

When the Liver Affects the Brain

Neuropsychiatric Features of Hepatic Encephalopathy

Ana Francisca Aires^{1*}; Mariana Lázaro²

^{1,2}Department of Psychiatry and Mental Health (DPSM) Unidade Local de Saúde do Algarve (ULS) Faro, Portugal

Corresponding Author: Ana Francisca Aires^{1*}

Publication Date: 2026/04/14

Abstract: Hepatic encephalopathy is a serious neuropsychiatric complication of acute or chronic liver dysfunction and represents a major cause of morbidity among patients with chronic liver disease. We report the case of a 51-year-old female patient from Guinea with a history of chronic hepatitis B since childhood who presented with progressive neuropsychiatric symptoms, including behavioral disturbances and auditory hallucinations, necessitating hospitalization in the psychiatric ward. Laboratory investigations revealed markedly elevated liver enzymes, including aspartate aminotransferase (AST) 250 U/L, alanine aminotransferase (ALT) 450 U/L, and gamma-glutamyl transferase (GGT) 2000 U/L, accompanied by hyperammonemia of 170 $\mu\text{mol/L}$. The patient was managed with lactulose and antipsychotic therapy (paliperidone), resulting in gradual improvement of mental status and behavioral symptoms. This case emphasizes the importance of early recognition and appropriate management of neuropsychiatric manifestations in hepatic encephalopathy.

Keywords: Hepatic Encephalopathy, Hepatitis B, Neuropsychiatric Symptoms, Hyperammonemia.

How to Cite: Ana Francisca Aires; Mariana Lázaro (2026) When the Liver Affects the Brain. *International Journal of Innovative Science and Research Technology*, 11(4), 443-444. <https://doi.org/10.38124/ijisrt/26apr071>

I. INTRODUCTION

This case report illustrates the clinical presentation, comprehensive diagnostic evaluation, and multidisciplinary management of a patient with hepatic encephalopathy secondary to chronic hepatitis B infection, highlighting the challenges in distinguishing neuropsychiatric symptoms of hepatic origin from primary psychiatric disorders. The patient's acute behavioral changes, including verbal hallucinations and disorientation, prompted psychiatric admission, yet laboratory and biochemical investigations, including markedly elevated aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), and hyperammonemia, confirmed the underlying hepatic pathology. The successful integration of supportive care, ammonia-lowering therapy with lactulose, and judicious use of antipsychotic medication (paliperidone) underscores the importance of timely recognition and targeted intervention in preventing further neurological deterioration. This report emphasizes the need for heightened clinical awareness of hepatic encephalopathy in middle-aged patients presenting with acute neuropsychiatric disturbances, particularly in the context of chronic liver disease, to facilitate early diagnosis, optimize treatment outcomes, and reduce the risk of long-term cognitive impairment.

II. CASE PRESENTATION

A 51-year-old female patient from Guinea with a history of chronic hepatitis B since childhood was admitted to the psychiatric ward due to progressive behavioral disturbances and the onset of verbal hallucinations. The patient demonstrated disorientation, altered mental status, irritability, and significant changes in personality and behavior. She had no prior psychiatric history, and these neuropsychiatric symptoms represented a new-onset condition.

Laboratory evaluation revealed substantially elevated liver enzymes, including aspartate aminotransferase (AST) 250 U/L, alanine aminotransferase (ALT) 450 U/L, and gamma-glutamyl transferase (GGT) 2000 U/L, indicating significant hepatocellular injury. Subsequent measurement of serum ammonia levels confirmed hyperammonemia of 170 $\mu\text{mol/L}$, consistent with hepatic encephalopathy. Imaging studies, including brain MRI and CT scan, showed no structural abnormalities, and no focal neurological deficits were identified.

The patient was treated with lactulose, administered orally to reduce intestinal ammonia absorption, and paliperidone, an antipsychotic agent, to control behavioral disturbances and hallucinations. Over the course of therapy,

the patient showed gradual improvement in cognitive function and psychiatric symptoms, supporting the diagnosis of hepatic encephalopathy with neuropsychiatric manifestations secondary to chronic hepatitis B.

