

Frequency and Severity of Thrombocytopenia with Liver Cirrhosis

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ABSTRACT

Background and Aim: Thrombocytopenia is a major complication of liver disease that impairs liver cirrhosis treatment by restraining administrative therapy and surgical or diagnostic planning delaying which in turn increases the bleeding risk. The present study aimed to evaluate the frequency and severity of thrombocytopenia in liver cirrhosis patients.

Materials and Methods: This cross-sectional study was carried out on 88 liver cirrhosis patients in the department of Gastroenterology, Shaikh Zayed Hospital, Lahore from November 2020 to June 2021. Individuals with cirrhosis disease were evaluated for the severity of thrombocytopenia. All the cirrhosis patients with age >14 years of either gender were enrolled while aplastic anemia cases, idiopathic thrombocytopenic purpura, drug therapy patients, osteopetrosis, cases of repeated blood or platelet transfusion history, myelodysplastic syndrome, and refusal to informed consent were all excluded. The demographic details, clinical examination, and treatment plan were assessed. A platelet count test was performed on all cirrhosis patients. Thrombocytopenia severity was divided into three categories: mild thrombocytopenia (50,000/l), moderate thrombocytopenia (20,000/l), and severe thrombocytopenia (10,000/l). SPSS version 20 was used for descriptive analysis.

Results: Of the total 88 cirrhosis patients, 62 (70.5%) were male and 26 (29.5%) were females. All the cirrhosis patients were assessed for thrombocytopenia. The overall mean age was 39.41±11.56 years. Out of 88 cirrhosis patients, the prevalence of thrombocytopenia was 63 (71.2%). Of the 63 thrombocytopenia diagnosed cirrhosis patients, 41 (65%) were male and 22 (35%) were females. The prevalence of mild, moderate, and severe thrombocytopenia was 21 (33.3%), 23 (36.5%), and 19 (30.2%) respectively.

Conclusion: Our study concluded that thrombocytopenia prevalence was 71.2% in liver cirrhosis patients. The assessment and monitoring of platelet count identified the thrombocytopenia among liver cirrhosis. Moreover, the severity of bleeding episode that might cause fatality were reduced.

Keywords: Thrombocytopenia; Liver Cirrhosis; Severity; Platelet

INTRODUCTION

Thrombocytopenia is a liver disease major complication, and it is often described based on platelet count. Spontaneous bleeding occurs in patients whose platelet count <10 109/L-20 109/L, which in turn needs surgical intervention during the treatment of cirrhosis patients [1, 2]. The thrombocytopenia presence can exacerbate traumatic bleeding, as well as ominously thwart routine patient, such as antiviral therapy, liver biopsy, and cirrhotic patients optional surgery, in turn cancelled therapeutic administration and operative treatment for a variety of circumstances such as chronic hepatitis C virus infection antiviral therapy. Certainly, the thrombocytopenia degree is an effective predictive marker for severe thrombocytopenia in liver cirrhosis patients [3, 4].

Furthermore, a low platelet count can be a sign of undiagnosed cirrhosis or the presence of esophageal varices [5, 6]. Multiple factors can contribute to the thrombocytopenia development in cirrhotic patients, including splenic sequestration, cirrhotic coagulopathy, and bone marrow for cirrhosis suppression by chronic HCV infection. The major mechanisms for thrombocytopenia in liver cirrhosis are (1) platelet sequestration in the spleen and (2) reduced TPO production in the liver. Historically, thrombocytopenia was thought to be caused by an increase in platelet pooling in splenomegaly. Splenomegaly can cause it to exceed 1000 mL. Repossession of Platelet is observed in splenomegaly caused by portal hypertension caused by cirrhosis and is characterized by a redistribution

of platelets from the circulating pool to the splenic pool [7, 8].

Platelets are megakaryocytes that have been cytoplasmically fragmented. Circulating inert platelets have smooth surfaces and are disc-shaped. They are approximately 2-3 microns in size and 1.5-4 lakhs in number per cumm. They have a shelf life of 10 days in circulation [9]. Platelets are produced by megakaryocytes and play an important role in hemostasis [10]. In chronic hepatic disease, thrombocytopenia and hypoalbuminemia were discovered [11]. Patients with hepatic cirrhosis have hemostatic impairment, which includes thrombocytopenia [12]. Other causes of thrombocytopenia include chronic hepatitis' suppression of bone marrow and a decrease in thrombopoietin activity level [13]. Taking this into account, the study determined the incidence and sternness of thrombocytopenia in liver cirrhosis patients.

