

Case Report

Rrefractory Metabolic Acidosis in a Diabetic Patient : A case of Unsuspected Methanol Intoxication

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Abstract:

We present a case of a 45 years old male, diabetic and hypertensive who was admitted into hospital with a history of restlessness, blurring of vision and breathlessness for one day. In spite of conservative treatment for 10 hours, his general condition gradually deteriorated and he was shifted to ICU. He was kept on mechanical ventilator. His laboratory results showed severe high anion gap metabolic acidosis not corrected by sodium bicarbonate and adequate fluid resuscitation. MRI of brain showed bilateral putamen lesion suspecting methanol intoxication. His acidosis persisted and after giving 3 sessions of Sustained Low Efficiency Dialysis (SLED), metabolic acidosis was corrected with normalization of renal function. After six months in a follow-up, patient complained of total blindness and fundal photography showed bilateral optic atrophy.

Keywords: Metabolic Acidosis, Sustained Low Efficiency Dialysis (SLED), Methanol intoxication, Diabetic Ketoacidosis (DKA).

Introduction

Severe methanol intoxication is rare but life-threatening event. Even ingestion of a small amount of methanol can be potentially lethal¹⁻². The symptoms of methanol intoxication are not very specific except for the visual disturbances and specially the so called "snowstorm vision"³. On the other hand, the presence of a high anion gap acidosis combined with a high osmol gap and normal Delta gap should raise the level of suspicion. Although rare, methanol poisoning is a serious medical problem. Diabetic ketoacidosis (DKA) is a complication of diabetes mellitus which is usually not difficult to diagnose. When a diabetic patient presents with severe hyperglycemia, ketonuria and high anion gap (AG) metabolic acidosis, the presumptive diagnosis is DKA. However, DKA should be differentiated from other causes of metabolic acidosis like methanol poisoning⁴. Both types of acidosis are characterized by high AG metabolic acidosis as it is easy to assume DKA while ignoring the possible presence

of methanol intoxication in a diabetic patient presenting with severe metabolic acidosis. But in case of DKA, metabolic acidosis is usually improved after adequate fluid resuscitation. This case warns us to keep in mind the possibility of methanol intoxication in case of refractory metabolic acidosis in a diabetic patient with a suspected diagnosis of DKA.

Case Presentation

A-45 years old Bangladeshi male, known COPD, diabetic and hypertensive was admitted into hospital with a history of restlessness, blurring of vision and breathlessness for one day prior to admission. The patient had no history of fever, headache, vomiting or convulsion. His blurring of vision was associated with pain in the eye but there was no history of trauma to eye or contact with any chemical. In spite of conservative treatment for 10 hours his general condition gradually deteriorated and his GCS began to deteriorate. He developed severe respiratory distress with falling of SPO₂ in spite of high flow Oxygen. So he was shifted to ICU for further management. There he was intubated and kept on mechanical ventilator. On examination before intubation his GCS was 5 and pupils were bilaterally normal in size but with poor reaction to light. Ophthalmoscopy revealed hyperemia of the optic disc with loss of physiological cupping. Initial blood pressure was 90/50 mm/Hg with a regular heartbeat of 122 beats/min and body temperature of 38° Celsius. Auscultation of the lung was normal except some coarse crepitations in right lung. The heart sounds showed no abnormalities. The remaining physical examinations were normal.

Laboratory results showed a severe high AG metabolic acidosis with a pH of 6.68 and HCO₃⁻ of 3.2 mmol/L, PCO₂ 21.3 mmHg and calculated osmolality 302 mOsm/kg, Na⁺ 145 mmol/L, K⁺ 4.7 mmol/L, Urea 5.8 mmol/L, Glucose 14.6 mmol/L, Cl⁻ 110 mmol/L and Lactate 12.2 mmol/L, Anion gap 36 mmol/L and a Delta gap of 3.2 indicating an almost pure high AG acidosis.

