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CASE SERIES

Case Series of Myxedema Coma Diagnosed in Patients Presenting to ICU in A Tertiary Care Medical College Hospital

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Abstract Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia, hyponatremia, and other symptoms as a result of slowing of function in multiple organs. Myxedema coma is an extreme complication of hypothyroidism in which patients exhibit multiple organ abnormalities and progressive mental deterioration. The term myxedema is often used interchangeably with hypothyroidism and myxedema coma Myxedema coma is a medical emergency with high mortality rate around 40 percent. Myxedema coma can occur as a result of severe long standing hypothyroidism,or can be precipitated by an acute event like infection, trauma, surgery, opiods, anaesthesia etc We report a series of cases presenting to ICU with myxedema coma. All cases in our series, presented to the ICU, needing mechanical ventilation. Patient 1 ,was a 54 year old male patient,presenting with history of fever and altered sensorium .Patient 2 was a 78 year old male patient presenting with fever,cough and breathlessness.Patient 3,was a 40 year old female,who underwent emergency laparotomy and had to be shifted to ICU in view of refractory hypotension .In all the 3 cases ,there were co existing conditions like Sepsis,COPD,Cardiac issues which made diagnosis of coexistent myxedema to be missed initially.All 3 patients were diagnosed as myxedema coma,co existing with other clinical conditions in view of high TSH values ,and low T4 values .All the 3 patients received combined treatment with T4 (levothyroxine) and T3 (liothyronine), ,corticosteroids,and other supportive medications .All of them improved clinically and were shifted out of ICU,and later discharged home.

Keywords: Hypothyroidism, medical emergency, hypothermia, ICU

INTRODUCTION

Myxedema coma was first described by Ord in 1879, and few subsequent reports followed that of Summers in 1953 until 1963 when Forester reviewed seventy seven cases [1]. An approximate incidence of 0.22 cases per million inhabitants/year is estimated[2].It is more common in females,and in older age groups .Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia,hyponatremia, and other symptoms as a result of slowing of function in multiple organs [3].As a result of widespread availability of thyroid stimulating hormone assays, it is now a rare presentation.[4,5].

In 2021, Yoshinaka A et al reported a case of sudden cardiac arrest associated with myxedema coma due to undiagnosed hypothyroidism. In his case report , a female patient was clinically diagnosed with myxedema coma, which was the cause of cardiac arrest. She was treated with thyroid hormone and hydrocortisone, resulting in improvement in her general condition, except for the neurological dysfunction [6]. In 2017, Takamura A et al, reported a case of myxedema coma complicated by renal failure. He described a rare case of myxedema coma with atypical features associated with renal failure. The patient presented with mild respiratory failure and a reduced level of consciousness without hypotension [7].

In the indian scenario,case reports from a tertiary care ICU have highlighted the importance of diagnosing myxedema coma,especially in the geriatric age group [8]In critical care set up diagnosis of myxedema coma is complicated by confounding factors like sepsis,renal failure,liver failure,multiple drug interactions etc.Diagnosis of myxedema coma can be easily missed ,leading to increase in mortality and morbidity[9].Myxedema coma ,although carrying a high mortality can be easily diagnosed with blood investigations,and can be easily treated

We report a series of cases presenting to ICU with myxedema coma

Patient 1

54 year old male patient, presented to the ICU in shock with history of fever for 3 days and altered sensorium for a day . He was hypertensive, on tab olmesrtan 40 mg OD, obese with a BMI of 36.No significant family history or past history elicited. He was hypotensive despite fluid resuscitation.He was drowsy ,but arousable ,with no neurological deficits and had bilateral pedal edema . Lab investigations revealed raised WBC count,hyponatremia.Urine routine showed plenty of pus cells suggestive of Urinary tract infection. 12 lead ECG

revealed bradycardia with a HR of 60 (Normal PR interval). CT brain was normal. 2D echo done on the day of admission showed concentric Left Venticular Hypertrophy,Normal systolic function with an EF of 50%, and normal diastolic function ..A provisional diagnosis of septic encephalopathy with shock was made. He was started on broad spectrum antibiotics, cefaperazone sulbactum 1.5 gm BD iv after sending cultures, fluid resuscitation with 30 ml/kg Ringer Lactate .

