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HEPATITIS B-RELATED DECOMPENSATED CIRRHOSIS WITH ASCITES PERMAGNA IN A 64-YEAR-OLD INDONESIAN FARMER

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Abstract

Hepatic cirrhosis increased from 36.9 million in 1990 to 58.4 million in 2021. However, the burden varies significantly by etiology. In 2022, hepatitis B caused 1.1 million deaths from cirrhosis and hepatocellular carcinoma (primary liver cancer). This study highlights how important it is to understand and treat cirrhosis in hepatitis B patients as soon as it develops, as it can be fatal if not treated. We reported a case of HBV-related decompensated cirrhosis of a 64-year-old Indonesian male with a two-week history of progressive abdominal swelling that had worsened significantly in the preceding 24 hours accompanied with dyspnea and epigastric pain. Physical examination revealed bilateral icteric sclera and conjunctival pallor. Abdominal examination was remarkable for a convex, extended abdomen with weakened bowel sounds. Percussion revealed dullness in all quadrants except the umbilical region, positive fluid waves, and shifting dullness. EKG, blood laboratory, chest X-ray, and abdominal ultrasound examinations were performed. The treatment approach addresses multiple aspects of the patient's condition, including diuretics, supportive care for liver function, gastroprotection therapy, and ursodeoxycholic acid. The patient also underwent ascites puncture and found 4400 cc of serous fluid.

Keywords: *Ascites, Hepatitis B, Hepatitis B associated with hepatic cirrhosis, Hepatic cirrhosis, Ascites puncture*

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INTRODUCTION

Hepatic cirrhosis represents one of the most significant liver diseases worldwide, imposing a substantial burden on global healthcare systems and causing considerable morbidity and mortality across diverse populations. The disease affects people across all socioeconomic levels and geographic regions, with incidence between 1990 and 2021, the number of people with hepatic cirrhosis rose from 36.9 million to 58.4 million. (Duo H et al., 2025). The Global Burden of Disease (GBD) studies indicating that the estimated number of deaths associated with cirrhosis reached 1,472,011 in 2019 (Wu XN et al., 2024), constituting 2.4% of all deaths worldwide (Huang DQ et al., 2023). However, the burden varies significantly by etiology, with hepatitis C, hepatitis B, alcohol-related cirrhosis, and non-alcoholic steatohepatitis (NASH) contributing 395.000, 331.000, 372.000, 134.000 deaths in 2019, respectively (Huang DQ et al., 2023). WHO estimates that by 2022, there would be 254 million individuals with a chronic hepatitis B infection and 1.2 million new cases of infection annually. Hepatitis B is estimated to have caused 1.1 million deaths in 2022 from cirrhosis and hepatocellular carcinoma (primary liver cancer) (WHO, 2025).

Specific country-level analyzes reveal even more pronounced variations in cirrhosis epidemiology, with several nations experiencing dramatic changes over recent decades. Egypt consistently demonstrates among the highest age-standardized mortality rates globally, reaching 126.7 per 100,000 population in 2019, while countries like Iceland and Singapore maintain rates as low as 3.3 per 100,000 (Xiao S et al., 2023). Asia-Pacific regions face particularly complex challenges in cirrhosis epidemiology, primarily driven by the high prevalence of viral hepatitis infections that serve as major risk factors for hepatic cirrhosis development. The region accounts for approximately 54.3% of global cirrhosis-related deaths, with hepatitis B causing 51.3% of cirrhosis deaths in the Asia-Pacific region in contrast to just 18.4% in the US and 24.3% in Europe (Sarin SK et al., 2020). An estimated 77–97 million people in China alone suffer from a chronic hepatitis B infection, with around 20–30 million of them having active liver disease (Sarin SK et al., 2020).

