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ORIGINAL ARTICLE

The Prevalence of Thrombocytopenia in Chronic Liver Disease Due to Hepatitis B and C Infection

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ABSTRACT

Background: *Chronic liver disease (CLD) is a major global health challenge, with hepatitis B virus (HBV) and hepatitis C virus (HCV) being the predominant causes. Thrombocytopenia is a frequent hematological complication of CLD, reflecting disease severity and influencing management. However, data regarding its prevalence in Bangladeshi patients remain limited.* ***Objective:*** *This study aimed to determine the prevalence of thrombocytopenia among patients with CLD due to HBV and HCV infection and to evaluate its distribution by age, sex, and disease severity.* ***Methods & Materials:*** *This cross-sectional observational study was conducted at the Department of Medicine of Dhaka Medical College Hospital and Anwar Khan Modern Medical College Hospital from June to November 2011. One hundred patients with CLD secondary to HBV or HCV infection, diagnosed using clinical, serological, and imaging criteria were enrolled. Demographic information, platelet count, and Child-Pugh classification were also recorded. Data were analyzed using SPSS version 16.* ***Results:*** *Among the 100 patients with CLD, 79 had HBV-related and 21 had HCV-related diseases. The mean age was 42.3 ± 11.4 years, with a male predominance. Thrombocytopenia (<150×10^9/L) was observed in 28 patients with HBV (35.4%) and nine with HCV (42.9%). Its prevalence increases significantly with disease severity, occurring in almost all Child-Pugh Class C patients.* ***Conclusion:*** *Thrombocytopenia is a common finding in HBV- and HCV-related CLD, particularly in advanced disease, underscoring its clinical and prognostic significance. Regular platelet monitoring should be integrated into patient management to improve risk assessment and the treatment outcomes.*

Keywords: *Chronic liver disease, Hepatitis B, Hepatitis C, Thrombocytopenia.*

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INTRODUCTION

Chronic liver disease (CLD) is a major cause of morbidity and mortality in the world, as a result of its progression to cirrhosis and hepatocellular carcinoma. Acute and chronic hepatitis B virus (HBV) and hepatitis C virus (HCV) infection are two of the most notable causes of CLD, and collectively they affect hundreds of millions of people across the world [1,2]. These infections place disproportionately heavy burdens in South Asia, where HBV is known to be the most frequent cause of cirrhosis, followed by HCV [3,4].

Chronic viral hepatitis follows the natural history of progressive hepatic fibrosis and may progress to cirrhosis. Together with structural and functional degradation of the liver, patients likely develop hematological abnormalities, one of which, thrombocytopenia, is especially frequent. Such a complication, besides being an indicator of a high stage of

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disease, also makes the clinical management of CLD complex [5]. Several processes are involved in thrombocytopenia related to chronic hepatitis: hypersplenism as a result of portal hypertension, failure of thrombopoietin synthesis by the diseased liver, bone marrow suppression and immune-mediated destruction of platelets [6].

Thrombocytopenia is highly prevalent in CLD patients and is greatly subject to study population, disease stage, and etiology. This is demonstrated by a systematic review, which showed that, based on a prevalence rate of HCV-related cirrhosis and chronic liver disease, it was about 24%. However, the prevalence rate rose considerably in cirrhotic or decompensated conditions [7]. Less prominent and more scarcely researched is a stronger association of HBV infection and thrombocytopenia in advanced fibrosis or cirrhosis [8]. Both causes of the virus etiologies thus constitute significant sources of the burden of platelet anomalies in the affected groups.

Thrombocytopenia is clinically and prognostically important in addition to its hematological presentation. The presence of low platelet counts is often used as a surrogate indicator of portal hypertension and progressive fibrosis, which clinicians use to determine the severity of the disease condition without invasive testing [9]. In addition, the occurrence of thrombocytopenia complicates all diagnostic and treatment approaches. Specifically, patients who have low platelets are more susceptible to bleeding during liver biopsies or endoscopies [10]. In the same way, antiviral agents, especially interferon-based regimens, once used to treat HCV, were frequently restricted by hematologic side-effects in thrombocytopenia patients requiring dose reduction or discontinuation of treatment [11].

