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HEPATIC ENCEPHALOPATHY PRESENTING WITH ACUTE NEUROLOGIC DEFICITS IN CIRRHOSIS: CASE REPORT AND PRACTICAL DIAGNOSTIC APPROACH

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Background: Overt hepatic encephalopathy (OHE) occurs in approximately 30–45% of patients with cirrhosis and remains a frequent cause of acute altered mental status requiring urgent evaluation [1, 2]. In the emergency setting, OHE can present as a stroke mimic (metabolic encephalopathy) and diagnostic delays are common; therefore, early recognition and prompt identification of precipitating factors are critical to initiate targeted therapy and avoid unnecessary stroke-directed interventions [3, 4].

Case report: A 58-year-old man with known alcohol-related cirrhosis (previously compensated, no history of stroke) was brought to the emergency department due to abrupt behavioral changes, disorientation, and slurred, incoherent speech that developed over 12 hours. Relatives reported progressive somnolence and reduced oral intake during the previous two days. On arrival, blood pressure and oxygen saturation were stable, temperature was normal. Neurological assessment revealed fluctuating attention and psychomotor slowing without persistent focal deficits; pupils were equal and reactive, cranial nerves were grossly intact, and there was asterixis. The Glasgow Coma Scale score was 13, consistent with West Haven grade II–III hepatic encephalopathy.

Given the acute onset, a stroke pathway was initially activated. Non-contrast head CT showed no acute ischemia or hemorrhage. Bedside glucose was normal. Further examination revealed melena, and laboratory tests demonstrated anemia with elevated urea, impaired liver function tests, and mild electrolyte disturbances; serum ammonia was elevated (supportive but not diagnostic). There was no leukocytosis and no meningeal signs. The clinical picture was interpreted as overt hepatic encephalopathy precipitated by upper gastrointestinal bleeding (likely variceal) with dehydration.

Topical sedatives and opioids were avoided. Treatment was initiated immediately with lactulose titrated to achieve 2–3 soft stools/day and rifaximin as add-on therapy due to moderate severity. Simultaneously, precipitating factors were addressed: intravenous fluids and electrolyte correction were started, and gastroenterology management for suspected variceal bleeding was initiated (hemodynamic monitoring and endoscopic source control as indicated). Within 24–48 hours, mental status improved markedly: asterixis resolved, orientation returned, and speech normalized.

By day 3, the patient returned to baseline cognition, and secondary prevention counseling emphasized trigger control (bleeding, constipation, dehydration, infections, sedatives) and early presentation if symptoms recur.

Conclusion: Hepatic encephalopathy should be considered early in any patient with cirrhosis who presents with acute confusion, dysarthria, or fluctuating consciousness, as it may closely resemble an acute cerebrovascular event. because the presentation can closely resemble an acute cerebrovascular event. Recognition of key bedside features, particularly asterixis and impaired attention with fluctuation, together with rapid exclusion of structural brain lesions and a prompt search for precipitating factors, enables timely targeted treatment. The most clinically relevant triggers include upper gastrointestinal bleeding, dehydration, electrolyte disturbances, constipation, infection, and sedative drugs. In this case, immediate initiation of lactulose therapy with add-on rifaximin combined with simultaneous control of suspected upper gastrointestinal bleeding resulted in rapid neurological recovery. The outcome highlights that clinical assessment and correction of triggers determine success more than serum ammonia values alone.

References:

1. Duah A, Agyei-Nkansah A, Osei-Poku F, Duah F, Ampofo-Boobi D, Peprah B. The Prevalence, Predictors, and In-Hospital Mortality of Hepatic Encephalopathy in Patients with Liver Cirrhosis Admitted at St. Dominic Hospital in Akwatia, Ghana. *Can J Gastroenterol Hepatol*. 2020;2020:8816522. doi:10.1155/2020/8816522. https://pubmed.ncbi.nlm.nih.gov/33425806/

2. Grønkjær LL, Sehsted TH, Norlyk A, Vilstrup H. Overt Hepatic Encephalopathy Experienced by Individuals With Cirrhosis: A Qualitative Interview Study. *Gastroenterol Nurs*. 2018;41(6):468-476. doi:10.1097/SGA.0000000000000286. https://pubmed.ncbi.nlm.nih.gov/33425806/

3. Okano Y, Ishimatsu K, Kato Y, Yamaga J, Kuwahara K, Okumoto K, et al. Clinical features of stroke mimics in the emergency department. *Acute Med Surg.* 2018;5(3):241-248. doi:10.1002/ams2.338. https://pubmed.ncbi.nlm.nih.gov/29988676/

4. European Association for the Study of the Liver. EASL Clinical Practice Guidelines on the management of hepatic encephalopathy. J Hepatol. 2022;77(3):807-824. doi:10.1016/j.jhep.2022.06.001. https://pubmed.ncbi.nlm.nih.gov/35724930/