The study from Cipto Mangunkusumo Hospital showed a 90-day mortality rate of 42.2% in hepatic cirrhosis patients (Hasan I et al., 2023). During the two-year follow-up 75.3% of the patients died, with infection accounting for 45.5% of all deaths (Gani, 2021). The etiological pattern in Indonesia reflects the country's classification as having intermediate to high hepatitis B endemicity, with hepatitis B representing the predominant cause of cirrhosis in 53.7% of cases, followed by hepatitis C in 22% of cases (Darnindro N et al., 2021). The prevalence of hepatitis B in Indonesia has shown encouraging improvement, declining from 7.1% in 2013 to 2.4% in 2023 according to national Basic Health Research (Riskesdas) data (Indonesian Ministry of Health, 2024). The prevalence of hepatitis B surface antigen in islands outside Java (8.5%) is significantly higher than in Java island (4.9%) (Mulyanto, 2016).

The evolving epidemiological landscape of hepatic cirrhosis demands urgent attention from the global health community, as current trends suggest that without comprehensive intervention strategies, the burden will continue to escalate. This study emphasizes how crucial it is to understand and treat cirrhosis in hepatitis B patients as soon as it develops, as it can be fatal if not treated.

METHODS

This study was based on a case report at K.M.R.T. Wongsonegoro Hospital in April 2024. Information was taken from the patient's inpatient medical records. Both ethics approval and the patient's informed permission were obtained.

CASE REPORT

A 64-year-old Indonesian male farmer presented to the emergency department of K.M.R.T. Wongsonegoro Hospital with a two-week history of progressive abdominal swelling that had worsened significantly in the preceding 24 hours. The complained accompanied with dyspnea and epigastric pain rated 5/10 on the visual analog scale (VAS). The dyspnea was particularly pronounced when lying supine, interfering with his sleep pattern. He denied fever, headache, chest pain, nausea, vomiting, or peripheral edema

The patient had no known history of hypertension, tuberculosis, diabetes mellitus, heart disease, or malignancy. He denied any history of blood transfusions, intravenous drug use, needle sharing, or alcohol consumption. Notably, he had no known allergies to medications. His occupation as a farmer involved regular physical labor, and he frequently used over-the-counter analgesics such as Bodrex® for work-related muscle pain. The patient was an active smoker with a 10-year history of consuming half a pack per day. His dietary habits included regular consumption of herbal medicines, fried foods, and coconut milk-based dishes, which are common in Indonesian cuisine.

On presentation, the patient appeared moderately ill with stable vital signs except for grade 1 hypertension (143/81 mmHg). His oxygen saturation was 95% on room air, with other vital parameters within normal limits. Physical examination revealed significant findings including bilateral conjunctival pallor and icteric sclera.

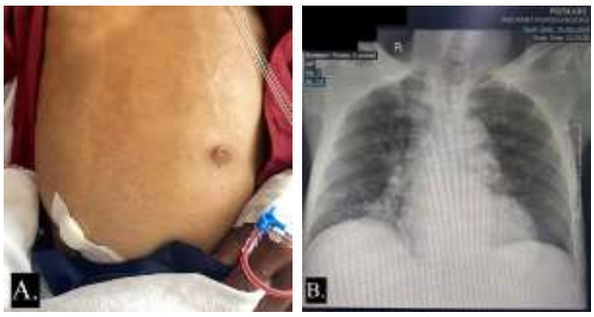

Figure 1. Examinations (a) Abdominal; (b) Chest X-ray

Abdominal examination was remarkable for a convex, extended abdomen with weakened bowel sounds. Percussion revealed dullness in all quadrants except the umbilical region. Positive fluid waves and shifting dullness. The abdomen was soft but demonstrated epigastric tenderness on palpation. Creatinine and other routine blood parameters were within normal limits. Chest x-ray shows bronchopneumonia (Figure 1).

Laboratory tests revealed a qualitatively positive HBsAg, thrombocytopenia, hypoalbuminemia (2.6 g/dL), elevated liver enzymes (SGOT 202 U/L; SGPT 65 U/L), and hypercholesterolemia.

