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Lost in the mix: A challenging diagnosis of myxedema coma in an elderly patient

Nidha Shapoo*; Arlen Ventura; Bryan Quintanilla; Armeen Poor

Department of Pulmonary and Critical Care, New York Medical College/Metropolitan Hospital, New York, USA.

***Corresponding Author: Nidha Shapoo**

Department of Pulmonary and Critical Care, New York Medical College/Metropolitan Hospital, New York, USA.

Email: shapoon@nychhc.org

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Abstract

Myxedema coma represents a severe form of hypothyroidism seen in patients with long-standing, untreated, or poorly controlled hypothyroidism. Myxedema coma has a high mortality, so immediate treatment is necessary based on clinical symptoms before the confirmation of diagnosis. Cardiovascular comorbidities often pose a challenge in recognizing the symptoms of myxedema coma, especially in patients with no prior history of hypothyroidism. The treatment could be delayed, leading to a fatal outcome. We present here an 86-year-old male who was admitted to the ICU with hypotension and hypothermia after an orthopedic procedure. The patient had chronic congestive heart failure with a heart block on a pacemaker and no prior history of hypothyroidism. The initial management focused on stabilizing the patient and addressing the underlying cardiac problems. Due to worsening mental status, hypoventilation requiring mechanical ventilation, and elevated thyrotropin levels, myxedema coma was diagnosed. The treatment was initiated promptly, but the patient deteriorated and succumbed to his disease. Our case highlights the challenges in identifying the symptoms of myxedema coma, which could be easily missed in elderly patients having comorbidities, especially cardiovascular. Due to the high mortality rate of myxedema coma, there is a need to revise the diagnostic criteria, particularly addressing this patient population.

Introduction

Myxedema coma is a rare, life-threatening complication of hypothyroidism associated with high mortality rates. The underlying cause includes severe deficiency of thyroid hormones due to long-standing or poorly controlled hypothyroidism, often precipitated by infections, trauma, surgery, myocardial infarction, or certain medications [1]. Early recognition of symptoms and aggressive treatment are imperative as the mortality approaches 30 to 60 percent even after optimal treatment [2]. Myxedema coma can affect virtually any organ system and may lead to multi-organ failure. The key features of myxedema coma are altered mental status and hypothermia, but hypotension, bradycardia, hypoventilation, and hypercapnia are also present [3]. The cardiovascular manifestations in myxedema coma are especially severe and can mimic the symptoms of congestive heart failure [4]. Myxedema coma in a setting of mild hypothyroidism is extremely rare, and only a few case reports exist in the literature [5-8]. We report here a patient of myxedema coma who had two complex issues: underlying congestive heart failure and no prior hypothyroidism, leading to a delay in diagnosis and a fatal outcome.

Case presentation

An 86 years old male with a past medical history of hypertension, diabetes mellitus, hyperlipidemia, chronic kidney disease, coronary artery disease, chronic congestive heart failure with reduced ejection fraction (25-30%), complete heart block s/p biventricular pacing, atrial fibrillation, and dementia was ad-

