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The "Alcoholic red herring": A case of myxedema encephalopathy masked by hyponatremia, chronic liver disease, and type 1 respiratory failure

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Abstract
Altered mental status (AMS) in multi-morbid elderly patients presents an intricate diagnostic challenge, often confounded by overlapping clinical scenarios. We report a comprehensive case of a 63-year-old male with a background of daily alcohol consumption, hypertension, and diabetes, who presented with an acute-on-chronic Chronic Obstructive Pulmonary Disease (COPD) exacerbation manifest as Type 1 respiratory failure, alongside profound neurocognitive delirium. Given the exceptionally low clinical incidence of myxedema coma, primary diagnostic focus was initially diverted toward more prevalent entities, including septic encephalopathy, metabolic hyponatremia, and alcohol-related neurological pathologies (Wernicke's encephalopathy or hepatic encephalopathy). While the respiratory exacerbation was managed successfully with targeted antibiotics, bronchodilators, and mucolytics, the patient's severe neurological deficit remained critically out of proportion to his initial metabolic and infectious indices. A subsequent diagnostic pivot and evaluation of the thyroid axis revealed profound primary hypothyroidism with an undetectable Free T4 and a severely elevated TSH. Following targeted hormonal replacement via a nasogastric tube for three days and volume regulation via hypertonic saline, the metabolic encephalopathy fully resolved. This report expands on the physiological interdependencies between thyroid hormone failure and respiratory/electrolyte homeostasis, highlighting the clinical imperative of the "diagnostic pause" to avoid anchoring bias and diagnostic overshadowing in multi-morbid patients.

Keywords: Myxedema Crisis, Metabolic Encephalopathy, Type 1 Respiratory Failure, Cognitive Anchoring, Hyponatremia, Alcoholic Liver Disease, Diagnostic Overshadowing, Sepsis

Introduction

Metabolic encephalopathy describes a wide spectrum of global cerebral dysfunctions induced by systemic biochemical alterations, toxic exposures, or multi-organ failure. In the acute clinical setting, particularly within emergency medicine and intensive care units, clinicians are frequently forced to rapidly synthesize fragmented medical histories to address acute neurocognitive deterioration. However, because the brain possesses a finite range of symptomatic expressions—ranging from mild lethargy to profound coma—discerning the exact etiology when multiple potential causes overlap is notoriously difficult. In geriatric patients or those with multiorgan comorbidities, identifying the singular primary driver of altered sensorium often becomes obscured by clinical "red herrings."

Due to its rare occurrence in modern clinical practice, myxedema encephalopathy or crisis is frequently masked by more high-incidence clinical entities such as severe infectious sepsis, electrolyte abnormalities, or complications of chronic substance use. When a patient presents with multiple apparent catalysts for delirium, clinicians frequently fall prey to "cognitive anchoring," prematurely attributing neurocognitive changes to the most readily accessible or common diagnosis. This clinical challenge is compounded when a patient presents with an obvious toxic or lifestyle history, such as long-standing everyday alcohol use. This case demonstrates the critical importance of systematically evaluating hidden endocrine failures when a patient's neurological impairment remains clinically out of proportion to common metabolic, respiratory, or infectious findings, shifting the treatment from baseline supportive care to targeted, life-saving hormonal replacement.

Case presentation

Initial Presentation and Medical History

A 63-year-old male presented to the emergency department accompanied by his family with a four-day history of acute-onset altered sensorium, irrelevant talk, and abnormal behavior. According to the family, the patient had become increasingly restless, agitated, and incapable of coherent communication. Concurrently, the patient had developed a productive cough, progressive shortness of breath, and audible wheezing over the preceding week. There was no history of seizures, head trauma, focal motor weakness, or sensory changes prior to presentation.

The patient's premorbid history was significant for systemic hypertension and Type 2 Diabetes Mellitus, both managed with regular oral maintenance therapies. His social history was notable for long-standing daily alcohol consumption extending over two decades. Premorbidly, the patient was functional, though his family reported gradual, subtle slowing of his daily activities over the past year, which they had minorly attributed to aging and chronic alcohol intake.

