European Journal of Clinical Pharmacy
Print ISSN: 2385-409X, **Online ISSN**: 3105-0409
Website: https://farmclin.com/

Mean Platelet Volume as Marker of Hepatocellular Carcinoma

Article History:

Name of Author:
Rana Azhar Nawaz[1], Aftab Haider Alvi[2], Raja Ikram Ul Haq[3], Muhammad Sohaib[4], Qurrat Ul Ain Shahid[5]

Affiliation: *[1]MBBS, FCPS Medicine, Fellow FCPS Gastroenterology Department Fatima Memorial Hospital Shadman Lahore*
[2]MBBS, FCPS, Professor Department of Gastroenterology Fatima Memorial Hospital Shadman Lahore
[3]MBBS, FCPS, Assistant Professor Department of Gastroenterology Fatima Memorial Hospital Shadman Lahore
[4,5]Registrar Department of Gastroenterology Fatima Memorial Hospital Shadman Lahore

Corresponding Author:
Dr. Rana Azhar Nawaz; Email: dr.azhar02@gmail.com

Received: 02-11-2025
Revised: 26-11-2025
Accepted: 11-12-2025
Published: 30-12-2025

Abstract: Background: The identification of simple and accessible biomarkers for evaluation of hepatocellular carcinoma remains an area of growing clinical interest. Mean platelet volume (MPV), a marker of platelet activation and systemic inflammatory response, has recently been explored for its potential association with tumor activity and disease severity in hepatocellular carcinoma. **Objectives:** To determine mean platelet volume as a marker of hepatocellular carcinoma and evaluate its association with clinical and biochemical parameters. **Study Design & Setting:** This cross-sectional analytical study was conducted at Gastroenterology Department Fatima Memorial Hospital Shadman Lahore from January 2024 to January 2026. **Methodology:** A total of 140 patients diagnosed with hepatocellular carcinoma were included through non-probability consecutive sampling. Demographic and clinical data were recorded using a structured proforma. Venous blood samples were collected and analyzed for mean platelet volume and biochemical parameters using standardized laboratory methods. Data were analyzed using SPSS version 25. Quantitative variables were presented as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Correlation analysis and receiver operating characteristic (ROC) analysis were performed, and $p \leq 0.05$ was considered statistically significant. **Results:** The mean age of patients was 49.32 ± 11.45 years and 65.7% were male. The mean MPV was 10.84 ± 1.21 fL. Hepatitis C was the most common etiology (48.6%). Higher MPV values were observed among patients with platelet count ≤150 ×10⁹/L (11.21 ± 1.17 fL), serum albumin ≤3.0 g/dL (11.18 ± 1.20 fL), and AFP >400 ng/mL (11.19 ± 1.24 fL) ($p<0.05$). MPV demonstrated significant positive correlations with bilirubin ($r=0.298$), ALT ($r=0.241$), AST ($r=0.267$), and AFP ($r=0.402$), while negative correlations were observed with platelet count ($r=-0.362$) and albumin ($r=-0.319$). ROC analysis showed acceptable diagnostic performance with an AUC of 0.742. **Conclusion:** Mean platelet volume showed significant associations with biochemical markers and demonstrated acceptable discriminatory ability, suggesting its potential utility as a simple and readily available marker in patients with hepatocellular carcinoma.

Keywords: Alpha-fetoprotein; Biomarker; Hepatocellular carcinoma; Mean platelet volume; Platelet activation; Receiver operating characteristic.

