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Case Report

Methanol poisoning induced chronic neurological sequelae

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ABSTRACT

Acute methanol intoxication can have serious neurological sequelae which can entail the course of illness. Patients generally present with acute neurological, visual, and gastrointestinal symptoms. Here we present a case of methanol poisoning who developed profound vision loss and severe brain damage with characteristic bilateral necrosis of the putamen shown on magnetic resonance imaging. This study highlights a unique undiagnosed case report of blindness associated with extrapyramidal symptoms, which was later found to be associated with methanol poisoning. A high index of suspicion is required for timely management of such cases, to prevent the long-term neurological sequelae.

Keywords: Methanol poisoning, Blindness, Putaminal necrosis, Extrapyramidal symptoms

INTRODUCTION

Methanol is an adulterant substance found in alcohol. Methanol ingestion can lead to toxic effects due to its metabolite formic acid, preponderantly on the retina, optic nerve, and other parts of the central nervous system (CNS), primarily in the putamen. The accumulation of formic acid results in metabolic acidosis, damage to the basal ganglia, and visual impairment. Here we discuss a similar case with history of sudden loss of vision following the consumption of an adulterated alcohol, followed by development extrapyramidal symptoms over time.[1]

CASE REPORT

A sixty-year old male was brought to the outpatient department, from a rehabilitation center, who was recovering from sudden loss of vision, following the consumption of adulterated alcohol (methyl alcohol) six months ago. History was suggestive of an episode of nausea, vomiting, headache, visual disturbances, and shortness of breath, which persisted for several hours after drinking an unknown amount of local liquor which occurred six months ago. For the episode, patient was conservatively managed at a local clinic and patient was discharged with acute blindness. Over the course of past six months, gradually patient developed a history of frequent falls, and difficulty in walking, which later progressed to inability to bear weight on his feet, following which the patient was brought to our hospital.

On examination

The patient had hypertension with a blood pressure (BP) of 160/100 mmHg, other vitals were normal. Patient was alert but had had mood disturbances, and deficits in recent and remote memory were reported. He had mask like facies, drooling of saliva and reduced eye blinking. Patient had generalized cogwheel rigidity.

The deep tendon reflexes were exaggerated at 3+, bilaterally in both upper and lower limbs. All sensory modalities were patent bilaterally. Ataxia was present.

Ophthalmological examination

Direct and indirect pupillary reflexes was impaired bilaterally. Visual acuity was decreased to finger counting

in both the eyes. The fundoscopic examination in both the eyes showed features of hypertensive retinopathy, in addition to optic disc atrophy.

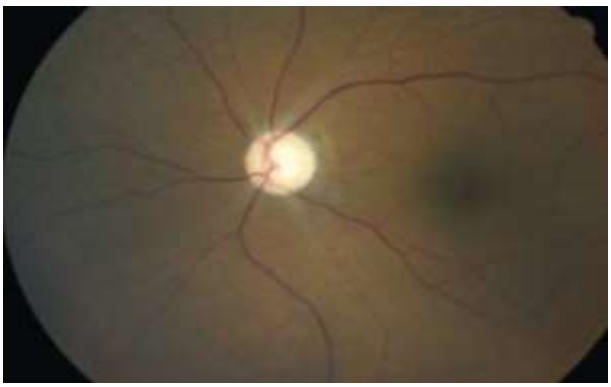

Figure 1: Optic disc on fundus examination.

In MRI brain, old infarcts were seen in bilateral basal ganglia.

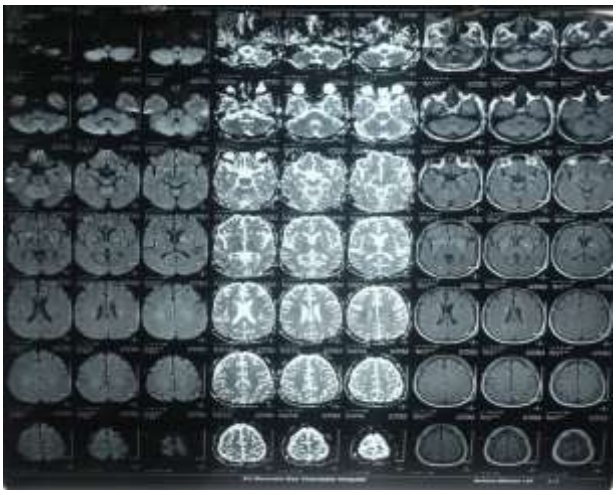

Figure 2: Hyperintensities in the bilateral basal ganglia.

