# Hepatic encephalopathy uncommonly presenting by epilepsy and hemiparesis: A case report


## ABSTRACT

**Hepatic encephalopathy (HE) is a serious complication of cirrhosis that presents with a variety of neuropsychiatric disorders, including disorientation, asterixis and coma. Such neurological disorders are because of hyperammonemia. However, hepatic encephalopathy with neurological symptoms resembling epilepsy and hemiparesis is uncommon. We present a case of decompensated liver cirrhosis manifesting initially by epilepsy and hemiparesis.**

**Case report: A 59-year-old male smoker known to be diabetic, which was well controlled, presented to our hospital with a chief complaint of epileptic attacks over the past 24 hours in addition to dizziness, sudden-onset left-sided weakness and disturbed level of consciousness with a Glasgow Coma Scale rating of 13. Brain CT scan and MRI revealed supratentorial white matter changes with no signs of stroke or bleeding. Electroencephalogram (EEG) showed diffuse slow wave rhythm. Initially, the patient was treated with antiepileptic drugs with no improvement. Laboratory examination suggested liver cirrhosis. Plasma ammonia levels upon admission were 2 times the normal value. Abdominal imaging showed chronic hepatopathy, portosystemic varices and splenomegaly.Upper endoscopy showed esophageal varices. Liver cirrhosis was confirmed by transient elastography. The aetiology of cirrhosis was considered metabolic. As a result, the diagnosis of HE was made. The symptoms were improved by adding lactulose and Rifaximin to antiepileptic treatment.**

**Conclusion: Though uncommon, hepatic encephalopathy, as a complication of liver cirrhosis, should be considered in patients presenting with epilepsy and hemiparesis. Antiepileptic drugs combined with lactulose are essential for treatment.**



*Keywords: Hepatic encephalopathy, liver cirrhosis, epilepsy, hemiparesis.*


***Abbreviations***

*HE: Hepatic encephalopathy*
*CT: Computed Tomography*
*EEG: Electroencephalogram*
*FLAIR: Fluid attenuated inversion recovery*
*DWI: diffusion-weighted image*
*ADC: apparent diffusion coefficient*
*MRI: magnetic resonance imaging*

## 1. INTRODUCTION

Liver cirrhosis is a chronic liver diseasein which hepatocytes become degenerated and necrotic[1].In addition, liver parenchyma is replaced by regenerative nodules and fibrosis[1].

Cirrhosis might remain asymptomatic for a long time and becomes patent by developing complications such as bleeding from esophageal or gastric varices, abdominal distension by ascites, neurological disorders named hepatic encephalopathy, etc [2]. . Hepatic encephalopathy (HE) occurs due toelevated levels of toxic metabolites, such as ammonia [3. It includes30%-45% of complications which develop from liver cirrhosis [3].HE can be easily diagnosed when neurological symptoms are overt and when it is associated with recognized presentations including changes in sleep pattern, hyperactive deep tendon reflexes, asterixis, disorientation and coma [2]..Focal neurological deficits and epilepsy are rare presentations of hepatic disease[4]. We present a case of decompensated liver cirrhosis manifesting initially by epilepsy and hemiparesis.

## 2. CASE PRESENTATION

A 59-year-old male smoker patient with a medical history of non-insulin-dependent diabetes and no alcohol consumption or drug intake, presented to the emergency department with a main complaint of sudden-onset epileptic attacks over the past 24 hours in addition to dizziness, sudden-onset left-sided weakness and decreased level of consciousness with a Glasgow Coma Scale rating of 13. The family denied any history of stroke, brain trauma, epilepsy, coma and hepatitis B/C virus infection. Vitalsigns on admission showed: blood pressure of 139/97, pulse of 93 beats/minute, and SpO2 of 99% on room air. On clinical exam, the patient was somnolent and slow in answering questions. He was confused but oriented to person and place, not to time. Neurological examination was remarkable for left-sided weakness with 3/5 strength in the right upper and lower extremities. Abdominal examination was unremarkable.

