Case 13776
MRI detected bilateral putaminal hemorrhagic necrosis due to methanol intoxication

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\textbf{Section:} Neuroradiology

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\textbf{Patient:} 30 year(s), male

\section*{Clinical History}

A 30 years man was referred to undergo Brain MRI from outside hospital. As per attendant's quote, this previously healthy man had history of unknown type and amount of alcohol intake one week ago. There after he had vomiting and lost bilateral vision. Now he is in unconscious state for last three days.

\section*{Imaging Findings}

Brain MR imaging performed with 1.5T Siemens machine.

Axial T1-Weighted Image (WI) at the level of basal ganglia shows slightly high signal component in bilateral putamina surrounding central low signal areas indicating hemorrhagic necrosis. (Figure 1a)

Axial T2WI demonstrate inverse image of TIWI, as high signal central area (necrosis) is surrounded by low signal rim (hemorrhage) in bilateral putamina. (Figure 1b)

Axial T2 FLAIR image confirms the necrosis by intense high signal area in bilateral putamina.
Axial T2* GRE image confirms bilateral putaminal hemorrhage by depicting peripheral hypointense areas due to susceptibility artefacts surrounding central necrosis. Diffusion weighted image (DWI) shows abnormal high signal in bilateral putamina with drop of signal on ADC representing diffusion restriction due to cytotoxic edema. Peripheral rim of intense contrast enhancement is noted bilaterally. The lesions are nicely limited to putamina. No significant brain edema or additional lesions are seen.

**Discussion**

**Background**

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

**Clinical Perspective**

Prompt diagnosis and treatment are essential. The clinical presentation may be variable in individuals. Usually a latent period of 12-24 hours precedes the clinical manifestation after ingestion. Visual disturbance is the first symptom in many patients. Other symptoms are headache, dizziness, malaise and gastrointestinal symptoms like nausea, vomiting and abdominal pain. Severe cases can result in dyspnea, seizure, coma, permanent neurological deficit and death. Respiratory arrest is often the terminal event. The diagnosis is made based on metabolic acidosis, high anion and osmolar gap and high serum methanol levels.

**Imaging Perspective**

Neuroradiological features of AMI are described in literature. Imaging helps in distinguishing AMI from other causes of acute unconsciousness in alcoholic patients such as hypoglycemic brain damage, carbon monoxide poisoning or head injury. The most characteristic finding of AMI is bilateral putaminal necrosis. The necrosis can be hemorrhagic or non-hemorrhagic however hemorrhage 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. Lesions of hippocampi, subcortical and deep white matter, cerebral and cerebellar cortex and midbrain are reported. Cerebral and intraventricular hemorrhage and diffuse cerebral edema can also occur. DWI can detect lesions that could not be depicted with CT or conventional T1 and T2WI sequences. 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 alkalinization. (1, 2) Survival depends on amount of methanol ingested and prompt start of treatment. (10) Mortality remains high, mainly because of often difficult and hence delayed diagnosis. (1)

Final Diagnosis

Alcohol intoxication

Differential Diagnosis List

Carbon Monoxide intoxication, hypoglycemic brain damage

Figures

Figure 1 T1WI and T2WI

Axial T1WI at the level of basal ganglia: Slightly high signal component in bilateral putamina surrounding central low signal areas indicating hemorrhagic necrosis.

Area of Interest: Neuroradiology brain;  
Imaging Technique: MR;  
Procedure: Diagnostic procedure;  
Special Focus: Acute;
Axial T2WI: High signal central area (necrosis) surrounded by low signal rim (hemorrhage) in bilateral putamina.

Axial T2 FLAIR image: Confirms the necrosis by intense high signal area in bilateral putamina.
Procedure: Diagnostic procedure;
Special Focus: Acute;

Axial T2* GRE image: Confirms bilateral putaminal hemorrhage by depicting peripheral hypointense areas due to susceptibility artefacts surrounding central necrosis.

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Area of Interest: Neuroradiology brain;
Imaging Technique: MR;
Procedure: Diagnostic procedure;
Special Focus: Haemorrhage;

Figure 2 DWI
DWI: shows abnormal high signal in bilateral putamina.

Area of Interest: Neuroradiology brain;  
Imaging Technique: MR-Diffusion/Perfusion;  
Procedure: Diagnostic procedure;  
Special Focus: Acute;

ADC map: Drop of signal representing diffusion restriction due to cytotoxic edema.

Area of Interest: Neuroradiology brain;  
Imaging Technique: MR-Diffusion/Perfusion;  
Procedure: Diagnostic procedure;  
Special Focus: Acute;

Figure 3 Post contrast
Axial contrast enhanced T1WI: Peripheral rim of intense contrast enhancement of the putaminal lesions is noted bilaterally.

Coronal contrast enhanced T1WI: Peripheral rim of intense contrast enhancement of the putaminal lesions is noted bilaterally.
References


Citation

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