

# Pseudo-SAH in a patient with methanol poisoning

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## Abstract

**Objective:** Methanol poisoning is a dangerous life-threatening event, manifested with various symptoms, sometimes very rare ones, that all should be addressed to prevent misdiagnosis of the methanol-poisoned patients.

**Case Presentation:** A 21-year-old young man was brought to the emergency department (ED) with a generalized tonic-clonic (GTC) seizure and he was diagnosed with methanol intoxication. A non-contrast computed tomography (NCCT) of the brain demonstrated findings similar to subarachnoid hemorrhage (SAH). After the brain CT, he had a Glasgow Coma Score (GCS) of 3 and all brainstem reflexes were absent. Neurology consultant agreed with the diagnosis of pseudo-SAH. Brain death was confirmed by a positive apnea test within 24 hours of presentation.

**Conclusion:** It is suggested that compression of dural sinuses due to severe brain edema, reduces the venous drainage and leads to venous engorgement, which appears high attenuated in the background of low-density edematous brain matter.

**Keywords:** Methanol, Brain edema, Pseudo-subarachnoid hemorrhage, COVID-19, Toxicity

## Introduction

Following the spread of coronavirus disease 2019 (COVID-19) in China and in almost all parts of the world, including Iran, a false rumor has been spreading that ingesting alcohol could protect from this new coronavirus infection (1). Besides, due to the legal prohibition of alcoholic beverages in Iran, these drinks are made in unauthorized workshops and distributed illegally. Methanol can contaminate these traditionally fermented alcohols or sometimes some bootleggers sell the available industrial alcohol which is mostly methanol as a drinkable alcoholic beverage. Consequently, emergency departments (EDs) have faced a surge of methanol intoxicated patients in early March 2020, in the midst of COVID-19 pandemic (2). Here we present a case of methanol poisoning in COVID-19 outbreak in Iran.

## Case Presentation

A 21-year old boy was brought in the ED by emergency medical service (EMS) with a generalized tonic-clonic (GTC) seizure. EMS was called when his father found him unresponsive in his room at late night. Last night, he returned home after engaging in binge drinking. He was a previously healthy person without any significant past medical history, drug abuse or addiction. EMS reported that at the scene he had a Glasgow Coma Score (GCS) of 8, findings of urinary incontinence and a blood sugar of 85. He had an episode of GTC seizure en route to the hospital,

lasting about 5 minutes and terminated with intravenous administration of 5 mg diazepam. On arrival to the ED, he developed the second episode of seizure, which was an upward gaze, generalized body stiffening with tonic-clonic movements of extremities and desaturation of oxygen to 55%. The tonic-clonic movements stopped after 10 minutes with 10 mg of intravenous diazepam, but myoclonic jerks of the eyelids continued. After termination of seizure, he had a GCS of 6, symmetrical dilated pupils on both sides, blood pressure of 95/50, pulse rate of 110, respiratory rate of 26, temperature of 36.5°C, oxygen saturation of 92% on 8 L/min of oxygen via face mask and Babinski sign was present bilaterally. The patient became intubated and mechanically ventilated.

The results of laboratory studies, five hours after arrival were: glucose= 70 mg/dL, serum sodium= 128 mEq/L, potassium= 3.8 mEq/L, ionized calcium= 5.6 mg/dL, urea nitrogen= 28 mg/d, creatinine= 1.3 mg/dL, serum pH=6.7, HCO<sub>3</sub><sup>-</sup>=5, urine drug screen= negative, ethanol level= 14 mmol/L, and methanol level= 78 mmol/L.

Based on the diagnosis of lethal methanol intoxication, repeated doses of fomepizole and sodium bicarbonate were administered, hemodialysis was planned and a double lumen dialysis catheter was inserted. Non-contrast computed tomography (NCCT) of the brain was performed before hemodialysis, which showed diffuse cerebral edema with a defect in differentiation of the gray white matter, decreased attenuation of brain parenchyma



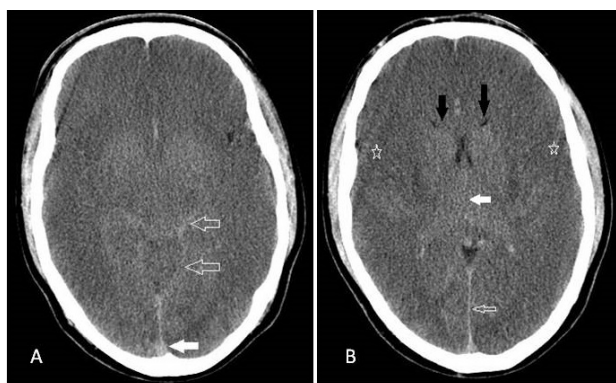
and hyper-attenuation of cerebral sulci, fissures and cisterns, and resembling subarachnoid hemorrhage (SAH) (Figure 1).

### Discussion

During March 20 and April 5, 2020, 136 patients with methanol intoxication presented to the ED of Imam Reza hospital. This hospital is the largest toxicology center in north-east of Iran. Out of these patients, 12 cases expired giving the mortality rate of 8.8%. This case was one of the mortalities. A repeated neurologic exam was performed after the brain CT: GCS was 3 and all brainstem reflexes were absent. Neurology consultant agreed with the diagnosis of pseudo-SAH based on the Hounsfield units (HU) of 40 at the basal cisterns and 41 at the tentorium and diffuse loss of gray white matter differentiation. Brain death was confirmed by a positive apnea test within 24 hours of presentation.

Pathologies mimicking SAH in Brain CT were first described by Spiegel et al in 1986 in 10 patients with diffuse brain edema without any evidence of SAH on autopsy (3). The mechanism for this phenomenon has not been fully recognized, but it is suggested that compression of dural sinuses due to severe brain edema, reduces the venous drainage of the brain and leads to venous engorgement, which appears high attenuated in the background of low-density edematous brain matter (4). Pseudo-SAH sign mostly arises in the base of a hypoxic-ischemic brain injury following resuscitation, but it has been reported in other contexts in the literature, including meningitis, subdural hemorrhage, polycythemia, or infarction (5,6). The phenomenon has been also reported in the literature after status epilepticus and its mechanism has been attributed to the brain damage, possibly on a hypoxia-ischemia basis or hypoperfusion (4).

Pseudo-SAH can be differentiated from SAH using the HUs on the CT. The HU values of high-density areas in



**Figure 1.** Non-contrast axial brain CT scan showing diffuse brain edema and loss of gray white matter differentiation. (A) Hypoattenuation of superior sagittal sinus (solid arrow) and quadrigeminal and ambient cistern (hollow arrows). (B) Hypoattenuation of inter-hemispheric fissure (hollow arrow), effacement of third ventricle (solid white arrow), both sides lateral sulcus ( ), frontal horns of lateral ventricles (solid black arrows)

pseudo-SAH are in the range of 30–40 HU, but in those with a true SAH, the HU values are higher (between 60 and 70 HU). Two other distinguishing features of pseudo SAH are symmetric densities, which are confined to the basal cisterns and generalized brain edema with basal cistern effacement (7,8).

### Conclusion

It is important for the emergency physicians to be familiar with the potential mimics of SAH and to recognize this phenomenon on the brain CT for a timely and accurate diagnosis and avoiding unnecessary workups and procedures.

### Authors' contributions

EP and MF conceived the idea and design of the article. EP, BZ and RH contributed to the preparation and review of the initial manuscript. All authors contributed in critical revision.

### Ethical issues

The patient and his family approved of using the data to be discussed in our case report without disseminating any personal information.

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