

Vision Loss After Accidental Methanol Intoxication : A Case Report NUURUL HUDA NIK PA¹, Azhana Hassan¹, Suraina M. Sulaiman¹ ¹Department Of Emergency, Hospital Tuanku Ja'afar, Seremban, NEGERI SEMBILAN, Malaysia

INTRODUCTION

Methanol intoxication often results in permanent problems such as visual deterioration, metabolic disturbances, neurological dysfunction and death. Indeed, small amounts of ingested methanol are sufficient to produce acute destruction of parts of the central nervous system leading to permanent neurological dysfunction and irreversible blindness.

CASE REPORT

A chronic alcoholic, 48 years old gentleman presented to Green Zone with gradually worsening loss of vision for one day. He also had vomiting and abdominal pain for 3 days. He had consumed cans of cheap alcohol; "Kingfisher Beer" bought at a fun fair a week before presentation. Examination revealed bilateral fixed and dilated pupils without light perception. Eye assessment by Ophtalmology team revealed normal fundus examination.

He had high anion gap metabolic acidosis. Patient was treated with oral whisky of 40% ethanol, purchased by his relative while waiting for 20% oral ethanol prepared by HTJS pharmacist. Our pharmacist had to prepare such dilution for the first time and it was mixed with fruit juice prior to ingestion. He was given intravenous infusion Folinic Acid, Sodium Bicarbonate, Parentrovite in ETD and underwent hemodialysis in the ward. Subsequently the acidosis resolved with normalization of renal function. However, he become permanently blind and was diagnosed as Methanol Poisoning with Metabolic Acidosis and Ocular Toxicity. Serum methanol sent was positive.

DISCUSSION

Methanol metabolic products are toxic and either managing these products or not permitting the formation of metabolic product is the strategy for treatment of humans with methanol ingestion. Production of methanol metabolic product is prevented by blocking or inhibiting the cytosolic alcohol dehydrogenase enzyme by fomepizol or by providing ethanol which is preferentially metabolized by alcohol dehydrogenase thus sparing methanol from metabolism.

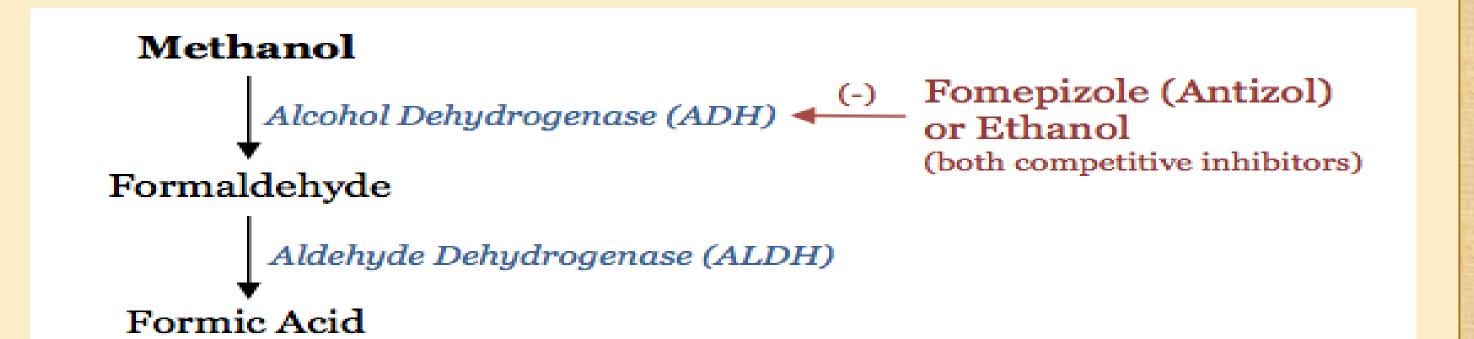
BLOOD INVESTIGATION

 Full Blood Count
 TWC: 14.69 x 10^9/L, Hb: 19.1 g/L, PLT: 308 x 10^9/L, Hct: 56.5%

 Venous Blood Gas
 pH: 7.241 HCO3: 6.8, Cl : 104, Lactate : 1.47

 Renal Function
 Na: 136 mmol/L, K: 3.3 mmol/L,

Oral ethanol is a lifesaving antidote due to its availability and cost-effective. The best antidote would be fomepizole, unfortunately it is reserved for special group of population such as paediatric and pregnant mothers due to cost constraint. Intravenous 10% ethanol is not yet available as it requires close monitoring by serum ethanol and only available in toxicology centers.



Renal Function	Urea 6.6 mmol/L, Creatinine 127 umol/L	
Liver Function	TP : 94 g/L, ALB : 53 g/L, ALT : 14 U/l, ALP : 69 U/l , TB : 17 μmol/l	
Coagulation Profile	PT 13.4 sec, INR: 1.04, aPTT: 39.3 sec	
Cardiac Enzyme	Troponin I : 0.03 ng/ml	
Dextrostick	11.7 mmol/L	
Serum Methanol	36 mg/dL (detected)	
Serum Ethanol	Not detected	
FIG	JRES	

Folic Acid (Vit B9)

CO2 + H20

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Figure 1: Kingfisher Beer was consumed by the patient. **Figure 2:** Alcoholic beverage with 40% alcohol (ethanol) content such as "Scotch Whisky" as a lifesaving antidote in Methanol Intoxication.

CONCLUSION

Methanol poisoning should be treated immediately as it causes high mortality and morbidity. Hence, it should not be missed and must be one of the differential diagnosis in any patient who presented with acute loss of vision.

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