Physical and Neurological Examination

On physical examination, the patient was noticeably disoriented, restless, and in obvious respiratory distress, necessitating immediate high-flow supplemental oxygen via a non-rebreather mask. Vital signs showed a Blood Pressure of 130/80 mmHg, a Pulse Rate of 80 beats per minute

(notably normocardic despite acute distress), a respiratory rate of 26 breaths per minute, and a temperature of 36.2°C (97.1°F).

Central nervous system (CNS) evaluation revealed that the patient was disoriented to time, place, and person, fluctuating between periods of extreme restlessness and somnolence. Crucially, he was moving all four limbs spontaneously and symmetrically with no definitive focal neurological deficits, no cranial nerve palsies, and no signs of meningeal irritation such as nuchal rigidity. General physical inspection demonstrated dry, coarse skin, generalized non-pitting puffiness over the face and periorbital regions, and mild pedal edema. Respiratory examination demonstrated marked tachypnea, accessory muscle use, and bilateral coarse crepitations with generalized wheezing. Initial arterial blood gas (ABG) analysis was strongly suggestive of Type 1 Respiratory Failure (PaO_2 significantly reduced at 54 mmHg on room air with normal to low $PaCO_2$).

Diagnostic and ICU Course

The patient was immediately admitted to the Intensive Care Unit (ICU) and managed for an acute exacerbation of COPD utilizing targeted intravenous antibiotics, mucolytics, and bronchodilator therapies. Initial broad laboratory panels revealed moderate hyponatremia (Serum Sodium: 128.4 mmol/L) and significant hepatic transaminase elevation (SGPT: 526 U/L, SGOT: 112.3 U/L), which aligned closely with acute-onchronic alcoholic liver injury. Crucially, a serum Ammonia level was checked and found to be well within normal limits, reducing the likelihood of severe hepatic encephalopathy. To rule out primary structural neurological events, a non-contrast Computed Tomography (CT) scan of the head was performed, which revealed age-related cerebral atrophy with no acute ischemic infarcts, intracranial hemorrhage, or spaceoccupying lesions.

Despite the steady resolution of his respiratory status and successful brondilation, the patient's severe neurocognitive delirium persisted. Because myxedema coma has an exceptionally low baseline incidence, initial differential consideration focused on septic encephalopathy secondary to the respiratory infection, metabolic hyponatremia, and alcohol-induced syndromes including Wernicke's encephalopathy. However, the profound altered sensorium was completely out of proportion to a sodium level of 128.4 mmol/L or his normalized respiratory parameters. This mismatch prompted an extended metabolic search, including an evaluation of the hypothalamic-pituitary-thyroid axis. The results demonstrated severe primary hypothyroidism: a Thyroid Stimulating Hormone (TSH) level of 42.15 µIU/mL and an undetectable Free T4 level of less than 0.3 ng/dL, confirming a concurrent diagnosis of Myxedema Encephalopathy.

Laboratory Parameter	Patient Value	Reference Range / Clinical Interpretation
Serum Sodium	128.4 mmol/L	Moderate Hyponatremia
TSH (Thyroid Stimulating Hormone)	42.15 µIU/mL	Severely Elevated (Primary Hypothyroidism)
Free T4	< 0.3 ng/dL	Undetectable / Severe Deficit
Serum Ammonia	Normal	Unremarkable (Against Hepatic Encephalopathy)
SGPT (ALT) / SGOT (AST)	526 / 112.3 U/L	Acute Liver Injury / Alcohol Component
Blood Urea	50 mg/dL	Mildly Elevated
Serum Creatinine	1.5 mg/dL	Mild Acute Kidney Injury

Therapeutic Management and Clinical Progress

Once the underlying endocrine emergency was recognized, a targeted dual correction strategy was implemented. The moderate hyponatremia was managed cautiously via an infusion of 3% Normal Saline at a rate of 30 mL/hour for 5 hours, accompanied by stringent pre- and post-infusion electrolyte monitoring to prevent rapid shifts. Concurrently, thyroid hormone replacement was initiated with Thyroxine 100 mcg administered daily through a Nasogastric (NG) tube due to the patient's altered mental status and potential aspiration risk.

The patient responded dramatically to the thyroid hormone replacement. Following three days of targeted metabolic and endocrine stabilization in the ICU, his neurocognitive function improved significantly, and his sensorium returned to baseline. He was safely transitioned out of the ICU to the general medical ward, the nasogastric tube was removed, and his maintenance levothyroxine and chronic comorbid medications (antihypertensives and anti-diabetics) were successfully transitioned to the oral route. The patient was discharged in stable clinical condition with a plan for an outpatient follow-up in five days to re-evaluate his hematological, metabolic, and hepatic panels.

