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CASE REPORT

Hypoglycaemic alcoholic ketoacidosis: a forgotten medical emergency

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Introduction

Hypoglycaemia is typically associated with diabetes and other metabolic disorders. Yet, hypoglycaemia associated with alcoholic ketoacidosis is much less common and significantly more dangerous if not recognised promptly. These patients are usually alert and oriented, which makes this diagnosis less obvious. The lack of a thorough workup and isolating these patients to detoxify could lead to irreversible brain damage and even death if not treated promptly. This article discusses a case in which a man presented with severe hypoglycaemia, an elevated blood alcohol level and an elevated anion gap.

Case report

A 64-year-old African American man with a past medical history of chronic alcohol abuse, hypertension and venous thromboembolism presented to the emergency room with complaints of alcohol intoxication and a 2-week history of shortness of breath preceded by drinking alcohol. He had had recent weight loss of about 5 kg and a decreased appetite over the last few months. His vital signs included a heart rate of 110 beats/minute, respiratory rate of 20 breaths/min, blood pressure of 155/100 mmHg, and he was afebrile. The physical examination was unremarkable. Computed tomography pulmonary angiogram was negative for pulmonary emboli. Lab findings were notable for blood glucose of 1.9 mmol/litre (normal range 4.0–5.5 mmol/litre), carbon dioxide 15 mmol/litre (normal range 22–31 mmol/litre) and anion gap 22 mmol/litre (normal range 3–11 mmol/litre).

A urine analysis revealed ketones >80 mg/dl (ref: negative), and his blood ethanol level was 124.1 mg/dl (legal level for intoxication 80 mg/dl). The remainder of the laboratory results, including complete blood count, brain natriuretic peptide, influenza A and B, and blood cultures, were unremarkable. Urine drug screen was positive for marijuana. Repeat blood glucose levels continued to demonstrate hypoglycaemia (2.3, 2.7 and 3.6 mmol/litre), while the patient received intravenous fluids containing dextrose. He was started on thiamine, multivitamin and folate supplementation and admitted for the treatment of hypoglycaemia and acidosis, observation for alcohol withdrawal, and optimisation of nutritional status.

Over the next 2 days, his hypoglycaemia resolved (4.3, 4.9 and 5.9 mmol/litre) and he was transitioned to a regular diet. On repeat lab tests, his sodium level was 137 mmol/litre, potassium 3.4 mmol/litre, chloride 102 mmol/litre, carbon dioxide 26 mmol/litre, and anion gap 9 mmol/litre. He was subsequently discharged home.

Discussion

This patient presented with a low blood glucose level and an elevated anion gap, secondary to alcohol intoxication and a prolonged malnourished state, indicating hypoglycaemic alcoholic ketoacidosis. While the historical data are limited, hypoglycaemia accompanying alcoholic ketoacidosis is relatively uncommon. In non-diabetic adult patients with blood alcohol levels above 0.10%, only 1–4% presented with hypoglycaemia (Sucov and Woolard, 1995; Ernst et al, 1996). Therefore, hypoglycaemia would only be anticipated in patients with alcohol use disorder who have poor nutritional status, depletion of liver glycogen stores and limitation of gluconeogenesis (Steiner et al, 2015), as seen in this case.

The liver itself contains enough glycogen to maintain euglycaemia for only 8–10 hours (Jain et al, 2002). Without the production of adequate glucose via gluconeogenesis in the presence of a fasting state, there is an increase in the breakdown of lipids to ketoacids. Normal physiological mechanisms allow ethanol to be metabolised by alcohol dehydrogenase (using nicotinamide adenine dinucleotide [NADH]), mitochondria oxidising systems (using nicotinamide adenine dinucleotide phosphate) and catalase (Ernst et al, 1996).

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Learning points

- Hypoglycaemia alcoholic ketoacidosis is a rare phenomenon that can occur in the setting of prolonged malnutrition alongside alcohol intoxication.
- It is crucial that hypoglycaemic alcoholic ketoacidosis remains on clinicians' differential diagnosis, as it can result in vital organ dysfunction, irreversible brain damage and even death if left untreated.
- Appropriate treatment regimen with glucose and nutritional supplementation should be initiated for the prevention of any possible vital organ dysfunction and death.

Alcohol intoxication causes an increase in the NADH/NAD+ ratio, resulting in decreased conversion of substrates such as alanine and glutamate to pyruvate and alpha-ketoglutarate, and subsequent ineffective gluconeogenesis resulting in hypoglycaemia (Hammerstedt et al, 2011). As such, the availability of intrahepatic gluconeogenic precursor decreases by 61% following alcohol consumption in overnight fasted men (Siler et al, 1998), further resulting in an increased conversion of acetoacetate into beta-hydroxybutyrate (a ketoacid) and development of an anion gap acidosis (Sucov and Woolard, 1995; Howard and Bokhari, 2020).

The treatment for alcoholic ketoacidosis is intravenous saline and dextrose. In patients with known alcohol use disorder, parenteral thiamine should also be administered to avoid Wernicke's encephalopathy (McGuire et al, 2006). Other underlying aetiologies, such as systemic infections, should be evaluated as these can cause increased gluconeogenic demand from the body. Furthermore, if left untreated, the ketoacidosis compounded with hypoglycaemia can result in clinically grievous effects secondary to the development of anion-gap metabolic acidosis. A previous case report detailed how a 50-year-old woman became hypoglycaemic and ketotic secondary to alcohol intoxication, developing irreversible encephalopathy, which resulted in a persistent vegetative state (Jain et al, 2002).

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