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Case Report

Patients with acute liver failure due to hepatitis B: a case report

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ABSTRACT

Hepatitis B is an infectious disease of liver cells by the hepatitis B virus (HBV). Hepatitis B is characterized by symptoms such as jaundice, decreased appetite, and abdominal pain. Acute liver failure (ALF) is one of the serious complications of hepatitis B. Previous studies reported that the incidence of acute hepatitis B patients reached 41.7% within 28 days after hospitalization, with high mortality rates on the 28th and 90th days, namely 43.9% and 70.4%. In these patients, they showed classic symptoms of persistent jaundice, nausea, vomiting, pain in the right upper quadrant, and progressive bloating. Extreme elevations in bilirubin and liver enzymes indicate a high level of hepatocyte necrosis, which is common in ALF due to acute HBV infection or reactivation of chronic infection. Patients with a history of hepatitis B develop ALF. ALF due to hepatitis B infection is a serious condition that requires immediate medical attention. Early diagnosis, including identification of classic symptoms such as jaundice, nausea, and abdominal pain, is essential. Management should include combination antiviral and supportive therapy to prevent further complications, including death. With better knowledge and understanding of the disease mechanism and clinical management, it is hoped that the prognosis of patients with this condition can improve in the future.

Keywords: Hepatitis B, Acute liver failure, Jaundice

INTRODUCTION

Hepatitis B is an infectious disease of the liver cells caused by the HBV, which has a significant impact on global health, including the potential development of liver cirrhosis and hepatocellular carcinoma, which can lead to ALF. It is estimated that more than 257 million people globally live with chronic hepatitis B infection, with high prevalence rates in Southeast Asia and sub-Saharan Africa.[1] In Indonesia, the prevalence of hepatitis B ranges from 7-10%, especially in high-risk populations such as injection drug users and patients with chronic diseases.[2]

Hepatitis B is characterized by symptoms such as jaundice, decreased appetite, and abdominal pain, with transmission mainly through contact with infected body fluids or blood, including sexual intercourse and vertical transmission from mother to child at birth.[3] Hepatitis B infection can cause chronic liver damage through immunological mechanisms, where an excessive immune response causes inflammation and hepatocyte necrosis, increasing the risk of complications such as cirrhosis, hepatocellular carcinoma, and ALF.[1]

ALF is a serious complication of hepatitis B. The development of ALF in hepatitis B patients can occur in patients with risk factors such as discontinuation of antiviral therapy, alcohol exposure, and corticosteroid use.[4] It is estimated that around 5-10% of patients with hepatitis B can develop into ALF, which is characterized by severe hepatocellular damage, with symptoms such as hepatic encephalopathy and coagulopathy, often requiring liver transplantation as a last resort.[5,6]

Previous studies have reported that the acute incidence rate in hepatitis B patients reaches 41.7% within 28 days post-hospitalization, with high mortality rates on days 28 and 90, which are 43.9% and 70.4%.[7] Because it has a high risk

of death if not treated immediately, rapid diagnosis and treatment through intensive monitoring and supportive therapy is essential to improve patient prognosis.[5]

This report is expected to improve clinical understanding of early diagnosis and management of ALF due to hepatitis B to prevent further liver failure.

CASE REPORT

A 46-year-old male patient came to Buleleng regional general hospital on August 15, 2023, complaining of yellow eyes and a yellow complexion that he had felt for 2 weeks. The yellow complaint was felt to be getting worse and did not go away. The complaint was accompanied by nausea and vomiting, pain in the upper right abdomen and bloating. In addition, the patient felt weak and had a decreased appetite. The patient had a history of hepatitis B since 2015 and routinely took medication. The patient's family history included hepatitis B in the patient's father and sister. Examination revealed vital signs within normal limits, sclera and entire body icteric, tenderness in the upper right abdomen and epigastrium, and abdominal distension. Complete blood examination within normal limits, total bilirubin 16.34 mg/dL, direct bilirubin 9.54 mg/dL, indirect bilirubin 6.80 mg/dL, SGOT 4693. 7 U/L, SGPT 3416.6 U/L, HBsAg rapid reactive, anti-HCV rapid non-reactive, PT 20.1 seconds, INR 1.60 seconds, APTT 38.9 seconds, AFP 16.44 ng/mL. In urinalysis, bilirubin +1, nitrite +1, ketones +1. On abdominal ultrasound, the impression is that there is no picture of hepatitis or cirrhosis of the liver. On an abdominal CT scan with contrast, the impression is cholecystitis secondary to hepatitis, and the liver morphology shows no abnormalities. The patient was diagnosed with ALF + hepatitis B, cholecystitis, and UTI. The management provided was IVFD as follows: aminoleban (2:1) 20 dpm, lansoprazole 1×30 mg IV, ondansentron 3×4 mg IV, ceftriaxon 1×2 gr IV, turmeric 3×1 PO, ursodeoxycholic acid (UDCA) 3×1 PO, tenofovir 1×300 mg PO.

