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Case Report: Analysis of Drug Related Problems (DRP's) in Patients with Hepatic Cirrhosis PNSD Ctp B, Chronic Hepatitis B, Dyspepsia Syndrome, Hypoalbuminemia and Hepatic Function Disorders

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Abstract

Background: Liver cirrhosis is a chronic liver disease characterized by fibrosis and replacement of normal liver tissue with abnormal nodules. The main etiologies include hepatitis B virus (HBV) and hepatitis C virus (HCV) infections, alcohol-related liver disease, and non-alcoholic fatty liver disease (NAFLD). The prevalence of liver cirrhosis based on autopsy findings is around 2.4% in Western countries, while in the United States it is estimated at 360 per 100,000 population, causing approximately 35,000 deaths annually. **Objective:** This case report aims to describe the treatment and identify drug-related problems (DRPs) in a patient diagnosed with PNSD CTP B liver cirrhosis, chronic hepatitis B, dyspepsia syndrome, hypoalbuminemia, and impaired liver function. **Method:** This study employed a case report design with primary data obtained from patient interviews and retrospective documentation review. Data were analyzed using the SOAP method to identify DRPs. **Results:** The analysis revealed minor drug interactions, including spironolactone–furosemide, tenofovir disoproxil fumarate (TDF)–morphine sulfate tablet (MST), and cefixime–furosemide. **Conclusion:** Patients with complex comorbidities are at increased risk of DRPs. Continuous pharmacotherapy monitoring is essential to minimize toxicity and adverse effects.

Keywords: *Cirrhosis, Chronic Hepatitis B, Dyspepsia Syndrome, TDF, Furosemide.*

Introduction

The word cirrhosis comes from the Greek word kirrhos, meaning "orange or yellowish-brown," and osis, meaning "condition." The World Health Organization (WHO) defines cirrhosis as a diffuse process characterized by fibrosis and changes in the normal architecture of the liver into structurally abnormal nodules, resulting in the loss of the normal lobular shape[1].

Hepatic cirrhosis is a pathological condition that represents the end stage of progressive hepatic fibrosis, marked by distortion of the liver architecture and the formation of degenerative nodules. More than 40% of patients with hepatic cirrhosis are asymptomatic and are often diagnosed during routine health examinations or autopsies[2].

There are many factors that have been proven to cause Hepatic Cirrhosis, including infections, toxins, hereditary conditions, and even autoimmune processes. Regardless of the cause, with each injury sustained, the liver forms scar tissue (a process called fibrosis), which initially does not eliminate the liver's normal function. However, when the injury occurs repeatedly, it leads to the expansion of fibrosis, resulting in the loss of normal liver function and the development of liver cirrhosis[3].

Liver fibrosis is the accumulation of scar tissue from extracellular matrix (ECM) proteins

following acute or chronic liver injury. The synthesis of ECM proteins is triggered by increased activity of Hepatic Stellate Cells (HSCs) as the main cells, Kupffer cells, pro-inflammatory cytokines, growth factors and their inhibitors, as well as various types of collagen. Determining the degree of fibrosis is very helpful for clinicians to understand the progression of the disease and to provide early and accurate treatment[4].

Epidemiological studies in developed countries show that liver cirrhosis is the third leading cause of death in patients aged 45–46 years (after cardiovascular disease and cancer). The incidence of liver cirrhosis based on autopsy findings is around 2.4% in Western countries, while in the United States it is estimated at 360 per 100,000 population and causes approximately 35,000 deaths per year[5]. According to reports from government general hospitals in Indonesia, the average prevalence of liver cirrhosis is 3.5% of all patients treated in internal medicine wards[6].

Liver cirrhosis is among the 14 leading causes of death worldwide, accounting for 1.3% of all global deaths, and is ranked among the top five causes of death in Indonesia[7]. In 2008, deaths caused by liver cirrhosis in the South-East Asia Region B (Indonesia, Sri Lanka, Thailand) totaled 51,715 cases, with 38,187 cases in men and 13,528 cases in women[7].

