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Hypoxic, Toxic, and Acquired Metabolic Encephalopathies at the Emergency Room: The Role of Magnetic Resonance Imaging

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Our purpose is to describe typical computed tomography and magnetic resonance imaging findings in encephalopathies in the emergency. The focus of this article are the most frequent toxic and acquired metabolic diseases and their preferential sites of involvement, such as hepatic encephalopathy, hypoglicemia, nonketotic hyperglycemia, osmotic demyelination, posterior reversible encephalopathy syndrome, uremia, illegal drug abuse, carbon monoxide poisoning, and hypoxic-ischemic encephalopathy. The radiologist must be able to identify the most usual patterns of lesion in computed tomography and magnetic resonance imaging in these settings.

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Introduction

Encephalopathy is a general term for any diffuse disease of the brain that alters brain function or structure, and may have several causes, including infectious diseases, metabolic and toxic derangement. The differential diagnosis of patients presenting with acute encephalopathy is broad, and clinical evaluation is often limited in these cases. In this critical emergency scenario, imaging plays an essential role not only in the diagnosis of the disorder, but also in the assessment of treatment response and prognostication. This article describes the more classic brain imaging features of some of the most common toxic and acquired metabolic encephalopathies in adults, and relates them to their specific pathogenic mechanisms. It is important to highlight that frequently, the diagnosis is not straightforward, and adequate correlation with clinical and laboratory data is essential for accurate assessment.

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Hepatic Encephalopathy

Hepatic encephalopathy (HE) is defined as a potentially reversible syndrome secondary to acute or chronic hepatic dysfunction or portosystemic shunt. It manifests as a wide spectrum of neuropsychiatric abnormalities, and motor disturbances. The most common clinical scenario is the development of mental disturbances or coma in a cirrhotic patient following an episode of gastrointestinal bleeding, concomitant infection, or acute superimposed hepatitis.[1,2]

Pathophysiology

The pathogenesis of HE is complex, multifactorial, and incompletely understood. Hepatic insufficiency leads to accumulation of several elements, such as manganese and ammonia, which then enter the brain and induce disturbances in astrocyte and neuron function.[1]

Hyperammonemia results in profound astrocyte changes, which can lead to brain edema and increased intracranial pressure.[2,3] The mechanism for astrocyte swelling induced by ammonia remains uncertain but is likely to include excessive generation of osmolytes, mainly glutamine.[3]

Brain manganese accumulation induces selective neuronal loss in basal ganglia structures, resulting in a progressive disorder of the extrapyramidal system.[3] These neurotoxic effects are more prominent in the globus pallidus.[1,3]

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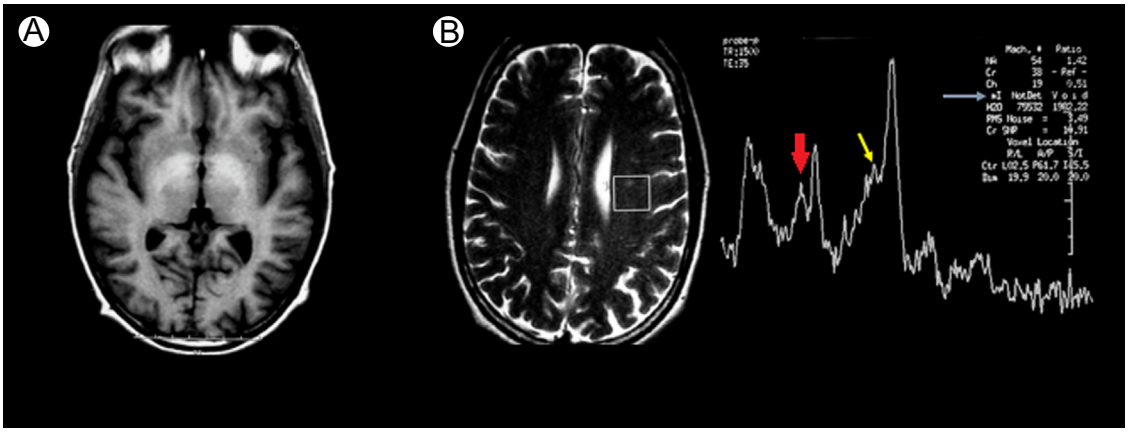

Figure 1 Hepatic encephalopathy. A 47-year-old patient with cirrhosis and cognitive alterations. (A) Axial T1W image shows bilateral, symmetric high signal intensity in basal ganglia (white arrows), owing to the rise in manganese concentration. (B) Axial T2-WI with an spectroscopic voxel at the level of the left corona radiate (left panel) and short-echo time MR-spectroscopy curve (right panel). There is increase in the glutamine or glutamate peak (yellow arrow) accompanied by myo-inositol depletion (blue arrow) and choline decrease (red arrow). (Color version of figure is available online.)

