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Myxoedema Coma: A Rare Case Report

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Abstract **Case Report**

Myxedema coma (MC) is a rare and life-threatening endocrine emergency resulting from severe hypothyroidism, with a high mortality rate. It can develop in patients with long-standing undiagnosed hypothyroidism or be precipitated by an underlying medical or surgical condition. Due to its nonspecific presentation, MC is often misdiagnosed, even in patients exhibiting key signs such as hypothermia. This case illustrates the diagnostic challenges and clinical management of a 43-year-old man with no prior medical history, who was admitted to the emergency department in a comatose state with severe hypothermia and bradycardia. Further biochemical testing revealed profound undetectable T4 levels, a TSH concentration exceeding 205 mU/ml, hyponatremia at 121 mEq/L and CK elevation at 28 960 UI/L. No precipitating illness was identified. The myxedema coma score was calculated at 135 points, highly suggestive of diagnostic of myxedema coma, while the sequential organ failure assessment (SOFA) score was at 9. Treatment consisted of supportive care, including fluid resuscitation, vasopressors, mechanical ventilation, passive rewarming, thyroid hormone replacement (levothyroxine) at a dose of 300µg and glucocorticoid administration (hydrocortisone 50 mg IV every 6 hours). This case underscores the critical need for clinicians to maintain a high index of suspicion for hypothyroidism in at-risk individuals to facilitate early diagnosis and timely intervention, potentially improving patient outcomes.

Keywords: Myxedema coma, Severe hypothyroidism, Hypothermia, Coma, Thyroid hormone replacement.

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INTRODUCTION

Myxoedema coma (MC) is a rare endocrine emergency and life-threatening presentation of severe hypothyroidism associated with a high mortality. This illness primarily affects women and the elderly due to the higher prevalence of hypothyroidism in these populations.

It can occur in patients with long-standing undiagnosed hypothyroidism or be precipitated by an underlying medical or surgical condition. The most common precipitating events are infections, myocardial infarction, or acute cerebrovascular events. However, other potential causes include hypoglycemia, hypothermia, congestive heart failure, gastrointestinal bleeding, trauma, and even certain medications such as anesthetics, beta-blockers, diuretics, lithium, rifampin, and amiodarone [1-3]. this disorder can be easily misdiagnosed, even in patients exhibiting key signs such as hypothermia [4]. The Clinical presentation can be non-specific, diagnosis relies on a combination of clinical evaluation considering symptoms of hypothyroidism, hypothermia, hyponatremia, hypercarbia, and hypoxemia along with laboratory assessment of thyroid-stimulating hormone levels [3, 4].

Treatment consists of thyroid hormone supplementation, along with supportive measures such as mechanical ventilation, management of hypotension, and correction of hypothermia. Nevertheless, recommendations for the treatment of myxedema coma are primarily based on expert opinions and case reports. In our country, patients with myxedema coma are treated with enteral LT4, with or without LT3, administered via a nasogastric tube. In contrast, in many other countries, these treatments are more commonly administered intravenously. This observation highlights the diagnostic challenges and clinical management of an elderly man who was eventually diagnosed with myxedema coma.

CASE PRESENTATION

A 43-year-old man with a non medical history was admitted to the emergency department for evaluation of coma. Upon admission on life-saving emergency unit, the patient was unresponsive, with a blood pressure of 84/60 mmHg, heart rate of 31 bpm,

oxygen saturation of 90%, and body temperature of 20 ºC. Physical examination revealed an obese middle-aged man with a flat affect who appeared older than his stated age. He had puffy face with macroglossia, dry and cool skin, coarse hair and an filling of the supra clavicular hollows. Laboratory tests revealed undetectable T4 and a TSH level exceeding 205mU/ml. His hemoglobin was 8 g/dl, white blood cell 9.2 x103/uL (4-10), lymphocytes 0.22 x103/uL (1-4), platelets 36 x103/ul (150-450),neutrophils 2.6x103/ul (2-7.5) total, aspartate aminotransferase (AST) 190 UI/l, alanine aminotransferase (ALT) 41 UI/l, CK 28960 UI/L, LDH 693 U/L, sodium 121 mEq/L, potassium 2.82 mEq/l, calcium 8,2 mg/dl, phosphorus 2.6 mg/dl and total CPK 28 960 (7 times a normal value), C-reactive protein 6 mg/dl , Urea 0.29 g/l, Creatinine : 8,18 mg :l .

The myxedema coma score was calculated at 135 points, highly suggestive of diagnostic of myxedema coma. The patient earned points in his myxedema coma score for hypothermia (20 points), obtunded state (30 points), anorexia/constipation before presentation (5 points), precipitating infectious event (0 points), bradycardia (30 points), low voltage complex (10 points), hypotension (20 points), hyponatremia (10 points) and Hypoxemia (10 points). The mean sequential organ failure assessment (SOFA) scores calculated at 9.

The endocrinology team was consulted, and the patient was treated for myxedema coma. Management includes supportive care with fluid resuscitation, vasopressors, mechanical ventilation, passive rewarming, and glucocorticoid administration (hydrocortisone 50mg IV hydrocortisone every 6h) to address potential concurrent adrenal insufficiency, followed by thyroid hormone replacement, the patient was treated with enteral thyroxine (300µg bolus followed by 100µg/day). By the 24 hours of admission, his clinical condition has not improved, and repeat laboratory tests showed T4<0,4 ng/dl and TSH at 113 mU/ml. Our patient died 3 days after admission from cardiogenic shock and deep hypothermia.