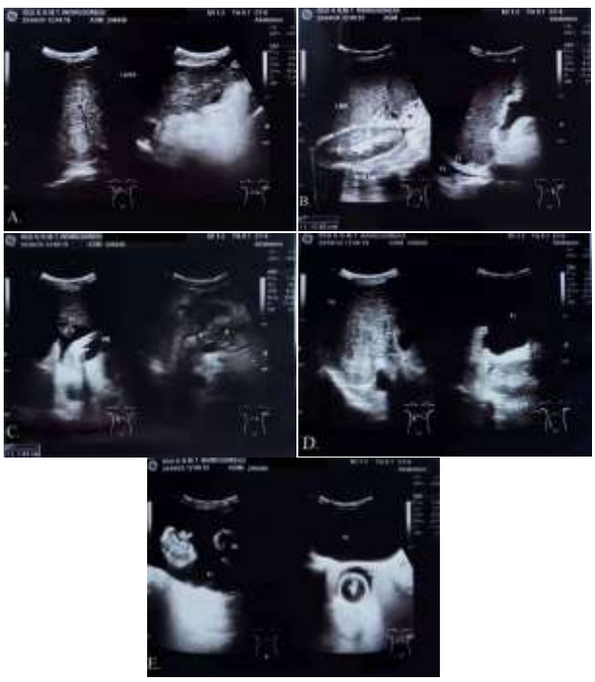

Figure 2. Abominal Ultrasound Examination

A whole abdominal ultrasound revealed a normal liver size but with a rough, inhomogeneous parenchymal structure, increased echogenicity, uneven edges, and blunt angles, which tend to be early signs of hepatic cirrhosis. Splenomegaly measuring 12.43 cm, ascites permagna, left pleural effusion, and cholelithiasis measuring 1.4 cm were also found (Figure 2).

The constellation of findings including positive HBsAg, characteristic ultrasonographic changes of cirrhosis, massive ascites, splenomegaly, and laboratory evidence of hepatocellular dysfunction supported the diagnosis of HBV-related decompensated cirrhosis. The treatment approach addresses multiple aspects of the patient's condition, including diuretic therapy for ascites (combination of furosemide and spironolactone), antibiotic treatment for concurrent infections (such as ceftriaxone and azithromycin), and supportive care for liver function (such as curcuma, aminofusin hepar), gastroprotection therapy, lowering cholesterol therapy, and ursodeoxycholic acid. The patient also underwent ascites puncture and found 4400cc of serous fluid.

DISCUSSIONS

The progression from chronic hepatitis B to decompensated cirrhosis involves complex interactions between viral replication, immune responses, and hepatocellular damage (Cheng JY et al., 2024). In this patient, the positive HBsAg indicates ongoing viral replication and chronic infection, which triggers persistent hepatocellular inflammation and subsequent fibrosis development. The pathophysiological cascade begins with HBV entering hepatocytes through the sodium taurocholate co-transporting polypeptide (NTCP) receptor, leading to formation of covalently closed circular DNA (cccDNA) in the nucleus (Cheng JY et al., 2024). This persistent viral template drives continuous viral protein synthesis and particle assembly, causing direct cytotoxic effects on hepatocytes while simultaneously triggering host immune responses involving CD4+ and CD8+ T cells. The chronic inflammatory milieu results in activation of hepatic stellate cells and excessive extracellular matrix deposition, ultimately leading to architectural distortion and functional impairment characteristic of cirrhosis (Cheng JY et al., 2024). The transition to decompensation occurs when the liver's synthetic and metabolic capacity becomes critically compromised, manifesting as the constellation of findings observed in this patient, including ascites, hypoalbuminemia, and elevated liver enzymes (Guan R et al., 2011).

The patient's presentation with progressive abdominal swelling, dyspnea, and epigastric pain over two weeks represents a classic acute decompensation episode in chronic liver disease (Huang YJ et al., 2025). The progressive abdominal distension over two weeks accompanied by worsening dyspnea reflects the pathophysiology of ascites formation, where splanchnic vasodilation brought on by portal hypertension triggers the renin-angiotensin-aldosterone pathway (Moore CM & Van Thiel DH, 2013). The development of dyspnea specifically indicates the presence of significant

fluid accumulation, as ascites displaces the diaphragm cephalad, reducing respiratory capacity and potentially contributing to the observed pleural effusion. The constellation of physical findings, including bilateral conjunctival pallor, icteric sclera, and massive ascites with positive fluid waves and shifting dullness, confirms significant portal hypertension and hepatocellular dysfunction (Guan R et al., 2011).

