CASE REPORT

A Case Report of Putaminal Hemorrhage Due to Methanol Toxicity; is Hemodialysis the Offender?

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Abstract

Introduction: Methanol toxicity is a life-threatening condition which is rare in developed countries but common in developing countries. Bilateral putaminal necrosis and hemorrhage are potentially two lethal consequences of methanol toxicity which may be due to direct neurotoxicity of methanol metabolites, especially formic acid, or the consequences of acidosis and hypoxemia in the course of poisoning. Hemodialysis is an important part of the treatment of methanol toxicity and some researchers believe that heparin which is administrated during the hemodialysis may be the cause of putaminal hemorrhage
Case report: We report a-32-year old man who presented with acute symptoms of methanol toxicity. A day after hemodialysis he suffered from seizure and Parkinsonism, and the neuroimaging revealed bilateral putaminal hemorrhage. Treatment with Levodopa-carbidopa was introduced for the management of Parkinsonism and finally the patient was discharged with marked improvement of symptoms and relative independency in daily activities
Discussion and Conclusion: Our patient suffered from a late manifestation of methanol intoxication, bilateral putaminal hemorrhage, and necrosis. This appearance along with subcortical white matter involvement are the most common abnormalities of methanol toxicity in the brain imaging which can be associated with peripheral enhancement. Based on the reported case and review of present evidences, it is suggested that putaminal hemorrhage in methanol toxicity can be due to anticoagulant agents used in hemodialysis

Keywords: Putamen hemorrhage, Toxicity, Hemodialysis, Methanol

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INTRODUCTION

Methanol toxicity is a life-threatening condition which is rarely observed in developed countries but it is more common in developing countries, especially those in which alcohol use is banned (1). It can cause a wide range of neurological complications including loss of consciousness, severe optic neuropathy, bilateral putaminal hemorrhage, delayed Parkinsonism, and seizure (2-4). Bilateral putaminal necrosis and hemorrhage are potentially the lethal consequences of methanol toxicity which may be due to direct neurotoxicity of methanol metabolites, especially formic acid, or the consequences of acidosis and hypoxemia in the course of poisoning (5, 6). Haemodialysis is an important part of the treatment of methanol toxicity and some of the indications include end organ damage (7), methanol level above 50 mg/dl , coma , seizure, new visual defect, and acidosis with pH below 7.15 (8, 9). Some scholars believe that heparin which is administrated during the hemodialysis may be the cause of putaminal hemorrhage (10, 11). We present a case of bilateral putaminal hemorrhage in the context of methanol toxicity and hemodialysis.

CASE REPORT

A-32-year old man referred to our hospital with Parkinsonism and gait disturbance. Twenty days before, he was admitted in another hospital with confusion and vomiting after methanol ingestion. On the first admission, the brain computed tomography (CT) scan was normal. He underwent hemodialysis for the treatment of methanol toxicity and immediately became conscious and responsive. On the next day, he presented a generalized tonic-clonic seizure and became progressively rigid in all four limbs and had some occasional abnormal jerky movements of the limbs (myoclonus perhaps).

During the examination, he was awake and aware. The vital signs and general examinations were all normal. The findings in focused neurologic examination were hypomimia, reduced eye blinking, positive Myerson sign, bilateral cogwheel rigidity, and prominent bradykinesia in four limbs. Speech was fluent but hypophonic and monotonous. Other neurologic examinations including fundoscopy and cranial nerves were also normal.

