Asian Australasian Neuro and Health Science Journal (AANHS J) Vol. 4, No. 2, 2022| 39 – 46

AANHS Journal
Journal of Medical Science

Liver Cirrhosis with Its Complications: Case Report In A Young Female Patient

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Abstract

Introduction: Liver cirrhosis is a chronic disease characterized by the presence of fibrosis and regeneration of nodules in the liver, the consequence of which is the development of portal hypertension and liver failure. Usually associated with infectious infectious diseases such as viral hepatitis, alcohol consumption, metabolic syndrome, autoimmune processes, storage diseases, toxic substances and drugs. Major complications include gastrointestinal variceal bleeding, ascites, spontaneous bacterial peritonitis infection, hepatorenal syndrome, hepatic encephalopathy, and hepatocellular carcinoma.

Case Report: A 23-year-old woman comes to the ER, dr. Soegiri Lamongan with complaints of vomiting blood. The patient also complained of black bloody stools. Referred patient from Intan Medika Hospital with the initial complaint of vomiting blood more than 5 times (± equivalent to one medium drinking bottle) four days ago. On examination also found anemic conjunctiva and found splenomegaly. On abdominal ultrasound examination, the liver was shrunken, the edges were obtuse angles, partially irregular, parenchymal echo intensity was heterogeneous, v.hepatica was normal, liver fibrosis staging was 11.81 kPa, no masses or nodules were seen. The conclusion on endoscopic examination was the presence of grade III esophageal varices, congestive gastropathy, and erosive gastritis.

Conclusion: Liver cirrhosis is a disease that has various etiologies. Early diagnosis and appropriate treatment in cirrhotic patients with complications is one way to improve patient survival

Keyword: Liver Cirrhosis, Esophageal varices, Complication, Anemia, Erosive Gastropathy

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Introduction

Liver cirrhosis is a chronic disease characterized by fibrosis and regeneration of nodules in the liver with the consequence of which is the development of portal hypertension and liver failure. [1]. Cirrhosis arises from various chronic diseases, which progress slowly over years or decades. It may arise as a consequence of toxic, infectious, autoimmune, exogenous vascular processes, by deposition or inborn errors of metabolism. Patients are diagnosed primarily when they are identified with changes in the anatomy and function of the liver through clinical examination, biochemical tests, imaging and/or histological findings. [1]

Liver cirrhosis is a public health problem. Usually associated with infectious infectious diseases such as viral hepatitis, alcohol consumption, metabolic syndrome, autoimmune processes, storage diseases, toxic substances and drugs. It is known that up to 40% of patients remain asymptomatic for a long time ; however, once complications develop, progressive deterioration occurs which results in death if the patient undergoes definitive treatment i.e. liver transplant. Many patients die of this disease in their fifth or sixth decade of life. [2]

The main etiology of cirrhosis in most developed countries is hepatitis C virus infection, alcohol abuse, and nonalcoholic steatohepatitis. Other causes include autoimmune hepatitis, Hepatitis B, primary sclerosing cholangitis, primary biliary cirrhosis, medications. Once complications of cirrhosis develop, the 5-year survival rate declines to less than 20%. Major complications include gastrointestinal variceal bleeding, ascites, spontaneous bacterial peritonitis infection, hepatorenal syndrome, hepatic encephalopathy, and hepatocellular carcinoma. Ascites is one of the most common complications of cirrhosis and is associated with an annual mortality rate of 20%. Infection causes 30% of deaths in one month and another 30% in one year. The most common diagnoses are spontaneous bacterial peritonitis, urinary tract infections, pneumonia and skin infections. Esophageal varices occur in 30 to 70% of patients with cirrhosis, with a bleeding risk of about 12% per year. When bleeding occurs, it can be fatal with a rate of mortality of at least 20% at 6 weeks 8. The development of hepatic encephalopathy is an ominous sign of cirrhosis because the mortality rate associated with it is up to 64% in a year. Another complication is hepatocellular carcinoma, which is diagnosed in more than 70% of cases as an unresectable tumor. The annual survival rate without treatment is 29%.[3], [4]

In this case, we will discuss a case report of cirrhosis with its complications in a 23-year-old woman.

