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Case Report

Severe Brain Lesions with Severe Clinical Outcomes in Collective Methanol Poisoning

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Abstract

Acute methanol intoxication is a severe affection associated to high rates of mortality and morbidity, we report the cases of two men in circumstances of fall in prices had consumed an adulterated whiskey. The first patient was 55-year-old man, he developed polyneuropathy and cognitive impairment after a fraudulent adulteration of alcoholic drinks (whisky). Neuroimaging findings showed typical bilateral lesions in basal ganglia and atypical hyperintensities in the occipital, temporal, and frontal areas which have been rarely reported. The second patient was a 29-year-old man, he developed confusion wish had been resolved in three days. We describe the clinical presentations and we highlight the importance of intensive detoxification protocols to improve clinical outcomes.

ABBREVIATIONS

MR: Magnetic Resonance; ADC: Apparent Diffusion Coefficient

INTRODUCTION

Acute methanol intoxication is a severe affection associated to high rates of mortality and morbidity [1]. Brain lesions in survivors of acute methanol poisoning are variable, imaging typically described in the literature involves putaminal necrosis, with or without hemorrhage and subcortical white matter lesions [2]. However, brain lesions at other sites may occur as we describe in this manuscript. Metabolic acidosis is the main biological feature of poisoning [2,3]. Two men in circumstances of fall in prices had consumed an adulterated whiskey.

We describe the case of the first patient which has been transferred in our department. He was a 55-year-old man who developed cognitive impairment, parkinsonism and polyneuropathy after fraudulent adulteration of alcoholic drinks (whisky). We will describe the MRI findings in a case of severe methanol poisoning and we will highlight the importance of intensive detoxification protocols to improve clinical outcomes. The second was a 29-year-old man, he was presented spatiotemporal disorientation.

CASES PRESENTATION

We report the first case, he was 55 year- old man without any past medical history. He was a chronic alcoholic. Following a fall in prices, he consumed a high dose of adulterated whiskey and consequently He suffered consciousness impairment. At admission on the emergency, the Glasgow Coma Scale rating was 3. Oxygen saturation was 96%, Blood pressure was 19/9 mmHg, pulse rate was 82 and pupillary assessment revealed bilateral meiosis. The patient was immediately mechanically ventilated and sedated.

Laboratory tests including serum glucose levels were normal. There was no signs of renal failure or rhabdomyolysis but a severe metabolic acidosis was noted with PH at 7,22. Blood and urine toxicological screenings were positive. Blood methanol level, measured approximately 6 hours after was 4.6 g/l.

CT scan done at H6 showed hypodensity in the putamina and caudate nucleus. He beneficiated of intensive care and correction of metabolic acidosis by chloride acid solution, vitamin therapy but no antidotal treatment or hemodialysis. After72-hour, he regained normal consciousness. In day 5 all laboratory studies were normal, and methanol level regained 0 g/l so he was addressed to our department of neurology. Clinical examination revealed temporo-spatial disorientation, apathy and memory disturbances. Motility exam showed Parkinsonian motor symptoms with rigidity in four limbs, shuffling gait, and hypokinesis without tremor. We noted also absent tendon reflexes and visual disturbances.

Magnetic resonance (MR) performed on day 15 after methanol poisoning, showed bilateral and symmetric hypointensity on the T1-weighted images in the striatum and the corona radiata. T2 FLAIR-weighted images (Figure 1) showed bilateral and symmetric hyper intense lesions in the striatum, globus pallidus, corona radiata and hippocampus with extensive

cortical involvement predominant in in the occipital regions. Diffusion sequence (B1000) confirmed the below described lesions (Figure 2a,2b) appearing as hyperintensities with mild Apparent Diffusion Coefficient (ADC) decrease. No hemorrhagic lesions were found on T2* sequences.

So, the patient was treated with vitamin B1, folic acid and physiotherapy. On day 25 he regained better neurologic state with less extrapyramidal symptoms, no gait disturbance. But he still had cognitive impairment, a difficulty with planning and organizing and a difficulty with coordination.

The second patient with his uncle, consumed a small amount of whisky, his parents reported that he had incorrect responses, he was disoriented to person, place and time. He was incoherent and had diminished concentration, he exhibited bizarre behavior but we hadn't noted any abnormalities in physical examination, the family decided not to pursue management and we were not able to make the explorations. Due to the uncle family, we have been able to track the evolution of the nephew, who recovered a normal conscience the third day.

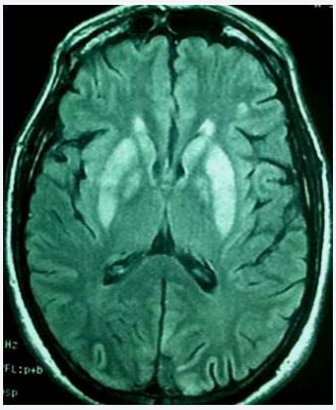

Figure 1 T2-FLAIR : The atypical lesions in case were those in corona radiata, hippocampus and occipital regions.

