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Myxoedema Coma Precipitated by Diabetic Ketoacidosis and Septic Shock: a Case Report

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Abstract—Myxoedema coma (or myxoedema crisis) is a severe and potentially fatal form of decompensated hypothyroidism with an underlying cause. A low index of suspicion and a search for triggering factors should be the first step in dealing with the condition at an early stage. Myxoedema coma should be suspected in patients who present with hypothermia, altered mental status, and coma, even if hypothyroidism has not previously been recognized. The symptoms of hypothyroidism decompensation may mistakenly be attributed to its precipitating factors, which may include sepsis, cardiac failure, myocardial infarction, cerebrovascular accident, anaesthetic, sedatives, antidepressant medications, or metabolic and electrolyte abnormalities. Here, we present a case of myxoedema coma with two major underlying precipitating causes: septic shock and diabetic ketoacidosis.

Index Terms—DKA, Hypothyroidism, Myxedema Coma, Septic Shock.

I. INTRODUCTION

Myxoedema coma is a severe and life-threatening form of decompensated hypothyroidism with an underlying precipitating factor. It can present with altered mental status, hypothermia, bradycardia, respiratory failure, and hyponatremia. Mortality rates may be as high as 25–60%, even with the best possible treatment [1-3].

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A low index of suspicion and a search for precipitating factors should be the initial step in dealing with myxoedema coma at an early stage [4]. Triggers may include poor medication compliance, thyroidectomy, acute events (including infection and cardiac infection among other stresses), and medical treatments that cause loss of thyroid homeostasis [5]. The mainstay of treatment includes hydrocortisone, intravenous liothyronine, levothyroxine, electrolyte correction, supportive therapies, and treating any underlying precipitant such as infection, surgery, or trauma [1,6].

Suspected myxoedema coma cases should receive intravenous hydrocortisone while awaiting laboratory results, as any delay can be life-threatening. The condition should ideally be managed in the intensive care unit (ICU) with pulmonary and cardiovascular support [7,8]. Here we report a case of myxoedema coma presenting with two main underlying precipitating factors: septic shock and diabetic ketoacidosis (DKA).

I. CASE PRESENTATION

The patient was a 42-year-old male with a history of Down syndrome, epilepsy, and type 1 diabetes mellitus (T1DM). One year prior, he had required a 28-day admission to the ICU where he received treatment for pneumonia, ultimately recovering without complications. In the current admission, he was transferred from a primary hospital to the emergency department (ED) on October 18, 2023 having been intubated for septic shock secondary to pneumonia and DKA. Prior to his transfer, the patient had experienced three days of fever, cough, sore throat, decreased level of consciousness, and loss of appetite.

Upon arrival at the Emergency Department, the patient was intubated for mechanical ventilation using a 7.5 mm endotracheal tube (ETT)

positioned at a depth of 22 cm. Ventilation was provided in the assist-control volume control (AC/VC) mode, with the following parameters: fraction of inspired oxygen (FiO₂) 100%, tidal volume (V_t) 480 mL, positive end-expiratory pressure (PEEP) 8 cmH₂O, and a respiratory rate of 20 breaths per minute. Oxygen saturation (SpO₂) was recorded at 92%, and bilateral inspiratory crackles were noted.

The patient was afebrile, with a blood pressure of 118/80 mm Hg, heart rate of 126 beats per minute, and random blood glucose level of 581 mg/dl. Venous cannulae (20 gauge) were secured, and the patient received ongoing sedation with fentanyl (100 mcg per hour) as well as norepinephrine (30 mcg per hour). He was following a protocol for the treatment of DKA, involving regular doses of insulin (7 IU), as well as potassium chloride at a dose of 15 meq in 500 ml fluid, administered intravenously at a rate of 250 ml per hour. Additionally, he had an indwelling urinary catheter (size F12 silicone), connected to a collection bag. However, despite this setup, only minimal urinary output was observed.

Management in the Emergency Department:

The patient was connected to a cardiac monitor and a bolus of 1000 ml normal saline was administered. Intravenous medications were also administered, including a single dose of 5 mg midazolam followed by a continuous infusion at 5 mg per hour, and an increase in fentanyl infusion to 200 mcg per hour. Hydrocortisone 50 mg and meropenem 1000 mg were also administered.

A right femoral central venous catheter (size 7) was inserted at a depth of 20 cm; vasopressin infusion was initiated at a rate of 0.04 IU per minute; and a 65-cm nasogastric tube (size F14) was inserted and confirmed via auscultation to be patent and intact. A urinary output of 200 ml was noted, which was amber in colour.

Chest X-ray revealed bilateral infiltrations, while urine dipstick analysis showed significant levels of protein, glucose, ketones, and blood. Table 1 illustrates the patient’s venous blood gases along with the chemistry panel.

