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ORIGINAL ARTICLE

Assessment of patients with platelet to white blood cell ratio associated with long term adverse outcomes in patients with acute deterioration of chronic liver disease presenting in Tertiary Care Hospital of Karachi, Pakistan.

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ABSTRACT… Objective: To determine the low and high values of platelet to white blood cell ratio (PWR) in patients experiencing acute deterioration of chronic liver disease (CLD) at a tertiary care hospital and assess their association with predicting long-term (28-day) adverse outcomes such as death. **Stud Design:** Cross-sectional study. **Setting:** Department of Gastroenterology, Liaquat National Hospital, Karachi, Pakistan. **Period:** April 2024 to December 2024. **Methods:** A total of 195 patients of both genders with age between 40 to 65 years, and admitted with acute deterioration of CLD were analyzed. Calculation of PWR was done within 24-h of admission according to clinical variables. Platelets counts were divided by WBCs findings to calculate the PWR. The 28-days adverse outcome (mortality) was evaluated for association with PWR level. Cox proportional hazard regression analysis was done to see association of PWR level with adverse outcome. P≤0.05 was considered as significant. **Results:** Among 195 patients, 115 (59.0%) were male. Significant associations of PWR with gender (p<0.000), hepatic encephalopathy (p<0.000), GI bleeding (p=0.010), bacterial infection (p<0.000), and survival status (p=0.019) were found. A higher probability of low PWR was observed in individuals with bacterial infections compared to those without (aOR=3.546, p=0.001). **Conclusion:** Association of 28-day outcome (death) was observed with low ratio of PWR among patients who are suffering with cirrhosis and acute decompensation.

Key words: Chronic Liver Disease, Infection, Mortality, Platelet, White Blood Cells.

INTRODUCTION

Chronic liver diseases (CLDs) defined as fibrosis and cirrhosis as a result of gradually destroying the liver parenchyma over a longer than six-month timeframe.[1] Cirrhosis disorders due non-alcoholic fatty liver disease, chronic viral hepatitis, and alcoholic liver disease, are the most prevalent forms of "chronic liver diseases (CLDs)". Globally, CLD affects around 1.5 billion people, making it the liver disease with the highest patient count and the leading cause of mortality for over 1.3 million people annually.[1-3]

Improving the prognosis of CLD requires early identification of disease changes and prompt therapy.[1] Typically the liver cirrhosis is classified in the form i.e. compensated and decompensated. The compensated cirrhosis advances asymptomatically until liver function deterioration and elevated portal pressure take place.[4] Acute liver function impairment due to direct or indirect hepatic insults occurs in patients with either recognized or undiagnosed chronic liver disease.[5] Patients with cirrhosis typically have thrombocytopenia as a result of complex circumstances. The severity of liver illness is directly correlated with the level of thrombocytopenia.[6]

Research on hematological indicators for chronic liver disease prognosis has recently increased.[7,8] Specifically, in several medical contexts, the ratio of "platelet-to-white blood cell (PWR)" is becoming more significant.[16] The systemic inflammatory

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response's hematologic measure is known as the ratio of platelet to white blood cell, or PWR. PWR derived from a full blood count, can be easily and affordably obtained. PWR is a trustworthy indication of infection.[5] Compared to the MELD score, the PWR is simpler and easier to compute because it is based on just two indicators.[9]

Based on white blood cell counts measured in certain studies, PWR, an affordable yet potent hematologic biomarker, has been shown to be related with the prognosis of patients with disease like liver failure, cirrhosis with acute decompensation associated with the chronic viral hepatitis, cirrhosis with intrahepatic cholangiocarcinoma, trauma and metabolic syndrome.[10,11] PWR has been demonstrated in several investigations to be connected with the degree of systemic inflammation. Leukocytes activating platelets to create leukocyte–platelet aggregates raises inflammation, WBC numbers, and excessive platelet consumption, which lowers PWR.[7] Some researchers have pointed out 13.5 as median of PWR among CLD patients.[12,13] Patients of studies having PWR >12.1 was comparatively younger among participants having PWR <12.1. Among study participants having low ratio of PWR were facing considerably worse overall unfavorable outcome (i.e., death) than those having higher ratio of PWR (P <0.001). The 28-days outcomes found significantly influenced by the following factors: bacterial infection, cirrhosis etiology, INR, bilirubin, albumin, Alt, ACLF, PWR level, and MELD score. Overall, 6.6% of patients passed away during the 28-day follow-up; although, 9.6% of those fatalities were recorded in low PWR, and 4.5% in high PWR. individuals with low PWR levels developed poor outcomes at a much greater rate than individuals with high ratio of PWR levels (p < 0.001) i-e a PWR ≤12.1 had a greater risk of poor outcomes compared to those with a PWR >12.1. One predictive factor for unfavorable 28-day outcomes was a decreased PWR level.[14]

Low platelet counts and noticeably elevated white cell counts were seen in CLD patients. It is not investigated, therefore, PWR—a platelet and white blood cell combination—can predict CLD survival. It is conceivable that PWR serves as a biomarker for systemic inflammation and that immunological disturbance might result from a very low PWR. Despite the fact that PWR is regarded as a significant illness biomarker, not many research on the subject have been published. This study was aimed to determine the low and high values of platelet to white blood cell ratio in patients experiencing acute deterioration of chronic liver disease at a tertiary care hospital and assess their association with predicting long-term (28-day) adverse outcomes such as death.