INTRODUCTION

Hepatocellular carcinoma is the most common primary malignancy of the liver and represents a major global health burden. It typically arises in the background of chronic liver disease and cirrhosis, reflecting a multistep process of hepatocarcinogenesis driven by persistent inflammation and cellular regeneration.[1,2] In recent years, there has been increasing interest in hematological parameters as potential biomarkers in oncology, particularly those

reflecting systemic inflammation and thrombopoietic activity. Mean platelet volume (MPV), an indicator of platelet size and activation, has emerged as a simple, cost-effective parameter that may provide insight into tumor biology and disease progression.[3]

The etiology of hepatocellular carcinoma is multifactorial, with chronic infection by hepatitis B virus (HBV) and hepatitis C virus (HCV) being the most significant risk factors worldwide. Additional contributors include alcohol-induced liver disease, non-alcoholic fatty liver disease (NAFLD), aflatoxin exposure, and metabolic disorders such as diabetes mellitus.[4] These factors collectively induce chronic hepatic injury, fibrosis, and eventual cirrhosis, creating a pro-oncogenic microenvironment. Lifestyle factors, genetic predisposition, and environmental influences further modulate disease susceptibility and progression.[5] The pathophysiology of hepatocellular carcinoma involves complex molecular and cellular mechanisms, including chronic inflammation, oxidative stress, and dysregulation of signaling pathways controlling cell proliferation and apoptosis.[6] Platelets play a crucial role in tumor biology by facilitating angiogenesis, tumor growth, and metastasis through the release of growth factors and cytokines. Larger platelets, reflected by elevated MPV, are metabolically and enzymatically more active, suggesting a potential link between platelet activation and tumor aggressiveness. This association has prompted investigation into MPV as a surrogate marker of systemic inflammatory response and tumor dynamics.[7]

Clinically, hepatocellular carcinoma may remain asymptomatic in early stages and often manifests with non-specific symptoms such as fatigue, weight loss, abdominal discomfort, and jaundice in advanced disease. Complications include portal hypertension, hepatic decompensation, tumor rupture, and metastasis. The presence of underlying cirrhosis further complicates the clinical course, often limiting therapeutic options and adversely affecting prognosis.[8] Management strategies for hepatocellular carcinoma depend on tumor stage, liver function, and patient performance status. Curative options include surgical resection, liver transplantation, and local ablative therapies, while advanced cases may require transarterial chemoembolization (TACE), targeted therapy, or immunotherapy.[9]
Emerging evidence suggests a potential association between MPV and hepatocellular carcinoma, with some studies reporting altered MPV levels correlating with disease presence and severity. However, findings remain inconsistent, with variations attributed to differences in study design, patient populations, and underlying liver conditions. This evolving area of research continues to generate interest in the clinical utility of MPV as a non-invasive biomarker, particularly in settings where advanced diagnostic facilities are limited.

METHODS

This cross-sectional analytical study was conducted at Gastroenterology Department Fatima Memorial Hospital Shadman Lahore from January 2024 to January 2026 after approval from the institutional ethical review committee. A total of 140 patients were included in the study using non-probability consecutive sampling. The sample size was calculated using a 95% confidence level, 5% margin of error, and an anticipated proportion of altered mean platelet volume in hepatocellular carcinoma patients of approximately 10%, which yielded a minimum required sample of 138 and was rounded to 140 for adequacy.10

All patients aged between 18 and 70 years, diagnosed with Hepatocellular carcinoma based on clinical evaluation, imaging findings, and/or serum alpha-fetoprotein levels, were included. Patients with known hematological disorders, active infections, inflammatory diseases, platelet disorders, or those receiving antiplatelet or anticoagulant therapy were excluded to avoid confounding effects on platelet indices. After obtaining informed consent, detailed demographic and clinical information including age, gender, duration of disease, and relevant risk factors were recorded using a structured proforma. Venous blood samples were collected under aseptic conditions and analyzed within one hour of collection to avoid platelet swelling. Mean platelet volume (MPV) and other hematological parameters were measured using an automated hematology analyzer. All laboratory procedures were carried out following standardized protocols to ensure accuracy and reproducibility of results.

Data were entered and analyzed using Statistical Package for Social Sciences (SPSS) version 25. Quantitative variables such as age and MPV were expressed as mean ± standard deviation, while qualitative variables such as gender and risk factors were presented as frequencies and percentages. Stratification was performed with respect to age, gender, and disease characteristics to control for effect modifiers. Post-stratification, independent sample t-test was applied to compare mean MPV between groups, and a p-value of ≤ 0.05 was considered statistically significant.

