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CASE REPORT OPEN ACCESS

ACUTE HEPATIC ENCEPHALOPATHY: ANALYSIS OF IMAGING FINDING IN A 15-YEAR-OLD PATIENT

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ABSTRACT

Hepatic encephalopathy is a neuropsychiatric syndrome due to severe liver failure. The clinical presentation is variable. MRI have a critical role in the diagnosis confirmation and understanding the neural mechanism of this disorder. We report the case of a 15-year-old patient, who is followed up for liver cirrhosis and who presents an acute hepatic encephalopathy quickly diagnosed with MRI.

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INTRODUCTION

Hepatic encephalopathy is the result of accumulation of compounds that are metabolized normally by the liver, in patients with liver dysfunction. Clinical presentation of the disease is variable and not specific. MRI is the key imaging tool for positive diagnosis and early detection of this pathological entity.

CASE REPORT:

A 15-year-old girl followed up for liver cirrhosis of unknown cause with portal hypertension, who presents with an acute right hemiparesis and speech disorder. MRI was performed and showed extensive hyperintensity in cerebral and cerebellar white matter, with restriction in Diffusion (Figure 1), and lack of contrast enhancement after gadolinium injection (Figure 2). The MRI aspect is compatible with an acute hepatic encephalopathy. Evolution was marked by spontaneous resolution of symptoms and disappearance of neurological signs. The liver transplant is scheduled for our patient.

Metabolic encephalopathies are neurological disorders caused by severe systemic illness such as liver disease, renal failure and diabetes. There are two major types: those due to lack of glucose, oxygen or metabolic cofactors (which are usually vitamin-derived) and those related to peripheral organ dysfunction (hepatic, uremic and dialysis encephalopathies) [1]. Hepatic encephalopathy occurs in patients with liver dysfunction that usually correspond to cirrhosis, portal hypertension and acute liver failure. It results of accumulation of compounds such as ammonia and manganese in blood, and which are metabolized by the liver in normal circumstances; these substances induce disturbances in neuron function [2, 3]. Clinical signs are nonspecific, consisting of a generalized depression of cerebral function, the most frequently consciousness disorders (range from subtle cognitive dysfunction to severe coma), and motor function changes (speech disorders, rigidity, tremor and hyper or hyporeflexia) [4]. Hepatic encephalopathy could be divided into three subtypes according to severity and mechanism of the disease: Type A is the result of an acute liver failure, type B is caused by a portosystemic bypass despite of normal hepatic function; and type C is related to cirrhosis associated or not to portal-systemic shunting.

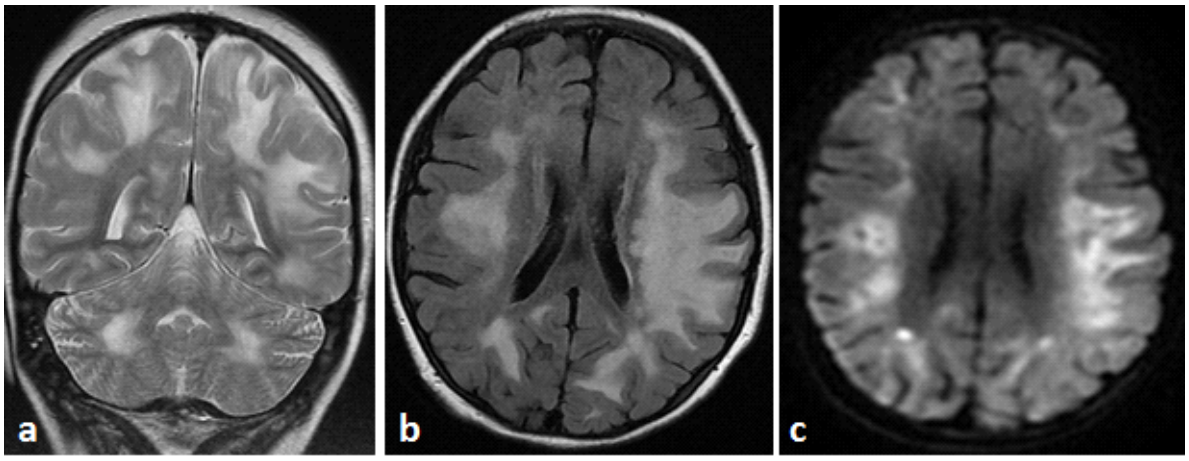

Figure 1. (a): T2-weighted image in coronal section and (b): T2 Flair -weighted image in axial section, Showing extensive hyperintensity in cerebral and cerebellar white matter. (c): Diffusion sequence that objectified a restriction of diffusion in the lesions