Several factors cause liver cirrhosis in Indonesia, primarily due to hepatitis B and C virus infections. Research in Indonesia indicates that hepatitis B virus accounts for 40%–50% of cirrhosis cases, hepatitis C virus for 30%–40%, while 10%–20% of cases have an unknown cause. Alcohol as a cause of liver cirrhosis in Indonesia is likely very rare, as there are no definitive research data available. The main etiologies of cirrhosis are hepatitis B virus (HBV) and hepatitis C virus (HCV) infections, alcohol-related liver disease, and non-alcoholic fatty liver disease (NAFLD)[8]. The Child-Turcotte-Pugh (CTP) score is used to assess the severity (Child A, Child B, Child C) of liver cirrhosis. This system also serves as a prognostic tool and is more commonly used in patients undergoing liver transplantation[9]. The Child-Turcotte-Pugh score, a modification of the original Child score, can assess the general condition of patients with liver cirrhosis and evaluate multiorgan changes caused by the disease[10].

Hepatitis is a public health problem in developing countries worldwide, including Indonesia. Hepatitis B is caused by the hepatitis B virus, a member of the *Hepadnaviridae* family, which can cause acute or chronic liver inflammation and may progress to liver cirrhosis or liver cancer. Infection with the hepatitis B virus has increasingly become a major and serious public health concern. In addition to its manifestation as acute HBV disease with its complications, hepatitis B can also occur in the form of chronic HBsAg carriage, which serves as a source of transmission to the community. Systemic hepatitis B virus infection can cause inflammation and necrosis of liver cells, leading to a series of clinical, biochemical, immunoserological, and morphological abnormalities[11].

Hepatitis is an inflammatory disease of the liver. In most cases, hepatitis B is caused by the hepatitis B virus (HBV). Hepatitis B infection is divided into two phases: acute infection and chronic infection. Acute hepatitis B is a short-term infection that occurs within the first six months after the incubation period. Prolonged hepatitis B infection is referred to as chronic hepatitis B infection[12]. According to the WHO, in 2019 an estimated 296 million people were living with chronic hepatitis B worldwide, particularly in low- and middle-income countries. In the same year, it was estimated that there were around 820,000 deaths, most of which were caused by cirrhosis and liver cancer[13]. This indicates that individuals with

chronic hepatitis B are at a higher risk of developing liver cirrhosis. Liver cirrhosis is damage to the liver characterized by scar tissue formation, which obstructs the normal flow of blood through the liver[14].

The progression of liver cirrhosis is slow, asymptomatic, and often undetected until complications of other liver diseases arise. Clinically, liver cirrhosis is classified into compensated cirrhosis, in which there are no apparent clinical symptoms, and decompensated cirrhosis, in which clear clinical symptoms are present. Most patients who come to the clinic are already in the decompensated stage with various complications[15]. The diagnosis of liver cirrhosis is established based on medical history, physical examination, and laboratory tests. The main and advanced clinical symptoms of liver cirrhosis occur due to two types of physiological disorders: liver cell failure and portal hypertension. Manifestations of liver cell failure include jaundice, endocrine disorders, hematologic abnormalities, peripheral edema, fetor hepaticus, and hepatic encephalopathy. Manifestations related to portal hypertension include splenomegaly, esophageal and gastric varices, as well as other signs of collateral circulation[16].

Laboratory examinations in liver cirrhosis often reveal a decreased platelet count. This reduction occurs due to chronic infection, which suppresses bone marrow activity, leading to lower platelet production. Patients with liver cirrhosis may experience thrombocytopenia caused by increased sequestration of platelets in the spleen due to splenomegaly, or by reduced production of thrombopoietin in the liver as a result of hepatic damage.

The prognosis of patients with liver cirrhosis can also be estimated using the Child-Pugh Score, in which the two-year survival rates for Child-Pugh classes A, B, and C are 85%, 57%, and 35%, respectively. To prevent disease progression and reduce poor prognosis, appropriate medical management is essential[17,18,19]. This case is relatively common among inpatients at Achmad Mochtar Hospital, with most cases already in advanced stages. Therefore, there was an interest in reporting a case of liver cirrhosis in a 54-year-old male patient who was hospitalized in the Ambun Suri ward at Achmad Mochtar Hospital.

The purpose of this case report is to examine the treatment provided to a patient diagnosed with liver cirrhosis PNSD CTP B, chronic hepatitis B, dyspepsia syndrome, hypoalbuminemia, and impaired liver function, as well as to identify the drug-related problems (DRPs) experienced by the patient at Dr. Achmad Mochtar Hospital, Bukittinggi.

Materials and Methods

Study Design

This study employed a case report design with primary data analysis obtained from interviews and retrospective documentation collected from the patient's medical records. The data were analyzed using the SOAP method to identify Drug Related Problems (DRPs).

