SHORT REPORT

Alcoholic ketoacidosis with multiple complications: a case report

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Alcoholic ketoacidosis is a poorly diagnosed medical emergency usually identified in chronic alcohol misusers following an abrupt cessation or reduction of alcohol consumption. A high index of suspicion should be maintained by acute physicians as response to treatment is rapid with complete resolution of metabolic derangements. Complications are usually the result of not instituting the correct treatment or not addressing associated conditions. We describe a case of alcoholic ketoacidosis with multiple complications at presentation.

Alcoholic ketoacidosis (AKA) is a poorly diagnosed medical emergency. It is a condition usually identified in chronic alcohol misusers following an abrupt cessation or reduction of alcohol consumption. It is characterised by metabolic acidosis with a high anion gap. Treatment is usually by rehydration and replacement of carbohydrate. We report a case of alcoholic ketoacidosis complicated by hypothermia and rhabdomyolysis.

CASE PRESENTATION

A woman in her 60s was found on the floor of her house unconscious by the London Ambulance Service. No history was available from the patient but collateral history excluded any history of diabetes mellitus, hypertension, cerebrovascular accidents or ischemic heart disease. She had a history of alcohol misuse and was not on any regular medication.

On initial examination she was bradycardic with a pulse of 44 beats/min regular, a blood pressure of 139/103 mm Hg, a respiratory rate of 22 breaths/min and a tympanic temperature of 26.6℃. Her Glasgow Coma Score (GCS) was 10/15 (E2M5V3) with no focal neurologic deficit. The rest of her physical examination was essentially normal. She underwent a series of biochemical and haematological tests with the following abnormal results: blood sugar, 1.1 mmol/l (20 mg/dl); urea, 22. mmol/l; creatinine, 298 mmol/l; creatine kinase, 4066 μmol/l; and amylase, 503 U/l. Arterial blood gas showed a metabolic acidosis with pH of 7.13, base excess of –20.0 and an anion gap of 41.

Her electrocardiogram (ECG) was compatible with hypothermia, showing J waves and confirming the bradycardia at a rate of 44 beats/min. A chest *x* ray was unremarkable and a computed tomographic (CT) scan of her abdomen did not show ureteric obstruction or pancreatitis. She was admitted to hospital because of hypothermia, hypoglycaemia, acute renal failure, acute pancreatitis and rhabdomyolysis.

She was initially treated with intravenous Pabrinex (high potency parenteral vitamins B and C), 50 ml of 50% dextrose solution, gentle rewarming and a subsequent infusion of 5% dextrose to maintain blood sugar above 5 mmol/dl. During the course of treatment she became hypotensive and oliguric. Repeat biochemical parameters showed a worsening urea and creatinine, 29.8 mmol/dl and 356 mmol/dl, respectively, and a rise in the creatine kinase concentrations to 26833 μmol/l. She was given inotropic support with intravenous epinephrine (noradrenaline) and subsequently mannitol infusion. She responded well to treatment with normalisation of all biochemical parameters after 10 days.

Our diagnoses were alcoholic ketoacidosis with hypoglycaemia, hypothermia with acute pancreatitis, and acute renal failure secondary to rhabdomyolysis.

DISCUSSION

AKA is a poorly diagnosed medical condition of chronic alcohol misusers attending accident and emergency departments in the UK. Presentation is usually following a period of alcohol bingeing with abrupt cessation or notable reduction in the consumption of alcohol as a result of stomach upset, abdominal pain or persistent vomiting.[1 2] These patients usually have an associated poor calorie intake, particularly while on the alcohol binge. The persistent vomiting leads to dehydration and acute starvation with consequent ketogenesis.

Following acute starvation, there is suppression of insulin with increased secretion of counterregulatory hormones such as catecholamines, cortisol, growth hormone and glucagons. This results in increased release of fatty acids into the circulation which are subsequently metabolised to ketoacids by the liver. This hormonal profile further enhances ketogenesis by inhibiting the hepatic metabolism of acetyl-co-A via the citric acid cycle. In AKA, β-hydroxybutyrate (BHBA) and acetoacetic (AAA) acids are produced with an increased ratio of BHBA/AAA.[3]

Alcohol metabolism may be contributory to the ketosis. Alcohol dehydrogenase and aldehyde dehydrogenase metabolise alcohol to acetaldehyde and subsequently to acetic acid in the mitochondria. These lead to the reduction of nicotinamide adenine dinucleotide (NAD) to NADH. The resulting increase in the ratio of NADH/NAD inhibits hepatic gluconeogenesis and elevates the ratio of BHBA to AAA.

Furthermore, as a result of persistent vomiting, there is intravascular volume depletion with consequent reduced renal excretion of ketoacids. The raised level of ketoacids accounts for the acidotic state and high anion gap characteristic of this condition.

Unlike diabetic patients, these patients have little or no glycosuria. Blood glucose values are usually low but could also be normal. Patients may also present other manifestations of excess alcohol consumption such as pancreatitis, alcohol withdrawal with delirium tremens, and Wernicke–Korsakoff encephalopathy.

Response to treatment is usually quite rapid. Rehydration and supply of carbohydrates is the mainstay of treatment.

Abbreviations: AAA, acetoacetic acid; AKA, alcoholic ketoacidosis; BHBA, β-hydroxybutyrate acid; NAD, nicotinamide adenine dinucleotide; NADH, reduced form of NAD

Intravenous dextrose solution and saline are the essentials of management. These should be given priority. Malnourished patients, particularly patients with a history of chronic alcohol misuse, should be given intravenous thiamine replacement to avoid precipitation of Wernicke–Korsakoff syndrome.[4] Insulin, bicarbonate and phosphate are usually not needed. The major cause of morbidity and mortality is not the acidosis but rather failure to adequately treat concurrent medical or surgical conditions.

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IMAGES IN EMERGENCY MEDICINE

A case of paraquat burns following an industrial accident

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Paraquat is a highly toxic chemical widely used as a herbicide. We present the case of a 27-year-old man admitted to hospital with suspected paraquat poisoning after falling in a toxic tank containing the solution at work. He lay in the solution for around a minute until his colleagues came to his aid. He was admitted under the care of the physicians via the emergency department with suspected paraquat poisoning. It was not until 4 days later, after this had been excluded, that the chemical burns he had sustained were noticed. He had superficial partial thickness burns to his lower back and gluteal region amounting to 7% total body surface area. These eventually healed with conservative management.

Fatalities have been reported from cutaneous exposure alone to paraquat.[1] Thorough inspection for evidence of skin exposure and prompt irrigation is therefore important both to reduce the severity of burns and to minimise transdermal absorption.

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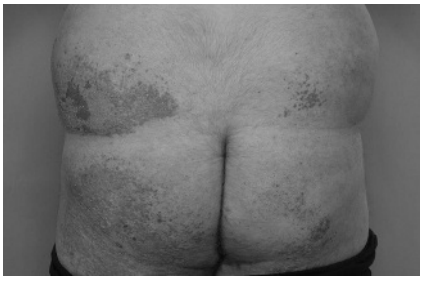

Figure 1 Superficial chemical burns to buttocks and back from exposure to paraquat solution. Informed written consent was obtained for the publication of this figure.

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Informed written consent was obtained for the publication of fig 1.

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1 **Wolfhart DJ**. Fatal paraquat poisonings after skin absorption. *Med J Australia* 1982;**1**:512–13.