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Case Report DOI: https://doi.org/10.3329/jacedb.v1i1.78365

Journal of Association of Clinical Endocrinologist and Diabetologist of Bangladesh ISSN (Online): 2959-8176
January 2022, Vol. 1, No. 1, pp. 31-34 ISSN (Print): 2958-0307

Two cases of severe hypothyroidism: myxedema coma

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Abstract

Myxedema coma is a severe life-threatening form of decompensated hypothyroidism which is associated with a high mortality rate. It is characterized by altered mental status, hypothermia, hypotension, hyponatremia, and hypoventilation. Infections and discontinuation of thyroid supplements are the major precipitating factors. Here, two cases of severe hypothyroidism are described. Although they were neither in 'coma' nor represent typical 'myxedema', both had altered mental status and lethargy along with typical sign-symptoms of hypothyroidism. Morbidity and mortality from myxedema coma are frequently due to a missed or delayed diagnosis. Early disease diagnosis and intensive supportive care have reduced the mortality rate. However, as the disease is rare and unrecognized, evidence-based treatment of myxedema has not yet been established in many countries. *[J Assoc Clin Endocrinol Diabetol Bangladesh, January 2022; 1 (1): 31-34]*

Keywords: Hypothyroidism, Myxedema, Pericardial effusion, Hypothermia

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Introduction

Hypothyroidism is a condition involving low thyroid hormone levels which can result in dysfunctions in heart rate, body temperature, and metabolism. The most common causes of hypothyroidism in the United States and worldwide are autoimmune thyroiditis and iodine deficiency, respectively.[1-2] Other possible causes are medication-induced, pituitary dysfunction, iatrogenic, infectious, postpartum, infiltrative, or congenital. Congenital hypothyroidism is rare due to universal newborn screening and early intervention with levothyroxine. If untreated, the condition presents with intellectual disability, stunted growth, and abnormal bone growth.[1] Myxedema coma is a life-threatening condition resulting from severe deficiency of thyroid hormone due to delay in diagnosis and treatment. It may also be precipitated by infection, trauma, or surgery.[4] A diagnosis of myxedema coma should be suspected in a patient with coma or altered mental status who has hypothermia, hyponatremia, and/or hypercapnia.[2] However, most patients do not present with myxedema or coma and altered mental status.[3] Due to the life-threatening severity of myxedema coma, treatment with thyroid hormone should begin while awaiting laboratory confirmation. The patient should be admitted to the ICU with intensive pulmonary and cardiovascular support.

Case description:

Case -1

A 35-year-old Bangladeshi male residing in Italy visited the outpatient department of Asgar Ali Hospital, Dhaka with complaints of severe generalized weakness, sleepiness, weight gain, feeling of cold even in summer, constipation, voice changes, and forgetfulness for the last 8 months.

On clinical examination, his body mass index (BMI) was 33.42 kg/m^2, blood pressure 110/70 mm of Hg, pulse rate 66 beats/min, body temperature 36.5°C, and oxygen saturation 93%. He was mentally alert but a bit drowsy and looking dull. He had dry skin, slight pitting edema, a puffy face especially the eyelids, and a hoarse voice [Figure-1]. He was unable to stand from a squatting position which denotes proximal myopathy. He had a moderate diffuse goiter and delayed relaxation of ankle jerk.

Laboratory findings are shown in table-I. Complete blood count, hemoglobin A1c (HbA1c), albumin creatinine ratio (ACR), aspartate aminotransferase (AST), tissue transglutaminase (tTG), calcium, phosphate, albumin, sodium, potassium, and Troponin-I were normal. Autoimmune hepatic markers were negative, and TCO_2 was 36 mmol/L (22-30).

