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CASE REPORT

METHANOL INDUCED INTRACRANIAL HEMORRHAGE: A RARE CASE REPORT

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Abstract:

Intracranial hemorrhage is a rare but fatal complication in methanol poisoning. We report a case of large bilateral basal ganglia hematoma in a 29-years old man with methanol poisoning. He drank alcohol, and was admitted to a tertiary level hospital 6 hours post-ingestion for depressed mental status, lower blood pressure, and high anion gap metabolic acidosis. After fourteen days of methanol exposure, he suddenly developed left sided weakness with one episode of seizure attack. MRI of brain was carried out and showed large intracerebral bleed basal ganglia, specially putaminal and brain edema with midline shift. The uncommon presentation of methanol poisoning is highlighted in this case report.

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Introduction:

Methanol tastes similar to ethanol and has a colorless, volatile, very toxic alcohol odor at room temperature. It is also easily soluble in water and bodily fluids.[1] It is found in a variety of commercial paint thinners, gasoline anti-freeze, windshield products, organic solvents, shellac varnish, washer fluid, photocopying fluids, perfumes, and in some eau de cologne.[2] Methanol is mainly metabolized in the liver and can be converted to formaldehyde with the participation of alcohol dehydrogenase. Formaldehyde dehydrogenase converts formaldehyde, which has a half-life of 1-2 minutes, into formic acid rather quickly. The half-life of formic acid in the human body is 20 hours, and it ultimately breaks down to CO2 and water.[3] A small amount of between 50 and 100 ml results in permanent blindness and severe neurological dysfunction leads to death.[4] More than half of methanol related morbidity and mortality is classified as accidental and therefore preventable. It can occasionally be brought on by the fraudulent adulteration of wine or other alcoholic drinks. Especially when there is a restriction on ethanol, it is widely used as an alcohol substitute (wartime or prohibition). When consumed, it results in high anion gap metabolic acidosis, which produces formic and lactic acids, as well as central nervous system disturbances as drowsiness, intoxication, obtundation, seizure, and coma. Selective toxicity of the optic nerve and basal ganglia are well-known features.[5] Intracranial hemorrhages, such as putaminal hemorrhages and subcortical necrosis, are among the consequences that are uncommon, have an unclear etiology, and have been linked to high mortality rates.[6] We report a rare case of late intracranial hemorrhage following methanol poisoning.

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Case report:

A 29 years old day labourer male addicted with illicit drugs was taken by his colleague to the emergency department of a tertiary level hospital with the complaints of breathlessness, feeling of discomfort followed by unconsciousness following ingestion of alcohol in around 500 ml the previous night with his peers. The symptoms occur about 6 hours after consumption of alcohol. Additionally he had also history of low urine output which started on the day of admission. He had no history of cardiovascular risk factors, no blood disorders, no thrombolytic drugs and no history of Autosomal dominant polycystic kidney disease (ADPKD) or Subarachnoid hemorrhage (SAH). He had a history of taking alcohol for last 6 years. On examination his Blood pressure was 100/70, pulse - 110/min, temperature -98 F, respiratory rate-25/min GCS was 8, bilateral dilated pupil, sluggish reacting to light and planter bilateral extensor and rhonchi presented all over the lung field. Fundus examination revealed oedematous optic disk with dilated peripapillary vessels. His conditions were gradually deteriorated and shifted to the ICU. On investigation showed polymorphonuclear leukocytocysis, random blood sugar was 12.0mmol/L, serum creatinine -2.8mg/dl, Blood urea 56mg/dl , HbA1C-6.3, His blood gas analysis showed severe metabolic acidosis (Ph6.78-7.12), PCO2(20.1-12.0),HCO3(3.0-4.4 mmol/L), serum electrolytes showed Na+ 122mmol/L and K+5.4 mmo/L. With these investigations patient was diagnosed as a case high anion gap metabolic acidosis with acute kidney injury (AKI) with electrolytes imbalance (Na+↓, K+↑). The patient was treated in ICU. With a presumed diagnosis of methanol poisoning as history of alcohol intake with visual disturbances with high anionic metabolic acidosis, he patient was started on intravenous sodium bicarbonate to correct acidosis. Ethanol was added to block formic acid production, while 2 units hemodialysis session without heparin was initiated to help correct the severe acidosis and eliminate both ethanol and its metabolites - formic acid. Follow-up arterial blood gases showed improvement of acidosis. Shortly after admission to Intensive Care Unit, he became hypotensive, fluid boluses given and vasopressor started. He remained in ICU for 7 days and his conditions were improved and discharged from hospital.

After 2 weeks he suddenly developed left sided weakness with one episode of seizure attack for which he was taken tertiary hospital. A CT scan of brain (Figure: 1) was done and diagnosed as a case of hemorrhagic stroke in bilateral basal ganglia (putamen, right > left) with slight midline shift to the left. MRI of brain shows (Figure: 2) that bilateral early subacute hemorrhage in the basal ganglia (putamen). Coagulation screening of patient Prothrombin time, INR, Protein C and Protein S levels were within normal range. Our patient was managed conservatively and survived with neurological sequelae with left sided hemiparesis (MRC grade 4/5 (Upper limb) and 2/5 (lower limb) and discharged from hospital from after 2 weeks with advised physiotherapy.

