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Case Report

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A Case of Hepatic Encephalopathy Masquerading as Parkinsonism Unmasked by Classic Brain Imaging Findings: A Case Report

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Case Report

Hepatic Encephalopathy (H.E), defined as a spectrum of neuropsychiatric abnormalities in patients with severe liver dysfunction. It is a most common complication of advanced liver disease with significant incidence and death rate [1].

The exact prevalence of hepatic encephalopathy is still unknown because it can be caused by different etiological factors, and have varying degree of severity based on age of the patient, type of H.E and has challenging clinical diagnosis [2]. some of the studies revealed that patients with chronic liver disease and hepatic encephalopathy effects their quality of life by about 70% [3, 4]. Other studies report that prevalence of HE is about 30% to 45% in the patients with liver cirrhosis and about 10% to 20% in patient's with portosystemic shunts. The mechanisms causing this brain abnormalities are still yet to be discovered [5, 6]. HE is classified into three different types based on how the liver is involved (Figure 1).

Case Presentation

We report a case of a 66 year old diabetic, hypertensive, non-alcoholic gentleman with disturbed sleep-wake cycle and episodic confused behavior, slowness of movements, hand tremors and falls for a few months prior to admission. Each episode lasted for 10-15 days. He had mild cognitive impairment with a Montreal Cognitive Assessment score of 24, bradykinesia, rigidity of the limbs, resting and flapping tremors of the upper limbs episodically. T1 bilaterally symmetric hyperintensities in the globus pallidus and substantia nigra were seen on MRI brain.T2/ FLAIR, diffusion weighted imaging showed periventricular hyperintensities and white matter changes respectively with no ADC mismatch. EEG showed grade 2 encephalopathy.

Serum ammonia was elevated (179.4) with thrombocytopenia and splenomegaly. Liver sonography and hepato-portal doppler were normal. Liver function tests and electrolytes were normal. Upper GI Most Comprehensive USMLE Career Program endoscopy showed mild Portal hypertensive gastropathy and Gastric Antral Vascular Ectasia suggestive of portal hypertension. He was treated with rifaximin, lactulose and supportive measures and he improved clinically. On follow up, liver elastography showed grade 2 fibrosis. When a patient has such episodic symptoms and normal LFT, HE can be suspected based on the classic MRI features of HE. MRI features lead to active search for the etiology of HE in our case.

Discussion

Hepatic encephalopathy (HE) is a neuropsychiatric condition presenting

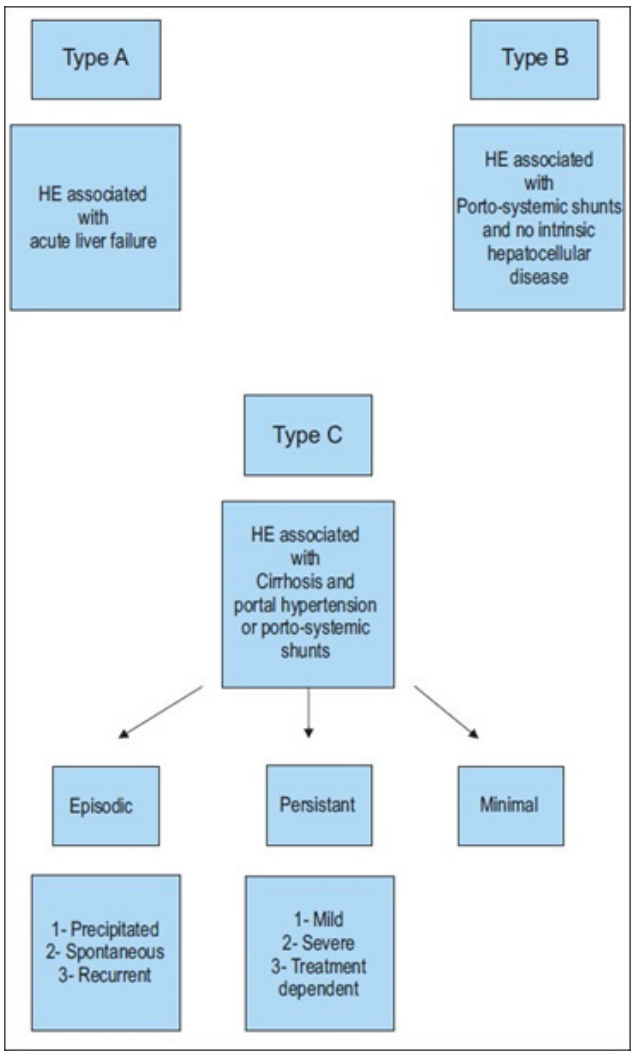
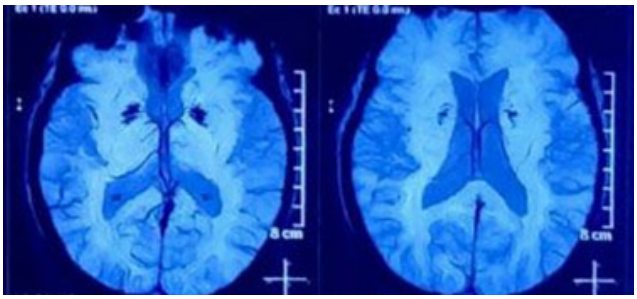
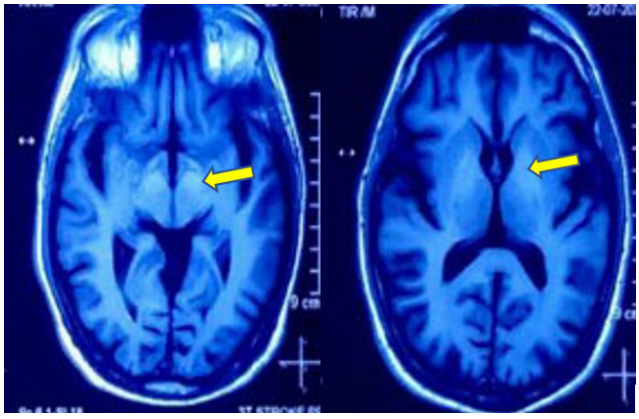