Case Report

A 23-year-old woman comes to the ER, dr. Soegiri Lamongan with complaints of vomiting blood. The patient also complained of black bloody stools. Referred patient from Intan Medika

Hospital with the initial complaint of vomiting blood more than 5 times (± equivalent to one medium drinking bottle) four days ago. The patient had black stools two days ago and colored stools as since six years ago. The patient also complained of pain in the left upper abdomen, intermittent since five years ago. The patient had difficulty sleeping and has been diagnosed with hepatitis B six years ago. It is known that the patient's grandmother also has hepatitis B. The patient has never drank alcohol, but the patient has smoked for four years, sometimes drinks coffee, and sleeps in the morning.

On physical examination, the GCS value was 456 with compos mentis consciousness, the patient's blood pressure was 102/55mmHg; 36oC temperature; Peripheral oxygen saturation (SpO2) 95%, respiratory rate 20 x/minute and pulse 84 x/minute. On examination also found anemic conjunctiva and found splenomegaly. Laboratory tests are shown in table 1.

Table 1. Laboratory examination results

Complete Blood Count	**Value**
Hemoglobin	6.00 g/dl
Leucocytes	1.240 x 10^3/µL
Erythrocyte sedimentation rate	35 – 50 Detik
Pack cell volume	18,9 %
Thrombocytes	470 X 10^3/µL
Neutrophil Absolut	600
Lymphocyte Absolut	420
SGOT	17 u/L
SGPT	14 u/L
Blood Glucose	117 mg/dL
Hbs-Ag	Positif

On abdominal ultrasound examination, the liver was shrunken, the edges were obtuse angles, partially irregular, parenchymal echo intensity was heterogeneous, hepatic vein was normal, liver fibrosis staging was 11.81 kPa, no masses or nodules were seen. The spleen looks enlarged, the parenchymal echo intensity is normal, the sinus cortex boundaries are clear, there are no stones or cysts visible. Gallbladder, pancreas, kidneys, bladder, and prostate are within normal limits. Conclusion Ultrasound showed hepatic cirrhosis with portal hypertension and splenomegaly.

On endoscopic examination of the patient (Figure 1), the esophagus was found to have a positive red color sign with grade III esophageal varices. In the gastric organs found congestive gastropathy. The gastric antrum found hyperemic mucosa, no ulcers or tumors. There were no abnormalities in the body, angle, pylorus and cardia. There were no abnormalities in the

duodenum. The conclusion on endoscopic examination was the presence of grade III esophageal varices, congestive gastropathy, and erosive gastritis.

One day after being in the ER, the patient was transferred to the inpatient room. The patient's complaints are still persistent but the patient's appetite increases while in the inpatient room. The patient received Asering 1500cc/24 hours infusion, injection of ceftriaxone (1g) single dose, vitamin K (10mg) 3x1, ranexamic acid (Kalnex) 3x1mg, ondasentron (4mg) 3x1, and, omeprazole (20mg) 2x1. The patient was scheduled for blood transfusion with PRC and gastric lavage was performed.

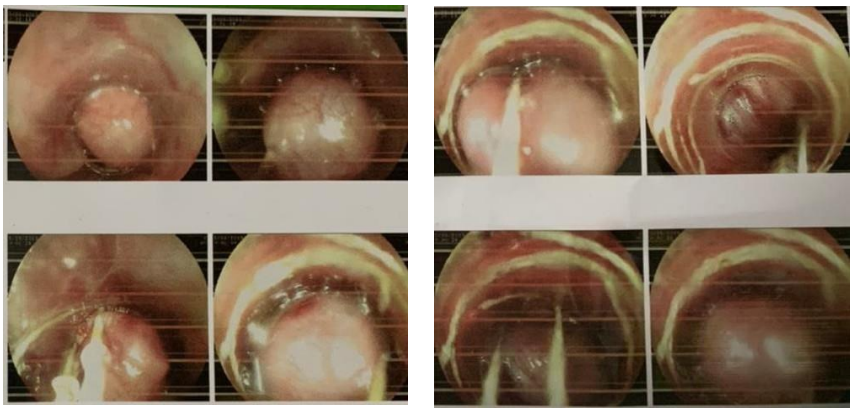

Figure 1. Endoscopy results. There is a picture of grade III esophageal varices, erosive gastritis and congestive gastropathy

