Case Report

Myxedema Coma: A rare presentation of hypothyroidism

Sahanie Liyanaarachchi[1], Charith Perera[2*]

[1]District General Hospital Chilaw, Sri Lanka

[2]Teaching Hospital Karapitiya, Sri Lanka

Abstract

Myxedema coma is an extreme manifestation of hypothyroidism presenting with altered level of consciousness, hypothermia, bradycardia, hypotension, hypoglycemia, and hypoventilation. It is an endocrine emergency. Now it has become a rare presentation due to early detection and treatment of hypothyroidism. The common precipitating factors like sepsis, myocardial infarction, hypothermia, sedative medications, and surgery should be addressed in a poorly controlled hypothyroid patient as early as possible. Treatment with high dose thyroid hormones and steroids should be started on clinical suspicion even before the laboratory confirmation. We present a case of an 85-year-old woman who presented to the emergency room with one day history of reduced level of consciousness, found to have bradycardia and hypothermia. Her symptoms significantly improved with oral levothyroxine therapy.

Keywords: Myxedema coma, Hypothyroidism, Oral levothyroxine

Funding: None

Competing interest: None

Received: 23.01.2023 **Accepted revised version:** 28.05.2023 **Published:**25.07.2023

*✉**Correspondence:** chulaguptha@gmail.com https://orcid.org/0000-0002-8431-4685

Cite this article as: Liyanaarachchi S.*et al*, Myxedema Coma: A rare presentation of hypothyroidism. Anuradhapura Medical Journal 2023; 17 (2): 29-32, DOI: http://doi.org/10.4038/amj.v17i2.7756

Introduction

Myxedema coma is a rare medical emergency with a high mortality rate ranging from 30 to 50 per cent [1-4]. Elderly females with hypothyroidism are often affected [1]. Patients usually present with reduced level of consciousness, coma and, sometime with psychotic features known as myxedema madness [5]. It is precipitated by an acute event such as infection, myocardial infarction, cold exposure, surgery, or sedative medications [6]. When myxedema coma is suspected, patients should be managed aggressively with thyroid hormone replacement.

Case presentation

Our patient was an 85-year-old female, bed-bound for the last 6 months due to neck of femur fracture. She had a medical history of diabetes mellitus, hypertension, and ischemic heart disease for 5 years. She was hospitalized because of reduced level of consciousness. Examination revealed Glasgow Coma Scale (GCS) of 10/15, heart rate of 56 beats per minutes, unrecordable blood pressure, and hypothermia at 35 °C. On general examination she was pale with dry skin, generalized non pitting edema and macroglossia.

Her Oxygen saturation was 90 % without clinical evidence of cardiac failure and the lungs were clear bilaterally. Initial blood test results are given in table 1. Electrocardiogram showed small complexes, and sinus bradycardia (figure 1). 2D echocardiogram revealed a moderate pericardial effusion, without evidence of tamponade. The liver functions were normal. Thyroid hormones were altered with thyroid-stimulating hormone (TSH) of >150 mIU/L and free thyroxine (FT4) of 0.64 ng/dl.

Table 1: Laboratory investigation results of the patient

Investigation	Day 1	Day 7	Day 12	6 weeks After discharged	Reference Range
Random Blood Sugar (mg/dL)	135	148	133		70 - 140
Venous blood gas					
pH	7.338				7.31-7.41
pCO_2 (mmHg)	26.8				41 -51
pO_2 (mmHg)	27				30-40
HCO_3 (mmol/L)	14.5				23-29
Lactate (mmol/L)	6				0.5 - 2.2
White Blood Cell (/µL)	17 770	14 630	8 650	8 100	4.0-11.0 x10^3
Hemoglobin (/dL)	8.1	10.4	10.9	11.1	11-16
Platelets (/L)	343 x 10^9	352 x 10^9	347 x 10^9	311	150-450 x10^9
C-Reactive Protein (mg/dL)	48	18	<5	-	< 5
Aspartate Transferase (U/L)	24	-	40	-	5 - 34
Alkaline Transferase (U/L)	16	-	14	-	0 - 55 u
Serum Sodium (mmol/L)	134	-	141	-	136 - 145
Serum Potassium (mmol/L)	4	-	3.6	-	3.5 - 5.1
Calcium (mg/dL)	8.8	-	-	-	8.4 - 10.2
Thyroid Stimulating Hormone (mIU/L)	>150	96	51	5.3	0.55 - 4. 78
T4 (ng/dL)	0.64	0.79	0.91	1.36	0.89 - 1.76
Anti thyroid peroxidase	Not available				
Anti thyroglobulin	Not available				
Serum Creatinine (mg/dL)	2.43	2.87	2.54	2.29	0.57 - 1.1
Blood urea (mg /dl)	16	20	18	18	7-18
Serum cortisol (µg/dL)	15	-	-	-	5 - 25
Urine Full Report					
Pus cells(per hpf)	30-40	10-15	<5	-	0-5
Red blood cells (per hpf)	0	0	0		<4
Urine Culture	Coliform bacilli isolated (> 10^5 colony forming units)	-	No growth	-	
Blood Culture	No growth	-	-	-	
Troponin I (ng/mL)	0.043	-	-	-	< 0. 016

