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The Incidence of Hypersplenism in Decompensated Liver Cirrhosis of Alcoholic Etiology

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Authors' contributions

This work was carried out in collaboration between both authors. Author GB designed the study, wrote the protocol and wrote the first draft of the manuscript. Author ZM helped perform the analysis of study with construcitive discussions. Both authors read and approved the final manuscript.

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Short Research Article

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ABSTRACT

Introduction: Liver cirrhosis is a terminal, morpho-functional entity of hepatic dysfunction, which is characterized by the replacement of the hepatic lobule connective tissue nodules. Splenomegaly is a common finding in patients with liver cirrhosis and portal hypertension can cause hypersplenism. Hypersplenism is the leading cause of thrombocytopenia.
Objective: The aim of this study was to investigate the association between platelet count and spleen size in patients with decompensated liver cirrhosis.
Materials and Methods: Retrospective study included 50 male patients with liver cirrhosis, alcoholic etiology, hospitalized in the University Clinical Center of Republic of Srpska, at the Department of Gastroenterology and hepatology. The degree of liver function we determined by using Child-Pugh score. Craniocaudal diameter of the spleen was determined by ultrasound, a criterion for splenomegaly has a diameter greater than 11 cm. Presence of thrombocytopenia is platelet count below 150,000 / ml.
Results: The average age of patients was 62 years. Splenomegaly was present in 62% of

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patients. The mean diameter of the spleen was 14.6 cm. The mean platelet count was 87,500 / ml. In this study, no significant correlation was observed between spleen size and platelet count ($p = 0.587$).

Conclusion: In this study, liver cirrhosis and thrombocytopenia may be present even in the absence of enlarged spleen, indicating the possible presence of other mechanisms that reduce the number of platelets.

Keywords: Thrombocytopenia; splenomegaly; hypersplenism; alcoholic liver cirrhosis.

1. INTRODUCTION

Liver cirrhosis is a terminal, morpho-functional entity of hepatic dysfunction, which is characterized by the replacement of the hepatic lobule connective tissue nodules. Cirrhosis is a condition that is defined histopathologically and has a variety of clinical manifestations and complications [1,2].

There are too many causes of the liver cirrhosis. Liver cirrhosis can be caused by: using of increased levels of alcohol, chronic viral hepatitis B or C, biliary disease, autoimmune disease, metabolic disorders and other, less frequent causes such as heart failure, etc. Chronic alcohol intake can cause: alcoholic fatty liver, alcoholic hepatitis, and alcoholic cirrhosis [3,4]. The alcohol liver cirrhosis is more frequent in male patients. It's associated with increased consummation of alcoholic drinks.

Portal hypertension is one of the first manifestations of decompensated liver cirrhosis. Pathological changes and proliferation of connective tissue invaded intra- and extra-hepatic blood vessels. It reduces the circulation throughout the liver and increase the blood pressure in portal artery. After that, the next step is forming the collateral circulation. Blood is going through the blood vessels from portal artery via lienal artery to the spleen. It causes the enlargement of the spleen and it is associated with hypersplenism [5]. But in fact, there is no hypersplenism without splenomegaly, but splenomegaly can occur without hypersplenism. Hypersplenism is a destructive ability of spleen to reduce the level of blood cells with normal function of the bone marrow. The hypersplenism can cause: anemia, leucopenia, thrombocytopenia and pancytopenia. The most common findings are thrombocytopenia.

Thrombocytopenia is often defined as a platelet count below the 150×10^9/ l and it is frequently manifests in decompensated liver cirrhosis. The etiology of thrombocytopenia is multifactorial [6]. In alcoholic liver cirrhosis it is associated with toxic effect of alcohol on bone marrow. But, chronic viral hepatitis or biliary disease jointly with consummation of alcohol drinks can cause several disorders in hepatic function and faster progression chronic hepatitis to liver cirrhosis and liver cancer [7].

This study was design to investigate the association between platelet count and spleen size in male patients with decompensated liver cirrhosis.

2. MATERIALS AND METHODS

The clinical tests were performed within the Department of Gastroenterology of the Clinic of Internal Medicine at the University Clinical Center of Republic of Srpska. Retrospective study included 50 male patients with decompensated liver cirrhosis, alcoholic etiology. Data source for this study is an archive of medical records, which contains case histories of patients.