Ultrasonography of abdomen revealed hepatomegaly with grade 3 hepatic steatosis, and bilateral renal parenchymal changes; Fibroscan revealed only

Table-I: Investigation profile of the patients

Investigations	1st patient		2nd patient		Normal range
	Initial	follow-up	Initial	follow-up	
TSH (mIU/L)	>100	5.2	62.8	1.99	0.4-4
Free T4 (ng/dL)	<0.7	0.92	0.8	1.48	0.9-2.3
Anti-TPO (IU/mL	>1000	-	>1000	-	-
Cortisol (mcg/dL)	10	-	11	-	3.7-19
Creatinine (mg/dL)	2.00	1.17	1.00	-	0.66-1.30
Uric acid (mg/dL)	10.3	6.0	7.0	-	3.5-8.5
ALT (U/L)	112	-	-	-	up to 50
Total cholesterol (mg/dL)	325	-	278	-	<200
HDL (mg/dL)	46	-	37	-	>40
LDL (mg/dL)	162	-	139	-	30
TG (mg/dL)	884	-	290	-	<150

TSH: thyroid-stimulating hormone Anti-TPO: Anti-Thyroid peroxidase
ALT: Alanine transaminase HDL high-density lipoprotein
LDL: low-density lipoprotein TG: triglyceride

steatosis without fibrosis while ECG showed sinus bradycardia [Figure-2]. Color Doppler echocardiography revealed moderate circumferential pericardial effusion (13 mm), left-ventricular ejection fraction (LVEF): 60%, hypertrophic obstructive cardiomyopathy (HOCM), left ventricular outflow tract (LVOT) gradient 35 mm at rest, and severe concentric left ventricular hypertrophy (LVH).

Figure-1: Physical appearance of the patient (Case-1)

After evaluation by an Endocrinologist, Cardiologist, Hepatologist, and Nephrologist, the patient was diagnosed as myxedema coma, moderate pericardial effusion, HOCM, non-alcoholic fatty liver disease (NAFLD) grade-3, chronic kidney disease (CKD), hyperuricemia, dyslipidemia, and obesity,

He was treated with levothyroxine 150 mcg daily, bisoprolol fumarate 5 mg once daily (OD), rosuvastatin 10 mg OD, ciprofibrate 100 mg OD, and febuxostat 40 mg OD, omega 3 fatty acid 1g twice daily, vitamin E capsule 400 mg OD.

He was followed up after 2 months. Follow-up investigations are also shown in Table-I. His pericardial effusion completely resolved without any intervention. ECG showed normal sinus rhythm.

Case-2

A 51-year-old Bangladeshi male residing in Saudi Arabia visited the outpatient department of Asgar Ali Hospital, Dhaka with complaints of severe generalized weakness, sleepiness, weight gain, feeling of cold even in summer, constipation, and hoarseness of voice.

On clinical examination, his BMI was 30.29 kg/m^2, blood pressure 100/60 mm of Hg, pulse rate 57 beats/min, body temperature 36.5°C, and oxygen saturation 96%. He was mentally alert but a bit drowsy and looking dull. He had dry skin, slight pitting

