

Methanol toxicity-an important deferential to consider in appropriate patients with high anion gap metabolic acidosis

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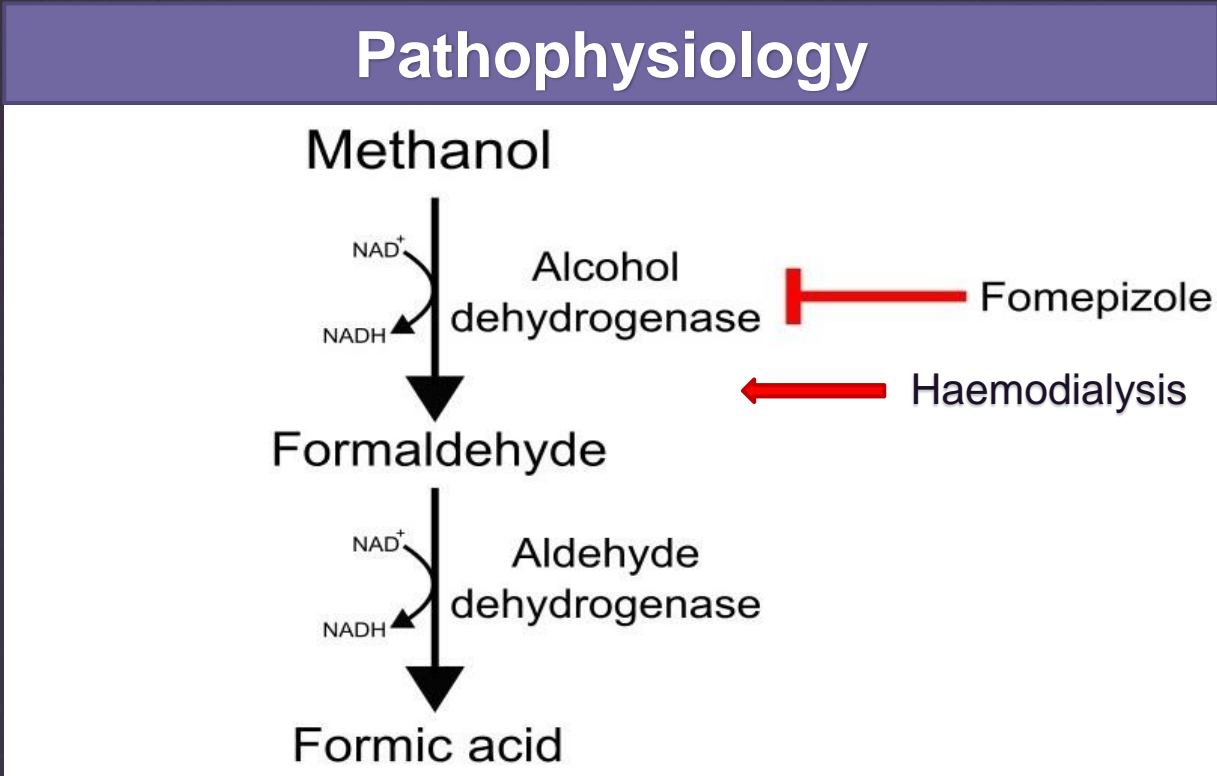
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Introduction

Methanol is a highly toxic substance and even a relatively small amount of accidental ingestion causes the formation of a toxic compound- formic acid which can lead to fatal outcomes or permanent neurological damage including blindness.

The main treatment modality is using fomepizole and/or intravenous alcohol to prevent the formation of lethal metabolites. If severe poisoning is suspected, early initiation of haemodialysis can lead to favourable outcomes by removing both parent alcohol and metabolites.

We are reporting four cases of young (23-36 years) male patients who presented to a tertiary care hospital in Bangladesh with high anion gap metabolic acidosis following accidental ingestion of methanol.



Case Presentation

All the four patients presented with the history of nausea and vomiting for 12-24 hours followed by dyspnoea, headache, blurring of vision and altered conscious level for few hours. Other than the history of recent alcohol ingestion, no definitive history was available. GCS were 7-8/15; pupils were dilated with minimal reaction to light. Bed side blood gas analysis revealed high anion gap metabolic acidosis (pH-6.5-7.1). Blood glucose and ketone were normal.

Diagnosis and Case Progression

As the toxicology screen was not readily available, the provisional diagnosis was reached by the process of elimination from presenting symptoms and signs and bedside arterial blood gas analysis. Brain imaging of two patients showed bilateral putamen necrosis.