There is still insufficient local evidence in Bangladesh to characterize the magnitude of thrombocytopenia in CLD patients, as the prevalence of HBV and HCV is very high there. Although the international studies can deliver precious information, the differences in epidemiology of the viruses, demographics of patients, and the availability of care point out the necessity of data that fit the specific context. The prevalence and the pattern of thrombocytopenia in Bangladeshi HBV- and HCV-associated CLD patients would be of special interest to clinical decision-making, risk stratification, and the structuring of the treatment plans.

This study was therefore designed to determine the prevalence of thrombocytopenia in CLD patients due to HBV and HCV infection in Bangladesh. By assessing platelet counts across different age groups, sexes, and Child-Pugh classes, the study provides valuable evidence to inform both clinical practice and future research in hepatology.

OBJECTIVE

The objective of the study was to determine the prevalence of thrombocytopenia among patients with CLD due to HBV and HCV infection and to evaluate its distribution by age, sex, and disease severity.

METHODS & MATERIALS

This cross-sectional observational study conducted at the Department of Medicine, Dhaka Medical College Hospital and Anwar Khan Modern Medical College, Dhaka, Bangladesh, from June 2011 to November 2011. A total of 100 patients with chronic liver disease due to Hepatitis B and Hepatitis C infection for more than 6 months were included in this study based on the selection criteria.

Inclusion criteria:

- All diagnosed patients of chronic liver disease.
- Serological evidence by presence of HBsAg and Anti-HCV.
- Patients and/or legally accepted guardians, who have given consent for the study.

Exclusion criteria:

- Patients of chronic liver diseases due to other causes (e.g. Alcohol, drugs)
- Evidence of any acute viral infection or acute febrile illness (e.g. Dengue)
- History of any blood diseases. (e.g. ITP, Leukemia, Aplastic anemia)
- History of taking any cytotoxic/myelotoxic drugs.
- Patients who are unwilling to take part in the study.

Data collection procedure: Patients admitted to the Department of Medicine of DMCH and Anwar Khan Modern Medical College Hospital with features of CLD due to hepatitis B or hepatitis C infection were initially seen by duty doctors in the corresponding department. The researcher was then informed via mobile phone. The researcher then evaluated the patients. Detailed history and clinical examination were performed with special attention to the hepatobiliary system and any clinical features of thrombocytopenia. After screening, if the patient was thought to be likely due to CLD, they were screened out, while the inclusion criteria were followed for enrollment. After enrollment, the patients or attendants were interviewed by the researcher using a structured case record form after obtaining written informed consent. Blood sample was taken for complete blood count with peripheral blood film, viral serology for HBsAg and Anti-HCV, serum bilirubin, serum alanine aminotransferase (S.ALT), serum albumin, prothrombin time (PT), USG of whole abdomen, endoscopy of upper GIT. All patients were assessed using the Child-Pugh classification. Patients aged <40 years constituted Group A aged >40 years were labelled as Group B.

Data Processing and Analysis: All collected questionnaire were checked very carefully to identify any error in the data. Using the statical package for social sciences (SPSS) version 16, data were processed.

Ethical Implications: Ethical approval for the study was obtained from the Ethical Review Committee of Dhaka Medical

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College. All patients were provided with detailed information regarding the objectives, procedures, potential risks, and benefits of the study through verbal explanation and printed handouts. Informed written consent was obtained before enrollment, following adequate counselling. Participants were assured of confidentiality, and their right to withdraw from the study at any stage without consequences was respected.

RESULTS

This study evaluated the prevalence of thrombocytopenia in patients with chronic liver disease (CLD) secondary to hepatitis B virus (HBV) and hepatitis C virus (HCV) infections. A total of 100 patients were included, of whom 79 had HBV-related CLD and 21 had HCV-related CLD. The results are presented in two tables and three figures, focusing on demographic characteristics, disease severity, and prevalence of thrombocytopenia.