He was started on vasopressors (noradrenaline 2 microgram/minute),nasogastric feeds,DVT prophylaxis,3% NS,and other supportive medications . Over the next 24 hrs,his clinical condition worsened needing mechanical ventilation and stepping up of vasopressors(increased noradrenaline to 4 mcg/min) Work up for hyponatremia was done on the second day of admission .TSH was reported as more than 100 mU/L. T4 Levels were low- 0.5 mcg/dl. Anti TPO antibodies was in normal levels.Endocrinology advice was sought immediately and he was initiated on steroids ,Inj. hydrocortisone 100mg TID for 2 days,tapered to 50 mg tid ,oral T3 (liothyronine) 10 mcg stat ,10 mcg bd ,oral thyronorm (T4) tablets (200 to 300mcg, increased to 400 mcg over 3 days).He was hypothermic(96F), managed with warm iv fluids, and warmer (Baer hugger).He was continued on antibiotics,RT feeds ,vasopressor support (noradrenaline 4 mcg/min) ,anticoagulation(Inj clexane 60 mg s/c OD) and other supportive medications .On the 7th day of admission,he was weaned off vasopressors.His sensorium remained low, and an elective tracheostomy was contemplated .MRI brain done on 8th day was normal. After 12 days on ventilator ,oral levothyroxine ,oral T3(liothyronine) and steroid supplementation, his sensorium gradually improved,he was alert ,obeying commands and tolerated spontaneous breathing trial. He was successfully extubated .He required intermittent Bilevel Positive Airway Pressure in view of persistent type 2 respiratory failure .His condition gradually improved, he was initiated on oral feeds, mobilization. He was shifted to wards after 18 days in ICU. He was later discharged home after a week with oral thyronorm.

Patient 2

78 year old male patient presented to the ICU with 3 days history of fever, cough and breathlessness. He was a known case of chronic obstructive lung disease. On examination his HR was 45 with prolonged PR interval, maintaining blood pressures. He was comatose and in type 2 respiratory failure with non-pitting pedal edema.

He was intubated and put on mechanical ventilation. Family gave history of constipation. Serum electrolytes was normal.TSH was 60.Anti TPO antibodies was in normal levels. T4 levels were low 2 mcg/dl.He was started on antibiotics, inj. hydrocortisone 100mg TID, oral levothyroxine and oral T3 . His condition gradually improved, he was weaned off mechanical ventilation and extubated .He was later shifted to step down ICU, wards and discharged .

Patient 3

40 year old female patient was taken up for emergency laparotomy for duodenal perforation closure under general anaesthesia .She had refractory hypotension during the procedure, and was managed with fluid resuscitation and vasopressors. She was found to be hypothermic, and was managed with warmer(baer hugger),warm iv fluids .She could not be extubated because of delayed recovery from anaesthesia and was shifted to icu and electively ventilated .CT brain done was normal.Her lab investigations revealed TSH > 100,T4 0.8 mcg/dl .she was continued on mechanical ventilation ,her cardiac enzymes were elevated.

2d echo showed hypokinetic changes in apical segment, with apical ballooning .Cardiology opinion taken, and started on antiplatelet ,and anticoagulation. She underwent CAG which was normal .Hence a diagnosis of tako -tsubo cardiomyopathy was made. She was started on inj hydrocortisone, oral liothyronine, oral levothyroxine,beta blocker,ACE inhibitors,diuretics ,antiplatelets,anticoagulation .She was extubated, and shifted to wards and later discharged home.

TABLE 1: THYROID FUNCTION TESTS

	At diagnosis			72 hrs post initiation of treatment		7 days post initiation of treatment		15 DAYS post initiation of treatment	
	TSH (mIU/L)	Free T4 (ng/dl)	Anti TPO antibodies (IU/ml)	TSH (mIU/L)	Free T4 (ng/dl)	TSH (mIU/L)	Free T4 (ng/dl)	TSH (mIU/L	Free T4 (ng/dl)
PATIENT 1	>100	0.001	5	92	0.08	30	0.4	9	1.1
PATIENT 2	60	0.10	8	57	0.16	40	0.2	11	0.6
PATIENT 3	>100	0.01	3	96	0.2	56	0.37	12	1.6

TABLE 2: ROUTINE INVESTIGATIONS

	Patient 1	Patient 2	Patient 3
Hb	11gm/dl	10gm/dl	9.8gm/dl
Total Leucocyte count	18000cells /mm^3	12000cells /mm^3	22000cells /mm^3
platelets	1.25lakh/microlitre	2.5 lakh/microlitre	3.72lakh/microlitre
Urea	28mg/dl	30mg/dl	42mg/dl
Creatinine	1.3 mg/dl	0.8mg/dl	1.2mg/dl
Serum Sodium	126meq/l	127meq/l	132meq/l
Serum potassium	5.4meq/l	3.9meq/l	4.2 meq/l
Serum chloride	105meq/l	111meq/l	110meq/litre
Liver Function Tests	within normal limits	within normal limits	within normal limits
Neuro imaging	Normal	Normal	Normal

DISCUSSION

Hypothyroidism is a condition where serum thyroid hormone levels are lower than the reference range. The typical normal reference range for Thyroid-Stimulating Hormone in non-pregnant adults is generally considered to be 0.4 to 4.0 milli-international units per liter

The interpretation of a result is highly dependent on individual factors, and the "normal" range is not absolute.

1. Variations in Reference Ranges

Lab-Specific: The exact normal range can vary slightly between different laboratories due to the different assays (testing methods) they use You should always refer to the range provided by the testing laboratory on your report.