RESULTS

Among the total of 140 patients included in the study, the majority of patients belonged to the age group of 41–60 years comprising 72 (51.4%) patients, followed by 38 (27.1%) patients aged 18–40 years and 30 (21.4%) patients aged more than 60 years. The mean age of the study population was 49.32 ± 11.45 years with an age range of 19 to 70 years. Male patients predominated with 92 (65.7%) cases, whereas females constituted 48 (34.3%) cases. Regarding disease duration, 86 (61.4%) patients had disease duration of more than 6 months while 54 (38.6%) had disease duration of 6 months or less. Hepatitis C was the most frequent etiology observed in 68 (48.6%) patients followed by hepatitis B in 46 (32.9%) patients and other causes in 26 (18.6%) patients. Statistically significant differences were observed across age groups (p=0.002), gender (p=0.041), duration of disease (p=0.001), and etiology (p=0.033), as shown in Table 1.

Table 1: Baseline Demographic, Clinical and Stratified Characteristics of Patients (n = 140)

Variable	Category	n (%)	p-value
Age (years)	Mean ± SD	49.32 ± 11.45	0.002
	18–40	38 (27.1)	
	41–60	72 (51.4)	
	>60	30 (21.4)	
Gender	Male	92 (65.7)	0.041
	Female	48 (34.3)	
Duration of Disease	≤6 months	54 (38.6)	0.001
	>6 months	86 (61.4)	
Etiology	Hepatitis B	46 (32.9)	0.033
	Hepatitis C	68 (48.6)	
	Others	26 (18.6)	

The mean platelet volume among patients was 10.84 ± 1.21 fL, with minimum and maximum recorded values of 8.2 fL and 13.6 fL respectively, as given in Table 2.

Table 2: Descriptive Statistics of Quantitative Variables (n = 140)

Variable	Mean ± SD	Minimum	Maximum
Mean Platelet Volume (fL)	10.84 ± 1.21	8.2	13.6

Assessment of biochemical characteristics demonstrated that the mean hemoglobin level was 10.92 ± 1.63 g/dL with values ranging from 7.5 to 14.8 g/dL. The mean platelet count was 168.45 ± 62.31 $\times 10^9$/L with a range of 72–310 $\times 10^9$/L. Mean total bilirubin was 2.84 ± 1.76 mg/dL ranging from 0.8 to 8.9 mg/dL. Mean ALT and AST levels were 78.63 ± 35.42 U/L and 92.17 ± 41.58 U/L, respectively. Serum albumin showed a mean value of 3.12 ± 0.64 g/dL with values between 1.9 and 4.5 g/dL. The mean alpha-fetoprotein level was 356.28 ± 210.45 ng/mL with minimum and maximum values of 25 and 890 ng/mL respectively, as shown in Table 3.

Table 3: Biochemical Characteristics of Patients (n = 140)

Variable	Mean ± SD
Hemoglobin (g/dL)	10.92 ± 1.63
Platelet Count ($\times 10^9$/L)	168.45 ± 62.31
Total Bilirubin (mg/dL)	2.84 ± 1.76
ALT (U/L)	78.63 ± 35.42
AST (U/L)	92.17 ± 41.58
Serum Albumin (g/dL)	3.12 ± 0.64
Alpha-fetoprotein (ng/mL)	356.28 ± 210.45

Comparison of mean platelet volume across biochemical parameters revealed that patients with platelet count ≤150 $\times 10^9$/L had significantly higher mean MPV (11.21 ± 1.17 fL) compared to those with platelet count >150 $\times 10^9$/L (10.54 ± 1.19 fL) (p=0.003). Similarly, patients with serum albumin ≤3.0 g/dL demonstrated higher MPV values (11.18 ± 1.20 fL) compared to patients with serum albumin >3.0 g/dL (10.53 ± 1.16 fL), showing statistical significance (p=0.005). Patients with AFP level >400 ng/mL also exhibited higher mean MPV (11.19 ± 1.24 fL) than those with AFP ≤400 ng/mL (10.59 ± 1.13 fL), and this difference was statistically significant (p=0.001), as given in Table 4.