Discussion

Liver cirrhosis has several etiologies which ultimately lead to the same process, namely liver fibrosis which causes impaired function. According to the stimulus, the breakdown can be faster or slower; for example alcohol and viral hepatitis produce early damage. Progressive degeneration of hepatocytes occurs and causes necrosis, and extracellular fibrotic scar material is formed to replace the parenchyma with regenerating nodules.[1]

The process of fibrosis is dynamic, and the early stages can be reversible, but despite new research, it is not yet possible to conclude at what point this process becomes irreversible or create drugs that stop this progression. It is considered that the main mechanism for the initiation of fibrosis is hepatic stellate cell activation; This happens in two phases. The first is called initiation or pre-inflammation produced by the body formed from cellular apoptosis, oxidative stress, stimulation of Kupffer cells, hepatocytes, platelets, and endothelium. Then the perpetuation phase begins where there is cell proliferation and fibrogenesis with an important inflammatory response.[5]

Hepatitis B virus (HBV) is an infectious disease that causes conditions ranging from anicteric hepatitis to life-threatening conditions such as fulminant hepatitis, or a chronic condition characterized by persistent inflammation that can progress to cirrhosis or

hepatocellular carcinoma. Worldwide, approx. 250 million people are chronically infected with HBV. Among the regions with the highest prevalence of HBV, with about 10%, are China, Brazil, Southeast Asia, and Africa. According to WHO data in 2018, four million new cases of acute HBV hepatitis are detected annually, and about 1 million people die of liver cirrhosis due to this infection.[6], [7]

In countries with a high prevalence between 70 and 90% of the population contracted the virus at an early age, under 40 years of age. If they are infants or children under 5 years, 90% from of them will present with chronic infection. In contrast, in adults, only 5% will persist chronically with infection. Of patients with chronic hepatitis B, 15 to 30% will develop cirrhosis of the liver, and 6% will develop hepatocellular carcinoma by 5 years. HBV is transmitted through direct contact with blood or body fluids such as saliva, semen and vaginal fluids of an infected person. This can occur through the use of non-sterile materials (needles, syringes, surgical instruments), sexual or perinatal or vertical. Hepatitis B vaccination is available and safe and prevents chronic hepatitis B. HBV infection in the acute phase does not require treatment. Treatment during the acute phase is only supportive and in cases progressing to the chronic phase, treatment with antivirals has been shown to improve survival to prevent disease progression, risk of decompensation and progression of hepatocellular carcinoma. [8]

Most of the patients with liver cirrhosis are usually asymptomatic or have nonspecific symptoms such as asthenia, weight loss, decreased lividness, among others, which delay the diagnosis. When the patient reaches the decompensated phase, symptoms vary from abdominal distension due to ascites and hepatomegaly, hematemesis and melena due to gastrointestinal bleeding, altered mental status in hepatic encephalopathy, hypoxaemia in cases of hydrothorax or hepatopulmonary syndrome, and/or jaundice. signs and symptoms that make you suspect liver disease. Cirrhosis in a patient with grade III esophageal varices and erosive gastritis. Patients with cirrhosis of the liver with esophageal varices have a one year survival rate of 50% and can cause death. Patients also complain of black stools. Black stools are usually caused by upper gastrointestinal bleeding, in this case caused by esophageal varices.[9]

It is important to investigate the personal history to look for pathologies that predispose to cirrhosis such as the presence of metabolic syndrome and autoimmune disorders. You should examine risk factors through a detailed history with a focus on personal and social history of alcohol consumption. Factors associated with hepatitis B and C infection (use of injection drugs, unprotected sex, tattoos, blood transfusions, vaccinations), use of hepatotoxic and/or herbal drugs, family history of genetic predisposing diseases such as hemochromatosis, Wilson's disease and alpha antitrypsin deficiency -1 should also be considered in the history. The patient was diagnosed with hepatitis B six years ago with the possibility of being diagnosed late because hepatitis B infection is usually asymptomatic. [9]

On physical examination, various manifestations can be found, the presence of which should be suspected as disease: asterixis, ascites, collateral circulation of the abdomen "jellyhead", spider veins and telangiectasia, palmar erythema, nail changes (striped and reddish nails). distal third of the nail), Dupuytren's contracture, gynecomastia, hepatomegaly, splenomegaly, bruising, testicular atrophy, and jaundice. [10]