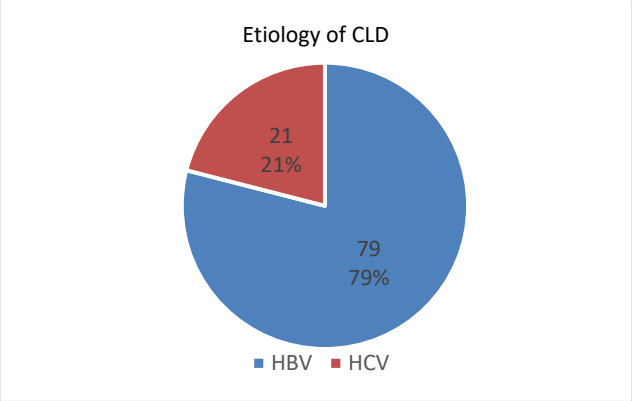

Figure – 1: Etiological distribution of chronic liver disease patients (*n*=100)

Figure 1 illustrates the relative proportions of HBV and HCV as causes of CLD. HBV accounted for the majority (79%) of cases, while HCV contributed 21%.

Table – I: Age and Sex Distribution of the Patients with Chronic Liver Disease (*n*=100)

Variable		Total (n=100)	HBV (n=79)	HCV (n=21)
Age	Mean ± SD (years)	42.3 ± 11.4	42.4 ± 11.4	44.9 ± 9.9
	<40 years, n (%)	64 (64.0)	52 (65.8)	12 (57.1)
	≥40 years, n (%)	36 (36.0)	27 (34.2)	9 (42.9)
Sex	Male, n (%)	72 (72.0)	65 (82.3)	7 (33.3)
	Female, n (%)	28 (28.0)	14 (17.7)	14 (66.7)

Table I presents the demographic distribution of CLD patients according to viral etiology. The mean age was 42.3 ± 11.4 years for all patients, with HBV cases averaging 42.4 ± 11.4 years and HCV cases averaging 44.9 ± 9.9 years. Among patients younger than 40 years, HBV was more frequent (65.8%) compared to HCV (57.1%). Males predominated overall (72%), particularly in HBV cases (82.3%), whereas HCV cases showed a higher female proportion (66.7%).

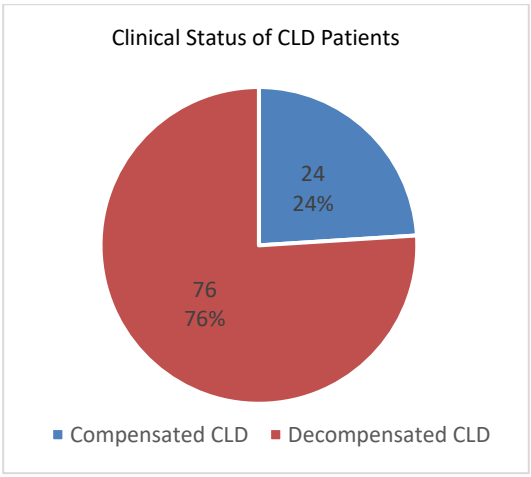

Figure – 2: Clinical status of chronic liver disease patients (compensated vs decompensated)

Figure 2 shows the distribution of clinical status among patients, stratified into compensated and decompensated CLD. The majority presented with decompensated disease, highlighting advanced clinical manifestations at the time of evaluation.

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Table – II: Child-Pugh Class and Prevalence of Thrombocytopenia by Etiology (*n*=100)

Child-Pugh Class	All CLD (n=100)	HBV (n=79) – with TCP n (%)	HCV (n=21) – with TCP n (%)
A	24	0 / 18 (0.0)	1 / 6 (16.7)
B	43	1 / 33 (3.0)	3 / 10 (30.0)
C	33	27 / 28 (96.4)	5 / 5 (100.0)
Total TCP	37	28 / 79 (35.4)	9 / 21 (42.9)

Table II describes the distribution of Child-Pugh classes and the occurrence of thrombocytopenia. Among all CLD patients, 24 were classified as Class A, 43 as Class B, and 33 as Class C. Thrombocytopenia was rare in Classes A and B but was highly prevalent in Class C. Specifically, 96.4% of HBV-related and 100% of HCV-related Class C patients exhibited thrombocytopenia. Overall, thrombocytopenia was present in 35.4% of HBV patients and 42.9% of HCV patients.

