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COMPARATIVE STUDY OF PLATELET COUNT AND THEIR INDICES IN PATIENTS WITH LIVER DISEASE AND NORMAL POPULATION AT TERTIARY CARE CENTRE.

Pathology

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ABSTRACT

Aims : To conduct a comparative study on platelet count and platelet indices (such as mean platelet volume (MPV) and platelet distribution width (PDW)) between individuals with liver disease and a healthy population, to identify potential hematological changes associated with liver dysfunction. **Objective:** To evaluate and compare platelet count in individuals with liver disease and the normal population. To analyze and compare platelet indices (MPV, PDW) between liver disease patients and healthy individuals. To identify any significant differences in platelet count and indices that may serve as potential biomarkers for liver dysfunction To contribute to the understanding of hematological alterations in liver disease and their clinical implications for diagnosis and management. **Materials And Methods:** This observational study was conducted between January 2024 to December 2023 at tertiary care centre. This study was included total one hundred persons among them sixty patients with liver diseases and forty were normal population. Platelet count, mean platelet volume (MPV), platelet distribution width (PDW) were reviewed and compared between the groups. **Result:** Platelet indices were significantly altered in patient with liver diseases compared to normal population. MPV and PDW were significantly higher in patient with liver diseases compared to control population. Platelet count was significantly lower in patient with liver diseases compared to control population. **Conclusion:** Platelet count and Platelet indices like MPV and PDW are useful parameters for monitoring of patient with diagnosed case of liver disease and also useful prognostic markers for them.

KEYWORDS

Platelet counts, MPV, PDW, Liver diseases.

INTRODUCTION:

Liver diseases is sometimes severe systemic disorder that can be life threatening. Various liver diseases includes alcohol-related liver cirrhosis (ALC), nonalcoholic fatty liver disease (NAFLD), chronic liver diseases, liver fibrosis and liver cirrhosis. Alcohol-related liver disease (ALD) and nonalcoholic fatty liver disease (NAFLD) belong to the most common hepatic pathologies with a global burden. Liver cirrhosis (LC), being a final stage of diverse chronic liver diseases (CLDs), constitutes a severe systemic condition with a broad range of life-threatening complications [1]. Its manifestation usually remains asymptomatic until the decomposition of the disease, when a fibrotic cascade in hepatocytes cannot be reversed [1]. From a clinical point of view, an early diagnosis of fibrotic rebuilding within the liver is of crucial importance, because it improves the outcome of patients diagnosed with CLDs. [2-5]

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension and end stage liver disease.[6]

Hematological indices (HI) are common in cirrhosis with a prevalence ranging from 6% to 77% in various studies.[7] Platelets are non nucleated cell fragments derived from megakaryocytes, most often present in the bone marrow. Studies show that platelets produced from megakaryocytes present in the microvasculature is governed by circulatory forces.[8] Thrombocytopenia was the most common and earliest HI abnormality to develop in cirrhosis.[7] MPV is the measure of average size of platelets in circulation, and PDW is an index reflecting the heterogeneity of platelets. Platelet activation leads to changes in platelet shape with increase in platelet size and anisocytosis leading to an increase in MPV and PDW. The etiopathogenesis of platelet abnormalities in cirrhosis include portal hypertension-induced splenic sequestration, alterations in thrombopoietin, bone marrow suppression mediated by toxins, consumptive coagulopathy due to low-grade disseminated intravascular coagulation, acquired intravascular coagulation, fibrinolysis and Increased blood loss.[7] Apart from their important role in hemostasis, it has become evident that platelets also are active players involved in inflammation and immunity. Direct interaction between platelets and bacteria leading to platelet activation has been reported in several studies in vitro and in vivo, recently reviewed by Fitzgerald et al.[10] Studies show that platelet indices are significantly altered in sepsis and septic shock due to increase in cytokines, endothelial damage, and bone marrow `suppression. Platelet count was decreased in sepsis and decreased platelet counts parallel the severity of infection. Higher MPV and increased PDW have been found in sepsis.

Alcohol-related liver cirrhosis (ALC) and NAFLD emerge as an important global burden, and a precise noninvasive assessment of the liver structure in their course is of crucial importance.

The most accurate solution would be an invention of noninvasive parameters, obtained from the blood. Even though indirect and direct indices of liver fibrosis were found to be reliable and practical laboratory tools, new noninvasive markers in this field of hepatology are of crucial interest [6]. Routinely obtained platelet (PLT) parameters (mean platelet volume (MPV), platelet distribution width (PDW), are potential indicators of liver fibrosis.

