HEPATIC ENCEPHALOPATHY MIMICKING STROKE IN A PATIENT WITH CHRONIC LIVER DISEASE

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ABSTRACT

Hepatic Encephalopathy usually presents with sleep disturbances, drowsiness and personality changes. Focal neurological loss (hemiplegia) is a very rare presentation of hepatic encephalopathy. A 55-year-old gentleman presented with irritability, drowsiness, right sided body weakness and constipation for 3 days. He was a diagnosed patient of Hepatitis C positive Chronic liver disease for 3 years. On examination, the patient was drowsy but arousable having GCS of 12/15. Power of grade 4/5 was noted in both left upper and lower limb with an extensor left plantar reflex. Flapping tremors and bilateral pitting pedal edema were noted. Abdomen was mildly distended with positive shifting dullness. CT scan (Brain) was done to rule out intracranial bleeding and the patient was admitted on lines of ischemic stroke and hepatic encephalopathy. He had thrombocytopenia, deranged LFTs and high serum ammonia. MRI Scan (Brain) showed foci of diffusion restriction in the left periventricular region and basal ganglia and T2WI/FLAIR hyperintense signals in the periventricular region, basal ganglia and deep white matter, however MR angiography demonstrated normal cerebral vessels. He was diagnosed with hepatic encephalopathy mimicking stroke. After initiating lactulose and Rifaximin, he passed stools leading to resolution of symptoms. His repeat serum ammonia was normal.

Keywords: Hepatic Encephalopathy, Liver Cirrhosis, Stroke, Serum ammonia.

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INTRODUCTION

Affecting up to 5 million patients yearly,[1] liver cirrhosis has a significant mortality burden causing up to 40,000 deaths per annum in the United States.[2] Liver cirrhosis also accounts to a high morbidity and disease burden especially in a developing country with limited resources such as Pakistan. In Pakistan, liver cirrhosis is the 5th most common cause of death and 11th most common cause of disability.[3] The complications of liver cirrhosis are numerous and multi-system including ascites, spontaneous bacterial peritonitis, fluid overload, portal hypertension, hepato-renal syndrome, esophageal and gastric varices, electrolyte imbalances especially sodium, pulmonary hypertension and hepatic encephalopathy.[4,5,6] Hepatic Encephalopathy is a potentially reversible life-threatening neuro-psychiatric complication of liver cirrhosis thought to be caused by deposition of ammonia and other toxins in brain astrocyte cells leading to brain edema and dysregulation of neurotransmitters.[7] It generally presents with sleep disturbances, asterixis, drowsiness, delirium, coma and personality changes. However, focal neurological loss in form of hemiplegia is a very rare presentation of liver dysfunction and hepatic encephalopathy.[8,9]

Herein, we present the case of a liver cirrhosis patient who presented with left sided hemiplegia which is a very rare presentation of hepatic encephalopathy.

CASE PRESENTATION

We report the case of a 55-year-old gentleman who presented with gradual onset of altered consciousness (irritability, drowsiness but arousable), right sided body weakness and constipation for last 3 days. There was no history of fever, abdominal distension, bleeding, vomiting, abdominal pain, cough, sputum, fits, joint pains or trauma. He was diagnosed with Hepatitis C positive Chronic liver disease 3 years ago. He had completed a 24-week course of oral anti-viral therapy (sofosbuvir 400mg and daclatasvir 60mg) and remained PCR negative since then. He was married with 3 children and worked as a tailor. He was a non-smoker, non-alcoholic and did not use illicit drugs. On examination, the patient was drowsy but arousable having a GCS of 12/15 (E3, V4, M5) and normal vital signs. Power of grade 4/5 was noted in both left upper and lower limb with an extensor left plantar reflex. There was no focal sensory, cerebellar or cranial nerve involvement. Flapping tremors were preset and bilateral mild pitting pedal edema was noted. There was no jaundice, pallor, clubbing, distended veins, gynecomastia or spider telangiectasia. Abdomen was soft, non-tender, mildly distended with positive shifting dullness, however no viscera were palpable. Cardiac and respiratory examination were unremarkable.