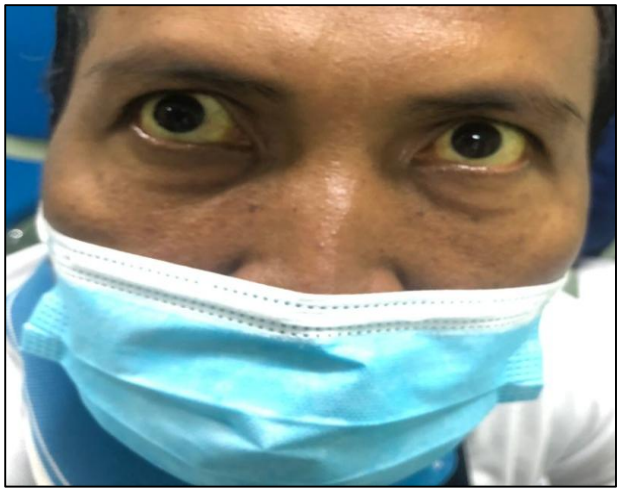

Figure 1: Jaundice on the sclera and face of the patient.

DISCUSSION

Definition

Hepatitis B is a liver infection caused by the HBV, with a prevalence of chronic infection reaching 300 million people. The disease is mainly spread through contact with infected body fluids, such as blood and other secretions, and through transmission from mother to child at birth.[8,9]

Etiology

Hepatitis B is caused explicitly by HBV infection, a DNA virus with a partial double structure from the *Hepadnaviridae* family. HBV has a capsid or core that surrounds its genome, coated with an envelope containing viral surface antigen (HBsAg), which plays an important role in the mechanism of infection and the body's immune response. After entering the human body, HBV attacks hepatocytes through specific receptors on their surface, namely sodium taurocholate co-transporting polypeptide (NTCP), which allows the virus to bind to and enter liver cells.[10,11]

Pathogenetically, HBV uses reverse transcriptase to replicate its genetic material, producing new virions in hepatocytes and spreading the infection to other liver cells. The body's immune response to HBV, which attempts to destroy infected cells, contributes to liver tissue damage and inflammation. Chronic infection can occur if the immune system fails to eliminate the virus, which increases the risk of liver fibrosis, cirrhosis, and primary liver cancer. In addition, HBV variants, such as specific genotypes and mutations, can affect the severity of the disease and response to treatment.[10]

Some risk factors that can increase exposure to HBV include lifestyle, sexual contact, and sharing of syringes. In addition, viral mutations and individual immune resistance also affect the clinical manifestations of hepatitis B. Hepatitis B is often asymptomatic in the early stages, so it is often diagnosed too late until the disease is in an advanced stage or other complications such as liver failure or liver cancer occur.[12]

The phases of hepatitis B

Hepatitis B develops through four clinical phases reflecting the immune response to infection and hepatocyte viral activity. These phases include the immunotolerant, immunoreactive, inactive, and reactivation phases. In the immunotolerant phase, the body does not effectively recognize or respond to HBV even though the virus is actively multiplying; HBV DNA levels are high, but liver enzyme (ALT) activity is normal or low. The immunoreactive phase marks an increased immune response to HBV, characterized by increased ALT, inflammatory potential, and liver damage. During the inactive phase, viral replication is suppressed, HBV DNA levels are low, and liver enzymes are stable. However, in

the reactivation phase, the virus becomes active again, often with a high risk of progression of liver damage.[13]

Progression to ALF can occur when the immune system fails to control HBV replication, or the immune response is very intense, causing massive damage to hepatocytes. This process involves an inflammatory response involving proinflammatory cytokines such as TNF-α and IL-6, which can trigger a "cytokine storm." This progression occurs more frequently in individuals who experience sudden viral reactivation or in cases of acute HBV infection. ALF usually occurs quickly and requires emergency medical attention, given the high risk of multiple organ failure and death.[14]

Table 1: Characteristcs of Hepatitis B phase.