Patient was put on physical rehabilitation therapy. The blindness and extrapyramidal symptoms were irreversible, which requires the need to bring forth such cases into attention.

DISCUSSION

Methanol poisoning is a dreaded condition which can occur via ingestion of adulterated alcohol, or via dermal exposure to paint thinners, hand-sanitizers and windshield-cleaner fluids which have methanol as a base. It carries a mortality rate ranging from 18 to 44%. The problem is more severe in the developing countries especially in the lower socio-economic strata where consumption of adulterated alcohol can lead to grave patient prognosis. It is similar to ethanol in physical properties, but cheaper, thus making methanol fluids a cheap alternative to adulterate ethanol, except that it is highly toxic and even lethal. Many outbreaks of methanol poisoning have also been encountered in the past.[2]

Symptoms of acute methanol intoxication begin 0.5-4 hours after ingestion and include gastrointestinal symptoms manifestations such as abdominal pain, vomiting and neurological depression with visual blurring. After a latency of 6-24 hours, decompensated high anion gap metabolic acidosis and a high serum osmolal gap is seen, with blurring of vision, progressing to early or late blindness. Blurred vision with normal consciousness should create a strong suspicion.[3] Vision loss occurs due to affinity of formic acid, which is a toxic metabolite of methanol, to the optic pathway. Blurred vision with normal consciousness should create a strong suspicion in our mind.[4]

Acute poisoning is managed by limiting the production of formic acid by treating with ethanol, or the antidote fomepizole which is an inhibitor of alcohol dehydrogenase. Hemodialysis is indicated in severe metabolic acidosis or end organ failure. The acute phase of methanol-induced optic nerve damage is believed to resemble optic neuritis in its pathophysiological course. Treatment includes the use of iv glucocorticoids, at a dose of 1 g methylprednisolone over 3 days, followed by oral prednisolone at 1 mg/kg body weight. Neuroimaging helps in distinguishing methanol poisoning from other causes of unconsciousness in alcoholics, which includes hypoglycemic brain damage, carbon monoxide poisoning and head injury. Bilateral basal ganglia necrosis accompanied with the typical fundus examination findings, associated with the typical history, is diagnostic for methanol poisoning.[5]

A study on thirty patients with methanol poisoning by Anderson et al showed that neurologic manifestations were seen in nineteen patients, which included coma, seizures and decreased visual acuity.[6] Fifteen patients developed serious neurologic sequelae or died which was directly related to the time from ingestion of methanol to presentation at the hospital. Initiation of treatment within eight hours of ingestion, was associated with a better clinical outcome.

A retrospective study was done on 50 sequential patients with methanol poisoning, between 2008 and 2014 by Galvez et al.[7] All these patients drank unbranded alcohol in last 2-3 days and developed profound, painless, bilateral visual loss. These patients had bilateral optic atrophy, Extrapyramidal symptoms which includes tremors and rigidity developed in 12 patients, and 11 of 30 patients which underwent MRI, had putaminal necrosis.

Blindness occurs due to bilateral optic atrophy and extrapyramidal symptoms develop due to putaminal hemorrhagic necrosis. Aggressive treatment which includes oral ethanol can treat acute poisoning, but the occurrence of permanent blindness is usually not preventable in cases with late presentation.[8]

Zakharov et al evaluated 50 patients of Czech mass methanol outbreak in 2012, out of which 14% developed visual sequelae, and 12% were discharged with both visual and central nervous system sequelae of poisoning.[9] However, patients with positive serum ethanol were 93% less likely to have optic nerve damage.

CONCLUSION

Rapid recognization and aggressive management have been emphasized to prevent grave outcome of blindness in cases of methanol poisoning. The concurrent occurance of bilateral optic nerve damage and bilateral putaminal necrosis in a young or middle-aged male is very suspicious for methanol poisoning. Management of acute poisoning in time, could prevent the long-term neurological sequelae from happening. Additionally, stringent measures need to be taken by the governing authorities to prevent distribution of adulterated alcohols in the society.

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