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Case Report

Hepatic cirrhosis causes pancytopenia: a case report

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ABSTRACT

Hepatic cirrhosis is a slow process liver disease, persisting over a long period of time, resulting in a progressive destruction, and is the final common pathological pathway. We report a case of 60-year-old man with hepatic cirrhosis and pancytopenia. He came to emergency room with chronic fatigue, light headed, and abdominal pain. In laboratory examination, hemoglobin 6.0 g/dL, red blood cell counts 2.8×106/mm3, platelets 58.000×103/mm3, leukocytes 3.800×103/mm3, and reactive HbsAg. Abdominal ultrasound revealed small liver with irregular edges, ascitic fluid, and enlarged spleen. The patient was treated with furosemide, spironolactone, lactulose and propranolol. The liver, or the hepatocyte constitutes a significant storage site for iron, produce erythropoietin and thrombopoietin. Hypersplenism causes retention of a large number of leukocytes, erythrocytes, and platelets in the spleen, and the number of retained blood cells can be 5.5-20 times higher than the normal level, thus facilitating capture, phagocytosis or destruction of blood cells by phagocytes resulting in peripheral cytopenias. This case illustrated that hepatic cirrhosis can cause pancytopenia. Therefore, we must look for any cause of pancytopenia in a patient with chronic liver disease. Lack of thrombopoietin and erythropoietin production, lack of iron storage in damaged hepatocyte, also splenic sequestration were the reason of pancytopenia in hepatic cirrhosis. Bone marrow examination is one of modalities that can be use, and still being recommended examination.

Keywords: Hepatic cirrhosis, Pancytopenia, Hypersplenism, Erythropoietin, Thrombopoietin, Chronic liver disease

INTRODUCTION

Liver is a key site for the lipid metabolism and synthesis of a number of proteins that important to the hematopoietic system.1 Physiologic synthetic functions performed by the liver, as well as its anatomic relationship to the spleen, facilitates appreciation of the resulting hematologic abnormalities that occur in association with liver disease.[1] Hepatic cirrhosis is a liver disease of slow process, persisting over a long period of time, resulting in a progressive destruction of the liver, and is the final common pathological pathway.[2,3] Alcoholism, chronic hepatitis B virus infection, chronic hepatitis C infection, and nonalcoholic fatty liver disease being the most common causes.[3]

Patients progress from compensated cirrhosis to decompensated cirrhosis when the liver is extensively scarred and no longer able to properly function, despite a prevalence of 844 million worldwide and a mortality rate of 2 million per year, the impact of cirrhosis on global public health is largely overlooked.[4] Of the 2 million deaths per year, 1 million were due to complications of cirrhosis and 1 million were due to viral hepatitis and hepatocellular carcinoma.[4] Mortality and morbidity of cirrhosis run high, due to resulting extrahepatic morbidities that reduce quality of life.[4]

This disease has many systemic features and has also hematological manifestations as bicytopenias and pancytopenias are frequently seen in these cases which is

present with lethargy, fatigue, infections and bleeding tendencies.[2] Pancytopenia, defined as a combination of white blood cell (WBC) count < 4.0×109/L, red blood cell (RBC) count < 3.5×1012/L, and platelet (PLT) count < 100×109/L.[5] Pancytopenia in chronic liver disease can be due to hypersplenism, megaloblastic anemia and primary marrow suppression and hypersplenism is the most common cause.[2] Hypersplenism is a clinical syndrome, enlargement of spleen, reduction of at least one cell line in the blood in the presence of normal marrow function and evidence of increased release of premature cells such as reticulocytes or immature platelets from the bone marrow into the blood.[2]

Cirrhosis and its complications not only impair quality of life but also decrease survival, and managing patients with cirrhosis can be a challenge and requires an organized and systematic approach.6 Here we presented a case report of 60-years-old man diagnosed with hepatic chirrosis which caused complication of pancytopenia.

CASE REPORT

We report a case of 60-year-old man diagnosed with hepatic cirrhosis and pancytopenia. He came to emergency room with chronic fatigue, light headed, and upper right abdominal pain. During the past 3 weeks he felt progressive increased of his abdominal perimeter which resulted in dyspnea. He also complained having lower limb edema since one week. Those were the reason he sought medical assistance. Hematochezia, melena, hematemesis, and hemoptoe were not present. He never consumed alcohol and drugs, either having a tattoo. The examination showed 21 time/minute respiratory rate, 120/80 mmHg blood pressure, 76 times/minutes regular heart rate, 37.0o C of temperature, and BMI 41 kg/mm2. Pale palpebral conjunctiva, spider naevus, prominent abdomen with water dullness, and bilateral lower limb edema was found. Thorax examination found normal.

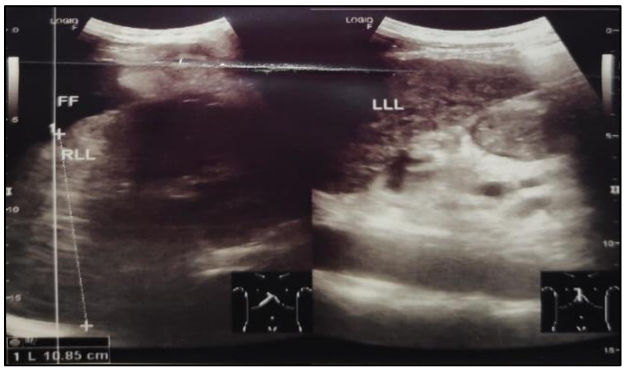

Figure 1: Abdominal ultrasound imaging showed small liver with irregular edges.