mitted in the ICU with hypotension and hypothermia after an orthopedic procedure. The patient underwent left femur intramedullary nailing for a left femur fracture and developed hypotension intra-operatively, for which he was put on nor-epinephrine. The patient was transferred to the ICU due to persistent hypotension requiring high doses of nor-epinephrine. There was no bleeding post-operatively. On examination, the patient was lethargic but conscious and oriented at presentation. There was bilateral pedal edema, raised jugular venous pressure, reduced air entry at the left base on lung auscultation, and apical systolic murmur on cardiac auscultation. There was no swelling in the neck and no prior surgical scar suggestive of any thyroid disease. The vital signs were Temp-97.7^{O}F, HR-70 bpm, RR-17/min, BP-97/52 mmHg, and SPO_2-98%. The laboratory tests yielded the following results: Hb-9.8 g/dl, WBC-4.99x10^3/mcL, BUN-50 mg/dl, creatinine-1.7 mg/dl, potassium-4.1 mEq/L glucose-187 mg/dl, magnesium-2.2 mg/dl, Pro BNP-27996 pg/ml, Troponins-negative. ECG showed no acute ischemic changes. The computed tomography pulmonary angiography was negative. The echocardiogram showed LVEF 25-30%, findings unchanged from the previous one. The patient was continued on nor-epinephrine with regular monitoring of vitals and intake/output. The patient improved hemodynamically, was weaned off the pressor support, and started furosemide. Despite hemodynamic stability, the patient showed deterioration in his mental status, progressing from obtundation to a stuporous state. Notable was hypothermia with a minimum temperature of 95°F (35°C), copious nasal and oral secretions, decreased urine output, and worsening renal functions. CT head without contrast showed old right MCA territory infarcts, no new findings. Chest X-ray showed bilateral atelectasis with left pleural effusion, no active infection. Blood cultures/sputum cultures/urine cultures were done and reported negative. Rewarming was started, and a trial of antibiotics was given. The patient did not show any signs of improvement and had worsening mental status and hypoventilation. Blood gas revealed hypercapnia with PCO_2-81. The patient was put on BIPAP. Thyroid functions done revealed: TSH-19.6 uIU/ml and free T4-0.5 ng/dl. Thyroid functions before admission were as follows: TSH-5.60 uIU/ml (05/09/23), 4 uIU/ml (02/03/23) with normal free T4-1.1 uIU/ml. Endocrinology was consulted. A diagnosis of myxedema coma was made with a diagnostic score of 85. The treatment was initiated with a loading dose of 200 mcg of levothyroxine followed by 50 mcg daily and hydrocortisone 50 mg IV 6 hourly. Lower doses of levothyroxine were chosen due to the underlying cardiac co-morbidities. The patient required mechanical ventilation due to worsening mental status, copious secretions, and hypercapnia. Over the course of a few days. The mental status improved slightly, and the patient was weaned off the ventilator. The patient was put on high-flow oxygen therapy at 30 L/min with a FiO_2 of 60%. The thyroid functions improved over the course of a few days, with TSH trending down to 5.14 uIU/ml with an increase in free T4 (Table 1). Despite improvement in thyroid functions, there was no clinical improvement. The patient became comatose, had cardiac arrest, and succumbed to his disease.

Table 1: The timeline of thyroid functions during admission.

Thyroid functions	08/25/23	08/27/23	08/28/23	08/30/2023	08/31/2023
TSH (0.27-4.20 uIU/Ml)	19.6	23.6	22.1	7.14	5.14
Free T3 (2.00-4.40 pg/mL)			1.08	0.77	0.86
Free T4 (0.9-1.8 ng/dL)		0.5	0.5	0.7	0.7

Table 2: Diagnostic score for myxedema coma.

Thermoregulatory dysfunction (Temperature in Celsius)	Points	Cardiovascular dysfunction	Points	Metabolic disturbances	Points
>35	0	Bradycardia		Hyponatremia	10
32-35	10	Absent	0	Hypoglycemia	10
<32	20	50-59	10	Hypoxemia	10
Central nervous system effects	**Points**	40-49	20	Hypercarbia	10
Absent	0	<40	30	Decrease in GFR	10
Somnolent/lethargic	10	Pericardial/Pleural effusions	10	**Gastrointestinal dysfunction**	**Points**
Obtunded	15	Pulmonary edema	15	Anorexia/abdominal pain/constipation	5
Stupor	20	Cardiomegaly	15	Decreased intestinal motility	15
Coma/Seizures	30	Hypotension	20	Paralytic ileus	20
Precipitating factors	Points	EKG changes: QT prolongation, or low voltage complexes, or bundle branch blocks, or non-specific ST-T wave changes, or heart blocks	10		
Absent	0				
Present	10				

Abbreviations: EKG: Electrocardiogram; GFR: Glomerular Filtration Rate.
A score of 60 or higher is highly diagnostic of myxedema coma.
A score of 25 to 59 suggests risk for myxedema coma, and a score <25 is unlikely to indicate myxedema coma.

