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CASE REPORT 2

Myxoedema coma with pancytopenia and diagnostic challenges

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Abstract

Myxoedema coma is a rare, life-threatening complication of severe hypothyroidism especially in geriatric population. We report a diagnostically challenging case of a 65-year-old man presenting with altered consciousness, bradycardia, anasarca, and hypothermia, consistent with myxoedema coma. Initial management included high-dose enteral thyroxine and IV hydrocortisone, but clinical improvement was minimal. Investigations revealed pancytopenia, deranged liver enzymes, and high anti-TPO antibodies. Severe constipation, meningoencephalitis, hepatic encephalopathy, Addisonian crisis, and other systemic illnesses were considered and excluded. The poor response to oral thyroxine suggested malabsorption, likely due to intestinal mucosal edema. Lack of IV thyroxine availability in a resource-limited setting posed a therapeutic challenge. The patient's condition deteriorated with cardiac arrest, pleuropericardial effusions, and ventilator-associated pneumonia, ultimately leading to death. This case highlights the atypical presentation, diagnostic dilemmas, and management challenges of myxoedema coma in low-resource environments. Early recognition and aggressive management are crucial to improving outcomes in this often-misdiagnosed endocrine emergency.

Keywords: Myxoedema coma, Pancytopenia, hepatic encephalopathy

Introduction

Myxoedema coma is an uncommon manifestation of severe form of untreated hypothyroidism [1]. This case represents a unique and diagnostically challenging presentation of myxoedema coma. While classical features such as hypothermia, bradycardia, altered mental status, and anasarca were present, the additional findings of pancytopenia and deranged liver enzymes are uncommon in hypothyroidism and significantly complicated the diagnostic process. During the management; poor response to oral thyroxine in the absence of IV thyroxine in a resource poor setting made the management a difficulty which we had to tackle. Only few cases of myxoedema presenting with pancytopenia in similar fashion are reported in the literature, yet the combination with liver derangement is quiet rare [1,2, 3, 4].

Case presentation

A 65-year-old man with no prior medical evaluation presented to the Emergency Treatment Unit (ETU) with a reduced level of consciousness. On arrival, he had a patent airway, SpO_2 of 94% on room air, heart rate of 40 bpm, blood pressure of 100/70 mmHg, respiratory rate of 24/min, and a Glasgow Coma Scale (GCS) of E3V3M4. He was hypothermic with cold peripheries, anasarca, Had madarosis, hypothyroidism facies and dry, coarse skin. An ECG (Figure 1) confirmed sinus bradycardia and bedside

Echocardiogram showed pericardial effusion with poor LV function (Figure 2). Capillary blood sugar was 120 mg/dL, and body temperature was 36°F.

The patient was initially managed as a case of myxoedema coma. He was rewarmed and given oral thyroxine 400 mcg via a nasogastric (NG) tube. Intravenous hydrocortisone 200 mg was administered stat after obtaining blood for basic investigations, cultures, random cortisol, and venous blood gas (VBG) analysis.

A retrospective history obtained from a family member revealed that he had previously been healthy but had developed gradually worsening edema, altered sensorium, and poor oral intake over the preceding week. The patient had been living alone without the care of his children, and no further history was available.

The patient was then transferred to the medical ward. Following the initial assessment, he developed bradycardic cardiac arrest. He was resuscitated with IV atropine and IV adrenaline and was subsequently intubated. During intubation, significant laryngeal edema was noted, which made securing the airway difficult. After successful intubation, he was transferred to the ICU. Basic investigations were reviewed and are summarized in Table 1.

Investigations revealed pancytopenia, deranged liver enzymes, low free T4 (fT4), high TSH levels, and normal random cortisol. High-dose thyroxine was continued via the NG tube, but improvement in GCS was very slow. Due to the poor clinical response, thyroid malabsorption secondary to probable gut oedema was suspected. Pre- and post-thyroxine fT4 levels were performed to assess absorption and response. Pancytopenia and liver enzyme abnormalities prompted further evaluation to exclude hepatic encephalopathy, especially as the patient had severe constipation and had not opened his bowels for three days. This required a stat dose of a Kleen enema, maximum-dose syrup lactulose, and increased oral fluids. Hepatic encephalopathy was excluded based on a normal portal venous Doppler study and absence of splenomegaly. The etiology of pancytopenia remained inconclusive.