DISCUSSION

Myxedema coma is a rare condition with high morbidity. The pathophysiology of myxedema coma is characterized by reduced intracellular T3 levels due to hypothyroidism, resulting in hypothermia and impaired cardiac function. Respiratory failure may occur due to decreased sensitivity to hypoxia, hypercapnia, respiratory muscle dysfunction and airway obstruction. Increased vascular permeability can lead to pericardial and/or pulmonary effusions and anasarca, while hyponatremia may develop secondary to renal dysfunction and excessive vasopressin secretion. Cardiac depression and hypotension can progress to cardiogenic shock, which is often resistant to vasopressors without appropriate thyroid hormone replacement. Additionally, hypoglycemia and central nervous system depression may contribute to seizures and altered consciousness.

This condition can often be misdiagnosed, this is due to the difficulty in establishing clear diagnostic criteria for the disease, given its rarity and sudden onset, which makes conducting large, prospective, well-controlled studies impractical [5]. The diagnosis of myxedema coma relies on a combination of clinical presentation and laboratory findings. A diagnostic scoring approach for myxedema coma was implemented to confirm clinical suspicion and enable earlier recognition and intervention [6, 7]. Geanina Popoveniuc *et al*., retrospectively assessed the frequencies of characteristics associated with MC in patients from their institutions and developed a semiquantitative diagnostic point scale. This scale was subsequently applied to selected patients whose data were retrieved from the literature. They concluded that a score of ≥60 in the proposed system is potentially diagnostic for MC, while scores between 45 and 59 could identify patients at risk for developing MC. This scoring was applied in our case and it showed highly suggestive/diagnostic of myxedema coma that validated our diagnosis. Additionally, the term "myxedema coma" itself may be misleading. Previous reports have highlighted that patients with myxedema coma can be difficult to identify in the early stages, as they are not always in a comatose state [8]. We believe that it is more important to treat patients with potential myxedema coma early stages proactively based on clinical judgment to prevent worsening severity and irreversible complications.

According to the American Thyroid Association, the initial management of myxedema coma should include intravenous levothyroxine, with the optional addition of liothyronine [9]. General recommendations suggest administering 300-500 mg of IV levothyroxine. Dosing may vary depending on different studies, with some recommending lower doses for older, frailer patients, particularly those with cardiovascular disease [10]. Other sources suggest a weight-based dosing approach, calculating the total body distribution of levothyroxine and administering a dose according to the distribution size [11]. The question of whether myxedema coma should be managed with LT4 alone or a combination of LT4 and LT3 remains controversial. A previous report indicated that higher doses of LT3 (≥75 µg/day) and LT4 > 500µg were associated with fatal outcomes [11-13]. The recommendations suggest using T3 with an initial loading dose of 10 to 20 µg, followed by 10 µg every 4 hours for 24 hours, then 10 µg every 6 hours for 1 to 2 days, until the patient can transition to oral therapy [13]. It is also important to look for potential drug interactions (such as proton pump inhibitors, iron, or calcium supplements) [13]. Our choice of therapy was limited to only oral T4 due to the lack of both IV preparations (LT4 and LT3). Although oral T4 via a nasogastric tube has proved to be equally effective, a major drawback has

been the poor absorption of orally administered drugs in MC [10].

Glucocorticoid therapy is recommended for all patients in stress doses before initiating thyroid hormone replacement, until coexisting adrenal insufficiency has been ruled out. However, the administration of catecholamines and steroids may have worsened myxedema coma and contributed to increased mortality. Published evidence supports this possibility, as dopamine and steroids have been shown to induce iatrogenic hypothyroidism by suppressing thyrotropin secretion, leading to decreased plasma T4 and T3 levels. Steroids also inhibit the peripheral conversion of T4 to T3 and are commonly used in the management of thyroid storm. Moreover, a previous study indicated that adrenal cortisol secretion was not significantly impaired in patients with hypothyroidism [14-16]. Additionally, Yosuke Ono and al reproted in their study that patients who received steroids and catecholamines had significantly higher mortality rates compared to those who did not require these treatments [5]. More research is needed to establish whether the use of steroids and/or catecholamines is beneficial in the treatment of patients with myxedema coma.

Pinaki Dutta and al analysed factors predictive of mortality in patients with CD and concluded in their study that low mean blood pressure, need for mechanical ventilation, precipitation of myxoedema coma by use of sedatives, concurrent sepsis, and baseline or mean sequential organ failure assessment (SOFA) scores ≥6 were predictive of mortality [17]. Given the challenges faced in his management and the presence of significant mortality predictors at presentation as SAFO score at 9 and mechanical ventilation, our patient tragically died 3 days after admission from cardiogenic shock and deep hypothermia.

CONCLUSION

Myxedema coma is a rare but life-threatening condition with a high mortality rate, even with appropriate treatment. Thyroxine levels should be assessed immediately in all patients presenting with bradycardia, bradypnea, elevated creatine phosphokinase (CPK), and hypoxemia, Early diagnosis and the implementation of appropriate management strategies are crucial for improving outcomes. The initial treatment involves supportive care, administration of intravenous levothyroxine, (the oral route being an alternative option) and avoid high doses for older, frailer patients, particularly those with cardiovascular disease.

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