

Methanol Poisoning Important Differential in a Refractory Metabolic Acidosis with A Diabetic Patient Suspecting Ketoacidosis: A Case Report

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Abstract

The incidence of methanol intoxication is less common in Bangladeshi population. Early and aggressive management with bicarbonate, ethanol and hemodialysis in patients having significant toxicity will decrease mortality and improve patient's outcome. Our experience of SLED in haemodynamically unstable patient with inotropic support may improve outcome.

Case presentation: A-45 years old male businessman, diabetic and hypertensive was admitted into cabin with history of restlessness, blurring of vision and breathlessness 1 day. In spite of conservative treatment after 10 hours his general condition gradually deteriorated, so he shifted to ICU and kept in mechanical ventilator. His laboratory results showed a severe high anion gap metabolic acidosis not corrected by sodium bicarbonate and adequate fluid resuscitation. His neurological condition of patient was deteriorating and MRI of brain showed bilateral putamenal lesion suspecting methanol intoxication. After conservative treatment, his general condition was deteriorated and acidosis was not corrected rather than he developed acute kidney injury so haemodialysis (SLED) was started after taking nephrology consultation. After giving 3 sessions of SLED, metabolic acidosis was corrected with normalization of renal function. His vital signs stabilized and he was extubated subsequently. After six months in a follow-up patient complained total blindness and fundal photography showed bilateral optic atrophy.

Conclusions: We conclude that in case of severe metabolic acidosis, methanol intoxication always should be kept in mind in a patient of Diabetes Mellitus suspecting DKA. Early initiation of haemodialysis is very effective to reverse renal and metabolic abnormalities in spite of haemodynamic instability. Metabolic improvements do not equal to healing the patient, especially permanent neurological deficit like blindness may persist.

Keywords: SLED (Sustained Low Efficiency Dialysis), Methanol Intoxication, DKA (Diabetic Ketoacidosis)

Introduction

Severe methanol intoxication is a rare but life-threatening event; even ingestion of a small amount of methanol can be potentially lethal [1-2]. Prompt action should therefore be taken when methanol intoxication is suspected, because delay can have deleterious consequences. Awareness of even the rare possibility of methanol ingestion is thus very important in emergency medicine. 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. Thus, although rare, methanol poisoning is a serious medical problem. Whereas Diabetic ketoacidosis (DKA) is a complication of diabetes that is usually not difficult to diagnose. When a diabetes 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 [4]. Both types of acidosis are characterized by high AG metabolic acidosis, 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 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 besides diagnosis of DKA.

Case Presentation

A 45 years old male businessman, known COPD, diabetic and hypertensive was admitted in hospital with history of restlessness, blurring of vision and breathlessness for same duration 1 day. The patient had no history of fever, headache, vomiting or convulsion. His blurring of vision was also associated with pain in the eye but there was no history of trauma or contact to any chemical in eye. In spite of conservative treatment after 10 hours his general condition gradually deteriorated and his GCS was falling and he developed severe respiratory distress with falling of SPO_2 in spite of high flow O_2 . So he shifted to ICU for further management. Immediately after arrival in ICU his respiration rate was 34/min, but shallow and SPO_2 gradually falling @ 78% with oxygen 10L/min, so he intubated and kept in mechanical ventilator. On nervous system examination GCS was 5 and pupils are bilaterally normal in size but poor reaction to light. Indirect 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 HCO_3^- of 3.2 mmol/L, PCO_2 21.3 mmHg and calculated osmolality 302 mOsm/kg [Serum Osmolality = $(2 \times (\text{Na} + \text{K})) + (\text{BUN} / 2.8) + (\text{glucose} / 18)$]. 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 $([\text{Na}^+] + [\text{K}^+] - ([\text{Cl}^-] + [\text{HCO}_3^-])) = (145 + 4.7) - (110 + 3.2) = 36$ mmol/L. Delta gap or Bicarbonate gap $(\text{AG} - \text{Normal AG}) - (\text{Normal bicarbonate} - [\text{HCO}_3^-]) = (36 - 12) - (24 - 3.2) = 3.2$ indicating an almost pure anion gap acidosis.