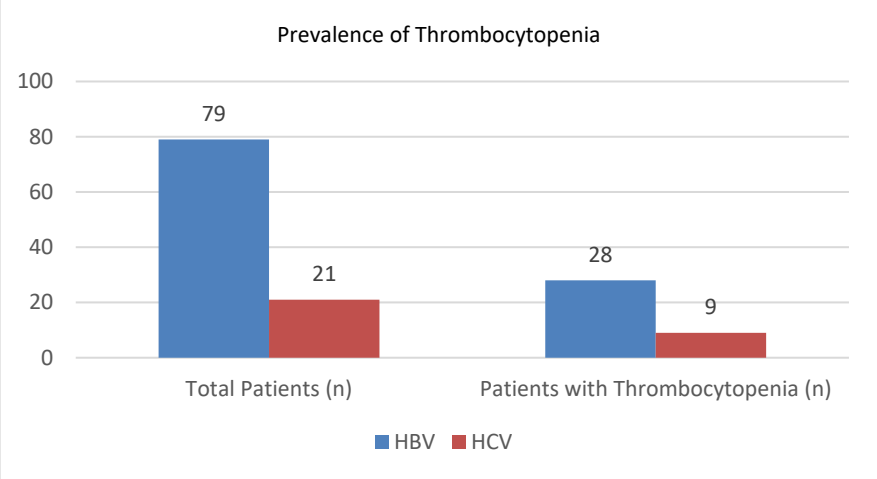

Figure – 3: Prevalence of thrombocytopenia in CLD due to HBV and HCV

Figure 3 highlights the prevalence of thrombocytopenia ($<150\times10^9$/L) among patients. Thrombocytopenia was detected in 28 of 79 HBV patients (35.4%) and 9 of 21 HCV patients (42.9%), demonstrating a considerable burden of hematological abnormalities in both viral etiologies.

DISCUSSION

This study assessed the prevalence of thrombocytopenia in patients with chronic liver disease (CLD) due to hepatitis B virus (HBV) and hepatitis C virus (HCV) infections. The findings demonstrated that thrombocytopenia was present in 35.4% of HBV-related CLD cases and 42.9% of HCV-related CLD cases. Moreover, its prevalence was significantly higher among patients with advanced disease (Child-Pugh Class C), highlighting the close association between disease severity and platelet reduction.

The findings of this study correlate with earlier studies that have also signified the association of viral hepatitis-related CLD with thrombocytopenia. A high correlation has been reported between HCV infection and decreased platelet counts in hyper additional evidence of this has also been reported in a hyperendemic community, showing that thrombocytopenia can be used as a barrier marker of disease progression [12]. Likewise, a central part of thrombocytopenia pathogenesis was also shown by Adinolfi et al., who further support our results, indicating that low platelet counts are more prevalent at advanced stages [13].

In HBV-associated CLD, we identified thrombocytopenia as present in more than one-third of the patients, and present almost universally in Child-Pugh Class C patients. These results share similar findings with those of Nwokediuko and Ibegbulam, who reported quantitative platelet alterations in HBV-related liver disease [8]. Karasu et al. also found close links between liver fibrosis and reduced peripheral platelet count in HBV and HCV patients on lending more credence to the logic behind the fact that disease progression is a cause of hematological abnormalities [14].

The pathophysiology of thrombocytopenia in CLD has a multifactorial nature. Portal hypertension-induced hypersplenism is well well-known contributor [15]. Decreased hepatic thrombopoietin production has also been a possible culprit, with Rios et al. pointing out its importance in the context of the cirrhotic patient [16]. Also, in viral hepatitis, there may be autoantibodies to platelets, altering the thrombocytopenia process. As Pereira et al. and Panzer et al. demonstrated, platelet autoantibodies are found in chronically diseased liver, especially in HCV, despite thrombocytopenia not being clinically significant [17,18].

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Our finding of increased prevalence of thrombocytopenia in HCV patients compared to that of HBV patients is similar to previous studies. A systematic review by Louie et al. observed that thrombocytopenia is quite common in chronic HCV, and it is commonly linked with advanced fibrosis and portal hypertension [7]. Furthermore, immune-mediated mechanisms might be more dominant in the HCV-associated cases since Pockros et al. reported immune thrombocytopenic purpura in chronic cases of HCV [19].