In the ICU, pleuro-pericardial effusion was detected along with persistent severe bradycardia. The patient was started on diuretics and inotropic support with adrenaline, which helped achieve hemodynamic stability. With ongoing management, the patient showed slow improvement, and tracheostomy was planned due to the anticipated prolonged GCS recovery.

On day 12 of ICU admission, the patient developed severe ventilator-associated pneumonia (Figure 2). Despite the infection, he remained afebrile (Fever Chart in Figure 3) due to persistent hypothermia associated with myxoedema. Unfortunately, he succumbed to overwhelming sepsis two days later despite maximal supportive care.

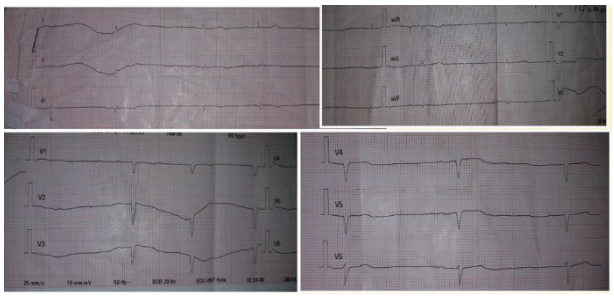

Figure 1. ECG.

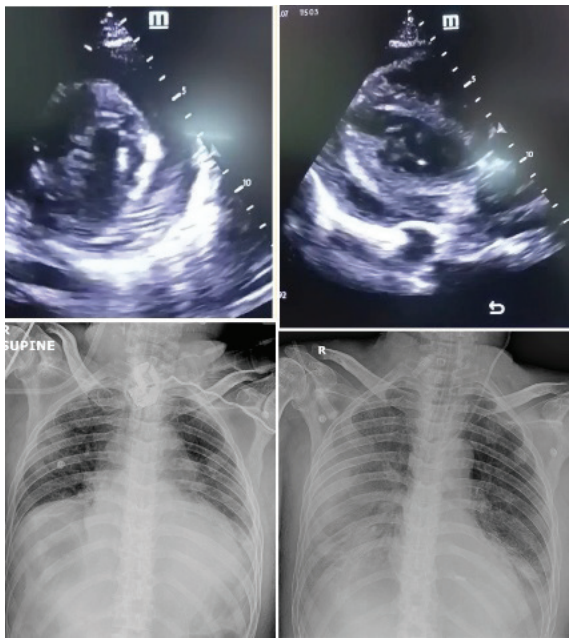

Figure 2. Initial Bed Side Point of Care Echocardiogram (Figure 2 A) & Post ICU admission Chest X rays (Figure 2B) (Initial one (Right) & Latest one before death (Left)).

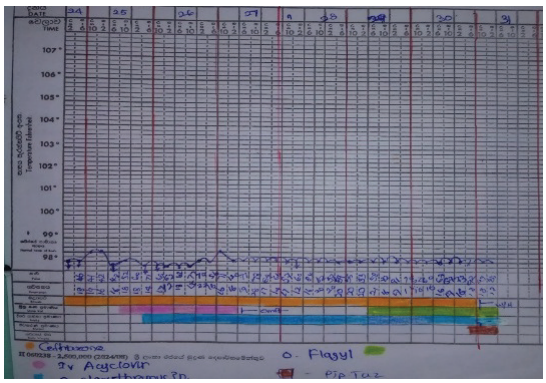

Figure 3. Fever chart.

