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Case Report: A Case of Myxedema Coma with Cardiovascular Complications

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Abstract

Myxedema coma is a critical endocrine condition with a high risk of death, caused by severe, long-term hypothyroidism and the failure of the body's normal mechanisms to maintain internal balance. Intravenous LT4 thyroid hormone replacement, combined with supportive care, is essential for the prompt recovery of neurological, cardiovascular, renal, pulmonary, and metabolic dysfunction. Coexisting comorbidities can affect the intensity of hormone replacement therapy and must be closely monitored during treatment. We present the case of a 48-year-old male who arrived at the emergency room unresponsive, with bradycardia, hypoxia, hypotension, found to have a complete heart block from inferior wall myocardial infarction. Initial treatment prioritized his cardiac resuscitation but , further investigation revealed severe hypothyroidism. The patient was started on levothyroxine, (oral followed by IV) parallel to the intricate management of complications including cardiomyopathy, shock liver, acute kidney injury and diabetes with gradual improvement in mental status and hemodynamics over a course of a week. This case underscores the importance of recognizing hypothyroidism as a critical etiology in patients presenting with multi-organ failure and the role of Levothyroxine in cardiac function .

Introduction

Myxedema coma in an endocrine emergency , caused by severe deficiency of thyroid hormone which impacts multiple physiological mechanisms leading to hemodynamic collapse and altered sensorium.(1) It can be due to long standing untreated hypothyroidism or may be the first presentation of hypothyroidism, with precipitating factors such as infections or cardiovascular events. The most salient feature of myxedema coma is deteriorating mentation. It has cardiac, neurological, gastrointestinal, renal, respiratory , electrolyte and hematological manifestations evident on clinical and biochemical exam and severity of which can be assessed by various scoring systems. Given high mortality , treatment should be commenced based on suspicion alone (2).Thyroxine replacement is mainstay of treatment . Factors such as the degree of clinical and biochemical hypothyroidism, active comorbidities, and details of

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administration of levothyroxine (e.g., dosage, timing, and other factors impacting absorption) are relevant considerations (3). This case illustrates a relatively uncommon and challenging presentation of myxedema coma in a middle-aged male with acute Myocardial infarction. We are aware that the two systems are interlinked and can exacerbate one another and there is limited data on interventional studies of Thyroid hormone replacement in acute Myocardial infarction. Fine tuning of thyroxine replacement , guided by Free T4 levels, along with critical management of cardiac decompensation resulted in good outcome.

Case Presentation

A 48-year-old male labourer was brought to the emergency room by his roommates due to generalized weakness, abdominal discomfort, and progressive lethargy starting 24 hours prior . The patient had a medical history of hypertension, type 2 diabetes mellitus, and asthma. Home medication included Glucophage . Upon presentation to the ER his vitals were as follows: heart rate 44 bpm, blood pressure 90/45 mmHg, SpO2 88%, and temperature 37°C. Upon arrival, the patient was unresponsive with a Glasgow Coma Scale (GCS) score of 12, later deteriorating to 3. ECG showed complete heart block without ST elevation, and echocardiography revealed inferior wall akinesis with an ejection fraction of 35%. The patient was initially stabilized in E.R with mechanical ventilation, transcutaneous pacemaker and transferred to the ICU. H.R improved to 62.

Blood work revealed blood glucose > 400, Lactate 6.6, bicarbonate 15 mmol, sodium 127 mg/dl , pH 7.2 , creatinine 1.66 , total bilirubin 1.3 ,Direct bilirubin 0.7 , alkaline phosphatase 148, serum protein 5.6 (g/dl) ,ALT 464 U/L ,AST 216U/L, INR 2.28 ,Troponin T 1.96ng/ml , D dimer 4.97.Coronary angiography showed triple vessel disease. PCI was not pursued due to delayed MI presentation.

Medical management was initiated with aspirin, Plavix, atorvastatin , atropine, dopamine, noradrenaline, IV fluids, antibiotics, IV insulin .Heart rate maintained on transvenous pacemaker. Nutritional support via tube feeds was started.

The patient's subsequent results (few hours after initial presentation) revealed severe hypothyroidism. TSH of 57 μIU/mL, a free T4 level of 0.127 ng/dL, and a free T3 level of 0.57 pg/mL. No prior Thyroid results for comparison. Myxedema score was >60 . Random cortisol was > 40. Levothyroxine was initiated at 250 mcg via NG tube for 2 days . Hydrocortisone was held given risk of wall rupture and good cortisol level.

