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A Rare but Fatal Cause of Seizure: Myxedema Coma

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BACKGROUND AND CLINICAL IMPORTANCE

Myxedema coma is a medical emergency with high mortality rate. As widespread availability to thyroid stimulating hormone (TSH) testing has become available, the diagnosis has become rare. Nonetheless, prompt diagnosis and treatment are essential to decreasing morbidity and mortality. Seizure is a rare, but highly fatal complication of myxedema coma. This case describes a medically complex patient who presented after new-onset seizure and was diagnosed with myxedema coma. This case illustrates the importance of having a wide diagnostic lens when thinking beyond the initial diagnosis. In this case, digging deeper to explore the cause of the patient's seizure uncovered a rare, medically emergent condition that potentially saved the patient's life.

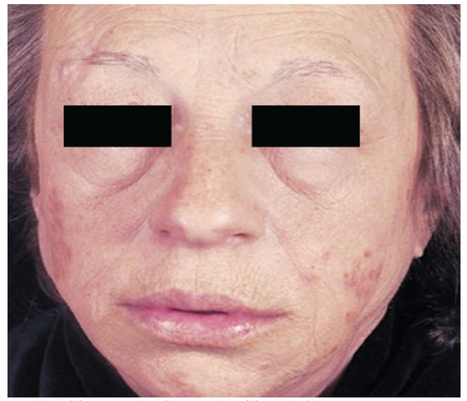
DESCRIPTION

A 57-year-old patient with past medical history including but not limited to cerebrovascular accident (CVA), coronary artery disease (CAD), type 2 diabetes, hypothyroidism, and tobacco abuse presented to the emergency department by emergency medical services (EMS) with new-onset seizure. Upon arrival to the emergency department (ED) the patient was minimally responsive and began seizing a second time. The patient required IV levetiracetam and lorazepam, was intubated and admitted to the critical care unit. Her course was further complicated by hypotension and bradycardia, requiring vasopressor support. Upon speaking with a family member, it was noted that the patient's cognitive status had been progressively declining since the time of a CVA about two years prior and considerably more in recent weeks to months. The family member believed it was highly likely that the patient was non-compliant with medications.

RESULTS

Physical examination revealed a morbidly obese patient with periorbital edema, a distinct puffiness to the face and lips, purplish discoloration of the skin, and non-pitting edema in the extremities. Respirations were slowed with prolonged expiratory phase. Glasgow Coma Score (GCS) of 10 was noted, reflecting the patient's best motor response, but this was variable. There was no withdrawal to noxious stimuli in the extremities and gag reflex was absent. The patient did, however open eyes to sternal rub for brief periods and intermittently follow simple one-step commands such as hand grip. Computed tomography of the head showed chronic ischemia in the right frontal lobe with compensatory dilatation of the right lateral ventricle and was negative for acute intracranial pathology. Initial complete blood count was unrevealing. Complete metabolic panel was remarkable for elevated creatinine 1.52 mg/dL, glucose 362 mg/dL, and alkaline phosphatase 134 U/L. Thyroid stimulating hormone was markedly elevated at 169.17 µU/mL and free thyroid hormones were undetectable. Arterial blood gas values were as follows: pH 7.21, PaCO2 84 mmHg, PaO2 89 mmHg, and HCO-3 of 32 mmol/L (performed on four liters nasal cannula oxygen). Endocrinology and Neurology were urgently consulted, and the patient was started on IV levothyroxine and liothyronine for myxedema coma and IV hydrocortisone for the possibility of concomitant adrenal insufficiency. Neurology recommended assessing for status epilepticus with electroencephalogram (EEG) monitoring and continuing IV levetiracetam. It was determined that the patient's seizure was potentially secondary to myxedema coma, precipitated by long-standing non-compliance for treatment of hypothyroidism.

FIGURE 1. FACIAL FEATURES OF MYXEDEMA COMA



Source: Sylvia C. McKean, John J. Ross, Daniel D. Dressler, Danielle B. Scheurer: Principles and Practice of Hospital Medicine, Second Edition, www.accessmedicine.com

DISCUSSION AND CONCLUSION

The mortality rate of myxedema coma is as high as 30-60% despite adequate treatment, making prompt diagnosis and treatment critical[1]. The most common precipitating factors are infection/sepsis and cold exposure, and many cases occur in undiagnosed hypothyroid patients[1,2]. Seizures are recognized as a rare manifestation of myxedema with high mortality rate[1,3]. Providers should consider myxedema coma in any patient with altered mentation and hypothermia, hyponatremia, and/or hypercapnia[4]. Additionally, though rare, due to the high mortality of patients with seizure secondary to myxedema coma reported in the literature, myxedema coma should be explored in the differential for a critically ill patient presenting with new seizures.

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