Table 1. Laboratory and Diagnostic Investigations

Investigation	Result	Reference Range
TSH	54.9 mIU/L	0.4–4.0 mIU/L
Free T4 (fT4)	<0.88 ng/dL	0.93–1.7 ng/dL
Free T3 (fT3)	<0.6 pg/mL	2.0–4.4 pg/mL
Sodium (Na^+)	127 mmol/L	135–145 mmol/L
Potassium (K^+)	5.0 mmol/L	3.5–5.1 mmol/L
Calcium (Total)	2.09 mmol/L	2.1–2.6 mmol/L
Phosphate (PO_4^{3-})	1.09 mmol/L	0.8–1.45 mmol/L
Magnesium (Mg^{2+})	0.6 mmol/L	0.7–1.0 mmol/L
White Blood Cells (WBC)	1.9–2.7 $\times 10^9$/L	4.0–11.0 $\times 10^9$/L
Neutrophils	85.8%	50-70%
Lymphocytes	11.7%	10-20%
Monocytes	1.7%	1-3%
Eosinophils	0.2%	0.5-1.5%
Hemoglobin (Hb)	7.9–8.1 g/dL	13.0–17.0 g/dL
Hematocrit (HCT)	22%	40–50%
MCV	101 fL	80–100 fL
MCH	33 pg	27–33 pg
MCHC	35 g/dL	32–36 g/dL
Platelet count	110–88 $\times 10^9$/L	150–400 $\times 10^9$/L
Serum Creatinine	75.9 µmol/L	60–110 µmol/L
CRP	1.4 mg/L	<5 mg/L
ESR	10 mm/hr	<20 mm/hr
Troponin I (Initial)	11 ng/L	<100 ng/L
Troponin I (Repeat Post cardiac arrest resuscitation)	478 ng/L	
Creatinine Phospo Kinase (CPK)	1100 U/L	30–200 U/L
Serum Cortisol (Random)	430 nmol/L	140–690 nmol/L
Anti-TPO Antibodies	629 IU/mL	<5.61 IU/mL
Total Protein	6.2	6-8 g/dl
Serum Albumin	3.2	3.5-5.0 g/dl
pH	6.999	7.35–7.45
PaO_2	66.2 mmHg	80–100 mmHg
HCO_3^-	24.2 mmol/L	22–28 mmol/L
Lactate	5.5 mmol/L	<2.0 mmol/L
CSF WBC	3 cells/µL	0–5 cells/µL
CSF Lymphocytes	100%	
CSF Protein	93 mg/dL	15–45 mg/dL
CSF Glucose	96 mg/dL	45–80 mg/dL
CSF LDH	512.8 U/L	<250 U/L
Blood & Urine Cultures	No growth	
NCCT Brain	Normal	
Bedside Echocardiogram	EF 60%, thin pericardial effusion, no RWMA	
Ultrasound Abdomen	No portal HTN, Grade 2 fatty liver, ascites Suggest to exclude CKD ± CLCD	
EEG (Electro Encephalogram)	Moderate encephalopathy	
Blood Film	Normochromic normocytic anemia, acanthocytes, neutrophilic predominance, giant platelets	

Discussion

Our case was managed as myxoedema coma though the presentation was atypical while the investigations are concerned. These atypical laboratory findings initially raised the possibility of hepatic encephalopathy, Addisonian crisis, polyglandular autoimmune syndrome, hypopituitarism, severe malnutrition, meningoencephalitis and other systemic illnesses, especially in the context of severe constipation and altered sensorium which are frequently encountered and oftentimes overlooked geriatric issues. However, the investigation panel, the absence of hepatosplenomegaly, normal portal venous Doppler, and preserved serum cortisol levels helped to exclude these differentials.

The differentiation of hepatic encephalopathy is important as it share similar patterns in clinical presentation and liver investigations and important to differentiate for better therapeutic outcomes [5]. Our patient had; very severe constipation which managed with Kleen enema, maximum doses of lactulose syrup and bisacodyl oral tablets. After exclusion of common causes; constipation was considered to be the precipitating cause of the myxoedema coma on an undiagnosed long standing hypothyroidism.

Clinically both conditions present similarly with altered sensorium, features of generalized fluid overload, anaemia, high LFTs and low sodium, hypoglycemia, deranged LFTs, triphasic EEG patterns. These similarities may masquerade and mislead clinicians [5]. Similar masquerading presentation has been documented in the literature [5,6].

