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ALCOHOL-ASSOCIATED ACUTE INTOXICATION AND ALTERED CONSCIOUSNESS: CHALLENGES OF DIFFERENTIAL DIAGNOSIS IN EMERGENCY TOXICOLOGY

Tamar Umekashvili[1], Eka Kurdadze[2]

[1]Clinical Toxicologist, Toxicology Department, TSMU First University Clinic, Tbilisi, Georgia.
[2]Assistant Professor, Department of Anesthesiology, Resuscitation and Toxicology, Tbilisi State Medical University; Head of the Toxicology Department, TSMU First University Clinic; Clinical Toxicologist, Tbilisi, Georgia

Background. Alcohol-associated intoxication is among the most frequent emergency toxicology presentations, and altered consciousness in these patients is often attributed to ethanol alone. This anchoring may delay recognition of alcoholic ketoacidosis, lactic acidosis, toxic alcohol poisoning, hypoglycemia, sepsis or acute kidney injury.

Case. A 59-year-old man with hypertension and about nine months of regular alcohol use, with markedly reduced food intake during the preceding month, was brought by ambulance with abdominal pain and distension, dyspnea, dizziness and vomiting after recent drinking. He was drowsy but in adequate contact, with fine horizontal nystagmus, tachycardia, tachypnea, SpO2 93–96% and a distended, diffusely tender abdomen. Blood gas analysis showed severe metabolic acidosis (pH 6.95, pCO2 14.2 mmHg, actual bicarbonate 3.0 mmol/L [standard 6.1], base excess –29.2) with an analyzer-calculated anion gap of 47.8 mmol/L, lactate 29.8 mmol/L (reference 0.9–1.7) and glucose 49 mg/dL. Other findings were macrocytosis (MCV 114 fL), thrombocytopenia, leukocytosis, hyperbilirubinemia (101 μmol/L), AST 320 U/L, GGT 715 U/L, creatinine 162 μmol/L and ketonuria. Contrast-enhanced abdominal CT excluded mesenteric vascular occlusion and perforation, and showed cirrhotic liver configuration, ascites, bilateral basal pulmonary infiltrates and delayed renal contrast excretion.

Course. Toxicology assessment favored alcoholic ketoacidosis with dehydration and malnutrition, and infusion therapy, glucose correction and supportive treatment were started. Acidosis improved only partially (pH 7.22), and the anion gap remained 44–46. Oliguria developed during fluid resuscitation, followed by coma (GCS 7–8), bradypnea, hypoxemia and hypotension (75/45 mmHg), requiring intubation, mechanical ventilation and vasopressors. Asystole occurred about 22 hours after admission, and resuscitation was unsuccessful. Final diagnosis: toxic effect of ethanol (T51.0) complicated by alcoholic liver failure, acidosis, renal failure, pneumonia and respiratory failure.

Conclusions. In patients with alcohol use, profound high-anion-gap acidosis with impaired consciousness requires structured differential diagnosis rather than attribution to ethanol. A gap that persists despite treatment should prompt early measurement of serum ethanol, β-hydroxybutyrate, osmolar gap and toxic alcohols, and assessment for sepsis, hepatic and renal failure and thiamine deficiency, alongside early intensive care involvement. Case-based reasoning of this kind is a valuable educational strategy in emergency toxicology training.
Keywords: ethanol; ketoacidosis; anion gap; consciousness; toxicology.