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Case Report

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Myxedema Coma, a Forgotten Clinical Entity in Medical Emergency: a Case Report

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ABSTRACT

Myxedema coma is a rare endocrine emergency and is often forgotten differential diagnosis in a patient with altered mental status. Despite its rare occurrence, clinical suspicion is needed in any patient with clinical features and biochemical evidence of hypothyroidism. It ideally requires intravenous levothyroxine for treatment. We report a case of 76 year old male, nonsmoker, non-alcohol consumer who presented with acute onset altered mental status and stupor, which was attributed to untreated severe hypothyroidism and successfully treated with oral levothyroxine despite unavailability of its intravenous form.

Keywords

Myxedema coma; levothyroxine; drowsiness; thyroid function test

INTRODUCTION

Myxedema coma is a clinical presentation of decompensated severe hypothyroidism, often triggered by infection, untreated hypothyroidism and abrupt discontinuation of treatment.[1] Early clinical detection and easy access to laboratory service may have contributed to decreasing the incidence of myxedema coma. However, some individuals with hypothyroidism infrequently present with life threatening severe hypothyroidism in emergency services as a true endocrine emergency.

We report a case of 76 year old male with presentation of acute onset altered mental status and stupor, which was attributed to untreated severe hypothyroidism and successfully treated with oral levothyroxine despite unavailability of its intravenous form.

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CASE PRESENTATION

A 76 years old non-smoker and non-alcohol consumer male with diabetes mellitus presented to emergency department with history of acute onset of altered mental status and stupor since morning. There was no preceding history of fever, headache, and drug intake. However, he had preceding lethargy, and malaise for months. On examination, he was drowsy. On Glassgow Coma Scale, it was E2M5V2. His Blood pressure was 110/70 mmHg, pulse rate 60 bpm, saturation of 88% in room air, respiratory rate 16/minute. His systemic examination was unremarkable.

Major differential diagnoses of such a patient with altered sensorium are meningoencephalitis, dyselectrolytemia, hypercapnia, uremia, stroke and hypoglycemia in emergency.

His Lab investigations are tabulated in Table 1. It showed hemogram with total leukocyte count 10,200/cumm, hemoglobin 9.4 g/dl, Neutrophil 86%, Lymphocyte 14% and platelet 234000/cumm. His random blood glucose was 236 mg/dl. His renal function showed sodium 107 meq/L, potassium 4.4 meq/L, serum creatinine 2 mg/dl, Urea 68 mg/dl. His arterial blood gas analysis showed PH 7.32, Pco_2 65mm Hg, HCO_3 30 meq/L. His urine routine examination showed albuminuria 2+. Absence of fever, headache, vomiting and meningeal signs of irritation did not favor meningoencephalitis. His urea level was not high enough to cause severe degree of altered sensorium. Carbon dioxide retention could be a cause of altered sensorium. However, degree of hypercapnia i.e. PCO_2 65 mmHg alone did not explain stupor in the patient.

His disorientation was attributed to severe hyponatremia, which was corrected gradually less than 8 meq/L in 24 hours with 3% Sodium chloride. His Sodium level reached 128 meq/L in three days. However, there was no significant clinical improvement in sensorium. On evaluation of hyponatremia, thyroid function test showed TSH >100 uIu/ml, free T4 0.12 0 ng/dl, free T 0.73 pg/ml at the time of admission. His serial thyroid function test is tabulated in Table 2. His MRI brain did not show any significant abnormalities, which ruled out any stroke. Myxedema coma diagnostic score was 60 points, including stupor (20 points), Hyponatremia (10 points), hypoxemia (10 points), hypercarbia (10 points), decreased GFR (10 points), which was highly suggestive of myxedema coma.

He was also treated with thyroxine 200 mcg via nasogastric tube from the next day of admission in ICU as intravenous thyroxine is not available in our country.