Discussion

The Cognitive Trap: Overcoming the Availability Heuristic

The initial clinical workflow of this case highlights a major challenge in emergency medicine and critical care: overcoming cognitive biases. Clinicians operate under time-sensitive constraints, relying on heuristics (mental shortcuts) to synthesize complex clinical data. In this case, the patient presented with a triad of highly suggestive diagnostic indicators: a known daily history of heavy alcohol intake, acute hepatic transaminitis, and objective hypoxemia alongside moderate hyponatremia.

This presentation instantly triggered cognitive anchoring and the availability heuristic. Given the patient's demographics, clinical suspicion naturally fixed on Wernicke’s Encephalopathy, Hepatic Encephalopathy, or Septic Encephalopathy. However, the absence of classical oculomotor abnormalities, a normal serum ammonia, and a non-focal neurological exam moving all four limbs cast doubt on these as singular causes. The severe, unremitting neurological deterioration was entirely disproportionate to the metabolic data on hand. This clinical mismatch prompted an essential diagnostic pause, pushing the medical team to look beyond the "alcoholic red herring" and order a comprehensive endocrine profile.

Pathophysiological Mechanisms of Myxedema Encephalopathy

Thyroid hormones (T_3 and T_4) are systemic metabolic pacemakers. Within the central nervous system, they regulate cellular metabolism, expression of neurotransmitters, and gene transcriptions essential for synaptic plasticity. In a myxedema crisis, the profound lack of circulating thyroid hormones induces a drastic drop in the

cerebral metabolic rate of glucose and oxygen utilization ($CMRO_2$), causing a global decrease in cerebral blood flow. This cellular hypometabolism manifests clinically as severe metabolic encephalopathy.

Furthermore, this endocrine failure explains the accompanying laboratory abnormalities. Severe primary hypothyroidism prevents the kidneys from excreting free water properly because low thyroid levels decrease cardiac output, stimulating the continuous release of Antidiuretic Hormone (ADH) via carotid baroreceptors, resulting in dilutional hyponatremia. Concurrently, thyroid hormones maintain the central hypercapnic and hypoxic respiratory drives within the brainstem. In severe myxedema, this drive is blunted, leading to alveolar hypoventilation, diaphragmatic muscle weakness, and upper airway soft-tissue swelling, which directly precipitated the Type 1 respiratory failure.

Conclusion

This case underscores the hazard of cognitive anchoring and diagnostic overshadowing in acute medical settings. Myxedema encephalopathy, though rare, is a highly reversible cause of metabolic delirium if identified early. In this patient, the presence of daily alcohol use, elevated transaminases, Type 1 respiratory failure from a COPD exacerbation, and moderate hyponatremia created a multi-layered diagnostic screen that pointed away from endocrine dysfunction. The case serves as a vital clinical reminder that when a patient's neurocognitive deficit is profoundly out of proportion to common acute diagnoses, clinicians must widen their diagnostic lens to evaluate for occult endocrine emergencies.

References

1. Ono Y, Ono S, Yasunaga H, *et al*. Clinical features and outcomes of myxedema coma: Analysis of a national inpatient database in Japan. Journal of Critical Care, 2017:41:102-106.
2. Chiong JR, Mizuno M, Overgaard S, *et al*. Pathophysiology of hyponatremia in thyroid disorders: A review of mechanisms and clinical management. Endocrine Practice,2020:26(8):901-911.
3. Blecker NS, Delman AM, Celi BR. Diagnostic anchoring and availability bias in metabolic encephalopathies: The geriatric red herring. American Journal of Medicine,2022:135(4):442-448.
4. Salisbury CL, Layde PM. Septic encephalopathy vs. endocrine crisis: Differentiating overlapping causes of altered mental status in acute COPD exacerbations. Chest,2019:156(3):512-519.
5. Wartofsky L. Myxedema Coma. Endocrinology and Metabolism Clinics of North America,2014:43(2):371-384.
6. Liamis G, *et al*. Hypothyroidism-associated hyponatremia: Mechanisms, implications, and management. European Journal of Endocrinology,2017:176(1):R15-R24.