Laboratory study including platelet count, prothrombin

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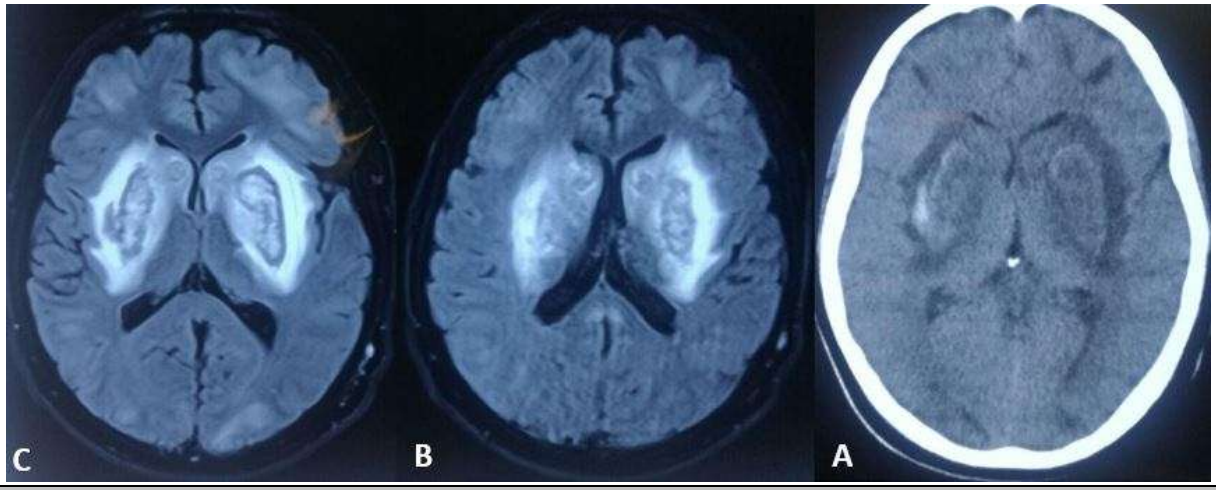

Figure 1.A. There are mixed hyperdense and isodense lesions in bilateral putaminal region with peripheral hypodensity in brain CT scan suggesting bilateral acute to subacute putaminal hemorhage. B&C: Axial plane of diffusion-weighted and FLAIR sequences demonstrates a heterogenouse signal change in favor of subacute hemorrhage, gliosis and surrounding edema.

time (PT), partial thromboplastin time (PTT), liver and kidney function tests, and electrolytes were all in normal ranges. The brain imaging revealed bilateral putaminal hemorrhage and necrosis with marked perilesional edema. The Brain CT and magnetic resonance imaging (MRI) are shown in figure 1. In the current case, a treatment with Levodopa-carbidopa was introduced for the management of Parkinsonism and finally the patient was discharged with marked improvement of symptoms and relative independency in daily activities.

DISCUSSION

Our patient suffered from a late manifestation of methanol intoxication, bilateral putaminal hemorrhage, and necrosis. Beside those findings, it was identified that subcortical white matter involvement is the most common abnormality of methanol toxicity in the brain imaging which can be associated with peripheral enhancement (12).

The damage to basal ganglia may be derived from direct effect of toxic metabolite of methanol, formic acid, and the neural cells (13). Why the putamen is more vulnerable to the toxic effects of formic acid? It is not clear, but high metabolic demand of putamen or its microvascular structure may play a role (11, 13). The main site for intracerebral hemorrhage (ICH) is the putamen and about 45% of hypertensive-related ICH (14). In addition, hypoxia and acidosis in the context of methanol intoxication maybe a precipitating factor (15). Moreover, some factors including anoxia, acidosis, electrolyte derangements, and coagulopathies may contribute to this catastrophe.

Some researchers have pointed to the role of heparin injection during hemodialysis as a causative factor of putaminal hemorrhage (10, 11). Likewise, the manifestations of putaminal damages in our patient began just after the hemodialysis, following a short period of symptoms remission. In this regard, Iseki et al. reported that the relative risk of cerebral hemorrhage in chronic dialysis patients is 10.7 compared to normal population (16). In addition to vascular changes in the course of chronic kidney disease, heparin is the preferred anticoagulant agent used in hemodialysis and may be a causative factor of ICH. We suggest that the heparin used in hemodialysis for the treatment of acidosis and toxic effects of methanol can induce the hemorrhagic infarction in the setting of putaminal necrotic lesions.

CONCLUSION

Bilateral putaminal necrosis is a rarely reported but characteristic finding of methanol intoxication, the anticoagulant can enhance the toxicity.

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