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Hepatic Encephalopathy in a -76 year-old Female with Reactivation of Chronic Hepatitis B due to Cytomegalovirus Infection: A Case Report

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ABSTRACT

Case Presentation: This case report documents a unique presentation of hepatic encephalopathy induced by the reactivation of chronic hepatitis B due to Cytomegalovirus (CMV) infection in a 76-year-old female patient. The patient was admitted with a diminished level of consciousness subsequent to a week of anorexia, intermittent fever, chills, postprandial nausea, and vomiting. Laboratory tests revealed significant hepatocellular injury, while imaging suggested cirrhosis. The presence of hepatitis B surface antigen and a detectable hepatitis B virus DNA qualitative analysis was noted, alongside an elevated CMV viral load, indicating the reactivation of chronic hepatitis due to CMV infection. Therapeutic interventions included Ganciclovir for CMV infection, Colistin for an Enterococcus urinary infection, and Tenofovir for hepatitis. Upon stabilization, the patient was discharged for continued medical management.

Conclusion: This case highlighted the significance of considering the reactivation of chronic viral hepatitis in the differential diagnosis of hepatic encephalopathy. It underscored the critical role of comprehensive diagnostic investigation in patients presenting with reduced consciousness, especially those with a history of liver disease. Moreover, it emphasized the importance of managing concurrent infections in the context of liver disease and served as a reminder of the potential for CMV to reactivate chronic hepatitis, leading to serious complications, such as hepatic encephalopathy.

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Introduction

Hepatic encephalopathy is a complex neuropsychiatric syndrome, associated with liver disease, characterized by a broad spectrum of neurological or psychiatric abnormalities ranging from subclinical alterations to coma (1). It is a major complication of cirrhosis and is associated with a poor prognosis (2). Hepatic encephalopathy can be precipitated by various factors, including infections, gastrointestinal bleeding, constipation, electrolyte imbalance, and medications (3).

Cytomegalovirus (CMV), a member of the Herpesviridae family, is known to be a major cause of morbidity and mortality in immunocompromised patients (4). The CMV hepatitis is usually asymptomatic or presents with a mild mononucleosis-like syndrome in immunocompetent hosts, but can be severe in immunocompromised patients, leading to fulminant hepatitis, hepatic failure, and death (5).

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This case report aimed to present a rare instance of reactivation of chronic hepatitis B due to CMV infection leading to hepatic encephalopathy in a 76-year-old female patient. This case underscored the importance of a thorough investigation when a patient presents with decreased consciousness level, particularly in those with a history of liver disease, as well as the role of managing underlying infections in the context of liver disease.

Case Presentation

A 76-year-old female, with a known medical history of hypothyroidism and hypertension, presented to Thamen Hospital with a decreased level of consciousness. The week preceding her admission was marked by intermittent fever and chills, anorexia, postprandial nausea, and vomiting, which resulted in overall weakness and diminished consciousness. She was diagnosed with hepatic encephalopathy and transferred to the ICU following intubation due to deteriorating consciousness after a two-day hospital stay.

Laboratory investigations at admission revealed hepatocellular injury as evidenced by abnormal liver function tests. The hepatitis B surface antigen was positive, and hepatitis B virus DNA was qualitatively detected, with a viral load of 10,000. Herpes simplex virus polymerase chain reaction (PCR), COVID-19, and Influenza PCR were negative. Moreover, autoimmune markers were negative, ruling out autoimmune hepatitis. Abdominal sonography indicated a nodular and heterogeneous liver, suggestive of cirrhosis [Figure 1]. Further investigations for Wilson's disease and hemochromatosis were negative. A liver fibroscan indicated a fibrosis grade of 4.

Interestingly, CMV was detected with a high viral load of 245,784 suggesting a reactivation of chronic hepatitis secondary to CMV infection. Other significant findings included a positive urine culture for Enterococcus, indicative of a urinary tract infection. The antinuclear antibodies of the patient were mildly elevated, which was attributed to the CMV infection. Serum protein electrophoresis showed a gamma peak at 23.5, consistent with cirrhosis and not indicative of autoimmune hepatitis.

The patient was started on Ganciclovir for the CMV infection, Colistin for the urinary tract infection caused by Enterococcus, and Tenofovir for hepatitis. After stabilization, she was extubated on the 10th day of her hospital stay and discharged with a final diagnosis of chronic hepatitis reactivated by CMV.

Follow-up laboratory investigations revealed some improvement, but with continued abnormalities in coagulation tests, a high ferritin level, and low creatinine (Table 1). Hematological evaluation suggested a form of anemia, and urine analysis revealed the presence of red blood cells and significant blood, indicative of a urinary tract issue.

The patient was scheduled for a follow-up plan, including a Magnetic Resonance Cholangiopancreatography and continued