MATERIAL AND METHODS

This cross-sectional study was carried out on 88 liver cirrhosis patients in the department of Gastroenterology, Shaikh Zayed Hospital, Lahore from November 2020 to June 2021. Individuals with cirrhosis disease were evaluated for the severity of thrombocytopenia. All the cirrhosis patients with age >14 years of either gender were enrolled while aplastic anemia cases, idiopathic thrombocytopenic purpura, drug therapy patients, osteopetrosis, cases of repeated blood or platelet transfusion history, myelodysplastic syndrome, and refusal to informed consent were all excluded. The demographic

details, clinical examination, and treatment plan were assessed. A platelet count test was performed on all cirrhosis patients. Thrombocytopenia severity was divided into three categories: mild thrombocytopenia (50,000/l), moderate thrombocytopenia (20,000/l), and severe thrombocytopenia (10,000/l).

Following that, the platelet count of all cirrhotic subjects was determined. Blood sample of 3 cubic centimeter venous blood sample was collected and forwarded for analyzing in the laboratory. The presence of ascites, albumin, encephalopathy, serum bilirubin, and prothrombin time were all factored into the child pugh score. Clinical examination (shifting dullness and fluid thrill) revealed ascites, which was confirmed on ultrasound. Blood biochemistry was used to assess serum albumin, PT/INR, and bilirubin levels in comparison to normal values, as well as hepatic encephalopathy based on history, clinical examination, and serum ammonia levels, with bleeding times greater than 6 minutes considered prolonged. The class was determined by adding the scores, i.e. class A has 5-6 points, class B has 7-9 points, and class C has 10-15 points. For data collection, a proforma was created. SPSS version 23.00 was used to analyses the data. The incidence and proportion of occurrences were computed. The categorical variables was

compared using chi-square test with <0.005 level of significance.

RESULTS

Of the total 88 cirrhosis patients, 62 (70.5%) were male and 26 (29.5%) were females. All the cirrhosis patients were assessed for thrombocytopenia. The overall mean age was 39.41±11.56 years. Out of 88 cirrhosis patients, the prevalence of thrombocytopenia was 63 (71.2%). Of the 63 thrombocytopenia diagnosed cirrhosis patients, 41 (65%) were male and 22 (35%) were females. The prevalence of mild, moderate, and severe thrombocytopenia was 21 (33.3%), 23 (36.5%), and 19 (30.2%) respectively. The mean value of platelet count in the overall population and thrombocytopenia cirrhosis patients was 131.45±7.23 and 69.57±7.34 respectively. Male and female patients had 69.98±8.32 and 63.56±4.96 respectively as shown in Table-1. Figure-I shows the age-wise distribution of cirrhosis patients. Prevalence of male and female in overall population and thrombocytopenia cirrhosis patients are shown in Figure-II and III respectively. Figure-IV illustrates the thrombocytopenia severity incidence among thrombocytopenia diagnosed patients. The incidence of mild, moderate, and severe thrombocytopenia are shown in Table-2.