Other researchers found a relationship between MPV and both: steatosis and liver fibrosis in patients diagnosed with HBV [7]. Of note, in another investigation, a decrease in MPV value within the HBV population of patients during the antiviral treatment was shown to be correlated with a regression of liver fibrosis [7].

MATERIALS AND METHODS:

A retrospective cohort study was conducted between January 2024 to December 2023 at tertiary care centre and study group includes patient data from medical records were examined retrospectively.

Study Group: Patients diagnosed with liver diseases based on a combination of clinical, radiologic and biochemical criteria. Among total 60 diagnosed liver disease patient there is 35(58%) Alcohol-related liver disease (ALD), 07(12%), nonalcoholic fatty liver disease (NAFLD), 10(17%) liver cirrhosis, 05(8%) liver failure and 03(5%) viral hepatitis.

Control Group; Age and sex matched patients were selected as controls. A total of 40 controls were selected for comparative study. Selection criteria for the control group were:

(1) No evidence for cirrhosis, active infection, systemic inflammatory response syndrome (SIRS) criteria;
(2) Normal ESR and C-reactive protein (CRP) level, leukocyte count in laboratory examination.
(3) Not on antiplatelet drugs or NSAIDS
(4) No DM, metabolic syndrome, CAD, CKD or stroke

The first complete blood counts (CBCs) performed after admission

was reviewed and platelet indices including platelet count, mean platelet volume and platelet distribution width were obtained. The CBCs were performed using a Mindray BC-6000. The normal range of PLT, MPV, PDW and PCT were 150–410×109/L, 7.2–11.7 fl and 9–17% respectively.

RESULT:

Baseline characteristics of the two groups are shown in Table 1. The age and sex variables are comparable in the two groups. Alcohol was the predominant etiology.

Table 1: Patient Characteristic Of The Two Groups.

	Liver diseases (n= 60)	Normal population (n=40)
Age (Average)	51	42
Sex	Male: 50 (83%) Female: 10 (17%)	Male: 20 (50%) Female: 20 (50%)
Etiology	Alcohol-related liver disease(ALD) :35(58%) Nonalcoholic fatty liver disease (NAFLD): 07(12%), liver cirrhosis: 10(17%), liver failure: 05(8%) viral hepatitis: 03(5%)	

The mean platelet count is significantly lower in liver diseases compared to normal population.

The mean MPV was significantly higher in liver diseases compared to normal population.

The mean MDW was significantly higher in liver diseases compared to normal population.

Table 2: Platelet Indices Of The Two Groups (Mean).

	Liver diseases (n= 60)	**Normal population (n=40)**
Platelet count (x1000)	75	250
MPV (fl)	13	8
PDW (%)	18.5	11

DISCUSSION:

Thrombocytopenia is the most common and first hematologic index abnormality to develop in cirrhosis.[7] Changes in platelet parameters accompany the progression of various forms of liver disease. This explains the use of platelet count as an indirect marker in some of the noninvasive assessments of hepatic fibrosis.[11] With availability of newer high performance design automated blood cell analyzers, platelet indices are also being estimated with better standardization and thus have greater clinical utility. Most important parameters among them are mean platelet volume (MPV) and platelet distribution width (PDW). We studied platelet indices in Different cause of liver diseases.

Enhanced breakdown of PLTs due to hypersplenism together with increased release of interleukin-6 shortens PLT life cycle. In the consequence, the production of PLTs by the bone marrow rises, promoting the release of larger, reticulated PLTs into the bloodstream. Theoretically, aforementioned disturbances should be reflected by an increase in MPV, PCT, and PDW. According to available literature, higher level of MPV might be even perceived as a prognostic factor of advanced fibrosis in primary biliary cholangitis patients, marker of hepatocellular carcinoma, and a parameter predisposing to the transformation of simple steatosis into steatohepatitis in the course of NAFLD. The elevation in MPV among patients with liver failure has been also suggested as an inflammatory marker due to the consumption of large active PLTs in the course of the disease.

Multiple studies have been identified significant difference between platelet count, MPV and PDW between patient with liver diseases and normal population. Mean platelet count in liver diseases and normal population (75×109/L versus 250×109/L) in our study was correlated with study by Payal Mukker et al[6] (75×109/L versus 292×109/L).