His metabolic acidosis was not corrected with sodium bicarbonate and adequate fluid resuscitation and his neurological condition of patient was deteriorating, we decided to do MRI of brain and it showed bilateral putamen lesions (Figure 1). So we suspected methanol intoxication, even though the patient initially did not give history alcohol consumption. Repeat history suggested occasional binge alcohol intake, but duration and amount was not known.

In ICU, his hypotension was successfully treated with normal saline and inotropic support. Ethanol was administered through nasogastric tube as Fomiprazol is not available in our country. Folate and thiamine were also administered. We also used intravenous Methylprednisolone for optic neuritis. Following these measures the hemodynamic condition of the patient improved markedly. 24 hours after admission, overall condition deteriorated and acidosis was not corrected rather than he developed acute kidney injury (s. creatinine 3.4 mg/dl), so haemodialysis (SLED) was started after taking nephrology consultation. After 3 sessions of SLED, metabolic acidosis was corrected, as indicated by pH 7.46, HCO_3^- 25.6 mEq/L, PaCO_2 36 mmHg, PaO_2 106 mmHg and s. creatinine 1.2 mg/dl with daily urine output increased to >1500 mL. So SLED was discontinued. His vital signs stabilized on the 5th day of admission and consequently, he was extubated. After six months of event, he went abroad for better management, there they did fundal photography and it showed bilateral optic atrophy (Figure 2).

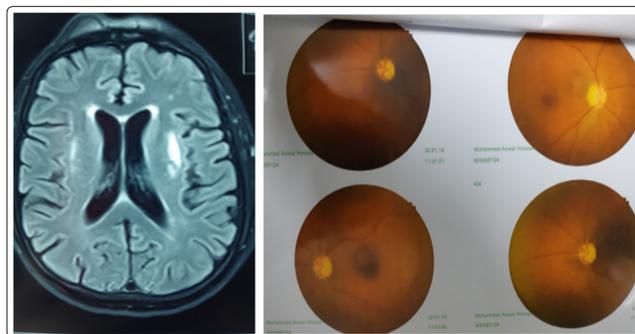


Figure 1

Figure 2

Figure 1: MR brain imaging showing bilateral putamen lesions. in diffusion-weighted image with restricted diffusion. Fundal photograph showed optic atrophy after six months (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^- . In the clinical judgement of metabolic acidosis, AG values are important. According to different AG values, metabolic acidosis can be divided into a high AG normal chloride type and a normal AG high chloride type.

In general, high AG metabolic acidosis caused by excessive endogenous acid is more common in the clinic [6]. Methanol poisoning also results in increased AG. Methanol is metabolized by alcohol dehydrogenase to formaldehyde and then transformed to formic acid by aldehyde dehydrogenase [7]. 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 blindness [8]. 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 clear [9]. Previous studies also showed that the degree of acidosis caused by methanol poisoning is positively correlated with the risk of permanent blindness in patients [10]. Residual visual disturbance due to toxic optic neuropathy has been shown to recover partially, and in some cases fully, in survivors of methanol intoxication [11]. Unfortunately, our patient developed complete blindness. The lesson of this report is that although it is to be hoped that methanol consumption is uncommon, it should be always kept in mind the possibility of methanol intoxication in a Diabetic patient presenting with refractory metabolic acidosis. Clinicians should perform a thorough history inquiry regarding potential alcohol intake and early recognition and treatment is essential for the management of methanol intoxication and minimization of morbidity and mortality. Particularly for diabetes patient with metabolic ketoacidosis, if the patient shows an atypical response to treatment, intoxication should be considered immediately. A toxicological scan should be made, and great care must be taken to prevent complications.

Conclusions

In case of differential diagnosis of severe metabolic acidosis, methanol poisoning should always be taken into account besides diabetic ketoacidosis. Methanol intoxication induced derangements are successfully treated with SLED even in a hemodynamic instable

patient. Metabolic improvements do not equal to healing the patient, especially permanent neurological deficit may persist inspite of metabolic improvement. A rapid and active management can save the life of methanol intoxicated patient

Consent for Manuscript and Figure

The patient and his wife gave written consent for the use of personal and medical information for the publication of this case report and accompanying images.

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