In our case only splenomegaly was found. Histologically, chronic portal hypertension-induced splenomegaly has an extensive white pulp and marginal zone area and is distinct from congestive splenomegaly, in which the red pulp is more pronounced and prominent. Clinically, splenomegaly has been associated with a poor prognosis in liver cirrhosis and is used during radioactive or acoustic examinations as an index for non-invasive assessment of esophageal varices and bleeding risk. Spleen Stiffness may also increase as splenomegaly develops. Portal system congestion is widely considered to be an early cause of splenomegaly during cirrhosis of the liver.[11]

Changes in serum glutamic oxaloacetic transaminase (SGOT) and glutamic-pyruvic transaminase (SGPT) indicate hepatocellular damage and in cirrhosis are usually high, although they can also be found in normal values. An SGOT/SGPT ratio greater than 1 is a strong predictor of cirrhosis except in alcoholic liver disease, and in advanced stages, the relationship may be reversed. In this case, the patient's SGOT and SGPT values were within normal limits but the SGOT/SGPT ratio was more than >1 indicates liver cirrhosis. [11]

Bilirubin values are usually normal in the compensated state but as the disease progresses they gradually increase, so it is considered an important parameter for assessing liver function in Child-Pugh. Albumin is produced exclusively by the liver; therefore, it is a marker indicating dysfunction of hepatic synthesis. The liver synthesizes several coagulation factors that interfere with the extrinsic pathway of coagulation. When prothrombin time is prolonged, this reflects changes in hepatic synthesis. Hyponatremia is a common finding in cirrhotic patients with ascites, with sodium and water retention by the kidneys, and is considered a poor prognostic finding.[11]

Anemia is caused by several factors such as folic acid deficiency, alcohol poisoning, chronic blood loss and/or hypersplenism. Thrombocytopenia with a count of less than 150,000 platelets per mm^3 is the result of platelet destruction due to portal hypertension with hypersplenism and is a sensitive and specific finding for the diagnosis of portal hypertension.[12]

Leukopenia is caused by portal hypertension with hypersplenism. It is important to remember that the value of the hepatogram can also be changed in pathologies other than the liver. For example, bilirubin in hemolysis, transaminases in thyroid, muscle and heart disease,

alkaline phosphatase in bone pathology. In our case, the liver was enlarged to Schuffner 3. The liver enlargement in our case was caused by portal hypertension in the patient. Anemia was also found in this case which was probably exacerbated by splenomegaly and upper GI tract bleeding. [12]

Ultrasound is the diagnostic method of choice because of its low cost, non-invasiveness and easy access. It has a sensitivity of 91.1% and a specificity of 93.5% for the diagnosis of liver cirrhosis. This allows evaluating the macroscopic appearance of the liver, portal vein and hepatic vein blood flow. Ultrasound can even detect ascites and can help find signs of portal hypertension. Findings of nodularity, caudate lobe hypertrophy, increased echogenicity and parenchymal atrophy are sonographic signs of cirrhosis. In our case, the liver was shrunken, obtuse angle of the margins Partly irregular, parenchymal echo intensity was heterogeneous, normal hepatic vein, liver fibrosis staging 11.81 kPa. The score of 11.81 kPa from ultrasound examination was classified as liver stiffness F3, which means the liver chirrosis had moderate severity. [13]

Patients with esophageal variceal bleeding require escalation of care to the intensive care unit for closer hemodynamic monitoring and consideration of intubation for airway protection. Initial steps in management include establishing large-bore intravenous access, fluid resuscitation with crystalloid solutions and checking blood groups for transfusion to achieve a hemoglobin level of 8 g/dL. Higher hemoglobin levels are counterproductive because excessive transfusion can worsen portal hypertension and outcome. Patients with severe coagulopathy and thrombocytopenia may benefit from platelet transfusions. Vasoactive therapy with octreotide, a splanchnic vasoconstrictor, should be initiated prior to endoscopic evaluation to reduce the incidence of active bleeding during endoscopy. Splanchnic vasoconstrictors should be continued for 5 days to reduce the risk of rebleeding. The ultimate goal is to control bleeding with endoscopic band ligation.[14]

Conclusion

It is very important to carry out a complete prospective study of the etiology of liver cirrhosis and its clinical history, because treatment and/or cessation of stimulation can slow the progression of the disease and keep it at the compensatory stage for a longer time increasing its survival. Multidisciplinary management both in primary care and for diagnostic evaluation from an early stage and close monitoring of pathology.