Laboratory abnormalities including elevated SGOT (202 U/L) and SGPT (65 U/L) indicate ongoing hepatocellular injury, while the AST:ALT ratio greater than 2:1 suggests advanced fibrosis (Saiman Y, 2024). The hypoalbuminemia (2.6 g/dL) reflects impaired hepatic synthetic function. This, irrespective of renal function status, is linked to a bad prognosis in cirrhotic patients, as well as higher short- and long-term mortality (Hung TH et al., 2023). Thrombocytopenia in this patient likely results from hypersplenism secondary to portal hypertension, a common complication that affects relates to a bad prognosis and affects 45-75% of cirrhotic patients (Zhuang YP et al., 2021). Hypercholesterolemia in this patient may seem paradoxical given advanced liver disease but can occur due to altered lipid metabolism and cholesterol synthesis pathways in cirrhosis (Mallick B et al., 2022). Serum bilirubin, creatinine, and INR are used to compute the Model for End-Stage Liver Disease (MELD) score, would provide valuable prognostic information, as scores ≥20 are associated with significantly higher short-term mortality in decompensated cirrhosis (Emenena I et al., 2023).

The chest X-ray finding of bronchopneumonia in this patient represents a serious complication that significantly impacts the prognosis. Pneumonia in cirrhotic patients carries substantial mortality risk, with studies reporting 18.7% and 31% as mortality rates in 30 day and 90 day (Xu L et al., 2018). The inflammatory response triggered by pneumonia can rapidly deteriorate liver function through excessive cytokine production and systemic inflammatory syndrome, creating a vicious cycle that compromises both pulmonary and hepatic function (Xu L et al., 2018). The ultrasonographic findings in this case demonstrate classic features of advanced cirrhosis with portal hypertension complications. The liver parenchyma showing rough, inhomogeneous structure with increased echogenicity and blunt angles represents the typical architectural distortion seen in established cirrhosis (Cheng JY et al., 2024). Splenomegaly measuring 12.43 cm indicates significant portal hypertension, as the spleen enlargement results from both passive congestion and tissue hyperplasia due to increased splenic blood flow (Bolognesi M et al., 2002; Yoshida H et al., 2023). Left pleural effusion, or hepatic hydrothorax, occurs in 5-16% of cirrhotic patients and represents direct movement of ascitic fluid through diaphragmatic defects into the pleural space (Wilkins H et al., 2024). The concurrent cholelithiasis (1.4 cm) is not surprising, as gallstone prevalence is significantly higher in cirrhotic patients compared to healthy individuals, with studies showing 4-5.5 times increased risk in HBV-related cirrhosis (Mallick B et al., 2022).

The comprehensive pharmacological approach outlined in this patient's treatment plan addresses multiple therapeutic targets critical for managing decompensated HBV cirrhosis and its complications. The management of massive ascites in this patient requires a comprehensive approach combining sodium restriction, diuretic therapy, and consideration for large-volume paracentesis. Spironolactone, an aldosterone antagonist, is preferred as initial therapy because the preferred diuretic for treating ascites is an aldosterone antagonist, which works better than a loop diuretic. If spironolactone alone fails to achieve adequate response, furosemide should be added at doses up to 160 mg daily, maintaining the traditional 100:40 mg ratio (spironolactone : furosemide) to preserve normokalemia (Shroff H, 2020; Moore KP et al., 2006). The combination approach may be preferred in patients with recurrent ascites like this case, as it provides more rapid natriuresis while maintaining electrolyte balance.

The combination antibiotic therapy of azithromycin 500 mg daily and ceftriaxone injection 2 grams daily appropriately targets the concurrent bronchopneumonia while addressing both typical and atypical respiratory pathogens (Kato H, 2024; Ito A et al., 2019). This combination is particularly relevant for cirrhotic patients who have compromised immune function and higher pneumonia-related mortality risk. Ceftriaxone monotherapy has demonstrated equivalent efficacy to broad-spectrum antibiotics in pneumonia treatment while being more cost-effective, but the addition of azithromycin provides enhanced coverage against atypical organisms including Mycoplasma and Legionella species (Kato H, 2024; Murter F et al., 2019). Research indicates that in individuals with severe community-acquired pneumonia who fulfill IDSA/ATS criteria, azithromycin combination therapy dramatically lowers 30-day mortality (OR 0.12, 95% CI 0.007-0.57) (Ito A et al., 2019).