Patient was evaluated for the etiology of pancytopenia as well because it's not common among the myxoedema cases. The pathophysiology of pancytopenia in myxoedema is not well understood [2]. thyroid hormones role in in erythropoiesis is well understood. Generally, it seems that hypothyroidism causes hypoplasia in all myeloid cell lineages and hyperthyroidism result in hyperplasia [4]. Though the hypothyroidism; can rarely cause pancytopenia; the evaluation needs many disorders to be ruled out making diagnosis a conundrum. The high LDH and CPK levels noted could be explained by increased muscle membrane permeability and overall showed metabolism [3]. The acanthocytes noted in the blood picture though seen commonly seen in liver dysfunction its seen rarely in severe hypothyroidism too [7]. The high TPO antibodies traced later possibly indicate autoimmune hypothyroidism due to Hashimoto's thyroiditis.

In the initial presentation of our patient; it was difficult to differentiate Addisonian crisis and myxoedema and he was managed as both and high dose of IV hydrocortisone given before the blood taken for serum cortisol made the diagnostic dilemma worse due to the unavailability of spot random cortisol in a resource poor setting and the inotrope dependence of our patient made omitting hydrocortisone a challenge. There are rarely reported cases of concurrence of both conditions and it carries high mortality even with proper treatment [8,9].

Respiratory failure and cardiac stunning may complicate the picture as in our case [8]. Another problem we encountered was the unavailability of IV levothyroxine in Sri Lanka and poor response for the oral levothyroxine. Treatment refractory hypothyroidism was considered and malabsorption was considered as the most likely etiology. Due to the early demise of the patient; exact etiology of malabsorption couldnt be ruled out. In literature various causes like coeliac disease, maldigestion related to hypochlorhydria, Proton Pump Inhibitor therapy, intraluminal factors are described. When levothyroxine needed more than 2.5 μg/kg daily, treatment-resistant hypothyroidism should be considered [10]. In our case the poor response to enteral thyroxine further suggested impaired gastrointestinal absorption, likely due to mucosal edema, which has also been rarely documented [11]. Again, geriatric patients are at higher risk of malabsorption due to age related gut changes as well. In our case this was tackle with very high difficulty by changing the diet completely to liquid-based diet and avoiding semisolid feed and increasing dosing frequency of oral levothyroxine via nasogastric tube in three divided doses with monitoring fT4 levels closely initially pre and post dose and later every other day basis. Though patient succumbed due to a severe ventilator associated pneumonia; patient improved from the myxoedema perspective. Frailty and immunosenescence likely contributed to the fatal trajectory [12].

Conclusion

Myxoedema coma is a fatal endocrine emergency with high mortality, that needs quick recognition and treatment. In certain scenarios it can be masquerading and make diagnosis difficult. Our case sheds light on tackling a similar complicated scenario in a resource poor setting.

List of Abbreviations: IV: Intra Venous, Anti-TPO: Anti Thyroid Peroxidase, ICU: Intensive Care Unit, LDH: Lactate Dehydrogenase, HTN: hypertension, CKD: Chronic Kidney Disease, CLCD: Chronic Liver Cell Disease.

Declarations

Ethics approval and consent to participate: Informed consent was obtained from the patient for the publication of this case report and any accompanying images. All ethical considerations were adhered to in accordance with institutional guidelines. No personal data has been displayed; all the information were de-identified.

Consent for publication: All authors unanimously decided and approved the final manuscript and consented for publication.

Data availability statement: The data supporting the findings of this case report are available from the corresponding author, upon reasonable request. Due to patient confidentiality and privacy concerns, detailed data may be shared in a de-identified format where appropriate and with relevant ethical approval.

Authors' Conflict of Interest Statement: The authors declare no conflicts of interest.

Author Contribution Roles: Conceptualization: NMMR, Case Management: NMMR, NA, IKJ, KADW Manuscript Drafting: NMMR, JFS Critical Review and Final Approval: NA, IJK.

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