Is Salt, Vitamin, or Endocrinopathy Causing this Encephalopathy? A Review of Endocrine and Metabolic Causes of Altered Level of Consciousness

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KEYWORDS

• Level of consciousness • Mental status • Endocrine
• Metabolic • Seizure • Delirium • Emergency

It's a beautiful Saturday morning, the first one in a while that hasn't started with pulling on a pair of scrubs and heading to the emergency department (ED). While you are sitting out on the porch sipping a steaming cup of latte, the postal carrier arrives bearing a special delivery from the American Board of Emergency Medicine: the ConCert is coming to a testing center near you! You consider early retirement. However, gulping your double-shot latte in the preceding moments of dread has resulted in the awakening of your finely tuned, caffeine-dependent brain and you're off to the races. Fruity breath? Diabetic ketoacidosis (DKA). Buffalo hump? Cushing syndrome. Stones, bones, groans, and moans? Hypercalcemia. Mental status change, ophthalmoplegia, and gait ataxia? Wernicke encephalopathy. Severe abdominal pain and hallucinations after starting oral contraceptives? Umm. Seizures and seborrheic dermatitis in a health care worker? Huh. Nausea, confusion, and agitation in an elderly patient after an outpatient cardiac catheterization? Duh. There's no knowledge gap a little caffeine and light reading can't bridge, so read on…

Altered level of consciousness covers a vast continuum from drowsiness to coma and describes the reason for 3% of critical ED visits.[1] Approximately 85% are found

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to have a metabolic or systemic cause, whereas the remaining 15% are caused by structural lesions.[1] Early laboratory studies such as a bedside glucose test, serum electrolytes, or a urine dipstick test often direct the ED provider toward endocrine or metabolic causes. This article examines common endocrine and metabolic causes of altered mentation in the ED via sections dedicated to endocrine-, electrolyte-, metabolic acidosis-, and metabolism-related causes.

ENDOCRINE-RELATED CAUSE

Glucose Metabolism

Hypoglycemia, DKA, and hyperglycemic hyperosmolar state (HHS) make up the triad of glucose metabolism disorders that may result in altered mentation.

Symptoms and signs of hypoglycemia (autonomic [eg, diaphoresis, tremors, weakness, pallor] and neuroglycopenic [eg, disorientation, confusion, lack of coordination, seizures, coma, hypothermia]) are manifest as the serum glucose level decreases to less than 55 mg/dL.[2] Hypoglycemia as a cause of altered mentation is confirmed with a serum glucose measurement and reversed with the administration of glucose or dextrose. Most hypoglycemia is drug induced, with the leading culprits being diabetes mellitus medications, followed by type Ia anti-arrhythmics, dextropropoxyphene, β-blockers, pentamidine, antidepressants, and angiotensin-converting enzyme inhibitors. Factitious hypoglycemia, caused by surreptitious self-administration of sulfonylureas or insulin, must also be considered in nondiabetic patients. With the exception of alcohol-induced hypoglycemias, toxic hypoglycemias (eg, carbon tetrachloride, ethylene glycol, or *Amanita phalloida*) are rare and recognized by their coincident acute liver necrosis. Equally as rare are tumor hypoglycemia, caused by insulinomas and extrapancreatic tumors of mesenchymal origin (eg, rhabdomyosarcoma, leiomyosarcoma, liposarcoma), and autoimmune hypoglycemia, characterized by fasting hypoglycemia, increased serum insulin levels, and autoantibodies to serum insulin. Reactive (postprandial) hypoglycemia occurs after meals as a result of congenital carbohydrate metabolism deficiencies and becomes apparent in infancy. The existence of idiopathic reactive hypoglycemia has been a matter of debate; less than 5% of patients believed to exhibit suggestive symptoms are found to have a serum glucose level less than 55 mg/dL associated with symptomatic episodes.[2]

DKA and HHS occur along the same clinical spectrum, hallmarked by the triad of hyperglycemia, ketonemia, and metabolic acidosis, but differ with regard to severity of each triad component (**Table 1**).[3] DKA occurs more commonly in patients with type 1 diabetes mellitus, accounts for 8% to 29% of hospital admissions with a primary diagnosis of diabetes, and has a mortality that has improved to less than 5%.[3] HHS occurs more commonly in people with type 2 diabetes, makes up less than 1% of diabetes-related hospital admissions, and continues to have a mortality exceeding 40%.[3] Whereas DKA symptoms develop over several hours, HHS symptoms develop in days to weeks and are more likely to include an alteration in mental status. Only 30% of patients with HHS progress to the classic coma; focal neurologic deficits and seizures may also constitute the neurologic findings associated with HHS.[3] Recognizing a history of diabetes, symptoms of hyperglycemia, physical signs of dehydration, and the laboratory triad mentioned earlier are keys to identifying alterations in mental status caused by DKA and HHS. An inverse relationship exists between level of consciousness and serum osmolarity in patients with HHS and in the nearly 30% of patients with DKA with an accompanying hyperosmolar state (**Fig. 1**).[3,4] Mental status changes are rare with a serum osmolarity of less than 320 mOsm/kg, whereas most