On basis of clinical findings of left sided hemiplegia, a CT scan (Brain) was done to rule out intracranial bleeding and the patient was admitted on lines of ischemic stroke and hepatic encephalopathy. On investigation, CBC revealed thrombocytopenia with platelet count 113 x 10^9/L but normal hemoglobin and TLC. LFTs revealed hemoglobin 1.8 mg/dl, ALT 434 IU, AST 486 IU and albumin 2.6 mg/dl. Serum ammonia levels were markedly raised at 1211 umol/l. Serum electrolytes, PT/APTT, RFTs and urinalysis were normal. USG abdomen revealed coarse shrunken liver and mild abdomin-pelvic ascites. MRI Scan (Brain) was done to show foci of diffusion restriction in the left periventricular region and basal ganglia and T2WI/FLAIR hyperintense signals in the periventricular region, basal ganglia and deep white matter as shown in Figure 1. It was followed by MR angiography which demonstrated normal cerebral vessels which helped to rule out thrombo-embolism. His ECG, echocardiography and chest X-ray were within normal parameters. Based on clinical and radiographic findings, he was diagnosed as having hepatic encephalopathy mimicking stroke and underlying liver cirrhosis Child-Pugh Class B (patient score 9). Furthermore, after being prescribed lactulose and Rifaximin during hospital stay, the patient passed stools which resulted in improvement in consciousness and resolution of the left sided body weakness. His repeat serum ammonia was within normal limits and the patient was asymptomatic by day 3 and discharged.

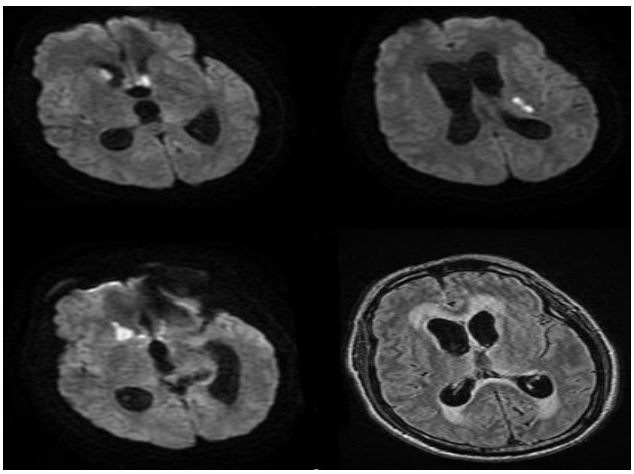

Figure 1: MRI Brain showing foci of diffusion restriction in the left periventricular region and basal ganglia and T2WI/FLAIR hyperintense signals in the periventricular region, basal ganglia and deep white matter

DISCUSSION

It is uncommon for hepatic encephalopathy and liver cirrhosis to produce focal neurological findings. Pearce et al.[10] reported focal neurological signs in 3% patients with liver decompensation having hepatic encephalopathy. Cadranel et al.[11] demonstrated focal neurological signs in 17.4% patients of hepatic encephalopathy but MRI changes were not present in any patient. Moreover, the symptoms resolved with treatment of hepatic encephalopathy. Younes et al.[12] reported a patient of hepatic encephalopathy presenting with features of middle cerebral artery infarct. However, there were no findings suggestive of infarct on MR angiography. The focal weakness was attributed to hepatic encephalopathy and resolved with treatment of hepatic encephalopathy.[12] Our patient presented with focal neurological signs characterized by left sided hemiparesis in addition to drowsiness and constipation. MR angiography was normal helping to rule out thrombo-embolism as a cause of hemiparesis in our patient. Subsequently his symptoms improved with treatment of hepatic encephalopathy and were completely resolved as the encephalopathy reversed.

The management of hepatic encephalopathy is based on the severity of encephalopathy, its causative agents, its complications and exclusion of other factors which may be contributing to altered conscious level such as stroke.[7,13,14] Lactulose (beta-galactosidofructose) and lactilol (beta-galactosidosorbitol) are two non-

absorbable disaccharides which are routinely used in treatment of hepatic encephalopathy. Lactulose and lactilol are degraded by the intestinal bacteria into lactic acid and other organic acids which appear to reduce intestinal production of ammonia.[15] A non-absorbable derivative of rifampin, rifaximin acts by reducing the population of gut microbiota as well as their metabolic function and has been shown to be as effective as lactulose or lactitol at improving hepatic encephalopathy symptoms.[7,16] Other oral antibiotics that have been used include neomycin, metronidazole, paromomycin and vancomycin. Treatments that increase ammonia clearance include L-ornithine L-aspartate (LOLA), zinc, Sodium benzoate, glycerol phenylbutyrate, sodium phenylacetate, sodium phenylbutyrate and L-carnitine.[17] Patients with end-stage liver cirrhosis may require liver transplantation.

Consent: Detailed informed consent was obtained from the patient and their attendant prior to data collection and manuscript writing.