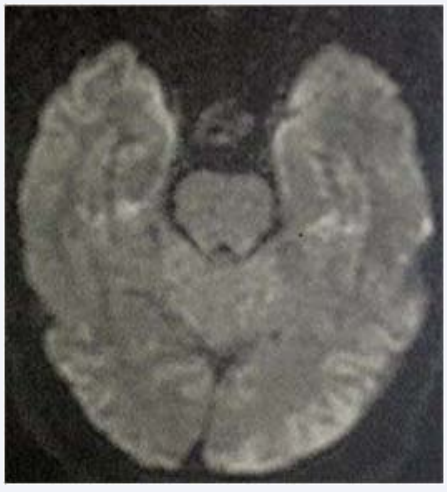

Figure 2a Diffusion (B1000), Bilateral hyper intense lesions in the striatum, globus pallidus, corona radiata and hippocampus with extensive cortical involvement predominant in in the occipital regions.

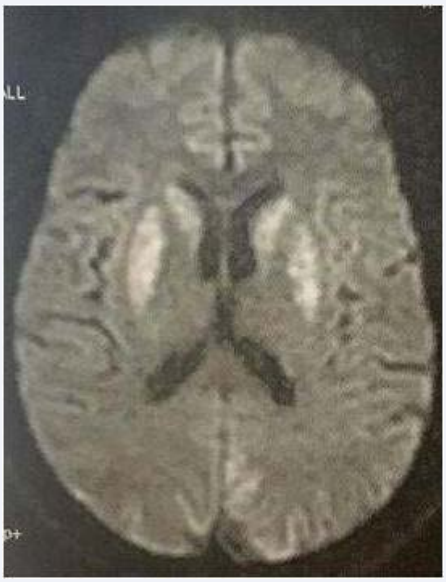

Figure 2b Diffusion (B1000), Symmetric hyper intense lesions in the striatum, globus pallidus, corona radiata and hippocampus with extensive cortical involvement predominant in the occipital regions.

DISCUSSION

Acute methanol poisoning is a relatively uncommon and dangerous form of intoxication. It generally occurs after suicidal or accidental events and can be potentially fatal if not diagnosed and treated promptly [4,5]. The main feature of this intoxication is metabolic acidosis due to formic acid, alcohol dehydrogenase metabolizes methanol to formaldehyde, which is then rapidly converted to formic acid by several enzyme systems in the body. Formate interrupts mitochondrial respiration by inhibiting cytochrome c oxidase activity, which leads to tissue hypoxia and lactate formation [4].

Symptoms may include a decreased level of consciousness, poor coordination, vomiting, abdominal pain, and a specific smell on the breath. Long term outcomes may include blindness and failure. The clinical findings initially observed in the first case were very similar to those previously reported [1,4,5]. The main feature was coma with severe metabolic acidosis and the diagnosis was confirmed by the presence of high serum methanol levels [1,4]. Progressive neurological deterioration as coma correspond to toxic level of methanol (20.00 mg/dl), in the second case the clinical presentation was clumsy, despite the absence of special blood tests we can explain this by the small amounts of whisky consumed,

In terms of radiologic findings, almost all of the patients who survived acute methanol poisoning had pathological findings by MRI [1,4]. The MRI lesions are variable; it can be hemorrhagic or non-hemorrhagic. Cerebral and intraventricular hemorrhage, cerebellar necrosis, and diffuse cerebral edema all have been described. Bilateral subcortical white matter necrosis, lesions in the brainstem, lesions in the region of the Globuspallidus have been also reported. But The predilection areas concerned bilaterally symmetric striatal and optic nerve lesions [1,2,4,5]. The basis for the selective vulnerability in these regions remains

unknown. It is probably a combination of factors, including cerebral microvascular anatomy and direct toxic effects of methanol metabolites [4]. The atypical lesions in our case were those in corona radiata, hippocampus and occipital regions (Figure 1). Clinically the first patient had presented several complications such as visual disturbances, extrapyramidal syndrome, neurogenic peripheral syndrome and dementia [1,3,4]. Progressive neurological deterioration observed in this case coincides with the advanced stages of poisoning [1], In our knowledge, there are few other reported observations of such severe Brain lesions following acute methanol intoxication. We can explain this severe outcome to the non-utilization of any antidote. Many therapeutic procedures have been described, include gastric lavage, detoxification with methanol, and secondary detoxification with hemodialysis Moreover, the uses of gastric lavage and activated charcoal in unconscious patients have been generally discouraged due to the high risk of aspiration pneumonia, so favorable outcomes depended on a prompt diagnosis of methanol poisoning, standard supportive care, and correction of metabolic acidosis, as well as the immediate institution of methanol detoxification protocols [1,3].

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