METHODS

This cross-sectional study was conducted at Department of Gastroenterology, Liaquat National hospital and Medical College, Karachi, Pakistan during April 2024 to December 2024. The research proposal was approved by the Research and Ethics Committee of Liaquat National Hospital and Medical College (0994-2024-LNH-ERC, dated: March 29, 2024). Participants were explained about the study purpose and its associated risk and benefits, before obtaining written and informed consent from them. The sample size was calculated by taking prevalence of Low platelet to white blood cell ratrio (PWR ≤12.1)=42.5%[14], margin of error=7%, and 95% confidence level. As per calculation, a total of 195 patients of both genders with age between 40 to 65 years admitted with acute deterioration of CLD were included. A non-probability consecutive sampling was applied for sampling. Patients without any chronic liver disease, hepatocellular carcinoma, and chronic extra hepatic disease were not included.

Data were collected about patient demographics, the etiology of liver disease, clinical and laboratory variables. Any kind of GI bleeding, bacterial infection, acute liver injury, and reactivation of viral hepatitis were also recorded as precipitating events. Blood sample was drawn and sent for calculating PWR. Calculation of PWR was done within 24-h of admission according to clinical variables. Platelets counts were divided by WBCs findings to calculate the PWR. The 28-days adverse outcome i.e. death was recorded. The date of death was used to censor the Person-

years. The evaluation of association between PWR level and 28-days outcome (death) was done.

Data analysis was done using "IBM-SPSS Statistics, Version 27.0". Mean and standard deviation (SD) were calculated for quantitative variables. Frequency and percentage were computed for qualitative variables. The Cox proportional hazard regression analysis was applied. P-value ≤ 0.05 was considered as significant in all analysis.

RESULTS

The current study included 195 patients in total, of which 80 (41.0%) were female and 115 (59.0%) were male. The patients' median age was 57(52–61) years old. Among 195, 188 patients (96.4%) had ascites, 29.8% had mild ascites and 70.2% had moderate ascites. Of the 195 individuals, 50.8% had HCV, 20% had HBsAg, and 3.6% had both HBsAg and HCV. Hepatic encephalopathy affected 65.1% of patients, gastrointestinal hemorrhage affected 44.6%, and bacterial infection affected 61.5%. In our investigation, the mortality rate was 9.7%, as presented in Table-I.

As seen, 48% patients in this study had a low ratio of platelet to white blood cell and 52% had a high one. According to Table-II, the median weight, sodium, hemoglobin, total bilirubin, albumin, creatinine, child-turcotte-pugh score, INR, and MELD-Na were 68 (61-80) kg, 129(125-134), 8.6(7.8-9.6) g/dl, 3.2(2.3-4.2) g/dl, 2.3(2-2.6) g/dl, 1.3(0.7-1.8) mg//dl, 11(10-12), 25(21-28), and 1.6(1.5-1.7) respectively. Based on the ratio of platelets to white blood cells, we discovered a significant difference for age (p=0.013) and INR (p=0.044). In Table-II, clinical characteristics are compared in detail based on the ratio of white blood cells to platelets.

Table-III shows the significant associations found between the platelet to white blood cell ratio and gender (p<0.001), hepatic encephalopathy (p<0.001), GI bleeding (p=0.010), bacterial infection (p<0.001), and survival status (p=0.019).

According to multi-variate logistic regression analysis, male patients are less likely than female patients to have low PWR, (aOR=0.325, p<0.001). Furthermore, a higher probability of low PWR was observed in individuals with bacterial infections compared to those without (aOR=3.546, p=0.001). Additionally, compared to patients who are still alive, expired patients had a higher likelihood of having low PWR (aOR=2.608, p=0.101). The Table-IV presents the specific odds.