Sample

The subject of this study was a 54-year-old male patient who was admitted to Dr. Achmad Mochtar Hospital, Bukittinggi, on June 30, 2024, with the chief complaint of abdominal discomfort for the past 2 days. On physical examination, his blood pressure was 116/72 mmHg, pulse 80 beats/minute, with abdominal pain localized to the right upper quadrant. The patient reported abdominal discomfort for approximately 2 months prior to admission, with symptoms of abdominal fullness after meals, jaundice for about 1 month, fatigue, and dark-colored urine resembling tea. He had no history of other chronic diseases but reported a history of alcohol consumption. Further physical examination revealed a compos mentis level of consciousness, blood pressure 116/72

mmHg, pulse 80 beats/minute, respiration rate 20 breaths/minute, and temperature 36.7°C. Other physical findings were within normal limits. Laboratory tests showed: MCH 34.5 pg, eosinophils 0.7%, neutrophils 80.2%, lymphocytes 11.3%, albumin 2.9 g/dL, SGPT 126 U/L, SGOT 624 U/L, and total bilirubin 4.95 mg/dL.

Data Collection Technique

Data for this study were collected through primary data from interviews and retrospective documentation from the patient's medical records.

Data Analysis Technique

Data were analyzed using the SOAP method (Subjective, Objective, Assessment, Plan) to identify and evaluate Drug Related Problems (DRPs).

Ethical Consideration

This study adhered to ethical principles, including respect for patient autonomy, confidentiality, and beneficence. Patient consent was obtained prior to data collection, and identifying information was anonymized to ensure confidentiality. Approval for the study was obtained from the Ethics Committee of Dr. Achmad Mochtar Hospital, Bukittinggi.

Results

A 54-year-old male patient presented to Dr. Achmad Mochtar Hospital, Bukittinggi, on June 30, 2024, with the chief complaint of abdominal tightness for the past two days. On examination, the patient's vital signs including respiration, pulse rate, and temperature were within normal ranges. Blood pressure readings fluctuated but remained within normal limits. The patient's condition included abdominal distension due to the presence of ascites.

Laboratory test results showed WBC: 8.35 × 10^3/µL, RBC: 3.97 × 10^6/µL, Hb: 13.7 g/dL, HCT: 38.2%, MCV: 96.2 fL, MCH: 34.5 pg, and MCHC: 35.9 g/dL. Liver function tests revealed elevated SGOT at 624 U/L (normal 0–40 U/L) and SGPT at 126 U/L (normal 0–41 U/L), with decreased albumin at 2.9 g/dL (normal 3.5–5.2 g/dL). HBsAg testing returned reactive. The patient was diagnosed with liver cirrhosis PNSD CTP B, chronic hepatitis B, dyspepsia syndrome, hypoalbuminemia, and impaired liver function.

Management for this patient included Aminofusin Hepar infusion: NaCl 1:1, lansoprazole injection every 24 hours, spironolactone 2 × 100 mg, curcuma 3 × 1 tablet, vitamin B complex 2 × 1, UDCA 3 × 250 mg, SNMC injection, ketorolac injection 30 mg every 8 hours, ceftriaxone injection every 24 hours, sucralfate syrup 3 × CI, lactulax syrup 2 × 15 cc, pantoprazole, inbumin 3 × 1 tablet, furosemide 1 × 40 mg, Lasix injection 1 × 1 ampoule, TDF 1 × 300 mg, cefixime 2 × 200 mg, MST 1 × 15 mg, Urinter 2 × 400 mg, vitamin K injection 3 × 2 mg/mL, and albumin transfusion 20% 1 vial/day until albumin > 3 g/dL.

Discussion

Mr. Z, a 54-year-old male, was admitted to Dr. Achmad Mochtar Hospital, Bukittinggi, and treated in the internal medicine ward with complaints of upper right abdominal pain and a feeling of tightness for approximately two months prior to admission. He reported abdominal bloating and fullness after eating or drinking, yellowing of the eyes for about one month, and general fatigue. The patient admitted to a history of alcohol consumption. The primary diagnosis was liver cirrhosis PNSD CTP B, chronic hepatitis B, dyspepsia syndrome, hypoalbuminemia, and impaired liver function. On admission, the patient's vital signs were: temperature 36.7°C, pulse 80 beats/min, respiratory rate 20 breaths/min, blood pressure 116/72 mmHg, and SpO_2 99%. Impaired liver function can be one of the major risk factors for the development of liver cirrhosis.

Liver cirrhosis has various causes; however, the majority of patients initially suffer from chronic liver disease due to viral hepatitis infection or alcohol consumption. Other etiologies of chronic liver disease include hemochromatosis, alpha-1 antitrypsin deficiency, Wilson's disease, biliary cirrhosis, cardiac cirrhosis, and other chronic inflammatory conditions[20].