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His metabolic acidosis could not be corrected with IV Sodibicarb and adequate IV fluid. His mental status was deteriorating, We decided to do MRI of brain and it showed bilateral putaminal lesions (Arrow in Fig-1). So we suspected methanol intoxication because on further quarry into history, occational binge alcohol intake by the patient was confirmed. Duration and amount of alcohol intake was not known.

In ICU, his hypotension was successfully treated with normal saline and inotropic support. Pure ehanol was administered through nasogastric tube as Fomiprazol was not available. Folate and IV thiamine were also administered. We used intravenous Mehtylprednisolone for optic neuritis. Following these measures, the hemodynamic condition of the patient improved markedly in 24 hours after admission. However overall condition deteriorated and acidosis was not corrected as desired. He developed acute kidney injury with serum creatinine 3.4 mg/dl. He was considered for urgent hemo dialysis. After 3 sessions of SLED in next 3 days, metabolic acidosis was corrected, as indicated by Arterial Blood Gas (ABG) of pH 7.46, HCO_3^- 25.6mEq/L, PaCO_2 36mmHg, PaO_2 106mmHg. Repeat serum creatinine was 1.2 mg/dl with daily urine output increased to >1500 mL. His vital signs were stabilized on the 5th day of admission and consequently, he was extubated. He appeared blind at discharge. Six months after discharge his fundal photography showed bilateral optic atrophy.

(Arrow in Fig- 2).

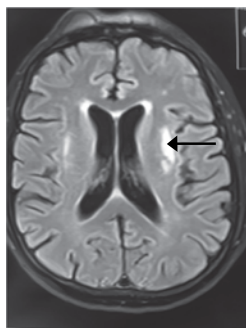


Figure: 1

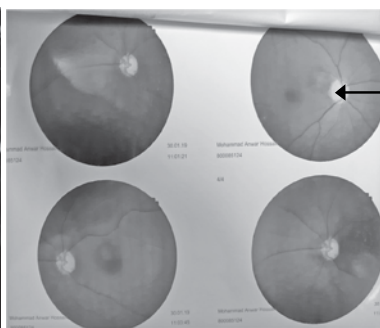


Figure: 2

Discussion

Metabolic acidosis is the most common disorder of acid-base balance and is characterized by primary HCO_3^- reduction and decreased pH caused by an increase in extracellular fluid H^+ or loss of HCO_3^- . To judge metabolic acidosis, AG values are important. According to different AG values, metabolic acidosis can be divided into a high AG (normochloremic) type and a normal AG (hyperchloremic) type. Our patient had slightly raised blood sugar (14.6 mmol/L) with evidence of severe metabolic acidosis. There are examples of euglycemic ketoacidosis on literature search⁵. In these cases, DKA is mislabeled because of euglycemia.

In general, high AG metabolic acidosis caused by excessive endogenous acid is usually more common⁶. Methanol is metabolized by alcohol dehydrogenase to formaldehyde and

then transformed to formic acid by aldehyde dehydrogenase⁷. Accumulation of formic acid can cause damage to the retina and optic nerve, resulting in changes in the histological morphology and function of the tissues and ultimately leading to blindness⁸. The organ most sensitive to methanol's toxicity is the optic nerve, as confirmed by both animal experiments and a large-scale clinical epidemiological survey in Cuba, although the mechanism is still not completely clear⁹. Previous studies also showed that the degree of acidosis caused by methanol poisoning is positively correlated with the risk of permanent blindness in patients¹⁰. Residual visual disturbance due to toxic optic neuropathy has been shown to recover partially, and in some cases fully, in survivors of methanol intoxication¹¹. Unfortunately, our patient developed complete blindness.

Conclusion.

Methanol intoxication should be kept in mind in a diabetic patient presenting with refractory metabolic acidosis. Clinicians should perform a thorough history regarding potential alcohol intake. Early recognition and treatment is essential for the management of methanol intoxication to minimize morbidity and mortality. Metabolic improvements do not equal to healing of the patient, especially permanent neurological deficit may occur despite metabolic improvement.

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