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(CASE REPORT)

Amiodarone induced myxedema coma presenting with cardiogenic shock and junctional rhythm

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Abstract

We describe the case of an elderly female, with no prior history of thyroid dysfunction, who presented with facial swelling and worsening fatigue. She was found to have myxedema coma, with thyroid-stimulating hormone (TSH) 98.90 uIU/mL and free thyroxine (fT4) 0.8 ng/dL, following two months of amiodarone therapy for paroxysmal atrial fibrillation. The patient developed cardiogenic shock and received liothyronine, levothyroxine, stress dose steroids, inotropic and vasopressor therapy while in the intensive care unit (ICU). She was weaned off vasopressors and switched to oral levothyroxine with a gradual steroid taper. Two months following hospital discharge, the patient remained asymptomatic while on oral levothyroxine. Literature lacks cases of amiodarone-induced myxedema coma and to our knowledge, myxedema coma secondary to amiodarone use has never been reported in patient on treatment for as short as two months. This case demonstrates the significance of early diagnosis and prompt treatment for rapid progression of signs and symptoms of amiodarone-induced myxedema coma to optimize outcomes. Although transient changes are seen while on amiodarone therapy, continuous monitoring is essential to examine for apparent thyroid dysfunction. Thyroid function testing prior to initiation of amiodarone and in cases of cardiac dysrhythmias or cardiogenic shock can help to avoid potentially fatal complication of hypothyroidism.

Keywords: Amiodarone; Myxedema Coma; Cardiogenic Shock; Hypothyroidism

1. Introduction

Nearly 15-20% of amiodarone-treated patients will have amiodarone-induced thyroid dysfunction [1]. Risk enhancement of myxedema coma, a life-threatening emergency, is seen in elderly female patients with severity being dependent upon the underlying status of the patient's thyroid gland [2]. Severe hypothyroidism is known to cause hemodynamic instability, including bradycardia, decreased myocardial contractility, a low cardiac output, and hypotension. In the absence of preexisting cardiac disease, overt congestive heart failure is a rare occurrence and thyroid hormone therapy can reverse the cardiac abnormalities.

2. Case Presentation

An 82-year-old female with no known history of hypothyroid disease presented with 2-day history of shortness of breath, swelling of the face, and generalized weakness. She endorsed medication compliance with amiodarone and apixaban for recently diagnosed paroxysmal atrial fibrillation two months prior to presentation. She was found to be hypotensive and bradycardic with a heart rate of 40 beats per minute. Initial vital signs also included temperature 36°C and pulse oximetry of 94% on 4 liters via nasal cannula. Physical examination noted facial swelling, non-pitting edema of bilateral lower extremity, cool extremities and no evidence of thyroid enlargement. Electrocardiogram (ECG) showed sinus bradycardia with first degree atrioventricular block (Figure 1). She received atropine with no improvement.

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Vasopressor and inotropic support with norepinephrine and dobutamine were initiated. Chest x-ray showed an enlarged cardiac silhouette and pulmonary edema. Chest computed tomography showed moderate sized left sided pleural effusion and ground-glass interstitial infiltrates in the right lobes. After thoracentesis removed 1300 mL, she was started on ceftriaxone and azithromycin. Laboratory investigations were significant for elevated thyroid-stimulating hormone (TSH) 98.90 uIU/mL, decreased free thyroxine (fT4) 0.8 ng/dL, free triiodothyronine (T3) 2 pg/mL, and cortisol 28 ug/dL. Close examination of the medical records revealed patient being euthyroid with a TSH of 3 uIU/mL before treatment of atrial fibrillation. Given findings on examination, myxedema coma was considered as the primary diagnosis.

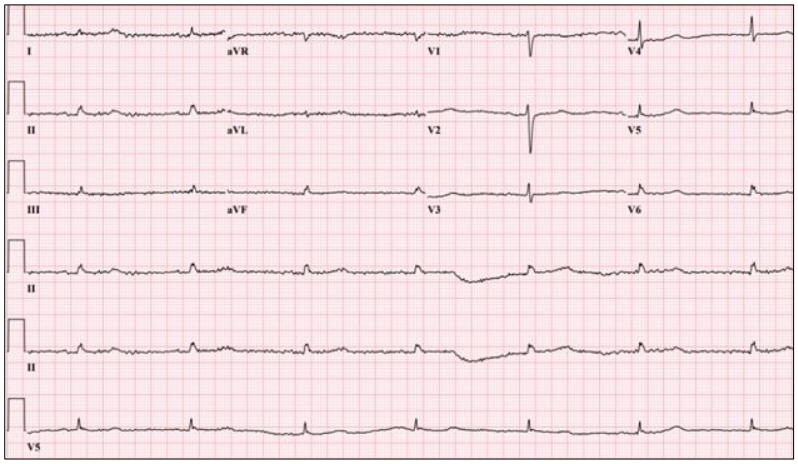

Figure 1 ECG on admission showing sinus bradycardia with first degree AV block

An emergent transthoracic echocardiogram revealed an ejection fraction of 45%, right ventricle systolic pressure 73.6 mmHg and a trivial pericardial effusion without hemodynamic compromise. Given the patient's worsening pulmonary symptoms, hypothermia, and decreased mentation, she remained in the ICU for close monitoring. Intravenous (IV) levothyroxine with initial dose of 200 ug, liothyronine 10ug, and 100 mg hydrocortisone Q8H were initiated for myxedema coma and critical-illness related corticosteroid insufficiency or until the coexistence of adrenal insufficiency was ruled out. Workup for autoimmune thyroiditis was negative.

