Case Report

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Case Report on Myxedema Coma Associated with End-Stage Cardiac Disease

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Abstract

Myxedema coma is a critical disorder with high mortality rates. Disruption of the compensatory mechanism for severe and long-term hypothyroidism by various causes leads to serious complications, including hypothermia, respiratory failure, circulatory failure, and central nervous system dysfunction. The most common causes of myxedema coma are thought to include infectious disease, stroke, myocardial infarction, sedative medicines, exposure to the cold and surgery in patients with poorly controlled hypothyroidism. We present a case of an 82-year-old female presented with complaints of loss of speech for 3 days, complaint of easy fatiguability for 3 days, altered sensorium since 1 day, weakness of both upper and lower limbs for 3 days and the patient attenders complaint of that patient is not taking food and difficult in swallowing since 3 days. In spite of the foregoing, patients with decreased mental status who have undergone thyroid surgery or hypothyroidism should be evaluated for myxedema coma. Additionally, this condition may be caused on by chronic hypothyroidism or by experiencing acute precipitating events like sepsis, a cerebrovascular accident, gastrointestinal bleeding, exposure to cold, trauma, or taking certain medications. A high mortality rate was noted, so patients whose myxedema coma is suspected should start receiving treatment right away.

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INTRODUCTION

Myxedema coma is a critical disorder with high mortality rates. One of the most common endocrinopathies worldwide is hypothyroidism. Depending on the demographic, the prevalence ranges from 1% to 10%, with a larger likelihood in females [1, 2].

Since myxedema coma is rare, with an incidence of 0.22 cases per million per year, it can have a high mortality rate (25–60%) if it is not detected and treated properly in its early stages [3, 4]. (3,4)Hypothyroidism's severe complication known as myxedema coma causes patients to have progressive mental decline as well as multiple organ problems [5, 6]. The patient may decompensate into myxedema coma if these homeostatic processes are overwhelmed by causes like infection. Septicemia and infections like pneumonia, urinary tract infections, and cellulitis are the leading precipitating factors. Patients with hypothyroidism frequently report fatigue, weight gain, constipation, and a sensitivity to cold temperatures [2]. Myxedema also describes the swollen skin and soft tissue that develops in hypothyroid patients [1].

Although we also see patients with severe hypothermia, in our experience, many patients with myxedema crisis have temperatures that are 2-3°F below normal. The primary underlying pathology in myxedema crisis, which results in hypothermia

and reduced heart activity, is low intracellular T3 related to hypothyroidism. Neurovascular adaptations include persistent peripheral vasoconstriction, mild diastolic hypertension, and decreased blood volume are ways the body tries to make up for the damage. To confirm the diagnosis of myxedema coma, laboratory investigations are crucial. The following outcomes are included: Most patients have high Thyroid-stimulating hormone (TSH), which is a sign of a primary thyroid disease, low levels of free triiodothyronine (T3) and free thyroxine (T4), low serum osmolality and hyponatremia, high serum creatinine levels result from reduced renal perfusion. Both an evaluation of adrenal function and a complete blood count (CBC) should be done. Leukocytosis may not be seen because of hypothermia. One of the few indicators of the presence of an infection may be a white blood cell differential. Cardiomegaly, pericardial effusion, congestive heart failure, or pleural effusion may all be shown on a chest radiograph.

Electrocardiography can detect arrhythmias, flattened or inverted T waves, low-amplitude QRS complexes, a prolonged QT interval, and sinus bradycardia.

This disorder can sometimes end in complications that cause cardiac arrest [7]. Myxedema coma, a potentially fatal condition, is caused by a severe thyroid hormone deficiency and long-term undiagnosed or untreated hypothyroidism. Additionally, myxedema crises may be triggered by cerebral hemorrhages, congestive heart failure, vehicle accidents, gastrointestinal bleeding, and a variety of sedative medications. Diuretics may obscure some myxedematous symptoms and exacerbate the hyponatremia brought on by myxedema crises.

