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**Case Report** 

# Severe Intracerebral Hemorrhage and Decompressive Craniectomy in a Case of Severe Methanol Intoxication - @

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#### ABSTRACT

Methanol poisoning is a life-threatening intoxication and a public health problem. Intra-cranial hemorrhage is a rare complication with this intoxication. We report a 21 years old male with a very rare case of severe intra-cranial hemorrhage complicating severe methanol intoxication and required decompressive craniectomy. The initial presentation was coma with high anion gap metabolic acidosis. Patient received initial resuscitative measures, antidote: 4 methyl-pyrazole and hemodialysis. The initial CT Brain showed bilateral basal ganglia hypodensities. The follow up MRI and CT Brain showed bilateral hemorrhagic transformation with mass effect and subfalcine herniation. Urgent decompressive craniectomy was done with evacuation of hemorrhage and placement of Endo Ventricular Drainage (EVD). Patient had prolonged mechanical ventilation and required tracheostomy at day 13. He was discharged from ICU after 23 days.

#### **INTRODUCTION**

Methanol is a rare but life-threatening intoxication. Coma, Visual impairment and severe metabolic acidosis are the most common complications of this intoxication [1,2]. Intra-cranial hemorrhage complicating basal ganglia ischemia is rare and only reported in some case reports.

We report in this case a very rare case of severe intra-cranial hemorrhage complicating severe methanol intoxication and required decompressive craniectomy.

#### **CASE PRESENTATION**

Twenty one-year-old male patient presented to our Emergency Room (ER) with six hours history of nausea, vomiting and diffuse abdominal pain with blurred vision and dizziness. Upon arrival to ER, he was found to be with Glasgow Coma Scale (GCS) less than 9 and hemodynamically stable: BP =113/76 mmHg, HR =130 bpm. There was a severe metabolic acidosis with pH 6.91 and bicarbonate 3.1 mmol/l. The family reported a history of taking multiple kinds of pills and local made alcohol from 24 hours.

He was afebrile, temperature 37.1° C with no signs of meningeal irritation. He was intubated in ER to secure airways. Initial CT brain done after stabilization and revealed bilateral basal ganglia (putamen) hypodensities (Figure 1).

The patient was shifted to ICU, connected to mechanical ventilation, and toxicology screening revealed high level of methanol with initial concentration of 228 mg/dl. Funduscopic exam was unremarkable. Patient was given Sodium Bicarbonate and Fomepizole loading dose 15mg/kg. Conventional hemodialysis was initiated and was conducted for two times within the first 24 hours, each session lasted for 2 hours and was heparin free. The significant laboratory findings on admission and after hemodialysis are represented in table 1.

On day 3, patient remained unconscious in spite of no sedation and improvement of clinical and metabolic markers. MRI Brain (Figure 2) with contrast was done and revealed bilateral basal ganglia hemorrhages with bilateral lateral and fourth ventricular hemorrhage. There was also midline shift, mild hydrocephalus and optic nerve neuritis. Neurosurgery was consulted and decided for medical measures in form of hypertonic saline and follow up MRI.On day 4, as the patient was not improving neurologically, a CT brain was done and revealed the same bilateral hemorrhagic transformation, more on right side with mass effect at the ipsilateral ventricle causing midline shift and subfalcine herniation of 8 mm to the left (Figure 3).

Right sided decompressive craniectomy, evacuation of intracerebral hemorrhage and Endo Ventricular Drain (EVD) insertion were done (Figure 4). Patient was kept after that heavily sedated and received all neuroprotection measures with prevention



Figure 1: CT brain on admission: Bilateral putamen hypodensities.