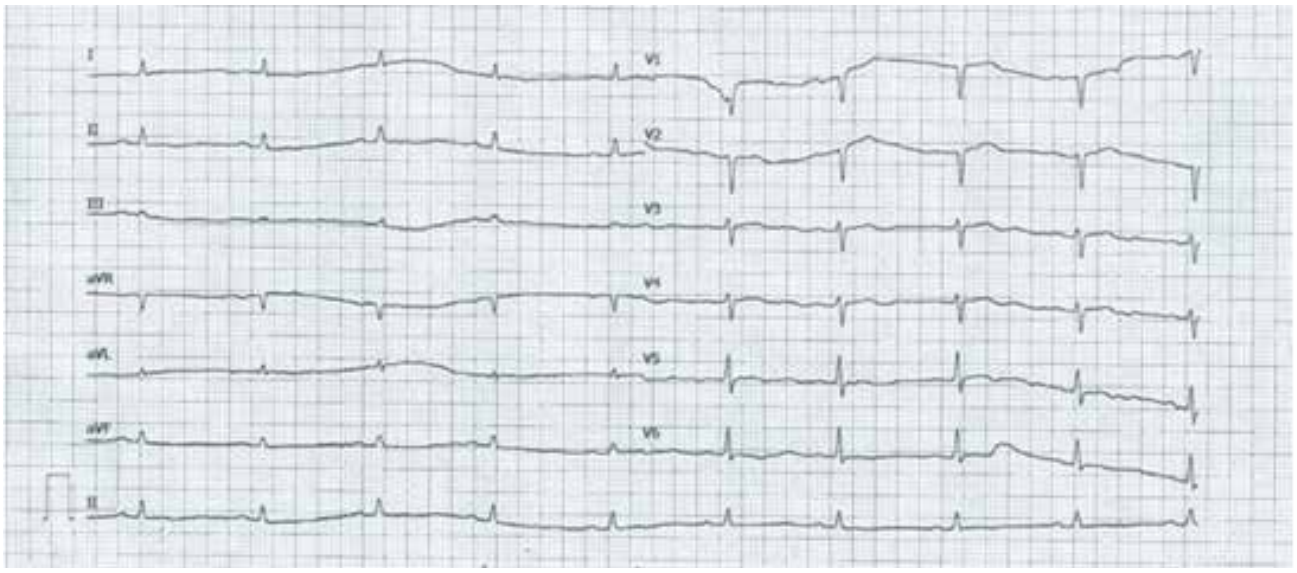

Figure-2: ECG of the patient showing sinus bradycardia (Case-1)

edema, and a puffy face, especially the eyelids. He was unable to stand from a squatting position denoting proximal myopathy. He had a moderate diffuse goiter and delayed relaxation of ankle jerk.

Laboratory findings are shown in Table-I. Complete blood count, HbA1c, ACR, liver function tests, calcium, phosphate, albumin, sodium, and potassium were normal. TCO_2 was 34 mmol/L (22-30 mmol/L). Ultrasonography of abdomen was normal, ECG revealed sinus bradycardia while Color Doppler echocardiography revealed minimal pericardial effusion, LVEF 60%. He was diagnosed with myxedema coma, minimal pericardial effusion, dyslipidemia, and obesity.

He was treated with oral levothyroxine 150 mcg daily, and rosuvastatin 10 mg daily. He was followed up after 2 months [Table-I]. Lipid profile became normal. Pericardial effusion completely resolved without any intervention. ECG showed normal sinus rhythm.

Discussion

The term myxedema coma is a misnomer as neither coma nor myxedema is required for diagnosis. There are no clear diagnostic criteria for the condition, however, patients typically present with altered mental status, confusion, and lethargy.[4] Besides, hypotension, hyponatremia, hypoglycemia, bradycardia, hypoventilation, hypercapnia, and anemia may also be found. Furthermore, hypothyroidism patients may have congestive heart failure and pericardial effusion even without preexisting cardiac diseases.[5] A retrospective study in Japan found the mortality rate of myxedema coma to be 30%, which has declined due to prompt diagnosis and treatment.[6] Cardiovascular instability, reduced consciousness, persistent hypothermia, and sepsis all contributed to a poorer outcome.[7] There is a universal consensus that thyroid hormone replenishment is an integral part of the treatment of myxedema coma; however, the debate lies in whether to use T4 alone or in combination with T3. Thyroid replacement should be initiated as early as possible with careful attention to hypotension, fluid replacement, and steroid replacement in an intensive care facility.

According to recent studies, combined therapy is suggested with T4 (levothyroxine) and T3 (triiodothyronine) rather than T4 alone.[8-11] However rapid correction may lead to myocardial ischemia, infarction, and arrhythmias.[9] Intravenous T4 200 to 400 mcg once, followed by 50 to 100 mcg daily until the patient can take T4 orally, and an initial dose of 5 to 20 mcg intravenous T3, followed by 2.5 to 10 mcg every 8 hours at the same time are recommended. T3 can be discontinued if clinical improvement and the patients are stable. Studies have shown that replacement of thyroid hormone through Ryle's tube with a loading dose and maintenance therapy is as efficacious as intravenous therapy. In many countries, T3 is not available and oral therapy with T4 can be used effectively without major significant differences in outcomes.[12]

Intravenous administration of levothyroxine is the best option due to the lower risk of enteric absorption which is inherent to the syndrome. Other supportive measures

including treatment in the intensive care unit, cardiovascular and ventilator support, passive rewarming, treatment of any concurrent infection, and correction of electrolyte imbalance are very important in the treatment of this threatening illness.

Our patients had almost all the clinical features of myxedema coma except coma which doesn't preclude the diagnosis. Although hyperuricemia in our first case is not associated with myxedema coma and might be explained by the metabolic syndrome. Delayed diagnosis in both cases reflects poor health care facilities they received abroad.

