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Case Report

An Unusual Case of Refractory Metabolic Acidosis

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ABSTRACT

Homeopathy is one of the most frequently used systems of complementary and alternative medicine. The patients who seek homeopathic treatment are primarily those suffering from long-standing, chronic diseases. These medicines may have considerable risk of severe side effects. Some homeopathic medicinal preparations use alcohol as a base and are frequently prescribed for common conditions. We hereby report an unusual case of refractory metabolic acidosis after homeopathic medicinal treatment.

Key words: Alcohol, homeopathy, intoxication, metabolic acidosis

INTRODUCTION

Homeopathy is one of the most frequently used systems of complementary and alternative medicine. General trends show a rise in the number of individuals utilizing naturopathic and homeopathic therapeutic methods. The patients who seek homeopathic treatment are primarily those suffering from long-standing, chronic diseases. It is based on the "principle of similars," where by highly diluted preparations of substances that cause symptoms in healthy individuals are used to stimulate healing in patients who have similar symptoms when ill.[1] Some of these medicines may have considerable risk of severe side effects. We hereby report an unusual case of refractory metabolic acidosis after homeopathic medicinal treatment.

CASE REPORT

A 25-year-old young male was brought to emergency department with history of headache, pain in abdomen, vomiting, difficulty in breathing, and multiple episodes of generalized tonic-clonic seizures, followed by loss of consciousness since last 10 h. There was no history of fever or head injury. He was chronic alcoholic and non-smoker. On examination, his general condition was low ($E_1M_3V_1$), he was tachypneic, his skin was cold and clammy, peripheral pulses were not palpable, and blood pressure was not recordable. Pupils were normal in size, not reacting to light and bilateral planter response was non-elicitable. Auscultation of chest revealed bilateral coarse crepitations. Rest of the examination was within normal limits. Arterial blood gas (ABG) analysis showed pH – 6.8, pCO2 – 23 mmHg, pO2 – 61 mmHg, plasma bicarbonate – 5 mmol/l, chloride – 96 mmol/l, serum lactate – 2.7 mmol/l, serum osmolality – 360 mmol/l, serum Na^+ – 151meq/L, serum K^+ – 4.6 meq/L and random blood sugar – 87 mg/dl. The patient was intubated and put on ventilatory support with 100% oxygen, intravenous fluids, dopamine, and noradrenaline infusion. Intravenous phenytoin loading was done for seizures.

Investigations revealed hemoglobin-12.9 gm%, total leukocyte count-14800/cumm, differential leukocyte count-P_{62} L_{38}, platelet count-2.34 lacs/cumm, blood urea-86 mg/dL, and serum creatinine-2.6 mg/dL. Urine examination was normal. Liver function test showed SGOT-675IU/L, SGPT-437IU/L, and normal serum bilirubin. Calculated osmolality was 336 mmol/L suggestive of high anion gap metabolic acidosis with high

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osmolar gap. X-ray chest showed bilateral infiltrates in basal lung fields. Computed tomography (CT) of head showed necrosis in bilateral basal ganglia suggestive of alcoholic intoxication. In view of CT findings, history of alcohol intake was reviewed in detail. Patient attendees denied any usage of alcohol in last 10 days because of family restrictions that he was imposed. They also gave a history of consumption of some homeopathic preparation for the cough for last 5 days and he was taking it in excess to normal prescription. No other medicine was being given to the patient. Attendants were asked to bring the homeopathic medicine and it was Causticum Hah having 90% alcohol by vol/vol [Figure 1]. Patient had taken three bottles (1350 mL) of homeopathic medicine in last 5 days out of which about 450 mL (one bottle) was consumed in 2-3 h. The patient was managed on the lines of alcohol intoxication with dextrose containing fluids, intravenous thiamine, bicarbonate infusion, and intravenous broad spectrum antibiotics. Blood ethanol level of the patient was not done because this facility is currently not available in our institution and patient's attendant refused to get it done from outside laboratory due to financial constraints. The repeated ABG analysis showed persistent acidosis with low serum bicarbonate. There was no improvement in blood pressure and general condition of the patient. The patient died on 3rd day of the admission.