	n (%)
Gender	
Male	115 (59)
Female	80 (41)
Ascites	
Yes	188 (96.4)
No	7 (3.6)
Ascites severity	
Mild	56 (29.8)
Moderate	132 (70.2)
Anti HCV	
Positive	99 (50.8)
Negative	96 (49.2)
HBsAg	
Positive	39 (20)
Negative	156 (80)
Anti HCV and HBsAg	
Positive	7 (3.6)
Negative	64 (32.8)
At least one positive	124 (63.6)
Hepatic encephalopathy	
Yes	127 (65.1)
No	68 (34.9)
Hepatic encephalopathy grade	
Grade-I	21 (16.5)
Grade-II	70 (55.1)
Grade-III	36 (28.3)
Gastrointestinal bleeding	
Yes	87 (44.6)
No	108 (55.4)
Bacterial infection	
Yes	120 (61.5)
No	75 (38.5)
Child-turcotte-pugh grade	
Grade A	2(1)
Grade B	28 (14.4)
Grade C	165 (84.6)
Survival status	
Expired	19 (9.7)
Alive	176 (90.3)

Table-I. Descriptive statistics of study population (n=195)

Variables	Overall	Platelet to White Blood Cell Ratio Median (IQR)		P-Value
		Low	High	
Age (years)	57(52-61)	55(51-60)	58(53-62.5)	0.013*
Weight(kg)	68(61-80)	68(61-80.25)	67(61-79)	0.996
Sodium	129(125-134)	128(124.75-132)	130(124.5-135.5)	0.196
Haemoglobin (g/dl)	8.6(7.8-9.6)	8.6(7.87-9.4)	8.7(7.65-9.6)	0.787
Total bilirubin (g/dl)	3.2(2.3-4.2)	3.2(2.3-4.55)	3.2(2.3-3.9)	0.301
Albumin (g/dl)	2.3(2-2.6)	2.3(2.07-2.60)	2.3(2-2.7)	0.574
Creatinine (mg/dl)	1.3(0.7-1.8)	1.4(0.7-1.9)	1.1(0.7-1.8)	0.200
Child-turcotte-pugh score	11(10-12)	12(10-13)	11(10-12)	0.084
International normalized ratio	25(21-28)	26(21-29)	24(20-27)	0.044*
MELD-Na	1.6(1.5-1.7)	1.6(1.57-1.80)	1.6(1.5-1.7)	0.116

Table-II. Comparison of clinical parameters according to platelet to white blood cell ratio

IQR; inter-quartile range; MELD-Na; model for end-stage liver disease-sodium; Mann-Whiteney U test was applied; *Significant at 0.05 levels.

Characteristics	Platelet to White Blood Cell Ratio		P-Value
	Low (%)	High (%)	
Gender			
Male	43 (45.7)	72 (71.3)	<0.001*
Female	51 (54.3)	29 (28.7)	
Ascites			
Yes	90 (95.7)	98 (97)	0.713
No	4 (4.3)	3 (3)	
Anti HCV			
Positive	48 (51.1)	51 (50.5)	0.937
Negative	46 (48.9)	50 (49.5)	
HBsAg			
Positive	20 (21.3)	19 (18.8)	0.667
Negative	74 (78.7)	82 (81.2)	
Anti HCV and HBsAg			
Positive	4 (4.3)	3 (3)	0.873
Negative	30 (31.9)	34 (33.7)	
At least one positive	60 (63.8)	64 (63.4)	
Hepatic encephalopathy			
Yes	70 (74.5)	57 (56.4)	0.010*
No	24 (25.5)	44 (43.6)	
Hepatic encephalopathy grade (n=127)			
Grade-I	10(14.3)	11 (19.3)	0.419
Grade-II	37(52.9)	33 (57.9)	
Grade-III	23(32.9)	13 (22.8)	
GI bleeding			
Yes	33(35.1)	54 (53.5)	0.010*
No	61(64.9)	47 (46.5)	
Bacterial infection			
Yes	72(76.6)	48 (47.5)	<0.001*
No	22(23.4)	53 (52.5)	
Child-turcotte-pugh grade			
Grade A	1(1.1)	1 (1)	0.977
Grade B	14(14.9)	14 (13.9)	
Grade C	79(84)	86 (85.1)	
Survival status			
Expired	14(14.9)	5 (5)	0.019*
Alive	80(85.1)	96 (95)	

Table-III. Association of Platelet to white blood cell ratio with demographic and clinical characteristics