The patient remained lethargic but showed stabilization of hemodynamics with extubation, and weaning off the pressors. Oral liquids were started, and agitation was managed with Seroquel and Haldol. After 2 doses of oral levothyroxine , on day 3 we procured intravenous Levothyroxine from outside facility. The patient was started on

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150 mcg IV levothyroxine for 3 days followed by 250 mcg IV .Free T4 was monitored every 2 days and increased from 0.127 to 1.14.

There was interval worsening of liver enzymes (3000U/L) and crp (148) . Patient was strictly monitored through central venous catheter and treatment resumed with inotropes(dobutamine , digoxin) and diuresis which improved end organ perfusion . Patient's mentation improved over next few days. His liver and renal function improved along with the blood pressure with successful weaning off the pressors .He was tolerating oral diet ,switched to oral Levothyroxine 150 mcg . The patient left against medical advice and moved back to his home country.

Date	TSH (µIU/mL)	Free T4 (ng/dL)
Day 1	57	0.127
Day 3	46	0.632
Day 5	34	0.663
Day 7	38	0.491
Day 10		1.14

Discussion

Myxedema is associated with a high mortality by itself and when you add coronary ischemia to the equation, you are dealing with a complex management scenario(3). It is well known that thyroid hormone (primarily T3) acts on cardiac myocyte, peripheral vasculature and red cell mass, to increase cardiac contractility, myocardial oxygen consumption, cardiac output and decrease systemic vascular resistance(4,6) . Consequentially Hypothyroidism results in decreased cardiac output, bradycardia and accelerated atherosclerosis and coronary artery disease largely via increased hypercholesterolemia and diastolic hypertension.(4)

Timing and Degree of hemodynamic support is priority. All measures for fluid resuscitation and correction of hyponatremia, hypothermia , and hypoglycemia, as well treatment of any underlying infections should be followed . Goal is to improve the oxygen supply to the ischemic myocardium before increasing the myocardial demand with T4 .

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Thyroid hormone replacement is the key. The usual loading of 200–400 μg of levothyroxine is recommended (2)However caution should be exercised as giving high-dose will exert an increase in metabolic demand and exacerbate the ischemia. (3)Also the cause of coma in some patients maybe be compounded by the precipitating comorbidity, therefore, not every patient will require a large loading dose(4)

We gave a lower dose of 150-200 mcg with good response. There is variable reports on the appropriate dose and route of administration for T4. IV levothyroxine may not be readily viable in all centers leaving us to rely on oral form. The literature suggests that the route of levothyroxine administration does not significantly affect outcomes, though intravenous administration is preferred in critically ill patients given rapid and predictable increase in concentration. (5)

Though Empiric glucocorticoid coverage should be employed as part of the initial therapy for myxedema coma, preceding levothyroxine administration, In our case given the risk of free wall rupture in background of myocardial infarction (MI) and good random cortisol , we avoided the use.

It was likely that our patient had a preexisting cardiomyopathy from long-standing, untreated and undiagnosed , hypothyroidism in addition to underlying ischemic disease. This was his first presentation of hypothyroidism. Also of note is his male gender and middle age , as opposed to previously reported incidence in elderly females. Patient's preexisting cardiac insufficiency may have been further exacerbated after the initiation of T4 therapy dose along with an additional iodine load from the cardiac catheterization. (1)

The therapeutic endpoints in myxedema coma should be, improved mental status, improved cardiac and pulmonary function as seen in our patient. The atrioventricular block resolved with levothyroxine treatment, thus avoiding the need for permanent pacemaker implantation.

Conclusion

This case of myxedema coma in a 48-year-old male with triple vessel disease underscores the importance of early diagnosis and prompt treatment. Aggressive levothyroxine therapy, in combination with supportive care, can lead to favorable outcomes even in critically ill patients. Important to consider thyroid dysfunction as a contributing factor in patients with cardiovascular instability. High-dose levothyroxine therapy, both oral and intravenous, can be life-saving, with careful monitoring of thyroid function and

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cardiovascular status for a favorable outcome. Given the rarity of myxedema coma, randomized clinical trials are not feasible, and so recommendations regarding the type, route, and outcomes measures for replacement are based solely on expert opinion and case reports.

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