Discussion

Myxedema coma, a complication of overt hypothyroidism, is highly unusual in the setting of mild thyroid dysfunction [1]. Mild elevation in thyrotropin may occur with aging, suggesting either a decline in thyroid function or a reset in the TSH set point [9-11]. Treatment is generally not indicated in asymptomatic patients with TSH less than 10 mU/L, who are followed up [12-16]. Our patient had mildly elevated Thyrotropin (TSH) 5.60 uIU/ml before admission with no prior history of hypothyroidism, thyroidectomy, or treatment with levothyroxine. Progression to myxedema coma, in these cases, is extremely rare, and only a few case reports have been described in the literature [5-8]. All the patients who have been reported before were on levothyroxine treatment, and often, there is a precipitating event like infection, surgery, or trauma that leads to physiological decompensation. In our case, the femur fracture surgery might have precipitated the event. There might be a possibility that in elderly patients with comorbidities, the threshold for progression to myxedema coma is low.

Myxedema coma is a multiorgan disease; however, the cardinal features include altered mental status and hypothermia. The decline in mental status may be subtle, manifesting as lethargy to more apparent changes of delirium, obtundation, or rarely coma [3]. The cardiovascular symptoms of myxedema coma tend to be severe and may include bradycardia, hypotension, pericardial effusion, or congestive heart failure [4]. The respiratory manifestations of myxedema coma can result from decreased ventilatory response to hypoxia and hypercapnia, leading to alveolar hypoventilation and respiratory failure. Our patient presented with hypotension and hypothermia with gradual deterioration in his mental status. Later, it progressed to alveolar hypoventilation and respiratory failure, requiring mechanical ventilation.

Myxedema coma, because of its rarity, is not always easy to diagnose when the patients have multiple comorbidities, especially cardiovascular [1]. The underlying congestive heart failure delayed the recognition of symptoms of myxedema coma in our patient. A diagnostic score was developed in 2014 based only on the clinical parameters, where a total score of >60 is considered diagnostic of myxedema coma [17] (Table 2). The diagnosis is then confirmed by an elevated Thyroid Stimulating Hormone (TSH) level and low free thyroxine [2,18]. Our patient had a diagnostic score of 85, a TSH level of 22.60 uIU/ml, and a free T4 level of 0.5 ng/dl.

Treatment in myxedema coma should be initiated based on clinical suspicion without waiting for the laboratory results, which includes intensive care, mechanical ventilation if necessary, intravenous fluids, correction of hypothermia and electrolyte imbalances, treatment of any underlying precipitating event and administration of thyroid hormone and glucocorticoids. Thyroid hormone therapy is initiated with an initial dose of 200 to 400 mcg of levothyroxine intravenously, followed by daily intravenous doses of 50 to 100 mcg until the patient can take oral levothyroxine. The lower end of the dosing range is preferred in older patients and those at risk for cardiac complications (myocardial infarction, arrhythmia). Intravenous hydrocortisone supplementation is also required to correct hypotension, which is preferred at 50 mg every 6 hours [19]. Once the diagnosis was made, the treatment was started promptly in our patient with steroid and levothyroxine therapy along with the supportive care that he was getting in intensive care. The clinical symptoms of myxedema coma were masked in our patient by the underlying cardiac comorbidities and dementia. The fact that the patient had subclinical hypothyroidism and was not on levothyroxine treatment also led to the delay in diagnosis in our case.

The factors associated with mortality in myxedema coma include hypotension, bradycardia at presentation, need for mechanical ventilation, hypothermia unresponsive to treatment, sepsis, intake of sedative drugs, lower GCS, high APACHE II scores, low Glasgow coma scale, advanced age and cardiovascular disease [20-22]. In our case, advanced age, underlying cardiac disease, low Glasgow coma scale, hypoventilation, and hypotension might have contributed to the fatal outcome.

Conclusion

Our case stresses the need for early recognition of myxedema coma symptoms, even in patients with normal or mildly elevated thyrotropin levels. All patients who present with hypotension and hypothermia should be evaluated for thyroid dysfunction. Myxedema coma can be highly fatal, especially in old patients who have underlying cardiac comorbidities.

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