Laboratory work-up upon admission showed : hemoglobin 6.0 g/dL, red blood cell count 2.8×106/mm3 , platelets 58.000×103/mm3, leukocytes 3.800×103/mm3 with 69% neutrophils, 26% lymphocytes, and 2% monocytes, hematocrit 21%, total protein 6.3 g/dL, albumin 3.4 g/dL, globulin 1.9 g/dL, total bilirubin 0.7 mg/dL, bilirubin direct 0.4 mg/dL, and reactive HbsAg. Peripheral blood morphology examination showed microcytic hypochromic red blood cell with cigar cell. Abdominal ultrasound imaging revealed small liver with irregular edges (Figure 1), ascitic fluid, and enlarged spleen. ECG 12-lead and posteroanterior chest x-ray were normal.

The patient was hospitalized for 6 days. He was treated with high protein diet, 750 cc transfusion of whole blood was given. He was given furosemide 20 mg intravenous three times a day, spironolactone 100 mg twice a day, and lactulose three times a day. Propranolol 10 mg once a day was also given to lower the blood pressure in esophageal varices and prevent bleeding. In the last day, the hematologic examination was normal and the patient, the patient was getting better. The patient was referred for bone marrow examination and hepatitis B infection treatment but refused. Thus, the patient regularly visit outpatient ward for blood laboratory checks and supportive treatment was given.

DISCUSSION

Chronic liver disease is characterized by a progressive destruction of the liver parenchyma or functional tissue for greater than 6 months, that may lead to fibrosis and ultimately, it is called cirrhosis.[4] Abnormalities in hematological indices are frequently encountered in cirrhosis, multiple causes contribute to the occurrence of hematological abnormalities.[7] Some studies suggest that the presence of cytopenias in chirrosis is in a poor prognosis.[7]

In this case, the patient came to the emergency room with chronic fatigue and light headed. He also had pale palpebral conjunctiva, with hemoglobin 6.0 g/dL, low count of red blood cell, and microcytic hypochromic red blood cell with cigar cell in peripheral blood morphology examination. Anemia is the most common complication of liver cirrhosis, and the etiology of anemia in liver disease is diverse and often multifactorial.[8] The common causes are acute and chronic blood loss due to upper gastrointestinal bleeding, malnutrition, hypersplenism secondary to portal hypertension, and impaired coagulation.[8] Contrast with the literature, in this case the patient never had bleeding history, including hematochezia, melena, hematemesis, and hemoptoe. He was obese and not lack of nutrition either. The liver, or the hepatocyte constitutes a significant storage site for iron, besides enterocyte and macrophage, which it receives through the portal circulation and, in case of increased demand, releases back to the systemic circulation.[9] It was supported by peripheral blood morphology examination that shown microcytic hypochromic. Beside that, erythropoietin is produced predominantly by the kidney but also by the liver, it protects RBCs from apoptosis and enhances the development of precursor red blood cells.[7]

In the examination we found thrombocytopenia, it is in line with other study. Moore, et al. found that thrombocytopenia is the most common hematological abnormalities and is often the first abnormality seen in patients with chronic liver disease.[10] Platelet production is largely associated with thrombopoietin which is predominantly synthesized in the liver in parenchymal and sinusoidal endothelial cells and in the kidneys and small amounts are also made in bone marrow stromal cells.[10] Thrombopoietin binds to the c-mpl receptor on megakaryocytes, to regulates the differentiation into platelets.[10] Another cause of thrombocytopenia, which is associated with platelet sequestration, is pseudothrombocytopenia which is a falsely low platelet count because of clumping platelet.[10] Similar to anemia and thrombocytopenia, leukopenia also caused by bone marrow suppression and sequestration.[1,2,5,7,10] Other causes of the decreased red blood cells, white blood cells, and platelet production includes reduced bone marrow production that can be a result of alcohol abuse and viral infections.[10] Qamar et al found that cytopenia caused by bone marrow suppression mediated by toxins, including alcohol, hepatitis B and C infection.[7] In contrary, this patient never consumed alcohol and drugs, either having a tattoo, but his hepatitis B infection increased the risk of pancytopenia.

Splenomegaly also found in this patient. Splenic sequestration and rapid destruction of platelets, white blood cells and red blood cells in the portal hypertension-induced enlarged spleen, it is called hypersplenism.[7,11] In patients with portal hypertension, caused by hepatic cirrhosis, the spleen can increase 8-10 times its normal size, then, splenic blood volume increases due to the increased venous pressure leading to congestive splenomegaly, or because of the increased splenic arterial blood flow induced by a variety of diseases, resulting in hyperemic splenomegaly.[11] As a consequence, there is retention of a large number of leukocytes, erythrocytes and platelets in the spleen, and the number of retained blood cells can be 5.5-20 times higher than the normal level, thus facilitating capture, phagocytosis or destruction of blood cells by phagocytes resulting in peripheral cytopenias.[11]

CONCLUSION

This case illustrated that hepatic cirrhosis can cause pancytopenia. Therefore, we must look for any cause of pancytopenia in a patient with chronic liver disease. Lack of thrombopoietin and erythropoietin production, lack of iron storage in damaged hepatocyte, also splenic sequestration was the reason of pancytopenia in hepatic cirrhosis. Bone marrow examination is one of modalities that can be use, and still being recommended examination to diagnose pancytopenia in hepatic cirrhosis. In this case, the patient was referred for bone marrow examination and hepatitis B infection treatment but refused. Thus, we did not underwent definitive diagnosis and treatment for hepatic cirrhosis and hypersplenism due to facility limitation. Recent study describe that most of the etiological causes of pancytopenia were associated with nonhematological diseases and were diagnosed with laboratory tests without the need of bone marrow examination. Correspond with literature, the outcome was good despite the examination and treatment was limited.

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