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Simultaneous Events and Subsequent Disabilities of Homemade Alcohol Consumption: A Case Report

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ABSTRACT

A 42 year old man with heavy drinking of homemade alcohol was referred to our hospital due to acute chest pain. At the onset of admission, he developed a cardio respiratory arrest and CPR was started immediately. Heart rate and respiratory rate returned to normal range after 5 minutes of CPR; however LBBB was detected in ECG. He underwent emergency coronary angiography and normal coronary arteries were reported. After CPR, he was unconscious. Vital signs on admission in emergency department of clinical toxicology were: BP: 100/60, PR: 112/min, and RR: 24/min. He was intubated and underwent mechanical ventilation. Pupils were mydriatic and nonreactive to light. Physical examination revealed no pathologic sign in lungs, heart and abdominal organs. Gasometry on admission revealed: pH: 7.13, p CO_2: 26.8, and HCO_3: 9. Serum methanol level was: 61 mg/dl. 300 ml of Ethanol 20% as loading dose and then 30 cc per hour was initiated via gavage, folic acid was prescribed; haemodialysis was performed due to loss of consciousness, high methanol serum level and severe metabolic acidosis. Serum Urea and Cr were: 43 and 2.5 mg/dl respectively on admission and increased to 72 and 3.1 mg/dl respectively on second day after admission. He underwent two other sessions of hemodialysis. Brain MRI was carried out and bilateral ischemic hemorrhagic lesions in basal ganglia were reported. He was a case of methanol and ethanol toxicity who developed acute chest pain, loss of consciousness, bilateral hemorrhagic necrosis of Basal ganglia, acute tubular necrosis and finally blindness as a consequence of optic neuritis following methanol toxicity.

Key words: Homemade alcohol, Events, Disability

1. INTRODUCTION

Alcohol poisoning is considered as one of the main preventable causes of mortality and morbidity in many countries. Although heavy drinking has been reported to be less than 1% in alcoholics in Iran, most of alcohol toxicity cases and their consequences are not reported because alcohol consumption is legally banned and despised based on religious and legal grounds (1, 2) Selling and drinking alcohol is also a punishable offence. However, homemade alcohols are made and traded illegally despite all protectiveness (2). In addition to numerous adverse consequences of ethanol addiction, such as liver dysfunction in different stages, a variety of malignancies, pancreatitis and etc., methanol as a probable compound in homemade alcohol can lead to optic neuritis, severe metabolic acidosis, acute renal failure and mild to severe grades of altered mental status (3-5). In previous studies, Glasgow Coma Scale (GCS) of less than 8 and a serum pH level of less than 7, particularly in the presence of inadequate hyperventilation, have been mentioned as predictors of poor prognosis in methanol poisoning cases (6). In this report, we will show a case of alcohol toxicity who suffered multiple complications over a short period of time.

2. CASE PRESENTATION

A 42 year old man who had consumed considerable amounts of homemade alcohol was referred to hospital due to acute chest pain. He described pain as a feeling of pressure in his chest. Likewise, he was suffering from nausea and vomiting at that time. At the onset of admission he developed a cardio respiratory arrest and cardio pulmonary resuscitation (CPR) was started immediately. Heart rate and respiratory rate returned to normal range after 5 minutes of CPR while left bundle branch block (LBBB) was detected in Electrocardiography. It was reported that he underwent emergency coronary angiography and normal coronary arteries. The consciousness was low and he responded only to painful stimuli at that time; he was transferred to emergency department of clinical toxicology (EDCT). Vital signs on admission in EDCT were: systolic/diastolic Blood Pressure: 100/60, Pulse Rate: 112/min, and Respiratory Rate: 24/min. He was intubated and mechanically ventilated. Pupils were mydriatic and nonreactive to light. Physical examination revealed no pathologic sign in lungs, heart and abdomen. Extremities were symmetric and normal in term of size and pulse condition. ECG pattern was normal and gasometry on admission revealed: pH: 7.13, P CO_2: 26.8, and HCO_3: 9. Serum methanol level was: 61.mg/dl. 300 ml of Ethanol 20% as loading dose and then 30 cc per hour was initiated via gavage, folic acid was prescribed in dose of 1mg/kg per 4 hours and haemodialysis was performed due to loss of consciousness, high methanol serum level and severe metabolic acidosis (7). Fomepizole is not accessible in our setting. Urea and Cr were 43 and 2.5 mg/dl respectively on admission and increased to 72 and 3.1 on second day after admission. Certain phosphokinase (CPK) was 13620 mg/dl on admission and turned back to 764 on the eighth day after admission. He underwent hemodialysis for next two other sessions due to persistent loss of consciousness; kidney function came back to normal on eight day. Brain MRI was carried out and bilateral ischemic hemorrhagic lesions in basal ganglia were reported (Figure 1. A, B).