Brain CT scan and MRI revealed supratentorial white matter changes with no signs of stroke or haemorrhage (figure 1). An Electroencephalogram (EEG) showed a diffuse slow wave rhythm consistent with HE (figure 2). His labs were significant for a GGT of 86, total bilirubin of 1.9, Albumin of 33, platelets 134x103, prolonged prothrombin time, blood ammonia 2 times the normal value and Hb A1C 7. All of his other labs were normal. The patient was treated with several antiepileptic drugs precisely,Carbamezepam, Levetiracetam, and Topiramate with no improvement. Because of elevated blood ammonia and high bilirubin, an abdominal CT scan was performed, showing an overall coarse and heterogeneous texture of the liver in addition to hypertrophy of segment I and hypotrophy of segment IV, in favour of liver cirrhosis. There were also signs of portal hypertension, precisely an enlarged portal vein (24 mm in diameter), an enlarged superior mesenteric vein (17 mm), an enlarged splenic vein (13 mm) and an enlarged spleen. Portosystemic collateral veins were seen in peri-gastric, peri-esophageal and peri-pancreatic areas.Moreover, a gastrorenal shunt was observed (figure 3).Upper endoscopy confirmed the presence of grade 2 esophageal varices with no signs of bleeding. Transient elastography (Fibroscan) value was 24 kPa confirming that liver cirrhosis was the cause of portal hypertension. Hepatitis B virus, hepatitis C virus and profile for autoimmune liver disease were all negative. The aetiology of cirrhosis was considered metabolic because of diabetes mellitus disease.
The patient was started on lactulose and Rifaximin. His neurological symptoms resolved without residual neurological deficits or epilepsy 36 hours after the last treatment. The patient was discharged on antiepileptic treatment, lactulose and Rifaximin.