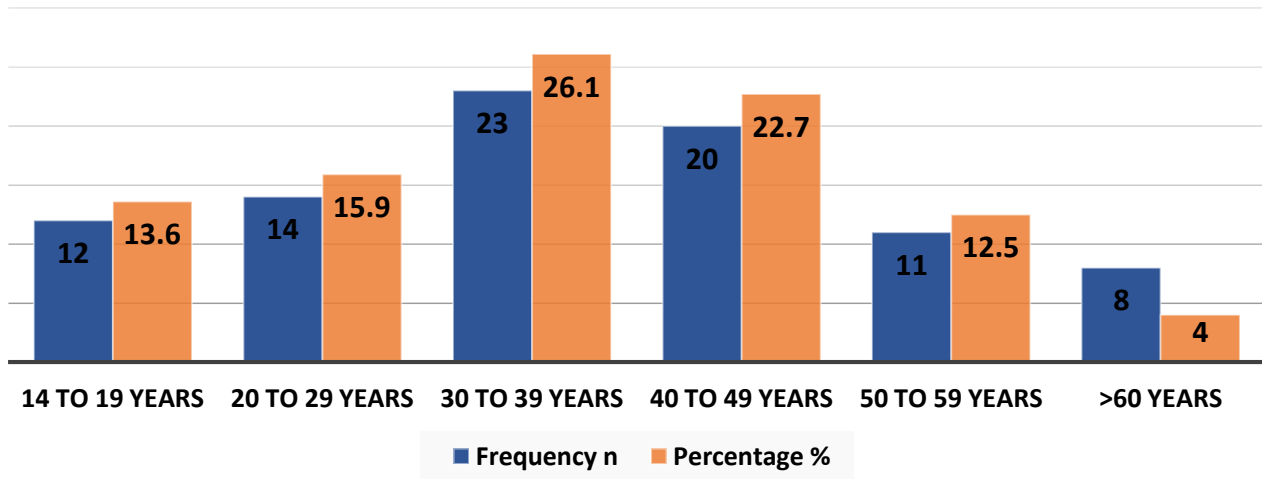


Figure-1 Age wise distribution of cirrhosis patients (n=88)

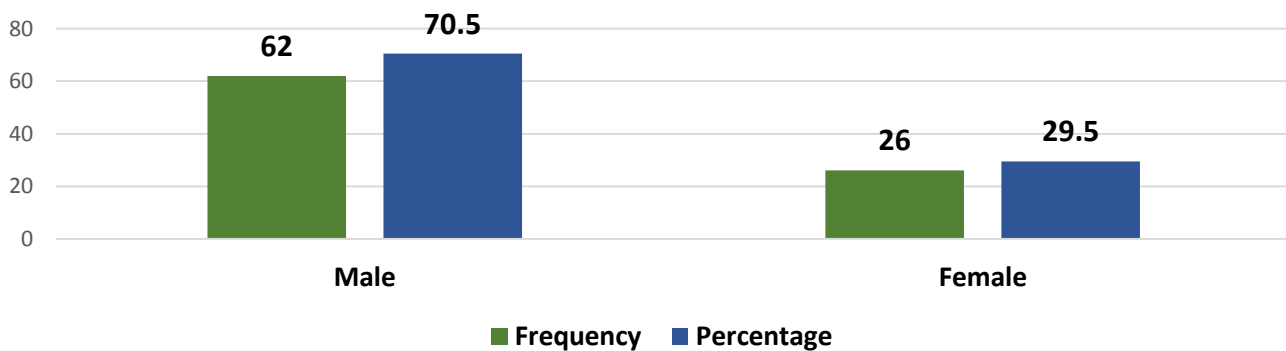


Figure-II Gender distribution of Cirrhosis patients (n=88)

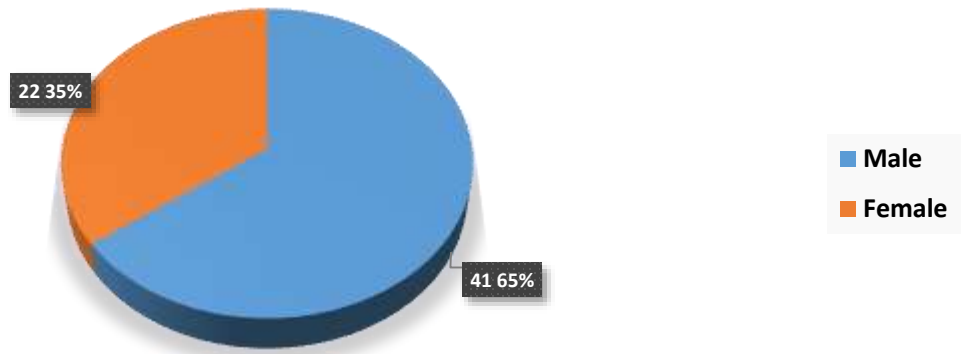


Figure-III Gender distribution of thrombocytopenia patients (n=63)

Table-1 Gender distribution with thrombocytopenia cases

Gender	Thrombocytopenia		Total N	P-value
	Yes	No		
Male	41(65%)	21 (84%)	62 (70.5%)	0.02
Female	22 (35%)	4 (16%)	26 (29.5%)	0.02
Total	63 (100%)	25	88 (100%)	0.02

Table-2 Prevalence of mild, moderate and severe thrombocytopenia

Thrombocytopenia	Frequency n	Percentage %
Mild	21	23.9
Moderate	23	26.1
Severe	19	21.6