The patient was admitted for DKA and septic shock secondary to pneumonia and showed no

Table 1. Venous blood gases and chemistry panel

Test	Result
Venous Blood Gases	
- pH	7.12
- Partial pressure of carbon dioxide (pCO ₂)	47 mmHg
- Bicarbonate (HCO ₃)	15 mmol/L
- Lactate	4 mmol/L
- Base deficit	-16 mmol/L
CBC	
- White blood cells (WBC)	8 x 10 ³ /μL
- Haemoglobin (Hgb)	14 g/dL
Chemistry Panel	
- Creatinine	235 U/L
- Urea	8 mmol/L
- Potassium	3.8 mmol/L
- Sodium	144 mmol/L
Urine Dipstick	
- Protein	++
- Glucose	+++
- Ketones	+++
- Blood	++

improvement within the first 48 hours of admission. Severe metabolic acidosis, hyperkalaemia, oliguria, and pre-renal acute kidney injury (AKI) secondary to septic shock all manifested simultaneously. Continuous renal replacement therapy (CRRT), anti-hyperkalaemia treatment, and furosemide infusion were initiated. Despite these interventions, the patient remained hypotensive. An arterial line was inserted and vasopressors were maximized, aiming for a 1-L negative fluid balance.

At that point, myxoedema coma was the main concern, especially when the thyroid-stimulating hormone (TSH) level came back very high; thus, T3 and T4 tests were ordered. The patient was not known to have suffered from hypothyroidism before (his history was taken from his family). Nonetheless, his thyroid function results confirmed the suspicion of myxoedema coma, which explains the masking of expected hyperthermia due to septic shock (the patient showed a temperature of 36°C). On that day, IV thyroxine was started at 200 mcg stat, and then reduced to 150 mcg daily.

The following day, the patient began to experience oro-nasal bleeding during suctioning, with secretions appearing moderately thick and bloody. Additionally, thrombocytopenia was observed, with a platelet count of $17 \times 10^3/\mu\text{L}$, along with acute liver failure indicated by elevated levels of aspartate aminotransferase (AST) at 4223 U/L and alanine aminotransferase (ALT) at 4248 U/L.

To address these complications, the patient received nebulized tranexamic acid, along with a transfusion of 4 units of fresh frozen plasma (FFP) and 6 units of platelets (PLT). Intravenous N-acetylcysteine was initiated at a dose of 12000 mg over 1 hour, followed by 4000 mg over 4 hours, and then 8000 mg over 16 hours.

Echocardiography revealed fair left ventricular systolic function with an ejection fraction (EF) of 50 %. Cardiac dimensions were within normal limits, valves were normal, and the right side size and function were also normal. No masses or thrombi were observed, and both the interatrial septum (IAS) and interventricular

septum (IVS) were intact. The pericardium appeared normal.

A few days later, the patient showed improvement in terms of mechanical ventilation parameters and vasopressor requirements. He was extubated and received dexamethasone 8 mg twice daily for 24 hours. He showed improvement, with a Glasgow Coma Scale (GCS) score of 13 out of 15. He continued to improve over the following days; after 2 days he was placed on a nasal cannula at 4 litres per minute (LPM).

The patient received a loading dose of intravenous levothyroxine (LT4) 200 mcg, followed by a maintenance dose of 150 mcg, initiated on October 19, 2023. His TSH levels decreased from 216 upon initiation, to 41 on 25 October, 6 days later. This drop of >50 % in less than one week indicated an appropriate replacement. His T4 levels showed improvement, but remained low.

At that time, the patient became tachypnoeic and desaturated while on a non-rebreather mask at 15 L, maintaining SpO₂ at 91 % with a GCS score of 15/15. CT chest revealed bronchiectasis. There was a rapid progression of bronchiectasis cavitation on the right side with new consolidation in the basal left side, likely attributed to Pseudomonas or Staphylococcus infection. Emphasis was placed on obtaining sputum and secretion cultures with antibiograms, IV colistin and IV imipenem for treatment.

15 days post-admission, the patient developed drowsiness, hypotension, increased tachypnoea, and desaturation despite the non-rebreather mask and BiPAP. His platelet count was $17 \times 10^3/\mu\text{L}$. Consequently, he was reintubated on the same day and received a transfusion of 6 units of platelets. Although a bronchoscopy had been planned, it was not performed due to the patient's deteriorating condition.

25 days post-admission, the patient was in pre-arrest status with refractory septic shock (despite norepinephrine and vasopressin infusion at high doses) and multi-organ failure. He arrested at 4:48 AM; as the rhythm showed bradycardia and pulseless electrical activity

(PEA), cardiopulmonary resuscitation (CPR) performed and return of spontaneous circulation (ROSC) was achieved after 12 minutes.