He had good clinical improvement on the third day with orientation to person. He had full clinical improvement with normal sensorium in a week. He had regular follow up in medicine

Table 1. Lab parameters of the patient with myxedema at presentation

Lab investigation	Patient's lab values	Reference range
Hemoglobin	9.4 g/dl	13-18 g/dL
Total Leucocyte count	10,200/cumm	4000-11000/cumm
Neutrophil	84%	45-70 %
Lymphocyte	14%	20-40%
Eosinophil	2%	1-6%
Platelet	234000/cumm	150000-400000/cumm
Random blood glucose	236 mg/dL	60-110 mg/dl
Sodium	107 meq/L	135-155 meq/L
Potassium	4.4 meq/l	3.5-4.5 meq/L
Urea	68 mg/dl	10-50 mg/dl
Serum creatinine	2 mg/dL	0.6-1.4 mg/dl
Arterial blood gas analysis	P^H 7.32	7.35-7.45
	PCO_2: 65 mmHg	35-45 mmHg
	HCO_3: 30 meq/L	22-26 meq/L

Table 2. Thyroid function test values

TFT	At admission	Day 10	Day 16	6 weeks	Reference
fT3	0.73 pg/ml	1.40 pg/ml	1.63 pg/ml	3.2 pg/ml	2.0-4.2 pg/ml
fT4	0.12 ng/dl	1.07 ng/dl	0.80 ng/dl	1.2 ng/dl	0.89-1.72 ng/dl
TSH	>100 ulu/ml	46.00 ulu/ml	21.00ulu/ml	4.1ulu/ml	0.3-4.5 ulu/ml

out-patient department. He was euthyroid. His mental state was good. His thyroid function test showed TSH 4.1 ulu/ml, free T4 1.2 ng/dl, free T3 3.2 pg/ml at six weeks.

DISCUSSION

Myxedema coma is a rare unrecognized medical emergency of untreated hypothyroidism. The gender preponderance is observed with two third cases occurring in females. The overall proportion of in hospital mortality in myxedema coma reaches as high as 29.5%.[2] Recognition of myxedema coma requires high degree of clinical suspicion.

Myxedema is a clinical diagnosis supported by biochemical evidence of hypothyroidism, hyponatremia, and carbon dioxide retention as seen in our case. Distinctive signs of hypothyroidism include generalized weakness, non-pitting edema, cold intolerance, sluggish mental and physical functions, dry skin, facial puffiness, and a husky voice. [2, 3] Common clinical manifestations include hypothermia (66%) followed by hemodynamic failure (57%) and coma (52%) in severe hypothyroidism. Thyroiditis and thyroidectomy are the primary causes of severe hypothyroidism accounting for 29% and 19%, respectively. The unrecognized cases of severe hypothyroidism account for 54% cases before admission to Intensive Care Unit. The leading triggers for severe hypothyroidism are discontinuation of levothyroxine (28%), sepsis (15%) and amiodarone (11%).[5]

Hyponatremia is a laboratory clue to suspicion of hypothyroidism. Severe hyponatremia is attributed to decreased capacity of free water excretion in severe hypothyroidism. So, measuring TSH levels in evaluation of hyponatremia is considered mandatory.[6]

The initial treatment involves promptly administering appropriate doses of thyroid hormone, either intravenously with levothyroxine or via a nasogastric tube with L-triiodothyronine.[3] Delay in return of warmth in skin, bradycardia, older age and high serum level of free T3 are determinants of fatal outcome in myxedema despite treatment.[7]

Despite the variation in oral absorption in critically ill cases, clinical resolution of oral Levothyroxine is satisfactory even in ileus. The exact dose and duration of oral Levothyroxine is still unconcluded. However, levothyroxine is used in higher initial doses in myxedema coma with watchful monitoring of cardiac effects. Levothyroxine remains mainstay treatment in severe hypothyroidism[8,9]. We treated our patient with initial dose of levothyroxine 200 mcg via nasogastric tube. Intravenous levothyroxine is unavailable in Nepal. We had a good clinical improvement with oral levothyroxine via nasogastric tube even in absence of intravenous liothyronine.

CONCLUSION

Myxedema should be a differential diagnosis in any patient with corroborative history and clinical features of hypothyroidism presenting as altered mental status. Prompt recognition and early treatment with only oral levothyroxine can be beneficial in case of unavailability of intravenous liothyronine.

CONSENT

Written informed consent was taken from the patient's guardian for this report publication.

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CONFLICT OF INTEREST

The author(s) declare that they do not have any conflict of interest with respect to the research, authorship, and/or publication of this article.

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