Hepatitis B phase	Characteristics	Risk of progression to ALF
Immunotolerance	HBV active, ALT normal or low	Low risk
Immunoreactive	Improvement ALT, hepatic inflammation	Medium risk
Inactive	HBV suppressed, ALT stable	Low risk
Reactivation	Virus reactivates, inflammation high	High risk

ALF

ALF due to HBV infection is a medical condition characterized by severe and rapid deterioration of liver function. This can occur in both individuals with acute and chronic HBV infection who experience viral reactivation. ALF is often characterized by coagulation disorders and rapidly progressive hepatic encephalopathy, usually in less than 26 weeks in individuals with no previous history of liver disease. The cause of ALF due to HBV involves a complex interaction between the host immune response and the viral infection itself, which can induce damage to hepatocytes and an intense inflammatory response, causing widespread apoptosis of liver cells.[14]

The development of ALF in patients with HBV infection is often exacerbated by the release of inflammatory cytokines such as interleukin-6, which can aggravate liver damage through the "cytokine storm" mechanism. In addition, the pathogenesis of ALF in HBV infection involves complex pathways involving immune activation, autophagy, and apoptotic processes in liver cells. Initial therapy for ALF patients includes respiratory support and management of hepatic encephalopathy. However, liver transplantation is still considered the primary treatment option for patients with poor prognosis or failure to respond to conservative therapy.[15]

Diagnosis of ALF

ALF in hepatitis B patients often begins with nonspecific symptoms such as malaise, anorexia, and nausea. More characteristic clinical manifestations appear when hepatic encephalopathy, jaundice, and severe coagulopathy occur, leading to decreased consciousness and even coma. In ALF, due to hepatitis B, there is a high risk of multiorgan failure and hypoglycemia, which worsens the prognosis.[16]

A thorough history is needed, including a history of hepatitis B virus infection, alcohol consumption, hepatotoxic drug use, and the presence of encephalopathy symptoms and signs of chronic liver disease. The physical examination is focused on signs of hepatic encephalopathy, such as asterixis and changes in consciousness. Icterus, hepatomegaly, and signs of coagulopathy are often found on physical examination. Neurological examination is key in assessing the degree of encephalopathy, which is an important predictor of prognosis in ALF.[17]

In our patient, he showed classic symptoms of persistent jaundice, nausea, vomiting, right upper quadrant pain, and progressive bloating. A history of chronic hepatitis B since 2015 increases the risk of these complications, especially given a family history of HBV infection. Similar cases often progress to acute-on-chronic liver failure (ACLF) due to immunological imbalance and acute hepatic inflammation, which in many cases requires intensive interventions such as liver transplantation for long-term improvement.[6,18,19]

Supportive examinations in patients with ALF include several aspects. First, laboratory tests that include liver function (ALT, AST), bilirubin levels, and coagulopathy (INR) form the basis for assessing laboratory conditions. In addition, hypoglycemia and hyperlactatemia are often found, indicating severe liver failure. Then, in the imaging aspect, an abdominal ultrasound is performed to detect hepatomegaly and help evaluate portal blood flow and the presence of portal vein thrombosis. Furthermore, virological examination through HBsAg and HBV DNA aims to confirm the etiology of hepatitis B in ALF. The prognostic assessment uses the model for end-stage liver disease (MELD) score and King's college criteria, which are internationally recognized prognostic tools to determine the need for immediate liver transplantation.[20]

In our case, the examination results showed classic signs of severe liver impairment, including hyperbilirubinemia with a total bilirubin of 16.34 mg/dL, as well as a significant increase in liver enzymes SGOT (4693.7 U/L) and SGPT (3416.6 U/L). The extreme increase in bilirubin and liver enzymes indicates a high level of hepatocyte necrosis, which is common in ALF due to acute HBV infection or reactivation of chronic infection. Similar case studies show that very high increases in SGOT and SGPT in hepatitis B patients are associated with progressive liver inflammation and damage, often requiring intensive

supportive therapy or further interventions such as liver transplantation for optimal recovery.[19]

Interestingly, imaging examinations such as abdominal CT scans and ultrasonography do not reveal structural abnormalities in the liver, which may indicate that damage occurs primarily at the cellular level without significant morphological changes. This is consistent with the literature, which mentions that in some cases of ALF due to hepatitis B, structural signs are not always visible despite severe biochemical and clinical dysfunction.[5,6]