Clinical implications of these findings are significant. Thrombocytopenia is not only a complication of routine management but also threatens in the event of bleeding during a diagnostic or therapeutic intervention. Findings in Seeff et al., noting a greater incidence of complications with liver biopsies in patients with advanced CLD, are of particular interest in the Bangladesh setting, where liver biopsy is commonly used in diagnosis and staging purposes [10]. Moreover, thrombocytopenia further restricts the application of antiviral therapy, namely, interferon-based regimens, which are on-going use even in poor-resource settings. McHutchison et al. showed that low platelet counts distorted treatment compliance and preserved successful virological management, prompting the change or interference of doses [11].

Other relevant issues are the prognostic importance of thrombocytopenia. Qamar et al. also emphasized hematological changes, including thrombocytopenia, as predictive factors of disease progression and poor outcomes of the patients with cirrhosis [9]. Predominantly, Nahon et al. reported that platelet count in combination with serum albumin was a predictor of mortality in patients with HCV-related cirrhosis [20]. Collectively, these experiments support the importance of platelet count as a non-invasive disease severity parameter.

The results we have obtained are quite comparable in terms of prevalence when compared to international statistics. Such as Benhava et al. presented data on thrombocytopenia in 39 percent of patients with CLD caused by HBV or HCV, which is very close to our data [21]. Nonetheless, epidemiology and access to care often vary locally and could affect the stage of presentation. Decompensated disease resulting in late presentation is a common problem in Bangladesh, which was observed in our study, where most of the patients were at advanced stages of the Child-Pugh classes and it possibly led to the increased prevalence of thrombocytopenia.

In summary, this study confirms that thrombocytopenia is a common hematological abnormality in CLD due to HBV and HCV, particularly in advanced stages. Its prevalence and association with disease severity highlight its role as a clinical marker and predictor of outcomes. Early recognition and monitoring of platelet counts are essential in optimizing the management of these patients, especially in resource-constrained settings where invasive diagnostics are not always feasible.

LIMITATIONS OF THE STUDY

The study's cross-sectional design and hospital-based sampling may limit generalizability, as patients presenting to tertiary centers often represent more severe cases. Additionally, the relatively small sample size and lack of bone marrow studies restricted exploration of other etiologies of thrombocytopenia. Despite these limitations, the research highlights an important clinical burden.

CONCLUSION

This study demonstrates that thrombocytopenia is a frequent complication in patients with chronic liver disease (CLD) due to HBV and HCV infection, with prevalence rates of 35.4% and 42.9%, respectively. The occurrence of thrombocytopenia was closely associated with advanced disease, particularly among Child-Pugh Class C patients, highlighting its value as a marker of disease severity. These findings emphasize the need for routine platelet monitoring in CLD management, especially in resource-limited settings where invasive diagnostics may not be feasible. Early recognition can facilitate risk stratification, guide therapeutic decisions, and reduce procedure-related complications in this vulnerable patient group.

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Ethical approval: The study was approved by the Institutional Ethics Committee.

REFERENCES

1. *Dufour MC. Chronic liver disease and cirrhosis. Digestive diseases in the United States: epidemiology and impact. US Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases. Washington, DC: US Government Printing Office. 1994:615-45.*
2. *Ganem D, Prince AM. Hepatitis B virus infection—natural history and clinical consequences. New England journal of medicine. 2004 Mar 11;350(11):1118-29.*
3. *Mahtab MA, Rahman S, Khan M, Kamal M, Karim MF, Ahmed F, Hussain MF, Podder PK. Aetiology of chronic hepatitis in Bangladesh. Indian J Gastroenterol. 2007;26(Suppl 2):142.*
4. *Das DC, Al Mahtab M, Rahim MA, Malakar D, Kabir A, Rahman S. Hepatitis B virus remains the leading cause of cirrhosis of liver in Bangladesh. Bangladesh Medical Journal. 2016;45(3):164-6.*
5. *Tripodi A, Mannucci PM. Abnormalities of hemostasis in chronic liver disease: reappraisal of their clinical significance and need for clinical and laboratory research. Journal of hepatology. 2007 Apr 1;46(4):727-33.*
6. *Peck-Radosavljevic M. Thrombocytopenia in liver disease. Canadian Journal of Gastroenterology and Hepatology. 2000; 14:60D-6D.*
7. *Louie KS, Micallef JM, Pimenta JM, Forssen UM. Prevalence of thrombocytopenia among patients with chronic hepatitis C: a systematic review. Journal of viral hepatitis. 2011 Jan;18(1):1-7.*