Despite the advanced stage of liver disease, antiviral therapy remains crucial for this patient and should be initiated regardless of HBV DNA levels, ALT values, or HBeAg status. Nucleos(t)ide analogues, specifically entecavir or tenofovir, are recommended as first-line therapy for patients with decompensated HBV cirrhosis (Guan R et al., 2011; Cao L et al., 2025). Gastroprotection such as intravenous omeprazole at 40-80 mg daily or continuous infusion at 8 mg/hour for 5-7 days is recommended for cirrhotic

patients with active bleeding risk, as proton pump inhibitors have demonstrated a protective effect in preventing secondary bleeding after endoscopic interventions (Xie C et al., 2025; Lin L et al., 2021). Meta-analyses show that PPI administration can reduce post-procedural rebleeding rates by approximately 50% (pooled OR 0.52, 95% CI 0.35-0.77) in cirrhotic patients (Lin L et al., 2021).

Curcuma (turmeric) supplementation at 3 times daily provides valuable hepatoprotective benefits through multiple mechanisms including antioxidant, anti-inflammatory, and antifibrotic properties (Salama SM et al., 2013; Farzaei MH et al., 2018). UDCA significantly improves liver function parameters by modulating bile acid composition, reducing toxic hydrophobic bile acids (deoxycholic acid, lithocholic acid), and increasing the proportion of hydrophilic, less toxic bile acids (Kim DJ et al., 2018). Meta-analyses demonstrate that UDCA treatment can reduce serum ALT, AST, and alkaline phosphatase levels by approximately 15-30% in patients with various liver diseases (Simental-Mendia M et al., 2020). Aminofusin Hepar represents a specialized branched-chain amino acid (BCAA) formulation specifically designed for patients with hepatic insufficiency (Ferenci P et al., 1980). Studies demonstrate that Aminofusin Hepar can significantly improve serum albumin levels in cirrhotic patients, with mean increase from 2.39 g/dL to 2.70 g/dL following treatment (p=0.002), reflecting improved hepatic protein synthesis and nutritional status (Novianti A et al., 2019).

The large-volume paracentesis yielding 4,400 mL of serous fluid represents both a diagnostic and therapeutic intervention with significant clinical implications (Haghighat M et al., 2023). The serous nature of the fluid suggests transudative ascites with a serum-ascites albumin gradient (SAAG) ≥1.1 g/dL, consistent with portal hypertension-related ascites rather than infectious or malignant etiology.

The prognosis for this patient with decompensated HBV cirrhosis is guarded, with multiple factors contributing to his overall risk profile (Huang YJ et al., 2025; Guan R et al., 2011). Decompensated cirrhosis carries a median survival of only 2-4 years, which is worse than many oncological diseases (Carvalho JR et al., 2018). The presence of ascites as the initial decompensating event indicates transition from compensated to decompensated cirrhosis, with studies showing 1-year, 2-year, and 3-year survival rates of 81.2%, 75.6%, and 69.5%, respectively, showing in decompensated patients compared to 100%, 98.5%, and 98.5% in compensated cirrhosis (Huang YJ et al., 2025). While the concurrent pneumonia substantially elevates risk, as infectious complications increase mortality four-fold in cirrhotic patients (Xu L et al., 2018).

CONCLUSION

The progression from chronic hepatitis B to decompensated cirrhosis involves complex interactions causing considerable morbidity and mortality across diverse populations. The complexity of this case report necessitates a multidisciplinary approach involving hepatology, infectious disease, pulmonology, and nutrition specialists. Immediate priorities include aggressive treatment of bronchopneumonia with appropriate antibiotics, careful management of ascites and portal hypertension complications, and initiation of antiviral therapy.

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