DISCUSSION

The current study assessed thrombocytopenia in patients with liver cirrhosis and discovered a 71.2% prevalence. Poor dad F’s study reported that thrombocytopenia is a foremost malady in liver cirrhosis patients. Tanaka M.’s investigation found that about 63% were the thrombocytopenia prevalence in cirrhotic subjects. [14, 15]. The majority of people in the current series were between the ages of 30 and 50, which was also observed in a 2011 study. In liver cirrhosis, thrombocytopenia is caused by increased platelet clearance and impaired thrombopoiesis [16]. Thrombocytopenia in cirrhotic patients now has numerous treatment choices such as embolization of splenic artery, surgical splenectomy, and platelet transfusion. Therapeutic options for safely and effectively increasing platelet levels can have a significant impact on cirrhotic patients’ care. Increased platelet levels, in particular, can ominously decrease the platelet transfusions need to cirrhotic patients.

Platelet counts <50 109/L may have advantage for patients with prophylactic transfusions to boost platelet counts prior to treatment [16]. Currently, no agreement about cirrhotic patient’s optimal onset standards for platelet transfusions. Platelet transfusion snags and restrictions include febrile no hemolytic, chronic transfusions, and infection risk [17, 18]. Additionally, transfusions of platelet gives no guarantee for platelet levels, particularly when higher risk for bleeding exist [19]. While about 120 days lifespan of red blood cells, a shorter lifespan has been observed of transfused platelets.

The male population predominates in the current study, which matched with results of Nasta P. [20].

Thrombocytopenia is a significant impediment in cirrhosis patients. In the Jan et al [21] research thrombocytopenic was 92% in cirrhosis patients, whereas earlier study stated 76%, and others found thrombocytopenia in cirrhotic patients. [22, 23]. In the current study, thrombocytopenia was mild, moderate, and severe in 23.9%, 26.1%, and 21.6% respectively. According to two previous studies, the prevalence of moderate thrombocytopenia is 22% and 27%, respectively. [24, 25]. Thrombocytopenia in cirrhosis is significant not only as an association, but also as a predictor of bleeding.

CONCLUSION

Our study concluded that thrombocytopenia prevalence was 71.2% in liver cirrhosis patients. The assessment and monitoring of platelet count identified the thrombocytopenia among liver cirrhosis. Moreover, the severity of bleeding episode that might cause fatality were reduced.

REFERENCES

1. Erdem MG, Cil EO, Tukek T, et al. Evaluation of platelet and mean platelet volume levels in patients with liver cirrhosis. Arch Clin Exp Med. 2018;3(1):18–21
2. Koganov ES, Carmichael SL, Forde EE, et al. Platelet function in thrombocytopenic patients with chronic liver disease. Blood. 2017; 130(Suppl 1):2314.
3. Cocero N, Bezzi M, Martini S, et al. Oral surgical treatment of patients with chronic liver disease: assessments of bleeding and its relationship with thrombocytopenia and blood coagulation parameters. J Oral Maxillofac Surg. 2017;75(1):28–34. doi:10.1016/j.joms.2016.08.03
4. Kalambokis GN, Oikonomou A, Christou L, et al. von Willebrand factor and procoagulant imbalance predict outcome in patients with cirrhosis and thrombocytopenia. J Hepatol. 2016;65:921–928. doi:10.1016/j.jhep.2016.06.002.
5. Golriz M, Ghamamejad O, Khajeh E, et al. Preoperative thrombocytopenia may predict poor surgical outcome after extended hepatectomy. CJGH. 2018:1275720. doi:10.1155/2018/1275720