Chi-square/fisher exact test was applied; *Significant at 0.05 levels

Characteristics	Un-adjusted		Adjusted	
	OR (95% CI)	p-value	OR (95% CI)	P-Value
Male gender	0.34 (0.19-0.61)	<0.001*	0.33 (0.17-0.61)	<0.001*
Ascites	0.69 (0.15-3.16)	0.632		
Anti HCV positive	1.02 (0.58-1.79)	0.937		
HBsAg Positive	1.17 (0.58-2.35)	0.667		
Anti HCV and HBsAg Positive	1.42 (0.31-6.62)	0.653		
Atleast one positive	Ref			
Hepatic encephalopathy	2.25 (1.23-4.14)	0.009*	0.73 (0.29-1.83)	0.497
GI bleeding	0.47 (0.27-0.84)	0.010*	0.63 (0.29-1.37)	0.241
Bacterial infection	3.61 (1.95-6.70)	<0.001*	3.55 (1.63-7.71)	0.001*
Child-Turcotte-Pugh Grade A	1.09 (0.07-17.70)	0.952		
Child-Turcotte-Pugh Grade B	1.09 (0.49-2.43)	0.835		
Expired	3.36 (1.16-9.73)	0.025*	2.61 (0.83-8.21)	0.101

Table-IV. Odds ratio for low Platelet to white blood cell ratio

Ref; reference category; Binary logistic regression was applied; *Significant at 0.05 levels

DISCUSSION

In our study results showed significant associations between the platelet to white blood cell ratio (PWR) with GI bleeding, bacterial infection, and survival status. It was further revealed that a higher probability of low PWR was observed in individuals with bacterial infections compared to those without. Further, in our study association of Low values of PWR in predicting long-term adverse outcomes in patients with acute deterioration of CLD was determined and associated more likely with 28-day unfavorable outcomes (i.e. death). Regarding the significance of PWR, thrombocytopenia plays a role in patients with CLD through a number of mechanisms, including decreased hepatic thrombopoietin production, splenomegaly brought on by elevated splenic vein pressure, destruction mediated by the immune system, and destruction brought on by an inflammatory response involving tumor necrosis factor.[15,16] There is a strong correlation between the severity of liver cirrhosis and a low platelet count.[17] Blood counts, particularly for neutrophils, indicate persistent inflammation. An elevated serum neutrophil count was brought on by the endotoxemia-related systemic inflammatory response.[18] The phagocytic and bactericidal capacities of circulating neutrophils are compromised, and these characteristics are predictive of death in Liver cirrhosis.[19]

A prospective research among patients with cirrhosis who were hospitalized for infection found that cirrhotic patients with bacterial infection had a considerably higher mean WBC count than cirrhotic patients without infection. However, because of leukopenia and splenomegaly, the majority of patients had WBC levels that were within the normal range.[20] In another multicentered cohort study which was called as the “Chinese Acute-on-Chronic Liver Failure (CATCH-LIFE)” research, lower platelet counts were linked to a higher risk of 90-day unfavorable outcomes, including mortality, in patients had acute-on-CLD, and these findings are showing similar trends to what we noticed.[21] Patients with and without cirrhosis as well as those with and without ACLF were included, and the emphasis was on the platelet count level. Irrespective of cirrhosis but not ACLF, individuals with decreased platelet counts had poorer unfavorable outcomes.[21] In AD with CLD, ACLF is a more accurate predictor of both short- and long-term mortality than MELD and CPS (Child-Pugh score) scores.[4] Acute phase reactants and WBC, neutrophil, lymphocyte, and platelet counts are examples of readily available, somewhat easy-to-use, and widely used inflammatory biomarkers in clinical settings. In a range of liver disorders, the combination of these markers for example the NLR (Neutrophil-to-lymphocyte ratio), MLR (lymphocyte-to-monocyte ratio), and PLR has been investigated for predictive significance previously.[22,23]

The limitations of this study include its cross-sectional design, which does not establish causality

between PWR and long-term adverse outcomes. The reliance on 24-hour PWR measurements may not account for temporal fluctuations in the ratio during the acute phase of liver disease. Potential confounders such as underlying comorbidities and variations in treatment protocols were not fully controlled. The study's focus on mortality as the sole adverse outcome may not capture other important long-term complications in patients with acute deterioration of CLD.

CONCLUSION

This study highlights the significant relationship between the PWR and key clinical outcomes, including GI bleeding, bacterial infections, and survival status. This study exhibits that patients with bacterial infections had a higher likelihood of having a low PWR. Low PWR values were predictive of poor long-term outcomes, such as a 28-day mortality, in cases experiencing acute deterioration of CLD. These findings suggest that PWR could be a valuable biomarker for assessing the severity and prognosis of these conditions.

CONCLUSION

This study concluded that the single dose corticosteroid improves wound healing in controlled diabetic patients after oral extraction and has no associated complications. The Landry healing index score for the corticosteroid treated group was higher as compared to the control group suggesting better healing outcomes associated with single dose of corticosteroid than giving no treatment modality at all.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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2	**Rajesh Kumar:** Conception and design, proof reading, critical revisions.
3	**Shahid Karim:** Conception and design, proof reading, critical revisions.
4	**Mir Nosherwan Khan:** Data collection, data analysis, initial manuscript writing.
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