REFERENCES

1. Blackwell DL, Lucas JW, Clarke TC. Summary health statistics for U.S. adults: national health interview survey, 2012. Vital Health Stat 10. 2014;(260):1-161.
2. Murphy SL, Xu J, Kochanek KD, Curtin SC, Arias E. Deaths: Final Data for 2015. Natl Vital Stat Rep. 2017;66(6):1-75.
3. Hyder AA, Morrow RH. Applying burden of disease methods in developing countries: a case study from Pakistan. Am J Public Health. 2000;90(8):1235-1240. doi: 10.2105/ajph.90.8.1235.
4. Khan S, Butt NI, Tareen MQK, Sami H, Ashfaq F, Mahmood K. Hyponatremia in liver cirrhosis patients presenting with hepatic encephalopathy. Professional Med J 2022;29(7):970-974. doi:10.29309/TPMJ/2022.29.07.7057.
5. Talwalkar JA. Prophylaxis with beta blockers as a performance measure of quality health care in cirrhosis. Gastroenterology. 2006;130(3):1005-1007. doi: 10.1053/j.gastro.2005.11.055.
6. Ahmad G, Butt NI, Ashfaq F, Khan AA, Nazir M, Raheem R. Prevalence of pulmonary hypertension in patients of chronic liver disease. Rawal Medical Journal. 2021;46(4): 792-794.
7. Butt NI, Butt UI, Kakar AATK, Malik T, Siddiqui AM. Is Lactulose Plus Rifaximin Better than Lactulose Alone in the Management of Hepatic Encephalopathy? J Coll Physicians Surg Pak. 2018;28(2):115-117. doi: 10.29271/jcpsp.2018.02.
8. Felipo V. Hepatic encephalopathy: effects of liver failure on brain function. Nat Rev Neurosci. 2013;14(12):851-858. doi: 10.1038/nrn3587.
9. Yamamoto Y, Nishiyama Y, Katsura K, Yamazaki M, Katayama Y. Hepatic encephalopathy with reversible focal neurologic signs resembling acute stroke: case report. J Stroke Cerebrovasc Dis. 2011;20(4):377-380. doi: 10.1016/j.jstrokecerebrovasdis.2010.01.011.
10. Pearce JM. Focal Neurological Syndromes in Hepatic Failure. Postgrad Med J. 1963;39(457):653-657. doi: 10.1136/pgmj.39.457.653.
11. Cadranel JF, Lebiez E, Di Martino V, Bernard B, El Koury S, Tourbah A, et al. Focal neurological signs in hepatic encephalopathy in cirrhotic patients: an underestimated entity? Am J Gastroenterol. 2001;96(2):515-518. doi: 10.1111/j.1572-0241.2001.03552.x.
12. Younes K, Gonzales NR, Sarraj A, Bonfante E, Jagolino-Cole A. Hepatic Encephalopathy Mimicking Acute Dominant Middle Cerebral Artery Ischemic Stroke: A Case Report. Case Rep Neurol. 2019;11(3):304-311. doi: 10.1159/000504017.
13. Habib O, Ashaq F, Butt NI, Mahmood K, Khan AA, Qureshi MA. Does Having a Chronic Liver Illness Make One More Likely to Become Melancholic and Anxious? PJMHS. 2022;16(07):227-229. doi: 10.53350/pjmhs22167227.
14. Zhan T, Stremmel W. The diagnosis and treatment of minimal hepatic encephalopathy. Dtsch Arztebl Int. 2012;109(10):180-187. doi: 10.3238/arztebl.2012.0180.
15. Campion D, Giovo I, Ponzo P, Saracco GM, Balzola F, Alessandria C. Dietary approach and gut microbiota modulation for chronic hepatic encephalopathy in cirrhosis. World J Hepatol. 2019;11(6):489-512. doi: 10.4254/wjh.v11.i6.489.
16. Arif K, Butt NI, Ali F, Ashfaq F, Habib O, Kakar AATK. Frequency of Recurrence of Hepatic Encephalopathy in Patients of Chronic Liver Disease Treated With Rifaximin. PJMHS. 2022;16(10):150-152. doi: 10.53350/pjmhs221610150.
17. Zacharias HD, Zacharias AP, Gluud LL, Morgan MY. Pharmacotherapies that specifically target ammonia for the prevention and treatment of hepatic encephalopathy in adults with cirrhosis. Cochrane Database Syst Rev. 2019;6(6):CD012334. doi: 10.1002/14651858.CD012334.pub2.

AUTHOR'S CONTRIBUTIONS

MSAG: Concept, study design, data collection, critical review
NIB: Concept, study design, Literature research manuscript writing
FQ: Concept, study design, data collection critical review
MUJ: Data collection, manuscript writing
AYA: Data collection, critical review
FA: Literature research, manuscript writing