Considering the above test results and the presentation with hypothermia and altered level of consciousness myxedema coma was diagnosed. 4 L/min oxygen was administered via a face mask with improvement of oxygen saturation to 96 %. She was aggressively treated with iv. crystalloids followed by iv. noradrenaline for hypotension. Simultaneously she was started on oral levothyroxine 150 ug daily. Blood was taken for random cortisol levels and intravenous hydrocortisone 100 mg 6 hourly was started, which was later found to be normal. She was started with intravenous Meropenem after taking blood and urine for cultures. The patient was rewarmed gradually. Two pints of blood transfused to correct anemia. Urine culture yielded positive results for coliform bacilli, which were found to be susceptible to meropenem. Blood pressure improved within the first 48 hours with the above treatment, and the inotropic support weaned off at the end of third day. The body temperature was back to normal, and her GCS improved up to 14/15 four days later. The dose of meropenem was reduced to 500mg every 12 hours due to the altered renal function tests, and it was continued for 7 days. Generalized edema gradually improved and the patient was able to take oral feeds by eighth day with full recovery of GCS up to 15/15. The urine culture was repeated and showed no growth of organisms. On the 13th day of admission, she

was discharged with the same dose of levothyroxine. She was reviewed in the medical clinic after 6 weeks, where most of the clinical features of myxedema coma was improved with TSH level of 5.3 mIU/L and free T4 level of 1.36 ng /dL. She was continued on the same dose of levothyroxine and monitored with TSH.

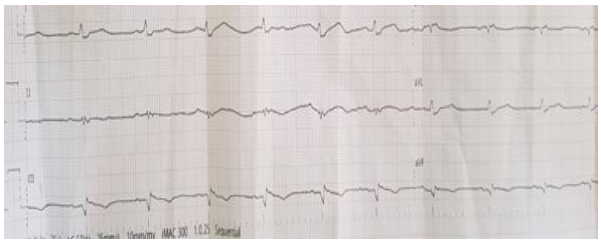

Figure 1: Electrocardiogram showing sinus bradycardia

Discussion

Severe hypothyroidism leading to decreased mental status, hypothermia, and other symptoms related to the slowing of function in multiple organs is termed myxedema coma [7]. Classic signs and symptoms include dry skin, hoarse voice, hypothermia, macroglossia, non-pitting oedema, decreased level of consciousness, hyponatremia, and hypoglycemia [8]. Although hypothyroidism is a relatively common condition, myxedema coma is a rare and potentially life-threatening complication that occurs in only a small proportion of patients with severe hypothyroidism [1-4, 9]. Factors contributing to its rarity include the effective management of hypothyroidism with thyroid hormone replacement therapy, the late-stage presentation of myxedema coma, and the potential under diagnosis or misdiagnosis of the condition due to its non-specific clinical features.

There are several precipitating factors that can lead to myxedema coma, including infection, medication use, myocardial infarction, cold exposure, noncompliance with thyroid hormone replacement therapy, and surgery or trauma [6]. Our patient presented with reduced level of consciousness, with an unrecordable blood pressure and found to have a culture positive urinary tract infection. Without a background history of hypothyroidism this presentation could have solely attributed to urosepsis. However, due to the presence of additional symptoms such as dry skin, non-pitting edema, macroglossia, hypothermia and bradycardia, the diagnosis was suspected to be myxedema coma precipitated by urosepsis.

Although the urosepsis, septic shock and myxedema coma may have some similar presentations, a comprehensive and thorough examination is crucial to differentiate between the conditions, and the presence of additional signs should prompt consideration of a possible myxedema coma diagnosis. A patient with shock due to sepsis, hypovolemia or cardiac failure usually presents with tachycardia, but our patient had bradycardia with hypotension. Septic shock usually manifests with warm peripheries, whereas our patient had hypothermia. These differences from other common causes for shock and the background examination findings of myxedema , helped to suspect the diagnosis of myxedema coma in this patient. It is important to initiate diagnosis and treatment promptly without waiting for laboratory confirmation.

The mainstay of treatment of myxedema coma is thyroid hormone replacement therapy. Glucocorticoids should be administered until coexisting adrenal insufficiency has been excluded [10]. Supportive measures including intensive care support, mechanical ventilation, gradual rewarming, judicious administration of intravenous fluid, treatment of underlying infections, and management of precipitating factors are all important in managing myxedema coma. The preferred treatment is combined therapy with T4 and T3. But it is uncertain whether to treat with T3, T4, or combined [9]. The therapy should be started without delay to prevent the risk of adverse outcomes and to reverse the effects of severe hypothyroidism. Because of hypotension and impaired gut absorption intravenous route is recommended over oral administration [11]. But our patient was started on oral levothyroxine due to the unavailability of the IV levothyroxine form. Oral levothyroxine is also an effective form of treatment where the iv form is not available [12]. This patient's thyroid hormone levels were regularly monitored every 3 to 4 days to evaluate the effectiveness of therapy and to facilitate adjustments to the dosage as needed. This patient was treated with IV hydrocortisone while waiting for cortisol level results and was also given IV meropenem at a dose of 500 mg every 12 hours for a total of 7 days to treat the urinary tract infection. Additionally, the patient was slowly rewarmed using blankets and received fluid resuscitation and inotropic support. Early detection and prompt treatment of myxedema coma is crucial to improve the mortality rate.

Conclusion

Myxedema coma is an endocrine emergency with a very high mortality rate. It is rare due to early detection of

hypothyroidism and availability of thyroid hormone assays. It is easily misdiagnosed due to nonspecific symptoms and under diagnosed due to its rarity. It is important for healthcare providers to maintain a high level of suspicion and consider myxedema coma in patients presenting with hypothermia, altered mental status, bradycardia, and other nonspecific symptoms, particularly in those with a history of hypothyroidism. Early diagnosis and prompt treatment are crucial in improving outcomes and reducing mortality even in very older patients in myxedema coma. Even though the recommended treatment is iv thyroid hormone therapy, in low resource setups where the iv form is not available oral thyroxine is also effective in improving patient outcomes.

Consent

Informed consent was taken from the patient for the publication of the case report.

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