On hospital day 2, oxygen requirements were increasing and respiratory status was severely compromised, on high flow nasal cannula at 100% FiO2 and 60 L/min, with SpO2 of 88%. Arterial blood gas was as follows: pH of 7.28, pCO2 37, pO2 75, and HCO3 17. Due to worsening renal function, she was started on sustained low-efficiency dialysis.

By hospital day 6, her mentation was not improving despite being off sedation. Urine culture grew *Enterococcus faecium* and she was transitioned from linezolid to daptomycin and meropenem. The following morning, the patient was awake and more alert than prior days. Ten days after starting IV thyroid hormone replacement, her TSH and fT4 improved to 7.18 uIU/mL and 0.9 ng/dL, respectively. Her heart rate improved, ECG changes resolved, and kidney function returned to baseline. She was weaned off vasopressors and switched to oral levothyroxine with a gradual steroid taper. The patient was discharged home with daily oral levothyroxine and close follow-up with her endocrinologist. Two months following hospital discharge, the patient remained asymptomatic while on oral levothyroxine.

3. Discussion

The most severe sequela of deficient thyroid hormone is myxedema coma. Amiodarone related myxedema coma was first reported in the early 1970s with fewer than twenty subsequent published cases. The scarcity of reported cases is largely due to readily available thyroid function tests allowing early diagnosis and treatment of hypothyroidism. This life-threatening clinical condition is defined by its cardinal manifestations of hypothermia, bradycardia, and altered mentation. Predisposing factors include pre-existing thyroid dysfunction, sepsis, hypothermia and certain medications with potential to affect thyroid function. Amiodarone, an iodinated antiarrhythmic, bears structural similarity to thyroid hormones which can have an adverse effect on thyroid function [3]. Given the rarity of amiodarone-associated myxedema coma, it is challenging to decipher the exact etiology of depressed mentation and peripheral edema in a patient with multiple comorbidities.

Myxedema coma is often suspected when history reveals antecedent symptoms of thyroid dysfunction followed by progressive lethargy, stupor, and coma [4]. If the state of decompensated hypothyroidism is suspected, blood investigations should be conducted prior to treatment for measurement of TSH, fT4 and cortisol. Majority of patients with myxedema coma will have primary hypothyroidism but those with central hypothyroidism may have associated secondary adrenal insufficiency [5]. Treatment is instituted without delay for laboratory confirmation as this endocrine emergency has a high mortality rate, even with treatment [6].

Thyroid hormone plays an important role in blood pressure homeostasis. For patients determined to have cardiogenic shock, such as our patient with sustained hypotension, the steps in management included hemodynamic support with norepinephrine and inotropic agent. Ventilator support was also required given our patient's deterioration in consciousness and acute respiratory failure to due pulmonary edema. Prompt management of hypotension with circulatory support and continuous hemodynamic monitoring allowed optimization of her volume status. Rapid employment of combined IV therapy with fT4, T3 and cortisol provided clinical and biochemical improvement within two weeks. The gradual decline of our patient's serum TSH was an indication of adequate therapy. Once she had regained consciousness and improved pulmonary and cardiac function, treatment solely with oral levothyroxine and steroid taper were able to maintain her stable clinical condition. For myxedema coma, the optimal mode of thyroid hormone replacement therapy remains unknown due to the rarity of the condition and lack of clinical trials comparing different treatment modalities [7]. We chose to co-administer T3 due to the greater biologic activity and more rapid onset of action compared to levothyroxine.

Given the non-specific clinical presentation, it is necessary to be vigilant in considering myxedema coma for patients presenting with bradycardia, respiratory failure, hypothermia, and altered mentation. Early recognition, prompt diagnosis and treatment with thyroid hormone therapy and supportive measures can help to improve outcomes.

4. Conclusion

Myxedema coma is a life-threatening emergency with very few reported cases in patients on amiodarone therapy. Although transient changes are seen while on amiodarone therapy, continuous monitoring is essential to examine for apparent thyroid dysfunction. Thyroid function testing prior to initiation of amiodarone and in cases of cardiac dysrhythmias or cardiogenic shock can help to avoid potentially fatal complication of hypothyroidism.

Compliance with ethical standards

Disclosure of conflict of interest

The above listed authors; Drs. Asllanaj; Sheikhan; Trad; and Darmal; have no conflicts of interest to declare.

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Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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