Case 13776

MRI detected bilateral putaminal haemorrhagic necrosis due to methanol intoxication
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Section: Neuroradiology
Area of Interest: Neuroradiology brain
Procedure: Diagnostic procedure
Imaging Technique: MR
Imaging Technique: MR-Diffusion/Perfusion
Special Focus: Acute Haemorrhage Case Type: Clinical Cases
Authors: Hidayatullah Hamidi1, Najibullah Rasouly2
Patient: 30 years, male

Clinical History:

A 30-year-old man was referred to undergo brain MRI from outside the hospital. According to the attendant, this previously healthy man had a history of intake of an unknown type and amount of alcohol one week ago. Thereafter he vomited and lost bilateral vision. At time of presentation he had been in an unconscious state for the past three days.

Imaging Findings:

Brain MR imaging was performed with 1.5T Siemens device.
Axial T1-Weighted Image (WI) at the level of basal ganglia showed slightly high signal component in bilateral putamina surrounding central low signal areas indicating haemorrhagic necrosis. (Fig. 1a)
Axial T2WI demonstrated inverse image of TIWI, as high signal central area (necrosis) was surrounded by low signal rim (haemorrhage) in bilateral putamina. (Fig. 1b)
Axial T2 FLAIR image confirmed the necrosis by intense high signal area in bilateral putamina. (Fig. 1c)
Axial T2* GRE image confirmed bilateral putaminal haemorrhage by depicting peripheral hypointense areas due to susceptibility artefacts surrounding central necrosis. (Fig. 1d)
Diffusion-weighted image (DWI) showed abnormal high signal in bilateral putamina with drop of signal on ADC representing diffusion restriction due to cytotoxic oedema. (Fig. 2a, b)
Peripheral rim of intense contrast enhancement was noted bilaterally (Fig. 3).
The lesions were nicely limited to putamina (Fig. 3b). No significant brain oedema or additional lesions were seen.

Discussion:

Background

Methanol is a potent central venous system (CNS) toxin. (1) Acute Methanol Intoxication (AMI) can occur as an accidental or suicidal event; however, fraudulent adulteration of wine is the common cause of AMI in developing countries. [2, 3]
Methanol is metabolized in the liver to formaldehyde and subsequently to formic acid, both of which are extremely toxic agents. In addition to systemic metabolic acidosis caused by accumulation of formic acid, AMI can cause optic nerve damage (necrosis and demyelination) [4] and CNS injuries (putamen being the most susceptible site [5]) [6],
hence AMI can cause a life-threatening condition and severe neurologic deficits. [3]

Clinical Perspective
Prompt diagnosis and treatment are essential. [6] The clinical presentation may be variable in individuals. [2] Usually a latent period of 12–24 hours precedes the clinical manifestation after ingestion. [7] Visual disturbance is the first symptom in many patients. [4] Other symptoms are headache, dizziness, malaise and gastrointestinal symptoms like nausea, vomiting and abdominal pain. Severe cases can result in dyspnoea, seizure, coma, permanent neurological deficit and death. [2, 4] Respiratory arrest is often the terminal event. [1]

The diagnosis is made based on metabolic acidosis, high anion and osmolar gap and high serum methanol levels. [2]

Imaging Perspective

Neuroradiological features of AMI are described in the literature. [8] Imaging helps in distinguishing AMI from other causes of acute unconsciousness in alcoholic patients such as hypoglycaemic brain damage, carbon monoxide poisoning or head injury. [3]

The most characteristic finding of AMI is bilateral putaminal necrosis [3, 4]. The necrosis can be haemorrhagic or non-haemorrhagic [1], however, haemorrhage is associated with poor prognosis. It should be kept in mind that bilateral putaminal necrosis is not specific to AMI, as it can also be seen in a variety of conditions, like Wilson and Leigh disease. [9]

Lesions of hippocampi, subcortical and deep white matter, cerebral and cerebellar cortex and midbrain are reported. [4] Cerebral and intraventricular haemorrhage [3] and diffuse cerebral oedema [2] can also occur. DWI can detect lesions that could not be depicted with CT or conventional T1 and T2WI sequences. [10]

Contrast enhancement is non-specific varying from non-enhancement to peripheral rim enhancement and even strong enhancement.

Outcome

Treatment consists of gastric lavage, administration of ethanol, fomepizole and cofactors such as folate, dialysis and alkalization. [1, 2] Survival depends on the amount of methanol ingested and prompt start of treatment. [10] Mortality remains high, mainly because of often difficult and hence delayed diagnosis. [1]

Differential Diagnosis List: Alcohol intoxication, Carbon monoxide intoxication, hypoglycaemic brain damage

Final Diagnosis: Alcohol intoxication

References:


Description: Axial T1WI at the level of basal ganglia: Slightly high signal component in bilateral putamina surrounding central low signal areas indicating haemorrhagic necrosis. Origin: French Medical Institute for Children
Description: Axial T2WI: High signal central area (necrosis) surrounded by low signal rim (haemorrhage) in bilateral putamina. Origin: French Medical Institute for Children
Description: T2Tirm Origin:
Description: Axial T2* GRE image:

Confirms bilateral putaminal haemorrhage by depicting peripheral hypointense areas due to susceptibility artefacts surrounding central necrosis. Origin: French Medical Institute for Children
Description: DWI: shows abnormal high signal in bilateral putamina. Origin: French Medical Institute for Children
Description: ADC map: Drop of signal representing diffusion restriction due to cytotoxic oedema.
Origin: French Medical Institute for Children
Figure 3

Description: Contrast Origin:
Description: Coronal contrast-enhanced T1WI: Peripheral rim of intense contrast enhancement of the putaminal lesions is noted bilaterally. Origin: FMIC