A patient with a coma or altered mental status who is also hypothermic, hyponatremic, and/or hypercapnic should have myxedema coma suspected. However, the majority of patients do not initially present with myxedema or coma, causing altered mental status as the key characteristic [8]. The stopping of thyroid supplements in severely ill patients is a background component in myxedema crises that is sometimes ignored. This may be because the presenting symptoms and triggering circumstances receive the majority of attention while the accompanying hypothyroidism is frequently disregarded. Due to myxedema coma is so serious it causes a life-threatening threat; thyroid hormone therapy should start even before laboratory confirmation. The patient must be admitted to intensive care unit (ICU) with intensive cardiovascular and pulmonary support. We describe a case of a typical myxedema coma that was quickly diagnosed and treated, resulting in a speedy recovery. Given the high death rate and severity with which the disease manifests, thyroid hormone replacement therapy should begin as soon as the presence of the condition is suspected, even before obtaining laboratory confirmation. Hydrocortisone doses for stress should be administered as well until concurrent adrenal insufficiency is ruled out.

Case Report

An 82-year-old female presented with complaints of loss of speech for 3 days, complaint of easy fatiguability for 3 days, altered sensorium since 1 day, weakness of both upper and lower limbs for 3 days and the patient attenders complaint of that patient is not taking food and difficult in swallowing since 3 days. Know case of carcinoma uterus (hysterectomy with chemotherapy was done), hypertension, CVA (2 years back), IHD, T2DM on regular medication and hypothyroidism (history of levothyroidectomy on tablet thyroxine 120mcg was done).

Admitted to medicine intensive care unit (MICU), AHRC, B G Nagara, Nagamangala, Mandya on 01 July 2023 (day 1) for the same complaints, the patient was conscious and not responding to oral commands. On investigations, we found her ESR- 42 mm/hr, RBS- 175 mg/dl. Her LFT showed SGOT – 43 U/L, RFT- was normal and NCCT scan of brain done, indicated small area of encephalomalacia changes with surrounding gliosis in left frontal lobe, multiple chronic lacunar infarct in bilateral capsulogangluonic regions and bilateral thalami, bilateral centrum semiovale and left cerebellar hemisphere, small vessel ischemic changes and age-related cerebral, cerebellar atrophic changes and ECHO report showed EF -30%. In view of patient condition primary treatment included IV pantoprazole 40mg, IV ondansetron 4mg, IV mannitol 100ml, oral ecospirinAV (Aspirin 150 mg+ atorvastatin 20 mg), oral cilnidipine 10 mg, S/C soluble insulin injection (injection human Atctrapid) 12-12-10 units and IVF NS @ 50ml/hr.

On 3 July 2023 (day 3), the patient was unconscious and difficulty in breathing so kept in mechanical ventilation and added nebuliser ipratropium bromide(500mg) +levosalbutamol(1.25mg) and budesonide (0.5mg). In view of thyroid hormone elevations, the thyroid test was done, the report values were TSH – 95.06μIU/ml, T3 - 0.391pg/ml and T4 - 0.736pmol/L work up done showed thyroid disease and by giving thyroxine the patient was responsive to the drug, hence the patient was confirmed suffering from myxedema coma.

The first-line treatment for myxedema coma is IV

levothyroxine, but in our case the patient had end-stage cardiac disease with EF - 30%, so if levothyroxine given in this condition, it may cause tachycardia and result in sudden cardiac arrest, so alternative to IV levothyroxine the patient was given oral thyroxine 125 mcg through ryles tube and IV hydrocortisone 100 mg STAT. Further considering the patient condition the same medication as day 1 continued adding to that IV amoxicillin potassium cluvanate 1.2gm.

The patient was good responsive to the medications and hence kept under observation. On 6 July 2023 (day 6), the patient regained consciousness. Thyroid test done on day 6 the vales were TSH – 32.61μIU/ml, T3 – 0.792pg/ml, T4- 6.53pmol/L. Hence the patient was good responsive to the medications, and the same were followed.

On 8 July 2023 (day 8), the patient feels better with no complaints. Hence the patient was shifted from MICU to Semi-special general ward and kept under observation for two days, on 10 July 2023(day 10), the thyroid report values were TSH – 8μIU/ml, T3 – 1.6pg/ml, T4 – 9.76pmol/L. The patient feels comfortable; hence the patient was planned to discharge.

On July 2023(day 10) 5:30 pm, the patient was discharged with advice on medication such as Oral thyroxine 125mcg 1-0-0, oral ecospirin A V (Aspirin 75mg+ atorvastatin 20mg) 0-0-1, IV human mixture (biphasic isophane insulin)10-0-8 units, oral cilnidipine 10mg 1-0-0 and initiated follow-up after 2 weeks.

The follow-up was done the patient had no complaints, feels comfortable and the vitals and lab reports were normal. Hence the same medication was advised to take and informed for regular check-ups (Table 1).