In this case, the likely cause of cirrhosis was the progression of chronic liver disease resulting from chronic alcoholism. The patient admitted to a habit of consuming alcohol from a young age, nearly every night, at a quantity of 1–2 bottles. Excessive alcohol consumption has an undesirable synergistic effect on several forms of chronic liver disease, particularly chronic viral hepatitis. The mechanisms of this interaction include faster fibrosis progression, increased risk of hepatocellular carcinoma, and higher mortality rates[21].

An international study by UNSW Sydney in 2017 found that among 31,924 people with HCV in Scotland, 1,375 (4.3%) patients presented to the hospital typically in the decompensated stage, often accompanied by complications such as variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome, or hepatic encephalopathy[22].

The clinical manifestations of liver cirrhosis include fatigue, loss of appetite, weight loss, muscle atrophy, jaundice, spider angiomata, splenomegaly, ascites, caput medusae, palmar erythema, white nails, gynecomastia, and loss of pubic and axillary hair in women[23].

Liver cirrhosis has various causes; however, the majority of cirrhosis patients initially suffer from chronic liver disease caused by hepatitis virus infection or are associated with habitual alcohol consumption. Other etiologies of chronic liver disease include hemochromatosis, alpha-1 antitrypsin deficiency, Wilson's disease, biliary cirrhosis, cardiac cirrhosis, and chronic inflammatory conditions[20]. In this case, the likely cause of cirrhosis is the progression of chronic liver disease resulting from chronic alcoholism. The patient admitted to having a habit of consuming alcohol since a young age, almost every night, drinking 1–2 bottles. Excessive alcohol consumption has undesirable synergistic effects on several forms of chronic liver disease, especially chronic viral hepatitis. The mechanism of this interaction includes accelerated fibrosis progression, increased risk of developing hepatocellular carcinoma, and higher mortality rates[21]. Based on the results of an international study by UNSW Sydney in 2017, it was shown that among 31,924 people with HCV in Scotland, 1,375 (4.3%) patients who came to the hospital were usually already in the decompensated stage, accompanied by complications such as variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome, or hepatic encephalopathy[22]. The clinical features of patients with liver cirrhosis include fatigue, loss of appetite, weight loss, muscle atrophy, jaundice, spider angiomas, splenomegaly, ascites, caput medusae, palmar erythema, white nails, gynecomastia, and even loss of pubic and axillary hair in women[23].

Liver function tests showed elevated SGOT and SGPT levels, along with decreased albumin. This condition of hypoalbuminemia is also one of the causes of ascites, which results in fluid accumulation in the peritoneum. Hypoalbuminemia, or the decrease in albumin, is caused by reduced synthesis in the liver parenchyma, in accordance with the degree of cirrhosis progression. Under normal conditions, the liver synthesizes plasma proteins, one of which is albumin, at a rate of 12–25 grams per day. However, due to the fibrosis and necrosis processes in cirrhosis, the synthesis rate of albumin is impaired, resulting in decreased serum albumin levels, a condition known as hypoalbuminemia.

The SGOT and SGPT enzymes function as fat metabolizers. However, if excessive fat

enters the body, the liver is unable to process it, leading to liver cell damage. The decline in liver function may be due to alcohol consumption since the age of 20. SNMC (Stronger Neo-Minophagen C) is a drug used to improve abnormal liver function in chronic liver disease. In addition, the patient was also given UDCA for primary biliary cirrhosis, according to *The Monthly Index of Medical Specialities*[24]. The dosage is 13–15 mg/kgBW/day given as a single dose or divided into 2–4 doses. Lactulose is intended to reduce ammonia production by bacteria in the intestines. If not administered, the drug could lead to the progression of hepatic encephalopathy. Its mechanism of action involves enhancing ammonia elimination, acidifying the intestine through the production of lactic acid and acetic acid, thereby significantly lowering the pH in the colon and reducing the absorption of ammonium ions and other toxic nitrogen compounds. This results in decreased blood ammonia concentration and improved mental function.

The liver cirrhosis was likely caused by a history of alcohol consumption and hepatitis B virus infection. To manage hepatitis B, the patient was given TDF (Tenofovir Disoproxil Fumarate) at a dose of 300 mg orally per day. This drug works by interfering with HIV DNA synthesis through competitive inhibition of reverse transcriptase and incorporation into viral DNA. It also inhibits hepatitis B virus polymerase, resulting in the suppression of viral replication[25].