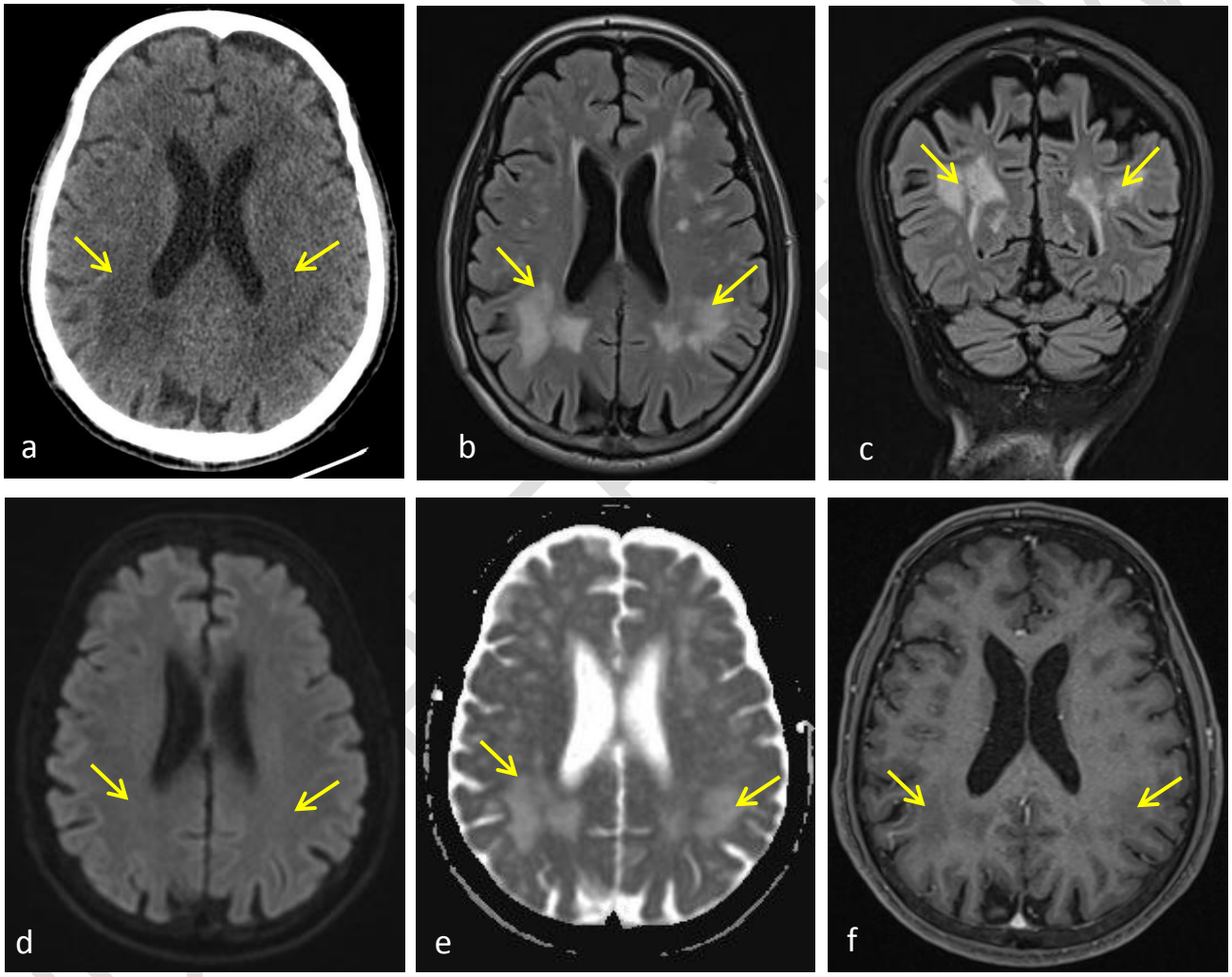


Figure 1: Brain CT (a) demonstrating frontal and parietal white matter hypodense areas (arrows), presenting as hyperintensities on axial and coronal T2-FLAIR (b & c), with no diffusion restriction on DWI and ADC map (d & e), and no enhancement after gadolinium administration (f).

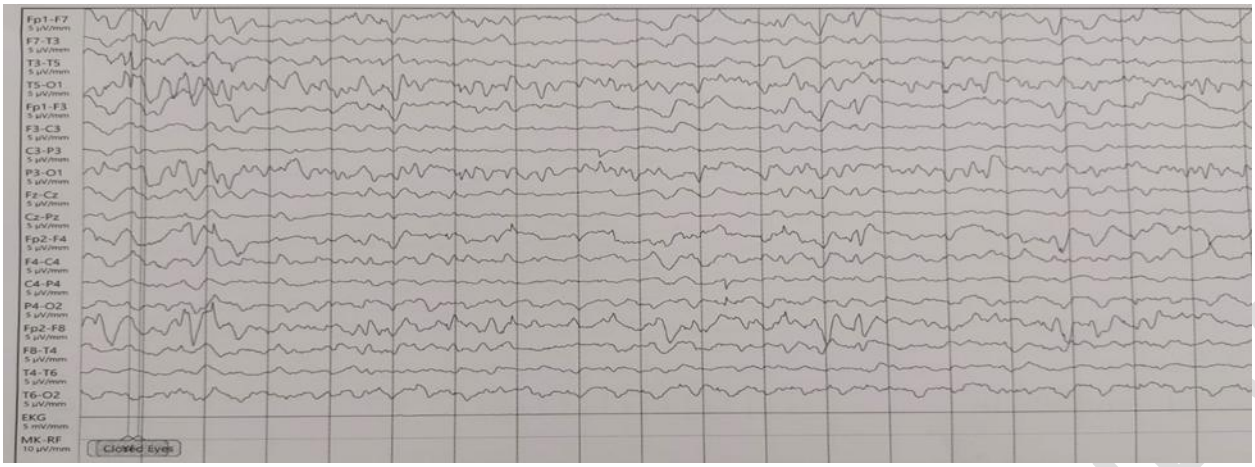


Figure 2: EEG showing diffuse slow wave rhythm.