Diagnostic criteria

ALF is generally diagnosed based on the rapid onset of encephalopathy in individuals with no previous history of liver disease, often accompanied by coagulopathy characterized by an INR ≥1.5. Several core criteria in the diagnosis include several factors. First, the rapid development of encephalopathy, which is characterized by neurological symptoms such as confusion or coma, is the main characteristic of ALF and is an important criterion in diagnosis, both globally and in Indonesia.[21] Second, coagulopathy and INR measurements with a value of 1.5 or higher are required for the diagnosis of ALF, according to the criteria of the American association for the study of liver diseases (AASLD) and the concerns of the Asia Pacific association for the study of the liver (APASL) in areas with limited laboratory access. Third, high transaminase levels, i.e. elevated ALT and AST levels, are a significant indicator, mainly due to the reactivation of hepatitis B, which is a common cause in endemic areas such as Indonesia.[22] Finally, excluding other causes is also an important part of the diagnosis, where practitioners in Indonesia often prioritize differentiating ALF caused by HBV from other causes, such as exposure to aflatoxin or other viral infections.[23]

Therapy

Therapy for ALF in hepatitis B patients aims to treat acute liver damage and increase life expectancy. One of the main approaches is to maintain antiviral treatment to prevent ALF, significantly since discontinuation of therapy can worsen the patient's condition. This is reinforced by findings showing that patients who stop hepatitis B treatment tend to experience serious complications, including the risk of liver transplantation.[5]

The therapeutic approach to ALF involves several strategies designed to address the cause, improve liver function, and prevent complications. First, antiviral therapy is an important step, especially in the case of hepatitis B. Antiviral agents such as Tenofovir or entecavir play a role in suppressing active viral replication and preventing further damage to liver cells. APASL and AASLD recommend this therapy as an initial stabilization effort in ALF patients associated with hepatitis B. Several studies show that the use of Tenofovir can improve ALF symptoms due to hepatitis B, such as jaundice and ascites, within 10 days, demonstrating the potential of this therapy in the management of acute hepatitis B infection.[19,24]

Second, corticosteroid therapy, especially glucocorticoids, has also been shown to be beneficial in reducing mortality in patients with acute-on-chronic hepatitis B liver failure (ACHBLF). Recent research shows that SOCS1 methylation levels can be a prognostic marker for response to glucocorticoid treatment, impacting overall life expectancy. Methylprednisolone (MP) is considered an effective clinical option in increasing the six-month survival rate in patients with ACHBLF caused by hepatitis B, adding to the evidence that this therapy can become the standard in the treatment guidelines for ALF in hepatitis B.[25,26]

Furthermore, supportive therapy and close monitoring are very important in managing ALF. Intensive monitoring of liver function, fluid balance, and possible complications such as hepatic encephalopathy are key to treatment. Supportive therapy is needed to maintain hemodynamics and other vital functions. In some cases, ALF treatment related to hepatitis B includes supportive therapy and liver transplantation as the primary option for patients with severe liver damage. Vaccination and monitoring for hepatitis B infection are also important preventive measures to reduce ALF cases.[6,27]

For severe cases where liver function does not recover despite intervention, liver transplantation is the last option. The criteria for transplantation are usually based on the inability of the liver to recover and the development of severe encephalopathy symptoms. In addition, MELD and other clinical criteria are also applied to determine transplant eligibility. Finally, symptom management and prevention of complications are an important part of treatment, where patients need to be given therapy to prevent secondary complications that commonly occur in ALF, such as electrolyte management, infection prevention, and therapy to prevent brain oedema due to hepatic encephalopathy.[28]

In the management of this case, a patient with ALF due to hepatitis B infection was given combination therapy that included the antiviral Tenofovir to suppress viral replication and ceftriaxone for prophylaxis or treatment of secondary infections, which often accompany ALF. Tenofovir is known as an effective antiviral agent for controlling hepatitis B in patients with ALF, showing improved liver function and resolution of clinical symptoms such as jaundice and ascites in various HBV-related ALF cases.[6,19]

In addition, UDCA and curcuma are added to support hepatoprotective functions, which can help reduce liver inflammation and support bile secretion, especially given the increase in bilirubin levels. Other supportive therapies, such as IV fluid infusion and ondansetron to control nausea, also follow standards to maintain hemodynamics and patient comfort. This comprehensive approach is

practical in several reported cases, especially in patients at high risk of complications from chronic hepatitis B and acute liver disorders.[5,18]

CONCLUSION

In this case report, a 46-year-old male patient with a history of hepatitis B developed ALF. ALF due to hepatitis B infection is a serious condition that requires immediate medical attention. Early diagnosis, which includes the identification of classic symptoms such as jaundice, nausea, and abdominal pain, is essential. Management should include antiviral and supportive combination therapy to prevent further complications, including death. Continuous monitoring and awareness of risk factors, such as discontinuing antiviral therapy and using hepatotoxic substances, are key to patient management. With better knowledge and understanding of the disease mechanism and clinical management, the prognosis for patients with this condition is hoped to improve.

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