The patient was given IVFD NaCl (1 bottle/12 hours) as therapy to balance electrolyte levels. The patient was also administered aminofusin hepar, indicated for patients with severe chronic liver insufficiency, decompensated liver cirrhosis, liver insufficiency caused by other factors, as well as serving as a hepatoprotector and enhancing cell regression. The patient received curcuma, vitamin B complex, and inbumin as supportive supplements to maintain liver function and prevent extensive damage to the liver. Meanwhile, Lasix (furosemide) and spironolactone were used as diuretics to reduce edema and ascites caused by fluid accumulation, as well as excess urea, sodium, and calcium. Spironolactone is a weak diuretic and is primarily used in combination with other diuretics to prevent hypokalemia.

Mr. Z was given intravenous lansoprazole injection indicated for dyspepsia syndrome. Dyspepsia syndrome is a collection of symptoms described as abdominal discomfort, such as a feeling of fullness, bloating, stomach pain, and epigastric pain. However, it should be emphasized that dyspepsia is not a disease but rather a symptom of an underlying illness or digestive disorder. On July 3, the lansoprazole injection was replaced with pantoprazole to reduce the patient's treatment costs. The administration of gastric mucosal protective drugs aims to prevent bleeding caused by portal hypertensive gastropathy erosion.

The patient received pantoprazole injection 1 ampoule/24 hours for six days. A double-blind study showed that intravenous pantoprazole helped relieve and stop dyspepsia symptoms within 30 to 60 minutes in the emergency unit. The use of the proton pump inhibitor (PPI) group is assessed based on its effectiveness, namely the recovery from vomiting, nausea, and relief from abdominal pain. A study by Khatir et al. reported that a comparison between the pantoprazole and intravenous ranitidine groups after treatment showed a significant difference in pain severity between the two treatment groups ($p < 0.001$). Pantoprazole was effective in improving initial epigastric pain. PPIs are effective against epigastric pain syndrome (EPS) and in evaluating symptom reduction in patients with functional dyspepsia[26].

On July 10, 2024, the patient complained of painful and scant urination. For this, the patient was given Urinter and cefixime as an

antibiotic for UTI. MST, which belongs to the opioid analgesic group, was administered to reduce the patient's pain. The patient also complained of nosebleeds, for which vitamin K injection was given to help accelerate blood clotting, ensure the body does not bruise easily, and promote faster healing in case of bruising.

On the 10th and 11th, the patient's vital signs showed a respiratory rate of 22 breaths/minute. The monitoring results categorized this as tachypnea, meaning the patient was breathing very rapidly (respiratory rate >20 breaths/minute). The patient's ineffective breathing pattern was caused by pressure on the diaphragm, leading to reduced lung expansion. This condition was due to fluid accumulation in the cavity between the membrane lining the abdominal wall and the internal organs. In addition, ascites, or fluid buildup, can cause nausea, bloating, and abdominal pain. The patient experienced bloating, nausea, weakness, and abdominal pain described as stabbing[27].

An interaction occurs between spironolactone and furosemide, where spironolactone increases and furosemide decreases serum potassium levels. The effect of this interaction is unclear; the drugs can still be used cautiously with proper monitoring. Another interaction is between TDF and MST, where Tenofovir DF increases morphine levels by reducing renal clearance. Monitoring of use is required due to the potential risk of increased toxicity. In addition, there is an interaction between cefixime and furosemide, in which cefixime increases furosemide toxicity through pharmacodynamic synergy. This is considered minor/with unknown significance but may increase the risk of nephrotoxicity. To minimize the occurrence of interactions, the drugs can be administered with an interval of approximately 30 minutes.

Overall, the therapy provided to the patient was appropriate and in accordance with the patient's condition. The choice of therapy, therapeutic indications, and prescribed dosage regimen were correct. However, monitoring of SGOT/SGPT levels, bilirubin, and abdominal enlargement due to fluid accumulation is necessary for the patient's recovery. In addition, education for the patient's family regarding the prescribed therapy and adherence to medication use is important to improve the success of the patient's treatment.

Conclusion

A patient diagnosed with Liver Cirrhosis PNSD CTP B with chronic Hepatitis B, Dyspepsia Syndrome, Hypoalbuminemia, and impaired liver function experienced DRPs in the therapy received. The DRPs in the patient's therapy involved drug interactions, although only minor ones, thus requiring monitoring to prevent toxicity and unwanted effects.

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