III. DISCUSSION

Hepatic encephalopathy is a major complication of chronic liver disease and may manifest primarily with neuropsychiatric symptoms. The pathophysiology involves elevated ammonia levels, neuroinflammation, and neurotransmitter imbalances, leading to cerebral dysfunction. In patients with chronic hepatitis B, ongoing liver injury can progress silently, culminating in episodes of HE that may present acutely with psychiatric symptoms such as hallucinations, confusion, and behavioral changes. Misdiagnosis as a primary psychiatric disorder is common, particularly when liver disease is not previously recognized or asymptomatic.

In this patient, laboratory findings, including AST 250 U/L, ALT 450 U/L, GGT 2000 U/L, and hyperammonemia of 170 $\mu\text{mol/L}$, provided clear evidence of hepatic dysfunction and metabolic derangement. Lactulose therapy, by reducing intestinal ammonia absorption, remains a cornerstone in the management of HE, while antipsychotics such as paliperidone may be used judiciously to control severe behavioral disturbances. Early recognition and management are critical to prevent progression to coma or irreversible neurological damage.

This case emphasizes the importance of including hepatic encephalopathy in the differential diagnosis of acute-onset neuropsychiatric symptoms in adults with chronic liver disease. Multidisciplinary collaboration among hepatologists, psychiatrists, and neurologists is essential for accurate diagnosis and effective treatment.

IV. CONCLUSION

Neuropsychiatric manifestations may often represent the earliest clinical indication of hepatic encephalopathy in patients with chronic liver disease, particularly in those with long-standing hepatitis B infection. These manifestations can range from subtle cognitive impairment, such as inattention or forgetfulness, to overt psychiatric symptoms, including acute behavioral changes, agitation, emotional lability, and both auditory and visual hallucinations. Such presentations are frequently misattributed to primary psychiatric disorders, which can lead to delayed recognition of the underlying hepatic pathology. Behavioral changes and hallucinations in this context should prompt immediate evaluation for liver dysfunction, including comprehensive assessment of liver enzymes - such as AST, ALT, and GGT - as well as measurement of serum ammonia levels, which serve as critical indicators of hepatic metabolic impairment. Early and timely initiation of ammonia-lowering therapy, including agents such as lactulose, in combination with appropriate supportive psychiatric care, can result in significant clinical improvement and may prevent further neurological deterioration.

Clinicians should maintain a high index of suspicion for hepatic encephalopathy whenever confronted with new-onset neuropsychiatric symptoms in patients with known or suspected liver disease, and a multidisciplinary approach involving hepatology, psychiatry, and internal medicine is essential to ensure accurate diagnosis, optimize treatment outcomes, and reduce the risk of long-term cognitive and functional impairment.

ACKNOWLEDGMENT

The authors declare no acknowledgments.

REFERENCES

- [1]. A. Schiff, *Diseases of the Liver*, 11th ed., Philadelphia: Lippincott, 2018, pp. 245-260.
- [2]. J. Ferenci, "Hepatic encephalopathy," *Gastroenterology*, vol. 123, pp. 726-742, 2002.
- [3]. M. Amodio, F. Montagnese, and R. Gatta, "Neuropsychiatric manifestations of hepatic encephalopathy," *Metab Brain Dis*, vol. 20, pp. 141–150, 2005.
- [4]. K. Bustamante, P. Rimola, and J. Ventura, "Clinical features and prognosis of hepatic encephalopathy in patients with cirrhosis," *J Hepatol*, vol. 20, pp. 565–572, 1994.
- [5]. R. Blei and M. Cordoba, "Current concepts in the management of hepatic encephalopathy," *N Engl J Med*, in press.
- [6]. L. Shawcross, and P. Jalan, "The pathophysiology of hepatic encephalopathy: recent advances," *Clin Liver Dis*, vol. 10, pp. 669–684, 2006.