Venous blood gas post-ROSC showed pH 7.0, pCO₂ 90 mmHg, HCO₃ 16 mmol/L, lactate 22 mmol/L, and potassium 6.5 mmol/L. Electrocardiogram (ECG) revealed no acute dynamic changes. Nonetheless, two hours later, the patient arrested again. CPR was performed for 40 minutes with no response, and death was declared.

II. DISCUSSION

We report a case of myxoedema coma caused by DKA and septic shock in a 42-year-old individual with a medical history including Down syndrome, epilepsy, and type 1 diabetes mellitus, who at the ED intubated for septic shock caused by pneumonia and DKA. This case is particularly unique in terms of the clinical course. To the best of our knowledge, there has been no mention in the literature of myxoedema coma triggered by DKA and septic shock. Previous small case-series investigations found the mortality rate for myxoedema coma to be 36% (4 out of 11 patients), 52% (12 out of 23 patients), and 25% (2 out of 8 patients) [9]. Due to the condition's rarity, the clinical presentation, prognosis, and outcome were poorly recognized.

Analysis of a nationwide inpatient database in Japan revealed that, out of 19 million inpatients, 149 were diagnosed with myxoedema coma, with a mean age of 77 years and two-thirds of them women [9]. A retrospective multicentre cohort study, by Simon Bourcier et al., of critically ill severe hypothyroidism patients in intensive care, found that the clinical presentation was hypothermia in 66% of cases, hemodynamic failure in 57%, and coma in 52%. Furthermore, 54% were undiagnosed with hypothyroidism prior to ICU admission. Severe hypothyroidism can be triggered by the termination of levothyroxine medication, sepsis, or amiodarone-related hypothyroidism. Although it is difficult to suspect hypothyroidism in the emergency department, especially in the presence of coexisting critical conditions such as sepsis, DKA, and

hypotension, diagnostic and management measures should be implemented as soon as possible for patients presenting with known hypothyroidism, thyroidectomy, hypothermia, and altered mental status, as mortality is high even with the best treatment [10].

Annually, an estimated 7.9 million infants are born with a serious birth defect caused by genetic or largely genetic origin. The most frequent severe aneuploid condition at birth is Down syndrome, which was initially reported in 1866 by the British physician Dr. John Langdon H. Down [11]. Despite their small and overweight look, most children with Down syndrome have normal thyroid function, while adults with Down syndrome tend to be more prone to both hypothyroidism and hyperthyroidism. Hypothyroidism may be caused by the late development of thyroid autoantibodies; testing thyroid-stimulating hormone (TSH) and blood thyroxine (T₄) levels, as well as triiodothyronine (T₃) and T₃ resin uptake, can assist in diagnosing the condition as well as weight loss, lethargy, heat sensitivity, and skin abnormalities.

In the present case, our patient had no history of hypothyroidism, despite Down syndrome patients having a high prevalence for thyroid diseases such as thyroiditis and congenital hypothyroidism [12,13]. He was suffering from pneumonia complicated by septic shock; there was no fever mentioned in the history prior to his arrival at the hospital, and his temperature during hospitalization was 36-37°C. We believe this was due to myxoedema and the absence of thyroid hormone thermogenesis.

Typical presentation of patients with myxoedema may include hypothermia, dry skin, hoarseness, and altered mental status; however, the absence of these symptoms does not rule out the diagnosis [14]. It is worth noting that sepsis or DKA may be atypical manifestations of myxoedema, and the condition should thus be considered in patients presenting with these symptoms. In the present case, early recognition and treatment, starting with supportive measures and levothyroxine, helped with the initial improvement of our patient's condition.

As his TSH levels dropped, the patient started to stabilize vitally, regained consciousness, and was extubated. Another important tool in the management of severe hypothyroidism is glucocorticoids [15,16], due to the risk of adrenal insufficiency that accompanies this condition. In our case, a dose of hydrocortisone was given in the ED.

In summary, myxoedema can easily be misdiagnosed, especially when symptoms are masked by other critical conditions — in the case of our patient, septic shock complicated by DKA. Thus, suspicion should be raised even where there is no known history of hypothyroidism. While the mortality of myxoedema remains high, early recognition and treatment can play a major role in improving patient outcomes.

III. CONCLUSION

Myxedema coma should be suspected in patients presenting with hypothermia, altered mental status, and coma, even where there is no history of hypothyroidism, and particularly in those at greater risk for the condition, such as, in our case, people with Down syndrome. Many factors can trigger myxedema, including cold exposure, sepsis, DKA, and cardiogenic shock. Those at high risk should be investigated, given that mortality is high even with the appropriate management.

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