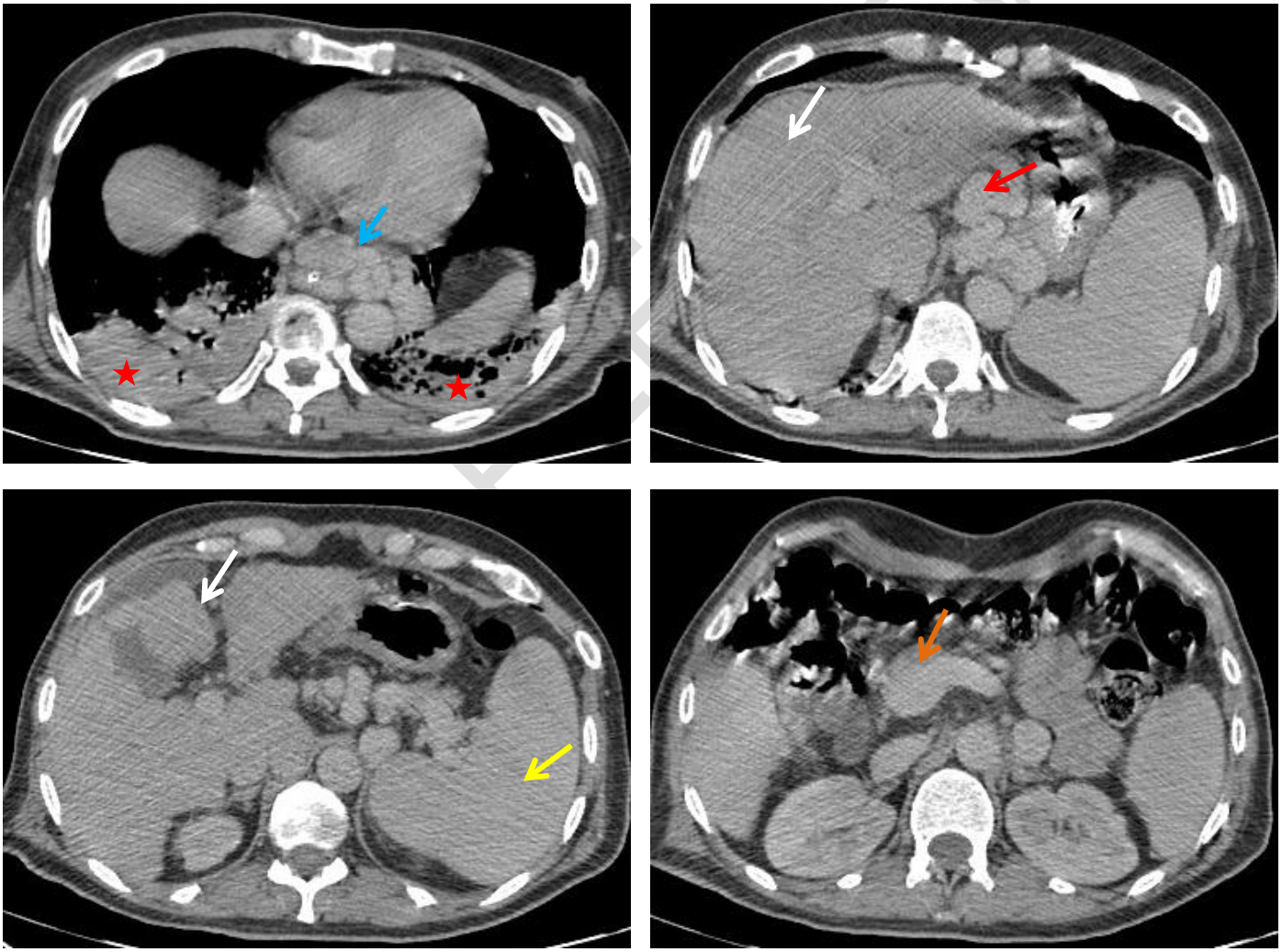

Figure 3: Abdominal CT showing signs of chronic hepatopathy (white arrows), paraesophageal varices (blue arrow), gastrorenal shunt (red arrow), enlarged superior mesenteric vein (orange arrow) and splenomegaly (yellow arrow). Note bilateral lung consolidation in favour of aspiration pneumonia (red stars).