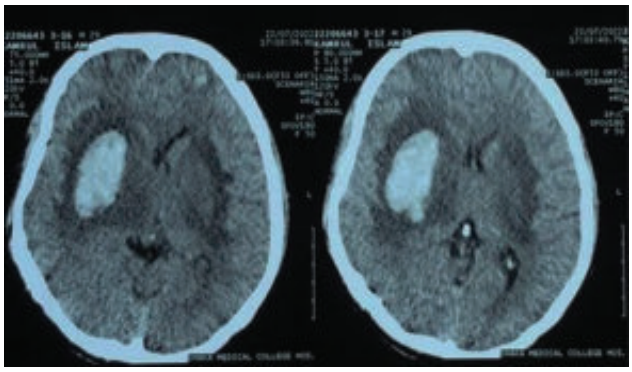

Fig.-1: *Computed tomography brain showing cerebral bleed in the basal ganglia; putamen (right>left) with midline shift.*

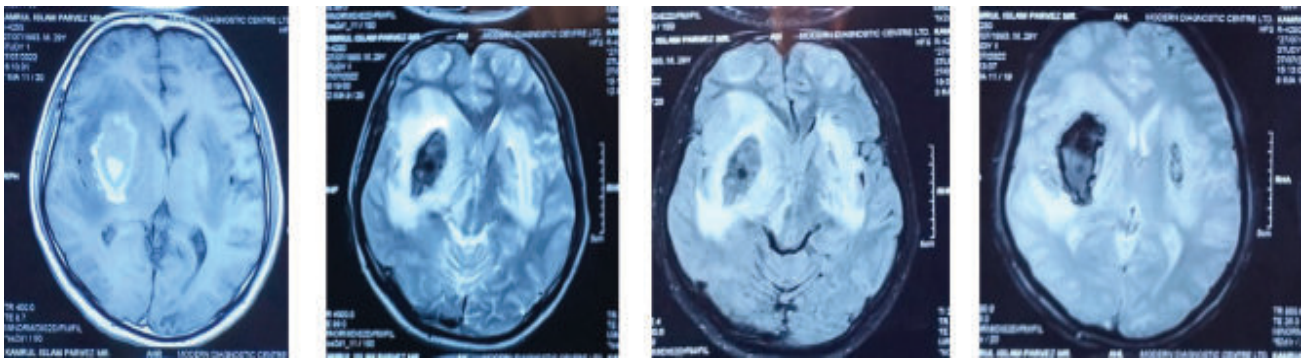

Fig.-2: *MRI of brain shows bilateral early subacute hematoma (right>left) which demonstrates hyperintense in T1WI (A), hypointense T2WI (B), hypointense in FLAIR(C) and hypointense in GRE (D).*

Discussion:

Bilateral putaminal hemorrhage, necrosis, and a late presentation of methanol intoxication were present in our patient. In addition to those findings, subcortical white matter involvement was discovered that the most prevalent anomaly of methanol toxicity in brain imaging connected to peripheral enhancement [7]. Toxic metabolites of methanol, formic acid, and neuronal cells may directly cause harm to the basal ganglia [8]. It is unclear, although the Putamen's high metabolic demand or its microvascular structure may be involved [8,9]. The Putamen is the primary location for intracerebral hemorrhage (ICH), which accounts for around 45% of all ICH cases [10]. In the setting of methanol intoxication, hypoxia and acidosis may also be contributing factors [4]. Additionally, several elements including anoxia, acidosis, electrolyte imbalances, and coagulopathies may be responsible for this fatal consequence. Certain researchers have linked putaminal hemorrhage to the administration of heparin during hemodialysis [9, 11]. Similar to how our patient's putaminal damages manifested after 2 weeks of hemodialysis and a brief period of symptom relief. A recent study showed that those chronic dialysis users have a 10.7 relative risk of cerebral hemorrhage compared to the general population's [12]. Heparin is the main anticoagulant agent used in hemodialysis and may cause ICH in addition to vascular alterations in the course of chronic renal disease. We propose that the anticoagulant techniques utilized in dialysis may be responsible for early CNS hemorrhage after a session. Usually, the prognosis is terrible with high mortality in basal ganglia hemorrhage related to severe methanol poisoning, and even autopsy case was reported [13]. Some other rare cases were published discussing the mechanism inducing intra-cerebral hemorrhage [14, 15]. However, because of the short half-life of heparin, and exclude the other coagulopathy methanol poisoning alone can cause delayed cerebral bleeding, as was the case with our patient.

Conclusion:

We reported the clinical and radiological manifestations of hemorrhagic stroke in day laborers after illegal ingestion of methanol. Cerebral infarction and hemorrhage are rare complications of methanol poisoning, but should be considered when treating such cases. Similarly, health care practitioners should consider the potential intake of methanol by young and middle-aged individuals who have had a stroke in societies where alcohol consumption is illegal.

Conflict of Interest:

The author stated that there is no conflict of interest in this study

Funding:

No specific funding was received for this study.

Ethical consideration:

The study was conducted after approval from the ethical review committee. The confidentiality and anonymity of the study participants were maintained.

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