MPV in in liver diseases and normal population (13 fl versus 8fl) was correlated with study by Payal Mukker et al[6] (9.12 fl versus 7.70fl). The platelets originate by fragmentation of larger precursor cells called megakaryocytes and thereafter interact with monocyte-macrophage system in spleen. In cirrhosis endotoxemia due to gut bacterial translocation, oxidative stress, hyperkinetic circulation, antiplatelet antibody formation, splenic sequestration all influence platelet activity. PDW in liver diseases and normal population (18.5% versus 11%) was was correlated with study by Payal Mukker et al[6] (18.23% versus 16.5%).

PDW is a more specific marker of platelet activation, since it does not increase during simple platelet swelling. Van Cott and coworkers also observed a decreased effect of storage time on PDW.[12] According to Luzzatto et al[13] increased proportion of giant platelets and platelet distribution width are better indicators of altered platelet homeostasis than mean platelet volume in liver cirrhosis.

Despite design advances, there are technical limitations to generation of platelet indices by automated hematology analyzers and these include imprecision in the identification of very small and very large platelets based on internal thresholds in the instrument software, variation in results among different analyzers even within the reference range and so comparison between different studies may be affected

CONCLUSION:

Platelet count and Platelet indices like MPV and PDW are useful parameters for monitoring of patient with diagnosed case of liver disease and also useful prognostic markers for them.

REFERENCES:

1) Agata Michalak, Plateletcrit and Mean Platelet Volume in the Evaluation of Alcoholic Liver Cirrhosis and Nonalcoholic Fatty Liver Disease Patients Hindawi BioMed Research International Volume 2021, Article ID 8867985, 9 pages https://doi.org/10.1155/2021/8867985
2) S. Sookoian and C. J. Pirola, "Review article: shared disease mechanisms between non-alcoholic fatty liver disease and metabolic syndrome - translating knowledge from systems biology to the bedside," Alimentary Pharmacology & Therapeutics, vol. 49, no. 5, pp. 516–527, 2019.
3) I. Grgurevic, K. Podrug, I. Mikolasevic, M. Kukla, A. Madir, and E. A. Tsochatzis, "Natural history of nonalcoholic fatty liver disease: implications for clinical practice and an individualized approach," Canadian Journal of Gastroenterology & Hepatology, vol. 2020, article 9181368, 10 pages, 2020.
4) T. Gustot and R. Jalan, "Acute-on-chronic liver failure in patients with alcohol-related liver disease," Journal of Hepatology, vol. 70, no. 2, pp. 319–327, 2019.
5) C. Moreno, S. Mueller, and G. Szabo, "Non-invasive diagnosis and biomarkers in alcohol-related liver disease," Journal of Hepatology, vol. 70, no. 2, pp. 273–283, 2019.
6) Payal Mukker1, Comparative study of platelet indices in cirrhosis, cirrhosis with sepsis and normal population International Journal of Research in Medical Sciences Mukker P et al. Int J Res Med Sci. 2016 May;4(5):1423-1428 www.msjonline.org
7) Qamar AA, Grace ND. Abnormal hematological indices in cirrhosis. Can J Gastroenterology. 2009;23(6):441-5.
8) Luff SA, Papoutsakis ET. Megakaryocytic Maturation in Response to Shear Flow is mediated by the Activator Protein 1 (AP-1) Transcription Factor via Mitogen-Activated Protein Kinase (MAPK) Mechanotransduction. J Biol Chem. 2016;291(15):7831-43.
9) Shah AR, Chaudhari SN, Shah MH. Role of platelet parameters in diagnosing various clinical conditions. Nat'l J Med Res. 2013;3(2):162-5.
10) Fitzgerald JR, Foster TJ, Cox D. The interaction of bacterial pathogens with platelets. Nat. 2006;4(6):445-57.
11) Chuks S, Nwokediuko, Ibegbulam O. Quantitative platelet abnormalities in patients with hepatitis b virus-related liver disease. Gastroenterology Research. 2009;2(6):344-9.
12) Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I.Hippokratia. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. 2010;14(1):28-32.
13) Luzzato G, de Franchis G, Fabris F, Gerunda GE, Girolami A. Increased proportion of giant platelets and platelet distribution width are better indicator of altered platelet homeostasis than mean platelet volume in liver cirrhosis. Folia Hematol Int Mag Klin Morphol Blutforsch. 1988;115(5):719-26.