receptor neprilysin inhibitors, spironolactone, and sodium-glucose cotransporter-2 (SGLT2) inhibitor therapies.

Our patient had severe hypothyroidism symptoms after recent thyroidectomy and RAIA. Her cardiac manifestations of hypothyroidism pertaining to the patient included prolonged QT interval and decreased cardiac contractility with associated ECG changes. Hypothyroid induced cardiomyopathy is a rare cause of cardiomyopathy that often presents without symptoms and does not increase all-cause mortality. As with our patient, it reverses with treatment of the underlying thyroid dysfunction.

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