Case Report

Bilateral Putaminal Hemorrhages: Serious Complication of Methanol Intoxication

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Abstract

Context: Methanol intoxication is a life-threatening condition. Hallmark of clinical presentations include severe wide anion gap metabolic acidosis with very high serum osmolar gap and visual complication. **Case Report:** We report a case of severe methanol intoxication with bilateral putaminal hemorrhage, an uncommon serious complication. A 56-year-old man presented with altered mental status. Fundus examination showed optic disc edema. Arterial Blood Gas (ABG) revealed severe anion gap metabolic acidosis with osmolal gap. Head computed tomography (CT) showed hypodense lesions in basal ganglia bilaterally. Hemodialysis and intravenous fomepizole were initiated. Serum methanol level was significantly elevated. Unfortunately, patient was lethargic 2 weeks after discharge. Repeated CT of head demonstrated new putaminal hemorrhages. **Conclusion:** Bilateral putaminal hemorrhage is an uncommon but serious complication in methanol intoxication. Clinicians should have high index of suspicion for putaminal hemorrhage when patients with recent methanol intoxication present with altered mental status.

Keywords: Basal ganglion hemorrhage, Metabolic acidosis, Methanol intoxication, Putaminal hemorrhage

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Introduction

Methanol or wood alcohol is commonly found as a component of household products including paint removers or windshield washer fluid. Due to catastrophic consequences of methanol consumption, it cannot be used as an alcohol beverage. We report a case of middle aged man presented with confusion, severe metabolic acidosis secondary to methanol intoxication. A repeat CT scan of brain revealed bilateral putaminal hemorrhage as a late complication of methanol poisoning.

Case Presentation

A 56-year-old man with history of episodic alcohol abuse and rectal adenocarcinoma status post-surgery and neoadjuvant chemoradiation therapy presented to emergency department with altered mental status. On admission, he was comatose with dilated pupils and minimal reaction to light. Fundus examination showed optic disc edema without evidence of retinal hemorrhage. Arterial Blood Gas (ABG) revealed severe metabolic acidosis; pH 6.89, pCO_2 13, pO_2 178, HCO_3 < 3. Blood tests also demonstrated anion gap and osmolal gap. Head computed tomography (CT) showed new hypodense lesions in basal ganglia bilaterally and old small vessel ischemia changes in periventricular matter [Figure 1a].

Emergent hemodialysis (HD) and treatment with intravenous fomepizole were initiated. Serum methanol level, obtained after hemodialysis, came back significantly elevated. Ophthalmologist was consulted for visual defect and diagnosed the patient with legal blindness without other focal neurological deficit prior to discharge. Type of alcohol, consumed by the patient, was still unknown. Unfortunately, patient developed confusion and lethargy 2 weeks after discharge. New CT of head without contrast demonstrated new hyperdensities in putaminal area compatible with the diagnosis of putaminal hemorrhages [Figure 1b].

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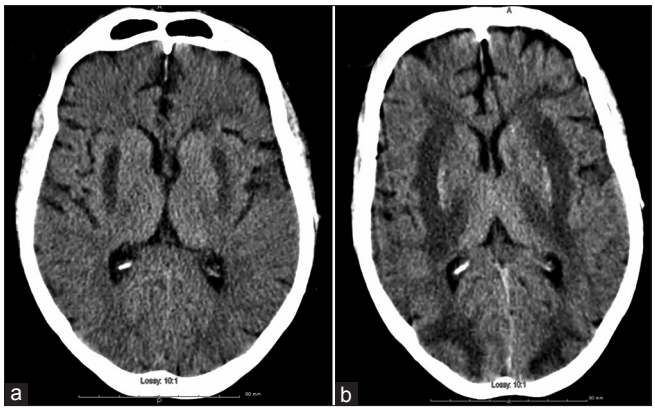

Figure 1: (a) Head computed tomography (CT) showed new hypodense lesions in basal ganglia bilaterally and old small vessel ischemia changes in periventricular matter. (b) New CT of head without contrast demonstrated new hyperdensities in putaminal area compatible with the diagnosis of putaminal hemorrhages

Discussions

Methanol intoxication is a life-threatening condition. Bilateral putaminal hemorrhages, complication carrying very high morbidity, have been described in methanol intoxication and can be found in other diseases including inborn error metabolism, renal failure with severe metabolic acidosis, and ethylene glycol intoxication.[1,2] The proposed underlying mechanism is acid-induced neural cell death.[3] In addition to putaminal hemorrhage, another important complication is visual loss. The mechanism is currently presumed to be formic acid-induced retinal damage from animal models.[4] There are no studies conducted in human for the pathophysiology of methanol optic neuropathy. There was one recent research demonstrating the role of intravenous erythropoietin with oral prednisolone to improve a visual acuity in the patients with optic neuropathy.[5]

Our case presentation demonstrates that even appropriate treatment, promptly initiated. The patient still developed putaminal hemorrhage. Clinicians should have high index of suspicion for putaminal hemorrhage when patients with recent methanol intoxication present with alter mental status, confusion or behavioral change.

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