Figure 1: Classification of hepatic encephalopathy [7].

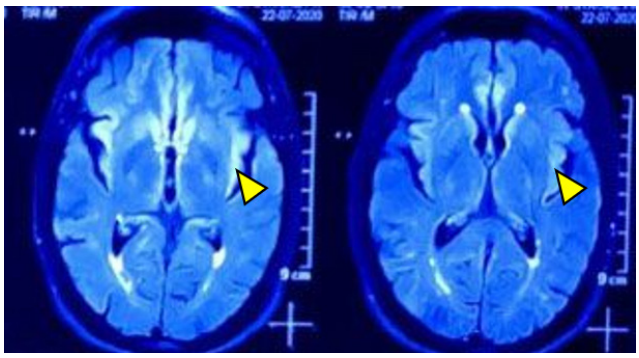
with cognitive impairment and neuromuscular dysfunction. The patient can have mild cognitive impairment or can present in coma. HE is associated with acute or chronic hepatocellular dysfunction or rarely with portosystemic shunts without intrinsic hepatocellular disease. Cirrhosis of the liver may not always be reflected by routine liver function test and USG abdomen. HE is predominantly a clinical diagnosis and other tests like EEG and MRI act as ancillary tests. Our patient was an elderly man who was not an alcoholic, had no overt abnormality on liver function tests so the diagnosis of hepatic encephalopathy was very doubtful. He showed episodic symptoms of confusion, memory loss, bradykinesia, tremors and falls. The episodic nature of his parkinsonian symptoms makes the arrival at a diagnosis even more difficult. EEG showed grade 2 encephalopathy. Serum ammonia was elevated (179.4) with thrombocytopenia and splenomegaly. Liver sonography and hepato-portal doppler were normal. Classic MRI features of T1 hyperintensities in the globus pallidus, substantia nigra due to manganese deposition and elevated ammonia levels helped us clinch the diagnosis. These MRI features are reported in 80-90% patients with chronic hepatocellular failure [8]. Elevated ammonia levels predict mortality rate in hepatic encephalopathy with 75-80% sensitivity and 80% accuracy [9].



GRE



T1 AXIALS



T2/FLAIR AXIALS

T1 bilaterally symmetric hyperintensities in the globus pallidus and *substantia nigra*.(arrow)

T2/ FLAIR images shows hyperintensities in bilateral insular cortex (arrow head). periventricular hyperintensities and white matter changes.

T1 hyperintensities in globus pallidus have sensitivity of 66% for predicting cirrhosis as seen in previous studies [10]. High degree of suspicion is required when a non- alcoholic, without any viral hepatitis or drug intake presents with episodic features of hepatic encephalopathy and fairly normal liver function tests. Fibroscan revealed liver grade 2 fibrosis and Upper gastrointestinal endoscopy showed mild portal hypertensive gastropathy and Gastric Antral Vascular Ectasia suggestive of portal hypertension. He was treated with rifaximin, lactulose and supportive measures and he improved clinically.

Conclusions

When a patient presents with episodic symptoms of secondary parkinsonism and normal LFT, HE can be suspected based on the classic MRI features of HE. MRI features led to active search for the etiology of HE in our

case and cirrhosis in our patient could be due nonalcoholic steatohepatitis leading to hepatocellular dysfunction keeping in mind his co-morbidities.

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