8. *Nwokediuko SC, Ibegbulam O. Quantitative platelet abnormalities in patients with hepatitis B virus-related liver disease. Gastroenterology Research. 2009 Nov 20;2(6):344.*
9. *Qamar AA, Grace ND, Groszmann RJ, Garcia-Tsao G, Bosch J, Burroughs AK, Ripoll C, Maurer R, Planas R, Escorsell A, Garcia-Pagan JC. Portal Hypertension Collaborative Group Incidence, prevalence, and clinical significance of abnormal hematologic indices in compensated cirrhosis. Clin Gastroenterol Hepatol. 2009 Jun;7(6):689-95.*
10. *Seeff LB, Everson GT, Morgan TR, Curto TM, Lee WM, Ghany MG, Shiffman ML, Fontana RJ, Di Bisceglie AM, Bonkovsky HL, Dienstag JL. Complication rate of percutaneous liver biopsies among persons with advanced chronic liver disease in the HALT-C trial. Clinical gastroenterology and hepatology. 2010 Oct 1;8(10):877-83.*
11. *McHutchison JG, Manns M, Patel K, Poynard T, Lindsay KL, Trepo C, Dienstag J, Lee WM, Mak C, Garaud JJ, Albrecht JK. Adherence to combination therapy enhances sustained response in genotype-1–infected patients with chronic hepatitis C. Gastroenterology. 2002 Oct 1;123(4):1061-9.*
12. *Wang CS, Yao WJ, Wang ST, Chang TT, Chou P. Strong association of hepatitis C virus (HCV) infection and thrombocytopenia: implications from a survey of a community with hyperendemic HCV infection. Clinical infectious diseases. 2004 Sep 15;39(6):790-6.*
13. *Adinolfi LE, Giordano MG, Andreana A, Tripodi MF, Utili R, Cesaro G, Ragone E, Mangoni ED, Ruggiero G. Hepatic fibrosis plays a central role in the pathogenesis of thrombocytopenia in patients with chronic viral hepatitis. British journal of haematology. 2001 Jun;113(3):590-5.*
14. *Karasu Z, Tekin F, Ersoz G, Gunsar F, Batur Y, Ilter T, Akarca US. Liver fibrosis is associated with decreased peripheral platelet count in patients with chronic hepatitis B and C. Digestive diseases and sciences. 2007 Jun;52(6):1535-9.*
15. *McCormick PA, Murphy KM. Splenomegaly, hypersplenism and coagulation abnormalities in liver disease. Best Practice & Research Clinical Gastroenterology. 2000 Dec 1;14(6):1009-31.*
16. *Rios R, Sangro B, Herrero I, Quiroga J, Prieto J. The role of thrombopoietin in the thrombocytopenia of patients with liver cirrhosis. Official journal of the American College of Gastroenterology| ACG. 2005 Jun 1;100(6):1311-6.*
17. *Pereira J, Accatino L, Alfaro J, Brahm J, Hidalgo P, Mezzano D. Platelet autoantibodies in patients with chronic liver disease. American journal of hematology. 1995 Nov;50(3):173-8.*
18. *Panzer S, Seel E, Brunner M, Körmöczi GF, Schmid M, Ferenci P, Peck-Radosavljevic M. Platelet autoantibodies are common in hepatitis C infection, irrespective of the presence of thrombocytopenia. European journal of haematology. 2006 Dec;77(6):513-7.*
19. *Pockros PJ, Duchini A, McMillan R, Nyberg LM, McHutchison J, Viernes E. Immune thrombocytopenic purpura in patients with chronic hepatitis C virus infection. The American journal of gastroenterology. 2002 Aug 1;97(8):2040-5.*
20. *Nahon P, Ganne-Carrié N, Degos F, Nahon K, Paries J, Grando V, Chaffaut C, Njapoum C, Christidis C, Trinchet JC, Chevret S. Serum albumin and platelet count but not portal pressure are predictive of death in patients with Child-Pugh A hepatitis C virus-related cirrhosis. Gastroentérologie clinique et biologique. 2005 Apr 1;29(4):347-52.*
21. *Behnava B, Alavian SM, AHMADZADASL M. The prevalence of thrombocytopenia in patients with chronic hepatitis B and C.*