ISSN: 2577 - 8005

Case Report

Medical & Clinical Research

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

U K Mallick[1], S M Hossain Shahid[2], Mohammad Asaduzzaman[3], Aflatun Asha[4], Sayem Mohammad Farhad[5], Supta Bardhan[6], Nahin Siddique[7], Nahid Sarker[8], Omar Faruq[9] and Ashikur Rahman[10]

[1]*Registrar, Dept. of Critical Care Medicine, National Institute of Neurosciences and Hospital, Dhaka*

[2]*Consultant & Head of ICU, Anwer Khan Modern Medical Collehe Hospital, Dhaka*

[3]*Mohammad Asaduzzaman, Assistant registrar, Dept. of CCM, National Institute of Neurosciences and Hospital, Dhaka*

[4,5,6,7,8,9,10]*RMO, ICU, Anwer Khan Modern Medical Collehe Hospital, Dhaka*

***Corresponding author**

Dr. Uzzwal Kumar Mallick, Registrar Dept. of Critical Care Medicine National Institute of Neurosciences and Hospital Dhaka, Bangladesh. Tel: 01712715180; E-mail: ukm1980@gmail.com

Submitted: 12 Nov 2019; **Accepted**: 04 Dec 2019; **Published**: 07 Dec 2019

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 inotrpic support may improve outcome.

***Case presentation**: A-45 years old male bussinessman, diabetic and hypertensive was admited into cabin with history of restlessness, blurring of vision and breathlessness 1 day. Inspite of conservative treatment after 10 hours his general condition gradually detoriated, so he shifted to ICU and kept in mechanical ventilator. His laboratory results showed a severe high anion gap metabolic acidosis not corrected by sodibicarb and adequatefluid resuscitation. His neurological codition of patient was detoriating 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 kept in mind in a patient of Diabetes Mellitus suspecting DKA .Early initiation of haemodialysis is very effective to reverse renal and metabolic abnormalities inspite of haemodymis unstability. Metabolic improvements do not equal to healing the patient, especially permanent neurological deficit like blindness may persit.*

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 kept 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 bussinessman, known COPD, diabetic and hypertensive was admited into cabin with history of restlessness, blurring of vision and breathlessness for same duration1 day. The patient had no history of fever, headache, voviting 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. Inspite of conservative treatment after 10 hours his general condition gradually detoriated and his GCS was falling and he developed severe respiratory distress with falling of SPO_2 inspite of high flow O_2. So he shifted to ICU for further management. Immediately after arrival in ICU his respiration rate was 34/, 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 opthalmoscopy 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 x (Na + K)) + (BUN / 2.8) + (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 ([Na]+ [K]) - ([CL]+ [HCO_3]) = (145+4.7) - (110+3.2) = 36 mmol/L.Delta gap or Bicarbonate gap (AG-Normal AG) - (Normal bicarbonate- [HCO_3]) = (36-12) - (24-3.2) = 3.2 indicating an almost pure anion gap acidosis.

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

In ICU, his hypotension was successfully treated with normal saline and inptropic support. Ehanol was administered through nasogastric tube as Fomiprazol is not available in our country. Folate and thiamine were also administered. We also used intravenous Mehtylprednisolone 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 25.6mEq/L, $PaCO_2$ 36mmHg, PaO_2 106mmHg and s. creatinine 1.2 ma/dl with daily urine output increased to >15 00 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 was done and it showed bilateral optic atrophy (Figure 2).

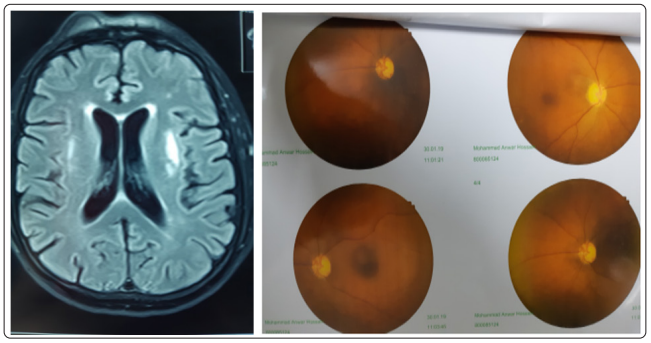

Figure 1 **Figure 2**

Figure 1: MR brain imaging showing bilateral putamen lesions. in diffusion-weighted image with restricted diffusion. Fundal photograph showed optic atrophyy 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 chlorine type and a normal AG high chlorine 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 patientt, especially permanent neurological deficit may persit inspite of metabolic improvement. A rapid and active management can save e 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.

References

1. ZieglerSL (1921) The Ocular Menace of Wood Alcohol Poisoning. Br J Ophthalmol 5: 411-417.
2. Bennett IL Jr, Cary FH, Mitchell GL Jr, Cooper MN (1953) Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. Medicine (Baltimore) 32: 431-463.
3. Jacobsen D, McMartin KE (1986) Methanol and ethylene glycol poisonings. Mechanism of toxicity, clinical course, diagnosis and treatment. Med Toxicol 1: 309-334.
4. Sanaei-Zadeh H, Zamani N, Shadnia S (2011) Outcomes of visual disturbances after methanol poisoning. Clin Toxicol (Phila) 49: 102-107.
5. Thawabi M and Studyvin S (2015) Euglycemic diabetic ketoacidosis, a misleading presentation of diabetic ketoacidosis. N Am J Med Sci 7: 291-294.
6. Salek T, Humpolicek P and Ponizil P (2014) Metabolic disorders due to methanol poisoning. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 158: 635-639.
7. Ng PCY, Long BJ, Davis WT, Sessions DJ, Koyfman A (2018) Toxic alcohol diagnosis and management: an emergency medicine review. Intern Emerg Med 13: 375-383.
8. Liesivuori J and Savolainen H (1991) Methanol and formic acid toxicity: biochemical mechanisms. Pharmacol Toxicol 69: 157-163.
9. Sadun A (1998) Acquired mitochondrial impairment as a cause of optic nerve disease. Trans Am Ophthalmol Soc 96: 881-923.
10. Desai T, Sudhalkar A, Vyas U and Khamar B (2013) Methanol poisoning: predictors of visual outcomes. JAMA Ophthalmol 131: 358- 364.
11. Nazir S, Melnick S, Ansari S and Kanneh HT (2016) Mind the gap: a case of severe methanol intoxication. BMJ Case Rep 2016.

Citation: *U K Mallick, S M Hossain Shahid, Mohammad Asaduzzaman, Aflatun Asha, Sayem Mohammad Farhad (2019) Methanol Poisoning Important Differential in a Refractory Metabolic Acidosis with A Diabetic Patient Suspecting Ketoacidosis: A Case Report. Med Clin Res 4(12): 1-3.*