Optimal Range: Some clinicians, particularly endocrinologists, suggest a narrower, more optimal range for the general population, such as 0.5 to 2.5 mIU/L, because levels even in the high normal range (3.0 to 4.0 mIU/L) may sometimes indicate the earliest stages of thyroid dysfunction.

Myxedema coma is defined as severe hypothyroidism leading to decreased mental status, hypothermia, and other symptoms related to slowing of function in multiple organs

Myxedema coma can occur as a result of severe long standing hypothyroidism,or can be precipitated by an acute event like infection,trauma,surgery,opiods [10] etc .The classical features of myxedema coma are decreased mental status,hypothermia, hyponatremia, hypoventilation,and delayed recovery from anaesthesia [11,12,13]

Neurologic manifestations: Most patients do not present with coma, but with varying degrees of altered consciousness [14,15].Some patients present with psychotic features ,also called myxedema madness ,if left untreated can progress to coma [14,16.17].Sometimes,focal or generalized seizures can occur due to co existent hyponatremia[9,18,19]

Hypothermia: Due to decrease in thermogenesis that accompanies the decrease in metabolism, severity of hypothermia is related to mortality [20,21]
Hypoventilation: Central depression of ventilatory drive with decreased responsiveness to hypoxia and hypercapnia[21]

Hypoglycaemia: Hypothyroidism alone, or concurrent adrenal insufficiency can cause hypoglycemia [22]
Cardiovascular abnormalities: Diastolic hypertension, reduced cardiac output, bradycardia, decreased myocardial contractility, pericardial effusion, low voltage complexes. All of the cardiac abnormalities are reversible with thyroid hormone therapy[23]

Diagnosis: Diagnosis to be considered in a patient with depressed mental status, along with hypothermia, hyponatremia, hypoglycaemia and or hypercapnia[24]
TSH: Serum TSH may be high in primary hypothyroidism, or low, normal, or slightly high in central hypothyroidism .Most patients with myxedema coma have primary hypothyroidism

T4: Very low [14]
Patients with central hypothyroidism may have associated hypopituitarism, and secondary adrenal insufficiency [25,26]

Treatment:
If myxedema coma is suspected, treatment is to be initiated without waiting for laboratory confirmation .It is an endocrine emergency with a very high mortality rate [23]

Thyroid hormone:
Combined treatment with T4 (levothyroxine) and T3 (liothyronine) is preferred, than T4 alone .It is preferable to give both because biologic activity of T3 is greater and its onset of action more rapid than T4 [9]
Dosing: T4, T3 to be given intravenously as a slow bolus, when available because of erratic gastric absorption [4,27]

T4 is administered in an initial dose of 200 to 400 mcg intravenously ,followed by daily iv doses of 50 to 100

mcg ,until patient can take T4 orally .Lower range of dosing is preferred in elderly patients, those at risk of developing myocardial ischaemia.The target is to raise T4 level by 2 to 4 mcg /dl [14]

T3 is given intravenously at the same time, initial dose of 5 to 20 mcg, followed by 2.5 to 10 mcg every 8 hrs.[28,12]

Monitoring: serum T4 or free T4 ,and T3 should be measured every one to 2 days ,and to avoid high T3 levels .Clinical, biochemical improvement are usually evident within a week .Ideally serum TSH levels should fall by 50 percent every week, who receive full replacement dose. If the TSH levels fail to drop, it is an indication of insufficient dosing.[14].Free T4 is more reliable

Once clinical improvement is attained, patient can be treated with oral T4 alone.

Glucocorticoids: Till coexisting adrenal insufficiency ruled out, patient must receive stress doses of glucocorticoids [14]

Supportive measures: Managing in an ICU, mechanical ventilation, fluid and electrolyte balance, antibiotics, anticoagulation, preventing hypoglycaemia, hypothermia etc. Passive rewarming with a blanket is preferred over active rewarming.

PROGNOSIS:

Myxedema is an endocrine emergency, and carries a mortality rate between 30 to 50 percent. Older age, cardiac complications, reduced consciousness, need for mechanical ventilation, persistent hypothermia, and sepsis were predictive of mortality [29,30]

CONCLUSION

In critical care setting patients present with multiple ongoing issues.Hypothyroidism can go unnoticed ,and can be easily missed .Hence this case series throws light on coexistent myxedema coma ,which can worsen the clinical outcome if not noticed and addressed Diagnosis of myxedema coma is to be considered in a patient with depressed mental status ,along with hypothermia,hyponatremia,hypoglycaemia and or hypercapnia In critical care setting,patients present to us with comorbidities,on multiple medications and with concurrent,multiple life threatening issues.Hypothyroidism If myxedema coma is suspected ,treatment is to be intiated without waiting for laboratory confirmation ,as it is an endocrine emergency with a very high mortality rate .

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