All the four patients were admitted to the critical care unit . As the definite antidotes, fomepizole and intravenous alcohol are unavailable in Bangladesh, haemodialysis was started immediately via temporary femoral or internal jugular catheters. Three patients recovered without any neurological and visual sequelae and one patient who presented with pH 6.5 expired despite dialysis.

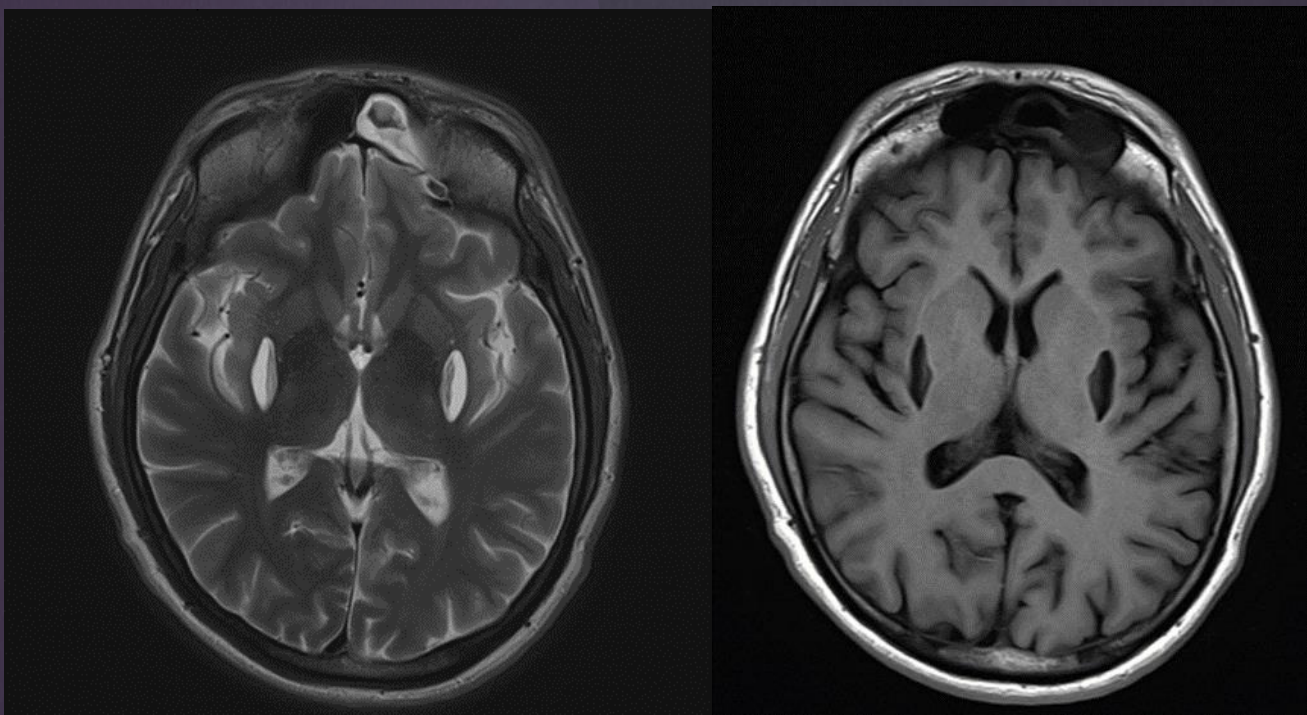


Figure 1: MRI brain of a patient showing bilateral putamen necrosis

Conclusions

In a resource constraint country, like Bangladesh, where the toxicology screen of methanol is not available, high clinical suspicion is crucial and diagnosis should be made from history and suggestive blood gas analysis. In the absence of definitive antidotes for methanol toxicity, early recognition and initiation of heamodialysis can be life and sight saving.

References

- Jacobsen D, McMartin KE. Methanol and Ethylene Glycol Poisonings; Mechanism of Toxicity, Clinical Course, Diagnosis and Treatment. Med Toxicol 1986;1:309-334.
- Barceloux DG, Bond GR, Krenzelok EP, et al. American Academy of Clinical Toxicology Practice Guidelines on the Treatment of Methanol Poisoning. J Toxicol ClinToxicol 2002;40:415-446.
- Kumar SS, Seerala Boopathy K, Bhaskar ME. Methanol poisoning--a Chennai experience. J Assoc Physicians India. 2003 Apr;51:425-426