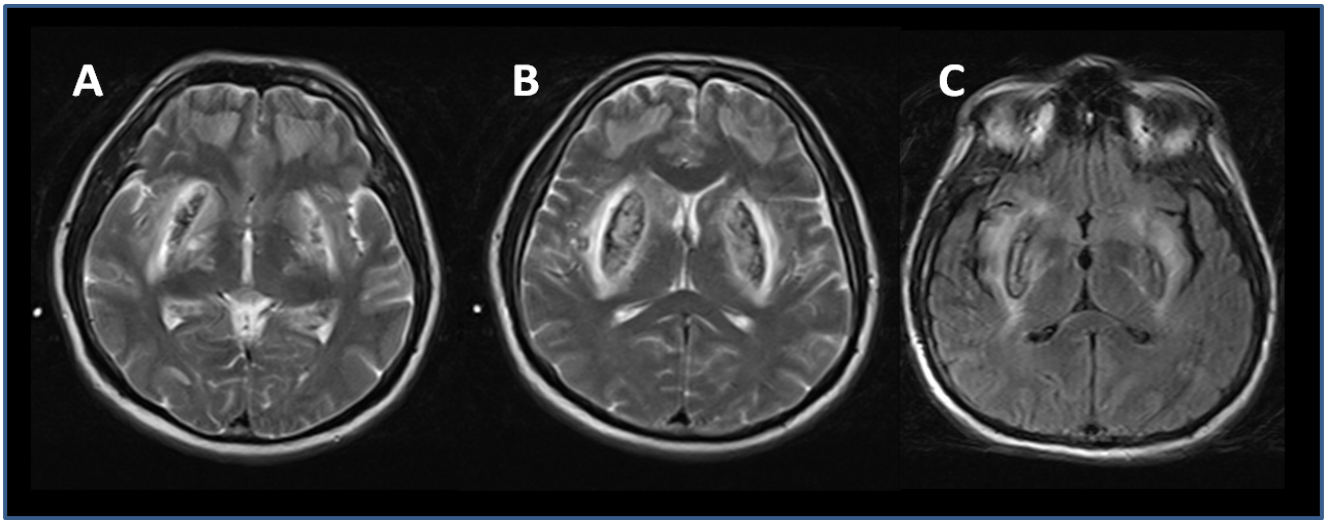

Figure 1. A, B: T2W images of brain MRI show massive ischemic hemorrhagic lesions in bilateral basal ganglia. C: flare image of brain MRI after two weeks shows previous findings without edema

After nineteenth day, he did not need mechanical ventilation support and on 21st day after admission, O_2 therapy by nasal canola simply provided appropriate arterial O_2 saturation. He was able to answer the questions partially, although had lost visual acuity. Ophthalmology consults revealed optic nerve atrophy secondary to toxin induced optic neuritis. Brain MRI (Figure 1, C) after two weeks represented previous findings while edema was resolved. This report provided evidence of a case with homemade alcohol toxicity that developed acute chest pain, loss of consciousness that was persistent for about 20 days, rhabdomyolysis and reversible acute renal failure, bilateral hemorrhagic necrosis of basal ganglia and finally blindness as a consequence of optic neuritis following methanol toxicity.

3. DISCUSSION

Several distinct complications were detected in our patient. Acute chest pain in this case might be due to some reasons; acute alcohol consumption leads to changes in esophagus and stomach motility and may be followed by gastroesophageal reflux and probably esophagitis. Gastric mucosa may be damaged and may cause hemorrhagic lesions following alcohol abuse (8). Our case didn't have any sign of bleeding in gastrointestinal tract and had no history of bleeding or other gastrointestinal problems before the recent admission; treatment by pantoprazol as a proton pump inhibitor for suppression of acid secretion was prescribed once he was admitted. Variant angina is a known consequence of ethanol consumption (9, 10). In this case, no evidence was found to confirm coronary artery spasm; although, transient spasm cannot be completely ruled out. Acute pancreatitis is a known complication of excessive alcohol consumption. The pathophysiology of alcohol-induced pancreatitis is complex. Some reasons such as gene expression changes in pancreas, changes in

homeostasis of cytosolic calcium and oxidative stress seem to have a role (11-13). In our case, amylase and lipase amounts were at normal range: 54 and 42 U/L respectively and patient had a typical chest pain and not pain or tenderness in epigastric region or other sites in abdominal examination. Impairment of adenosine triphosphate pump function, alteration in the sarcoplasmic reticulum and muscle membrane breakdown are mechanisms by which ethanol causes rhabdomyolysis (13). Acute renal failure is also considered as one of complications of methanol toxicity although it is not common (7, 14). The case of this study had an increased serum level of urea and Cr in addition to decrease in urine output on first and second day after admission but after one session hemodialysis followed by hydration and close monitoring of electrolyte balance, kidney function tests showed normal results on fifth day. Likewise CPK turned back to 764 mg/dl and 93 mg/dl on eighth and fourteenth day respectively. There are few studies on prognosis of acute renal failure in patients with alcohol toxicity (13). A study in 2013 reported two cases of acute renal failure following binge drinking in absent of any document for rhabdomyolysis or NSAID co-ingestion. In the aforementioned investigation, renal function was turned back to normal by conservative treatments (15). Putaminal necrosis which might be with or without hemorrhage is considered as the most characteristic finding in brain MRI of cases with methanol poisoning (16-18). In the present study the consciousness of patient decreased in few hours after alcohol consumption and brain MRI revealed bilateral ischemic hemorrhagic lesions in basal ganglia in the first day of admission. Brain MRI on discharge time presented the same picture. Visual sequela due to methanol toxicity is a known and disabling characteristic of methanol exposure (19, 20). Only 10 milliliter of methanol may result in blindness (19). Many cases are admitted in EDMT in Mashhad due to complications of methanol toxicity following consumption of homemade alcoholic beverages each year. Unfortunately, optic neuritis and loss of visual acuity was a persistent sequela in our case.

4. CONCLUSION

Although alcohol consumption is banned in Iran, homemade alcoholic beverages impose a burden of events and disabilities in individuals who illegally consume them. Unrecorded and noncommercial alcohol remains a considerable concern that needs special attention.

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AUTHORS CONTRIBUTION

This work was carried out in collaboration among all authors.

CONFLICT OF INTEREST

The authors declared no potential conflicts of interests with respect to the authorship and/or publication of this article.

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