Despite the reported efficacy of triiodothyronine at low doses in conjunction with levothyroxine, our patients received only levothyroxine alone because triiodothyronine is not available in our country and they continued to improve clinically and biochemically with levothyroxine alone.

Meticulous clinical observation of the patient's status is critical to recognize the treatment's efficacy. If the patient still fails to improve, triiodothyronine should be added at low doses. This approach is especially important in the treatment of elderly patients and those with underlying cardiac or respiratory conditions. We believe the judicious use of triiodothyronine can avoid catastrophic outcomes and improve the mortality rate of myxedema coma.[5-7]

Conclusions

Due to the widespread use of screening tests for thyroid dysfunctions and hence the early diagnosis even at the subclinical state, myxedema coma has become rare in Western countries. However, in developing countries, recognition of this entity is delayed by its slow onset, lack of awareness among physicians and patients, and absence of cost-effective guidelines to screen for subclinical thyroid diseases. So high index of suspicion is mandatory to avoid grave consequences.

Acknowledgement

None

Conflict Of Interest

The authors have no conflicts of interest to disclose

Financial Disclosure

The author(s) received no specific funding for this work.

Data Availability

Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

Written informed consent was obtained from all the study participants. All methods were performed in accordance with the relevant guidelines and regulations.

How to cite this article: Basak RC, Basak M. Two cases of severe hypothyroidism: myxedema coma. J Assoc Clin Endocrinol Diabetol Bangladesh, 2022; 1 (1): 31-34

Publication History

Received on: 7 October 2021

Accepted on: 24 December 2021

Published on: 1 January 2022

References

1. Worth C, Hird B, Tetlow L, Wright N, Patel L, Banerjee I. Thyroid scintigraphy differentiates subtypes of congenital hypothyroidism. Arch Dis Child. 2021, 106:77-79.
2. Kwaku MP, Burman KD. Myxedema coma. J Intensive Care Med. 2007, 22:224-231.
3. Wall CR. Myxedema coma: diagnosis and treatment. Am Fam Physician. 2000, 62:2485-2490.
4. Rodriguez I, Fluiters E, Perez-Mendez L, Luna R, Páramo C, García-Mayor RV. Factors associated with mortality of patients with myxoedema coma: prospective study in 11 cases treated in a single institution. J Endocrinol. 2004, 180:50.
5. Chaudhari, V. Gangadharan, T. Forrest. Heart failure presenting as myxedema coma: case report and review article. Tennessee Medicine. 2014,107:39–41,
6. Ono Y, Ono S, Yasunaga H, Matsui H, Fushimi K, Tanaka Y. Clinical characteristics and outcomes of myxedema coma: analysis of a national inpatient database in Japan. J Epidemiol. 2017, 27:117-22.
7. Dutta Pinaki, Bhansali Anil, Masoodi Shriq, Bhadada Sanjay, Sharma Navneet, Rajput Rajesh. Predictors of outcome in myxoedema coma: a study from a tertiary care centre. Critical Care. 2008;12(1).
8. Biondi B, Wartofsky L. Combination treatment with T4 and T3: toward personalized replacement therapy in hypothyroidism?. J Clin Endocrinol Metab. 2012, 97:2256-2271.
9. McCulloch W, Price P, Hinds CJ, Wass JA. Effects of low dose oral triiodothyronine in myxoedema coma. Intensive Care Med. 1985, 11:259-262.
10. Ueda K, Kiyota A, Tsuchida M, Okazaki M, Ozaki N. Successful treatment of myxedema coma with a combination of levothyroxine and liothyronine. Endocr J, 2019 18-0469.
11. Shakir MKM, Brooks DI, McAninch EA, Fonseca TL, Mai VQM, Bianco AC, et al. Comparative Effectiveness of Levothyroxine, Desiccated thyroid extract, and levothyroxine+Liothyronine in hypothyroidism. J Clin Endocrinol Metab 2021, 106:e4400–13.
12. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, et al. Guidelines for the treatment of hypothyroidism: Prepared by the American Thyroid Association Task Force on Thyroid Hormone Replacement. Thyroid 2014, 24:1670–751.