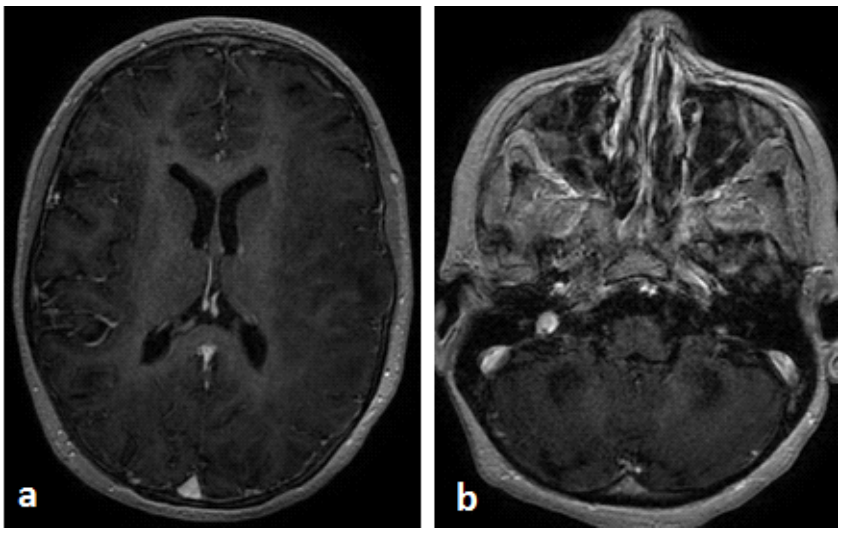

Figure 2. T1-weighted image in axial section after Gadolinium showing absence of pathological contrast enhancement in brain (a) and cerebellum (b)

This last type is classified into 3 main groups: episodic, chronic, and minimal according to the duration and characteristics of clinical signs [4]. Imaging plays an important role in the diagnosis, assessment of treatment response, prognostication of the disorder, and also in the evaluation of the mechanisms, in correlation with clinical and laboratory data. In acute hepatic encephalopathy, MRI objectifies T2 and T2 Flair weighted high signal-intensity within the brain indicating the diffuse cerebral edema, this last is the outcome of hyperammonemia.

In Diffusion sequence, we find also a high intensity in the lesions with decreased ADC value that reflect an accumulation of water in the extracellular compartment. The mechanism of the incrise in extracellular fluid may be explained by the extracellular migration of the macromolecules, since hyperammonemia may induce an increase in blood-brain barrier permeability [3, 6]. In addition to these mechanisms, the massive intra-astrocytic increase of glutamine contributes to the appearance of cerebral edema in the acute hepatic encephalopathy. Then, it is important to underline the possibility of the existence of both types of cerebral edema: intracellular and interstitial [3]. Hyperammonemia causes the increase of intracellular osmolality, then astrocytes release osmolytes such as choline, and an important amount of water enters the astrocyte. All this changes are the origin of impairment of cellular metabolism and disorder of neuronal function [7, 8, 9]. Cerebral edema can be observed also in the chronic hepatic encephalopathy but of lesser degrees, and in several patients cerebral edema is minimal and is not associed to MRI abnormalities [3]. MR imaging shows classically in most patients with cirrhosis or portal-systemic shunts, a bilateral symmetric high signal intensity at the globus pallidus and substantia nigra inT1-waidgned sequence, this signal abnormality is due to a rise in manganese concentration in the central nervous system that deposit preferentially in the globus pallidus [10]. The high signal T1 in the globus pallidus is observed also in any other pathology likely to cause a rise in manganese such as noncirrhotic portal vein thrombosis, Alagille syndrome, and in patients with total parenteral nutrition [3, 10]. We note that, in our patient, MRI did not show signs of manganese deposition in the globus pallidus. Spectroscopy in hepatic encephalopathy shows typically a rise in Glx/Cr ratio with lower mIns/Cr and Cho/Cr in all examined brain regions, associated to hyperammonia in patients with cirrhosis [6, 8, 9]. Advanced multimodality MRI methods are indispensable for the detection of mild brain deterioration in patients with chronic hepatic encephalopathy; such as magnetization transfer Imaging (MT), cerebral morphological analysis via voxel-based morphometry (VBM) and cortical

morphological analysis, diffusion tensor imaging (DTI), MR perfusion imaging, and blood oxygen level dependent functional MR imaging (BOLD fMRI) [4]. Some differential diagnoses must be ruled out in patients with liver failure and acute neurological disorder: Infectious encephalopathy associate meningitis, prospective brain abscess and empyema, with a severe clinical and biological infectious syndrome. Toxic encephalopathy per CO causes bilateral hyperintensities on T2 and DWI in the cerebral white matter in the acute phase (demyelination) , and in globus pallidus in the chronic phase (necrosis). Autoimmune encephalopathy include cerebellar degeneration, encephalitis of brain stem and striatum, and leukoencephalopathy, associated to serum auto-antibodies [11]. MR abnormalities in hepatic encephalopathy are typically reversible after successful liver transplantation attesting the reversibility of brain edema in patients with cirrhosis. Idem for the chronic rise in manganese concentration in the central nervous system [6, 12]. The reversibility of acute signal abnormalities and spectroscopy disorder precedes the disappearance of pallidal hyperintensity and correlates with favorable improvements in the neurologic manifestations [12].

Conclusions

Acute hepatic encephalopathy appears as diffuse brain edema associated with functional changes. MR imaging offers an increasingly important role in early diagnosis and play a crucial role for understanding structural and functional derangements of the brain with hepatic encephalopathy.

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