Table 1: Laboratory findings on admission and after hemodialysis.			
	At admission	After First Hemodialysis	After Second Hemodialysis
рН	6.91	7.14	7.36
HCO3- (mmol/l)	3.1	14	21
Methanol (mg/dl)	228	124	undetectable
Anion Gap (mmol/l)	52.6	29.8	20.9
Lactate (mg/dl)	81.2	48.4	16.2



Figure 2: MRI brain on day 3 axial T2 showing bilateral lateral ventricular intraventricular hemorrhage with fluid-fluid level..



Figure 3: Follow up CT brain on day 4: Bilateral hemorrhagic transformation more on right side with mass effect.



of secondary systemic brain injury. The EVD was removed ten days after surgery. On day 13, tracheostomy was done as patient remained ventilator dependent. On day 15, he was weaned off mechanical ventilation.and discharged to medical ward after total of 23 days with left sided hemiparesis.

#### DISCUSSION

Our patient presented with severe methanol poisoning complicated by severe intracerebral hemorrhage. Methanol intoxication is usually underdiagnosed and occurs in outbreaks as it was reported before in Libya and Kenya [1]. The toxicity of methanol is due to its metabolite formic acid which is the result of metabolism of methanol into formaldehyde by the enzyme alcohol dehydrogenase. Twelve to 24 hours are needed for the production of formic acid which explains the usual latent period as observed in our case [2]. The formic acid is responsible for high anion gap metabolic acidosis. Increase in formic acid diffusion across cell membranes causes ocular toxicity, Central Nervous System (CNS) depression with coma and seizures as in our patient [2,3]. The most effective antidotes to treat methanol poisoning are ethanol and 4-methyl-pyrazole (Fomipizole). These antidotes act as a competitive inhibitor of the enzyme alcohol dehydrogenase found in the liver which transforms methanol to its toxic metabolite: formaldehyde which is metabolized rapidly into formic acid [2]. 4-methyl-pyrazole is more expensive but seems to be more effective than ethanol. It was the only available antidote in our facility. Intermittent hemodialysis is the modality of choice of extracorporeal treatment in the management of acute methanol poisoning. It has clinical and metabolic indications including coma, visual impairment, renal failure, severe metabolic acidosis with initial pH < 7.0 and initial plasma methanol concentration more than 50 mg/dl [4].

Our patient presented initially with basal ganglia ischemia and then later on hemorrhage. Ischemia and hemorrhage of basal ganglia and subcortical region especially putamen were described in several case reports. The exact mechanism is debated [5]. It could be due to the direct effect of formic acid in the most metabolic dependent area in the brain or due to the ischemia reperfusion injury. The heparinization during hemodialysis was also thought to be the origin of intra cerebral hemorrhage reported by Phang and al and Hassanian-Moghaddam, et al [6,7]. However, during the Czech mass poisoning outbreak between 2012 and 2014, 106 cases were reported and in this study, the authors did not find any association between systemic anticoagulation during hemodialysis and brain hemorrhages. In addition, the hemorrhagic lesions were more frequent than the nonhemorrhagic intra cerebral lesions [8]. Usually the prognosis is very bad with high mortality in case of basal ganglia hemorrhage related to severe methanol poisoning and even autopsy case was reported [9]. Some other rare cases were published with discussion of the mechanism inducing intra-cerebral hemorrhage [10-12]. Our patient had decompressive craniectomy with hemorrhage evacuation. His life was saved but patient developed neurological sequelae with left sided hemiparesis. To our knowledge, no cases of high intra cranial pressure requiring decompressive craniectomy related to severe methanol poisoning were published in recent literature. The role of decompressive craniectomy after stroke or traumatic brain injury was well debated in literature but in the particular context of methanol intoxication, no data are available in recent publications [13].

#### **CONCLUSION**

Methanol intoxication is still the silent metabolic killer. This is to our knowledge, one of the first cases of severe methanol poisoning complicated by severe intracerebral and basal ganglia hemorrhage that required decompressive craniectomy. The awareness of the severity of methanol intoxication, implementation of local protocols of management and the availability of antidote therapy are required to prevent mortality and significant neurological sequelae.

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