6. Li L, Wang H, Yang J, et al. Immediate postoperative low platelet counts after living donor liver transplantation predict early allograft dysfunction. *Medicine*. 2015;94(34):1–7
7. Rowley MW, Agarwal S, Seetharam AB, et al. Real-time ultrasound-guided paracentesis by radiologists: near zero risk of hemorrhage without correction of coagulopathy. *J Vasc Interv Radiol*. 2019;30:259–264. doi:10.1016/j.jvir.2018.11.001
8. Mitchell O, Feldman DM, Diakow M, et al. The pathophysiology of thrombocytopenia in chronic liver disease. *Hep Med*. 2016;8:39–50.
9. Basili S, Raparelli V, Violi F. The coagulopathy of chronic liver disease: is there a causal relationship with bleeding? *Yes. Eur J Intern Med*. 2019;21(2):62–64. doi:10.1016/j.ejim.2010.01.005
10. Giannini EG, Moscatelli A, Brunacci M, et al. Prognostic role of mean platelet volume in patients with cirrhosis. *Dig Liver Dis*. 2016;48(4):409–413. doi:10.1016/j.dld.2015.10.018
11. Chin JL, Hisamuddin SH, O'Sullivan A, et al. Thrombocytopenia, platelet transfusion, and outcome following liver transplantation. *Clin Appl Thromb Hemost*. 2016;22(4):351–360. doi:10.1177/1076029614559771
12. Kurokawa T, Ohkohchi N. Platelets in liver disease, cancer and regeneration. *World J Gastroenterol*. 2017;23(18):3228–3239. doi:10.3748/wjg.v23.i18.3228
13. Garcia-Tsao G, Abraldes JG, Berzigotti A, et al. Portal hypertensive bleeding in cirrhosis: risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology*. 2017;65(1):310–335. doi:10.1002/hep.v65.1
14. Colli A, Gana JC, Yap J, et al. Platelet count, spleen length, and platelet count-to-spleen length ratio for the diagnosis of esophageal varices in people with chronic liver disease or portal vein thrombosis. *Cochrane Database Sys Rev*. 2017;4:1–210
15. Abraldes JG, Bureau C, Stefanescu H, et al. Noninvasive tools and risk of clinically significant portal hypertension and varices in compensated cirrhosis: the “Anticipate” study. *Hepatology*. 2016;64(6):2173–2184. doi:10.1002/hep.v64.6
16. Angeli P, Bernardi M, Laleman W, et al. EASL clinical practice guidelines for the management of patients with decompensated cirrhosis. *J Hepatol*. 2018;69:406–460. doi:10.1016/j.jhep.2018.03.024
17. Stine JG, Niccum BA, Zimmet AN, et al. Increased risk of venous thromboembolism in hospitalized patients with cirrhosis due to non-alcoholicsteatohepatitis. *ClinTranslGastroenterol*. 2018;9(3):140.
18. Ferre AC, Nunez GL, Tellez VL, et al. Epistaxis in the cirrhotic patient: a complication to be considered. *Gastroenterol Hepatol*. 2019;42(1):11–15.
19. Morgan J, Hinz EK. Conservative management of endometrial intraepithelial neoplasia in a patient with cirrhosis undergoing orthotopic liver transplant. *Menopause*. 2019;26:1068–1070. doi:10.1097/GME.0000000000001342
20. Noronha Ferreira C, Marinho RT, Cortez-Pinto H, et al. Incidence, predictive factors and clinical significance of development of portal vein thrombosis in cirrhosis: a prospective study. *Liver Int*. 2019;39:1459–1467. doi:10.1111/liv.14121
21. Yang LS, Alukaidey S, Croucher K, Dowling D. Suboptimal use of pharmacological venous thromboembolism prophylaxis in cirrhotic patients. *Intern Med J*. 2018;48(9):1056–1063. doi:10.1111/imj.2018.48.issue-9
22. Ardevol A, Ibanez-Sanz G, Profitos J, et al. Survival of patients with cirrhosis and acute peptic ulcer bleeding compared with variceal bleeding using current first-line therapies. *Hepatology*. 2018;67(4):1458–1471. doi:10.1002/hep.v67.4
23. Jimenez-Rosale R, Valverde-Lopez F, Vadillo-Calles F, et al. Inhospital and delayed mortality after upper gastrointestinal bleeding: an analysis of risk factors. *Scand J Gastroenterol*. 2018;53(6):714–720. doi:10.1080/00365521.2018.1454509
24. Ramos GP, Binder M, Hampel P, et al. Outcomes of endoscopic intervention for overt GI bleeding in severe thrombocytopenia. *Gastrointest Endosc*. 2018;88(1):55–61. doi:10.1016/j.gie.2018.01.028
25. Madhwani R, Hanif FM, Ul Haque MM, et al. Noninvasive clinical predictors of portal hypertensive gastropathy in patients with liver cirrhosis. *J Transpl Int Med*. 2017;5(3):169–173. doi:10.1515/jtim2017-0025