Imaging Findings

Computed Tomography

In the acute phase, Computed tomography (CT) scans can appear normal or show diffuse cerebral edema, with no contrast enhancement. In the chronic phase, it may show unspecific brain atrophy.[2] Although CT is not specific for the diagnosis of HE, it can be useful for the evaluation of other common conditions in the cirrhotic patient, such as brain hemorrhage secondary to coagulations abnormalities.

Magnetic Resonance Imaging (MRI)

Manganese accumulation can be demonstrated in T1-weighted sequences owing to its paramagnetic effect as bilateral, symmetric high signal intensity of the globus pallidus and substantia nigra, and less frequently, in putamina, subtalamic regions, and adenohypophysis (Figs. 1 and 2).[2] More rarely, white matter (WM) of both cerebral hemispheres may also be compromised. The signal intensity may increase after a transjugular intrahepatic portal-systemic stent shunt surgery and reverses after normalization of liver function, liver transplantation, or after occlusion of congenital portal-systemic shunts.[4,5]

In acute, severe hepatic failure, diffuse high signal intensity on T2-weighted and FLAIR sequences compromising the cerebral cortex may be observed, sparing the perirolandic and occipital regions.[2] Symmetric signal-intensity abnormalities with associated restricted diffusion in the insula and cingulate gyrus have been reported either.[6,7] High signal intensity along the hemispheric WM in or around the corticospinal tract may be also seen.[2]

WM foci hyperintensity T2-weighted lesions may be encountered in patients with liver cirrhosis with or without HE. These lesions resemble those commonly seen in patients with cerebrovascular small-vessel disease and in the healthy

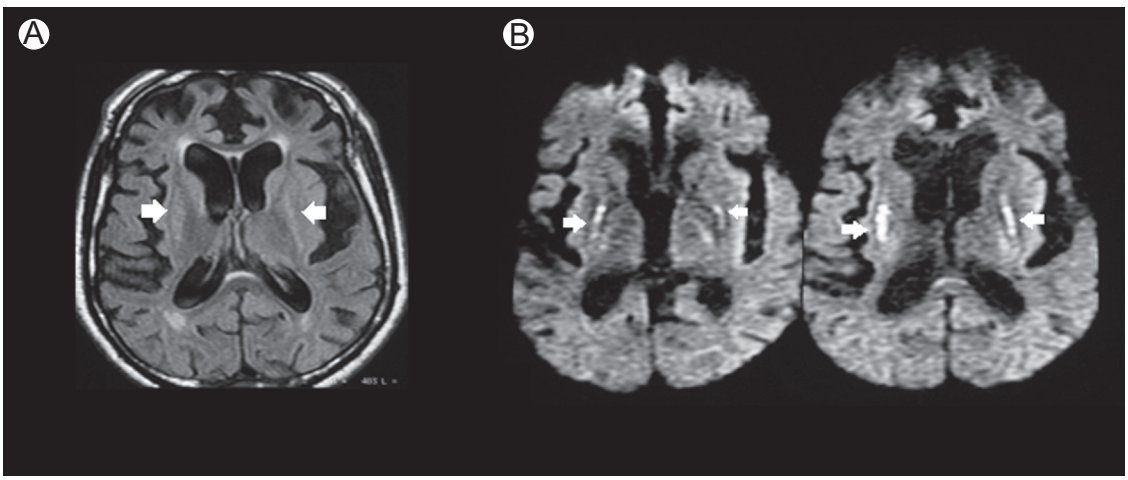

Figure 2 Hepatic encephalopathy. (A) FLAIR imaging shows bilateral high signal intensity in putamen (arrows) with correspondent DWI restriction (B) in a patient with hepatic failure.