## 3. DISCUSSION

Hepatic encephalopathy (HE) is a reversible consequence of advanced liver disease and/or portosystemic shunting. Impaired neurologic function,including altered mental status, asterixis, and possible coma, is an important characteristic of HE[7]. It is also associated with neuromuscular impairment and cognitive disorders [2]. However, focal neurologic deficits and epilepsy are unusual presentations of HE[13,14].

Focal neurologic deficits and unilateral weakness are typically associated with acute cerebrovascular accidents. Prior studies reported patients presenting with hemiparesis, however, their CT scan was normal initially and when repeated[5]. In these studies, the initial T2-weighted MRI of the brain showed a high-intensity lesion of 5-6 cm in the right frontal lobe. In addition, parietal and occipital lobes had multiple lesions because of hypoperfusion [5]. It was later hypothesized that the neurotoxicity and neurotransmitter derangements of hepatic encephalopathy may have created focal neurologic signs which is because perfusion is diminished and neurotransmitter function is impaired within the subclinicalstable lesions [5Another study with data,which was prospectively collected,revealed that 13% of patients who were hospitalized for HE had hemiparesis or hemiplegia as an initial presentation [6]. The initial or follow-up CT or MRI findings of these patients showed no significant findings. Moreover, their symptoms fully improved after the regression of HE [6].

Epilepsy is also a rare but life-threatening manifestation of HE [8,13].There is uncertainty regarding the frequency of seizures in HE.There is a study showing that up to 33% of patients with HE presented with seizures but this happened in the late stages of their liver disease.On the other hand, others are suggesting that seizures are an uncommon manifestation [9]. EEG tracings in patients hospitalized for HE were reviewed. It was found that patients with epileptiform abnormalities had poorer prognosis [9].

Many factors play a role in the pathophysiology of HE. Changes in ammonia levels, amino acids and inflammatory cytokines are included [10]. Hyperammonemia leads to an increased level of glutamine production which accumulates in astrocytes and creates an osmotic gradient resulting in the swelling of astrocytes. High level of glutamine causes more production of reactive oxygen species which plays a role in the dysfunction of brain cells in HE [10].

The benefit of EEG recordings is to identify the hidden cause of altered mental status in cirrhotic patients. Studies showing the usage of EEG to differentiate between HE and conditions such as non-convulsive status epilepticus (NCSE) are few. These conditions,which can be a hidden disorder in the presence of HE, need an alternative course of treatment [11]. The most common EEG abnormality in HE patients was slow wave rhythm [12,15].

Our case demonstrates a patient presenting with epileptic attacks and focal lesions which was not explained by a brain CT scan, MRI and EEG. Accordingly, the patient was treated with antiepileptic drugs with no improvement. Due to the presence of abnormal liver tests, elevated blood ammonia and radiological signs of liver cirrhosis with a large gastro-renal shunt, the diagnosis of hepatic encephalopathy due to liver cirrhosis was made. The patient showed great improvement after the addition of Rifaximin and lactulose. Therefore, HE should not be overlooked in patients with unexplained focal neurological signs and epilepsy resistant to treatment.

**Consent**

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

## 4. CONCLUSION

Focal neurological lesions and epilepsy are unusual andprobably serious complications in cirrhotic patients having HE. Other etiologies of altered mental status in patients presenting with convulsions and unilateral weaknesscan be ruled out by brain imaging and EEG recordings.Common anti-convulsant treatment might not be enough for patients experiencing epilepsy secondary to HE, as a result, the underlying liver diseases must be managed.

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