DISCUSSION

Metabolic acidosis is one of the commonest acid base disorder, faced by the physicians in day to day practice. It can occur because of an increase in endogenous acid production (lactate and ketoacids), loss of bicarbonate (diarrhoea and renal tubular acidosis), accumulation of endogenous acids (renal failure), or ingestion of toxins.[2] Metabolic acidosis can be normal anion gap or high AG (anion gap) acidosis. There are four principal causes of a high AG acidosis: (1) Ketoacidosis, (2) lactic acidosis, (3) ingested toxins, and (4) acute and chronic renal failure.[2] Ketoacidosis is commonly seen in diabetes mellitus, chronic alcoholics, and starvation. Lactic acidosis is seen in clinical settings like cardiac decompensation, respiratory and hepatic failure, septicemia, and occasionally in metformin treated patients. Among the toxins which cause high anion gap metabolic acidosis are methanol, ethylene glycol, and salicylates.[2] A probe into the history is required in case of toxin ingestion.



Figure 1: Bottle of homeopathic medicine Causticum Hah containing 90% alcohol

Ethanol, methanol, and ethylene glycol causes high AG and high osmolar gap metabolic acidosis.[2] Ethanol is commonly ingested as beverages. It is also used as a solvent and is found in many cosmetic and antiseptic preparations. In general, ethanol intoxication does not cause high AG metabolic acidosis.[2] Metabolic acidosis may be compounded by respiratory failure. Methanol is used in the industrial production of many synthetic organic compounds and is a constituent of a large number of commercially available solvents.[3] Toxicity usually occurs from intentional overdose or accidental ingestion and results in metabolic acidosis, neurologic squeal, and even death.[4]

The potentially lethal dose of methanol is variable; the lowest reported is 30 mL.[4] Following ingestion, there is typically a lag period of about 12-24 h before toxic manifestations occur.[3] The most important initial symptom of methanol poisoning is visual disturbance. Complaints of blurred vision with a relatively clear sensorium strongly suggests the diagnosis of methanol poisoning. Headache, dizziness, nausea, vomiting, and abdominal pain may accompany visual disturbances. The development of bradycardia, prolonged coma, seizures, and resistant acidosis indicates a poor prognosis.[3,4] Diagnosis is confirmed by history, clinical examination, and by measuring the blood alcohol levels. Other laboratory tests that suggest alcohol intoxication are high AG metabolic acidosis, high osmolar gap, and rarely ketoacidosis. Emergency treatment for acute ethanol intoxication strives to stabilize the patient and maintain a patent airway and respiration while waiting for the alcohol to metabolize.[5] Dextrose solution is given for hypoglycemia and thiamine is administered to prevent Wernicke-Korsakoff syndrome. If clinical suspicion of methanol or ethylene glycol poisoning is high, treatment with ethanol should not be delayed pending the report of blood methanol levels. Fomepizole is a direct alcohol dehydrogenase antagonist which is FDA approved for treatment. Hemodialysis whenever available should be used as a treatment modality in classified patients such as those having persistent refractory high anion gap metabolic acidosis, visual and mental obtundation,

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>30 mL of methyl alcohol ingestion or blood levels of >50mg/dL, deteriorating vital signs despite intensive supportive care, renal failure and significant electrolyte disturbances unresponsive to conventional therapy.[4]

The homeopathic medicinal preparations use alcohol as a base and are frequently prescribed for some of the common conditions.[1,6] Causticum Hah is a homeopathic medicinal preparation given for chronic rheumatological conditions and some of the respiratory symptoms.[6] We managed this as a case of alcohol intoxication as the homeopathic medicinal preparation was having 90% vol/vol of alcohol and he had consumed it in excessive amount (1350 mL). It was not clear to us that the alcohol which was used in the preparation was ethyl or methyl alcohol. We presume it to be an ethyl alcohol as it was a medicinal preparation. Though the patient's attendees denied any usage of alcohol by the patient for last few days, we are not sure whether this was solely due to ingestion of homeopathic medicine or combined effects of chronic alcohol use as well as recent homeopathic medicine use.

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