DISCUSSION

As a result of severe hypothyroidism, the symptoms of myxedema coma include decreased mental status, hypothermia, and other symptoms of diminished organ functions [9], and should be taken into consideration in patients, particularly elderly females with a history of thyroid surgery or hypothyroidism [4]. Myxedema coma may be triggered by an acute event, such as sepsis, a stroke, a myocardial infarction, gastrointestinal bleeding, exposure to the cold, trauma, or certain medications, particularly lithium, amiodarone, and opioids [4, 10]. Despite there being no definitive diagnostic criteria for the condition, patients frequently present with confusion, lethargy, and altered mental status [11]. According to a retrospective study conducted in Japan, prompt diagnosis and treatment have resulted in a 30% reduction in the in-hospital mortality rate for myxedema coma over the previous few years [12].

There is disagreement over whether to use T4 alone or in conjunction with T3, even though thyroid hormone replacement is widely acknowledged to be essential for the treatment of myxedema coma. According to reports involving animal studies, thyroidectomized rats who only received T4 replacement therapy were unable to obtain adequate tissue perfusion of T3. However human studies show that T4 can still exert the necessary biologic effects for recovery in myxedema patients even in the absence of T3 [8].

Although it is indisputable that T3 is more effective than T4 at enhancing thyroid hormone status, some risks must be taken into account before administration. Increased oxygen consumption by tissues is one way that T3 raises metabolic rate. If the patient is unable to elicit a compensatory increase in cardiac output to meet these increased demands, this poses a problem. An imbalance between oxygen delivery and consumption can cause tissue hypoxia, which ultimately leads to death. For elderly patients and those who have cardiac or respiratory comorbidities, this is a crucial factor to take into account [13].

Every patient may experience life-threatening complications from overly aggressive attempts to reverse hypothyroidism, so when T3 is indicated, it should be administered at a low and gradual dose. Due to the lack of objective diagnostic standards for myxedema coma, the effectiveness of treatment should be assessed using clinical judgement. Improvement in mental status and stabilization of vital signs are indicators of a proper response to treatment. An objective way to see whether patients with myxedema coma are getting better is by monitoring their T4 and TSH levels. TSH levels, however, are a reflection of how your thyroid has been functioning over the previous 6–8 weeks, so they might not be an accurate indicator of how well your thyroid treatment is working. This makes clinical improvement and T4 status potentially more useful in determining how well a treatment is working.

Our patient had a long-term history of hypothyroidism with intellectual disability, and abnormal facies and given thyroid hormone elevations, the thyroid test was done, the report values were TSH – 95.06microIU/ml, T3 - 0.391pg/ml and T4 - 0.736pmol/L work up done showed thyroid disease and by giving thyroxine the patient was responsive to the drug, hence the patient was confirmed suffer-

Table 1: Thyroid tests value

Test (reference range)	Day 3	Day 6	Day 8
T3 (2-4.4pg/ml)	0.391pg/ml	0.792pg/ml	1.6pg/ml
T4 (10-22pmol/L)	0.736pmol/L	6.53pmol/L	9.76pmol/L
TSH (0.27-4.2μIU/ml)	95.06μIU/ml	32.61μIU/ml	8μIU/ml

ing from myxedema coma. In our case, the patient received oral thyroxine 125 mcg through a ryles tube in addition to IV hydrocortisone 100 mg STAT.

IV levothyroxine is the standard treatment for myxedema coma, but because the patient had end-stage cardiac disease and an EF of only 30%, giving levothyroxine could have resulted in tachycardia and sudden cardiac arrest.

Patients who are elderly or who have underlying heart or respiratory issues should receive additional attention using this method of care. We believe the judicious use of oral thyroxine can avoid catastrophic outcomes and improve the mortality rate of myxedema coma.

CONCLUSION

Given the broad availability of TSH test and the regular monitoring by primary care physicians, myxedema coma is currently a rare occurrence, but it is still a serious condition that needs immediate attention. If myxedema coma is associated with end stage cardiac disease, it is advisable to avoid treatment using IV levothyroxine to prevent complications such as tachycardia, sudden cardiac arrest. In this condition, the patient should be treated with oral thyroxine through a ryles tube. In suitable clinical situations, we kindly request physicians to keep this distinction in mind. We also encourage our readers to conduct additional research comparing triiodothyronine and thyroxine as monotherapy versus combined therapy.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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