When testing was used to separate, specially designed test protocol in which they recorded all relevant information regarding research: anamnesis of patients, medical history, data on subjective complaints of patients, data on complications and clinical manifestations, laboratory tests, diagnostic procedures (standard chest radiography of the heart (RTG), abdominal ultrasound, esophagogastroduodenoscopy, any additional time (computerized tomography (CT) and / or nuclear magnetic resonance (NMR) abdominal doppler ultrasonography of the liver, spleen and portal system) immediately upon receipt of the conducting a detailed physical examination, blood taken for complete analysis, underwent chest X-ray, abdominal ultrasound and upper digestive endoscopy. Results were analyzed using standard statistical methods and presented in tables.

The factors determined were the degree of liver function, splenomegaly and hypersplenism.

The degree of liver function was determined by using Child-Pugh score. Child-Pugh score is used to assess the prognosis of chronic liver disease, mainly cirrhosis. The severity of liver disease was assessed in each patient based on five clinical features: 1) total bilirubin level, 2) serum albumin, 3) prothrombin time (now measured as the INR), 4) the degree of ascites, and 5) the grade of hepatic encephalopathy. The total point score was then used to determine the patient's Child-Pugh class. Craniocaudal diameter of the spleen was determined by ultrasound. A criterion for splenomegaly has a diameter greater than 11 cm [8,9]. The presence of thrombocytopenia is platelet count below 150,000 / ml.

3. RESULTS

Splenomegaly was observed in 62% of patients. The average age of patients with splenomegaly was 62 years. The most patients were aged over 60 years (Table 1).

Table 1. Age groups of patients with splenomegaly

40 - 49	4
50 - 59	13
60+	14

Total number of 34 patients were observed to have a B Child-Pugh score, while 8% of the studied population were rated as A Child-Pugh score (Table 2).

Table 2. Results of Child-Pugh score

Child-Pugh score	
A	4 (8%)
B	34 (68%)
C	12 (24%)

The mean platelet count was 87,500 / ml. Only 3 patients with splenomegaly had the platelets count below 50×10^9 / l. It was presented by tables (Table 3).

The most patients had a spleen diameter between 12 and 14 cm (62%). The mean diameter of the spleen is 14,6 cm (Table 4).

In this study we did not find any significant correlation between spleen size and platelet count (p=0.587).

Table 3. Patients group selection by thrombocytopenia count

Thrombocytopenia	
<50	3
50 - 100	9
100 - 150	8

Table 4. Spleen size groups of patients with splenomegaly

Spleen size (cm)	Splenomegaly (number of patients)
12 - 14	15
14 - 16	12
16 - 18	2
18 - 20	2

4. DISCUSSION

This study included 50 male patients with decompensated liver cirrhosis, alcoholic etiology. The average age of patients with splenomegaly was 62 years. Literature available data show predominance of male and elderly patients with liver cirrhosis [8]. Thrombocytopenia has been thought to arise from the increased pooling of platelets in an enlarged spleen (splenomegaly). Splenomegaly was observed in 62% of patients. The mean diameter of the spleen is 14,6 cm. These results are consistent with the literature data [9,10]. Child-Pugh score has undergone modifications and is currently used to assess the severity and prognosis of chronic liver disease and cirrhosis. Total numbers of 34 patients were observed to have a B Child-Pugh score, while 8% of the studied populations were rated as A Child-Pugh score. Thrombocytopenia is one of the most common complication in liver disease, and liver disease-related thrombocytopenia is often defined as a platelet count $< 150 \times 10^9$/ l. Two mechanisms which can cause the thrombocytopenia in liver cirrhosis are: accelerated platelet destruction in the spleen and low level of thrombopoietin in the liver [11,12]. The mean platelet count was 87,500 / ml. In this study we did not find any significant correlation between spleen size and platelet count (p=0.587).

5. CONCLUSION

Liver cirrhosis, portal hypertension and thrombocytopenia could be present even in the absence of splenomegaly. It means that other mechanisms could be important in reduction of platelet counts. For example, in alcoholic liver

cirrhosis thrombocytopenia is associated with toxic effect of alcohol on bone marrow. But, chronic viral hepatitis or biliary disease jointly with consummation of increased levels of alcohol drinks can cause several disorders in hepatic function and faster progression chronic hepatitis to liver cirrhosis and liver cancer.

CONSENT

Both authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this paper.

ETHICAL APPROVAL

Both authors hereby declare that all documentation approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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