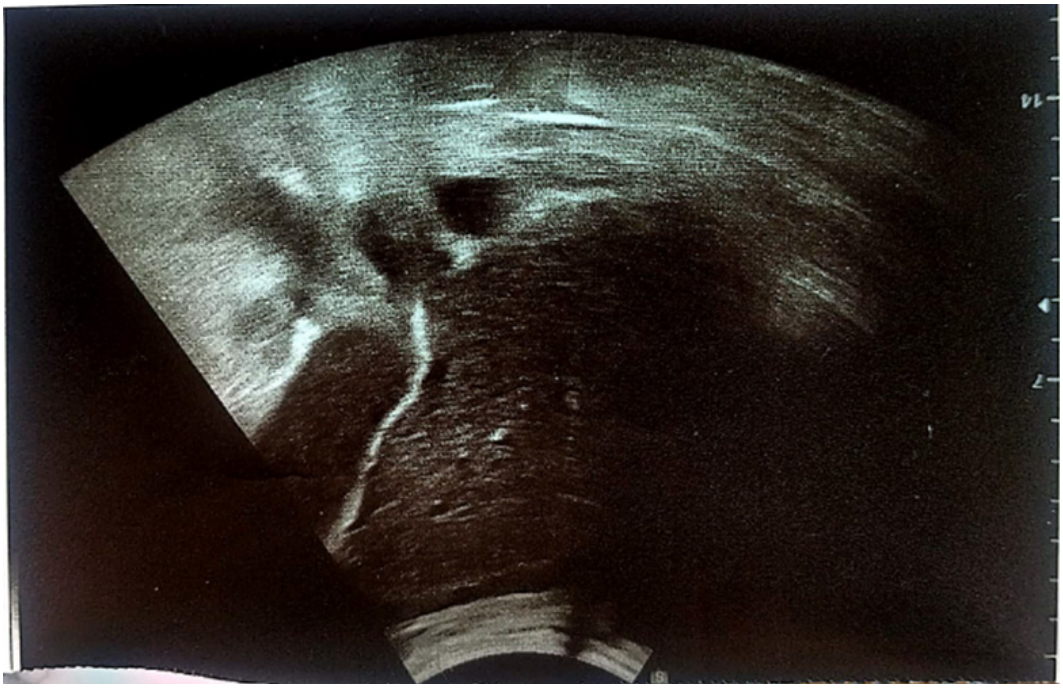

Figure 1. Abdominal sonography indicated a nodular and heterogeneous liver, suggestive of cirrhosis

Table 1. Laboratory Investigations

Test	Results	Normal range for adult females	Interpretation
Liver-related tests			
Liver-function test	Abnormal	Normal	Indicates hepatocellular injury
HBsAg	Positive	Negative	Suggests active Hepatitis B infection
HBV DNA	10,000	Undetectable	Indicates active Hepatitis B infection
CMV test	(High) 245,784	Negative/low	Indicates active CMV infection
Autoimmune markers	Negative	Negative	Rules out autoimmune hepatitis
ANA	Mildly elevated	Negative	Can be due to CMV infection
Liver Fibroscan	Grade 4	Grade 0-1	Indicates cirrhosis
Wilson's disease test	Negative	Negative	Rules out Wilson's disease
Hemochromatosis test	Negative	Negative	Rules out Hemochromatosis
Serum Protein Electrophoresis	Gamma peak at 23.5	No Gamma peak	Consistent with cirrhosis
Viral Tests			
HSV PCR	Negative	Negative	No Herpes Simplex Virus infection
COVID-19 PCR	Negative	Negative	No COVID-19 infection
Influenza PCR	Negative	Negative	No Influenza infection
Urinary tract-related tests			
Urine culture	Positive for enterococcus	No growth	Indicates urinary tract infection
Urine analysis	Presence of RBCs and blood	Negative	Indicates urinary tract issue
Blood and hematological tests			
Coagulation tests	Abnormal	Normal	Indicates impaired blood clotting
Ferritin	High	ng/mL 15-200	Possibly indicates inflammation or iron overload
Hematological evaluation	Suggests anemia	g/dL 12.0-15.5	Indicates anemia
Creatinine	Low	mg/dL 0.57-1.0	May suggest decreased muscle mass or impaired liver function

HBsAg: hepatitis B surface antigen HBV: hepatitis B virus, HSV: herpes simplex virus, ANA: antinuclear antibodies, PCR: polymerase chain reaction, CMV: Cytomegalovirus, RBC: red blood cell

treatment with Tenofovir and ursodeoxycholic acid (UDCA). The rationale for the use of UDCA was the possibility of biliary sludge due to the immobility of the patient during her hospital stay.

This case highlighted the importance of considering CMV infection in the differential diagnosis of hepatitis, particularly in patients not responding to initial treatments. Furthermore, it underscored the possibility of biliary sludge in immobile patients, emphasizing the role of mobilization in the prevention of such complications. The patient was advised for a follow-up to monitor the improvement in bilirubin levels and overall condition.

Discussion

In this report, we presented a case of a 76-year-old woman with chronic hepatitis B reactivated by CMV infection, leading to hepatic encephalopathy. This case highlighted the complexity of managing chronic liver disease, particularly in the context of infections, which are known precipitants of hepatic encephalopathy (3).

The CMV infection is common, with a seroprevalence of 40-100% in adults (6). In immunocompetent individuals, it is usually asymptomatic or presents as a mononucleosis-like syndrome. However, CMV can reactivate and cause severe disease in immunocompromised individuals, such as those with HIV/AIDS, transplant recipients, and apparently in our patient with chronic hepatitis B (7).

In this case, CMV infection appeared to have reactivated chronic hepatitis B, causing hepatic injury that led to hepatic encephalopathy. This added to the emerging body of evidence indicating that CMV can trigger exacerbations of chronic hepatitis B (8).

Detection of a high CMV viral load in our patient underscored the importance of considering CMV infection in patients with chronic liver disease who presented with hepatic encephalopathy. Early detection and treatment of CMV infection can potentially prevent the progression of hepatic encephalopathy and improve outcomes (9). After a one-month follow-up, the icteric condition of the patient was resolved, and their overall health was good.

Conclusion

In conclusion, this case demonstrated the importance of considering CMV infection as a potential trigger for hepatic encephalopathy in patients with chronic liver disease. It highlighted the necessity of thorough investigations, including screening for CMV, in patients with decreased consciousness and a history of liver disease. Future studies are needed to further explore the mechanisms underlying the exacerbation of chronic hepatitis B by CMV and to develop effective strategies for the prevention and treatment of this potentially lethal combination.

Conflict of interest

The authors declare no conflicts of interest.

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