Progression of fibrosis to cirrhosis can lead to worsening of portal hypertension and complications including variceal bleeding. Given the high morbidity and mortality of this complication, it is important to identify and refer these patients early for prompt and appropriate management and therapy.

References

[1] W. C. Zhou, Q. B. Zhang, and L. Qiao, "Pathogenesis of liver cirrhosis," *World J. Gastroenterol.*, vol. 20, no. 23, pp. 7312–7324, 2014, doi: 10.3748/wjg.v20.i23.7312.

[2] I. A. Rowe, "Lessons from Epidemiology: The Burden of Liver Disease.," *Dig. Dis.*, vol. 35, no. 4, pp. 304–309, 2017, doi: 10.1159/000456580.

[3] S. Piano *et al.*, "Epidemiology and Effects of Bacterial Infections in Patients With Cirrhosis Worldwide," *Gastroenterology*, vol. 156, no. 5, pp. 1368-1380.e10, 2019, doi: 10.1053/j.gastro.2018.12.005.

[4] P. Rawla, K. C. Thandra, A. Vellipuram, and C. D. M. Ali, "Efficacy and safety of megestrol in the management of hepatocellular carcinoma: a systematic review of the literature.," *Contemp. Oncol. (Poznan, Poland)*, vol. 22, no. 4, pp. 209–214, 2018, doi: 10.5114/wo.2018.82641.

[5] M. Sun and T. Kisseleva, "Reversibility of liver fibrosis," *Clin. Res. Hepatol. Gastroenterol.*, vol. 39 Suppl 1, pp. S60-3, 2015, doi: 10.1016/j.clinre.2015.06.015.

[6] N. A. Terrault, N. H. Bzowej, K.-M. Chang, J. P. Hwang, M. M. Jonas, and M. H. Murad, "AASLD guidelines for treatment of chronic hepatitis B.," *Hepatology*, vol. 63, no. 1, pp. 261–283, Jan. 2016, doi: 10.1002/hep.28156.

[7] A. Schweitzer, J. Horn, R. T. Mikolajczyk, G. Krause, and J. J. Ott, "Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.," *Lancet (London, England)*, vol. 386, no. 10003, pp. 1546–1555, Oct. 2015, doi: 10.1016/S0140-6736(15)61412-X.

[8] P. Nannini and E. M. Sokal, "Hepatitis B: changing epidemiology and interventions.," *Arch. Dis. Child.*, vol. 102, no. 7, pp. 676–680, Jul. 2017, doi: 10.1136/archdischild-2016-312043.

[9] E. A. Tsochatzis, J. Bosch, and A. K. Burroughs, "Liver cirrhosis.," *Lancet (London, England)*, vol. 383, no. 9930, pp. 1749–1761, May 2014, doi: 10.1016/S0140-6736(14)60121-5.

[10] F. F. Poordad, "Presentation and complications associated with cirrhosis of the liver.," *Curr. Med. Res. Opin.*, vol. 31, no. 5, pp. 925–937, May 2015, doi: 10.1185/03007995.2015.1021905.

[11] L. Li *et al.*, "The spleen in liver cirrhosis: revisiting an old enemy with novel targets.," *J. Transl. Med.*, vol. 15, no. 1, p. 111, May 2017, doi: 10.1186/s12967-017-1214-8.

[12] P. W. Marks, "Hematologic manifestations of liver disease.," *Semin. Hematol.*, vol. 50, no. 3, pp. 216–221, Jul. 2013, doi: 10.1053/j.seminhematol.2013.06.003.

[13] E. M. M. Kelly, V. A. Feldstein, M. Parks, R. Hudock, D. Etheridge, and M. G. Peters, "An Assessment of the Clinical Accuracy of Ultrasound in Diagnosing Cirrhosis in the Absence of Portal Hypertension.," *Gastroenterol. Hepatol. (N. Y).*, vol. 14, no. 6, pp. 367–373, Jun. 2018.

[14] I. C. Perez, F. J. Bolte, W. Bigelow, Z. Dickson, and N. L. Shah, "Step by Step: Managing the Complications of Cirrhosis.," *Hepat. Med.*, vol. 13, pp. 45–57, 2021, doi: 10.2147/HMER.S278032.