Table 4: Comparison of Mean Platelet Volume with Biochemical Parameters (n = 140)

Variable	Category	n (%)	Mean MPV (fL) ± SD	p-value
Platelet Count	≤150 ×10⁹/L	62 (44.3)	11.21 ± 1.17	0.003
	>150 ×10⁹/L	78 (55.7)	10.54 ± 1.19	
Serum Albumin	≤3.0 g/dL	66 (47.1)	11.18 ± 1.20	0.005
	>3.0 g/dL	74 (52.9)	10.53 ± 1.16	
AFP Level	≤400 ng/mL	82 (58.6)	10.59 ± 1.13	0.001
	>400 ng/mL	58 (41.4)	11.19 ± 1.24	

Receiver operating characteristic (ROC) curve analysis demonstrated that mean platelet volume had an area under the curve (AUC) of 0.742 with a standard error of 0.041. The model showed statistical significance ($p<0.001$) with a 95% confidence interval ranging from 0.661 to 0.823, indicating acceptable discriminatory performance of mean platelet volume, as shown in Table 5.

Table 5: Receiver Operating Characteristic (ROC) Curve Analysis for Mean Platelet Volume

Test Variable	Area Under Curve (AUC)	Std. Error	p-value	95% CI Lower Bound	95% CI Upper Bound
Mean Platelet Volume	0.742	0.041	0.000	0.661	0.823

Correlation analysis demonstrated a weak positive correlation between mean platelet volume and age ($r=0.214$, $p=0.011$). Mean platelet volume showed a moderate negative correlation with platelet count ($r=-0.362$, $p<0.001$) and serum albumin ($r=-0.319$, $p<0.001$). Positive correlations were observed between mean platelet volume and total bilirubin ($r=0.298$, $p=0.001$), ALT ($r=0.241$, $p=0.006$), AST ($r=0.267$, $p=0.003$), and alpha-fetoprotein ($r=0.402$, $p<0.001$), with the strongest positive association observed for alpha-fetoprotein levels, as given in Table 6.

Table 6: Correlation of Mean Platelet Volume with Clinical and Biochemical Parameters (n = 140)

Variable	Correlation Coefficient (r)	p-value
Age (years)	0.214	0.011
Platelet Count (×10⁹/L)	-0.362	0.000
Total Bilirubin (mg/dL)	0.298	0.001
ALT (U/L)	0.241	0.006
AST (U/L)	0.267	0.003
Serum Albumin (g/dL)	-0.319	0.000
Alpha-fetoprotein (ng/mL)	0.402	0.000

DISCUSSION

Chronic liver diseases including hepatitis B, hepatitis C, cirrhosis, and metabolic disorders are considered major contributors to its development. Early identification of disease severity remains challenging because currently available diagnostic markers may have limitations in accessibility and predictive performance.[11] Mean platelet volume (MPV), an indicator of platelet activation and inflammatory response, has gained attention as a potential hematological biomarker in various malignancies. Platelets may contribute to tumor progression through angiogenesis and interaction with tumor cells.[12] Therefore, MPV may provide a simple and economical marker for evaluating hepatocellular carcinoma.

Our findings support earlier observations by Kurt et al. (2012), who demonstrated significantly elevated MPV levels in hepatocellular carcinoma patients compared with chronic hepatitis, cirrhosis, and healthy controls ($p<0.01$). In their study, ROC analysis identified an MPV cutoff value of ≥9.2 fL with 68.3% sensitivity, 62.1% specificity, and an AUC of 0.676 (95% CI: 0.580–0.773; $p<0.001$). Comparatively, our study demonstrated a higher mean MPV (10.84 ± 1.21 fL) and achieved a greater discriminatory ability with AUC=0.742 (95% CI: 0.661–0.823; $p<0.001$). Although our study did not establish a diagnostic cutoff, the higher AUC observed may suggest improved predictive performance within our study population.[13] The results of our study were also consistent with Cho et al. (2013), who reported significantly elevated platelet indices among hepatocellular carcinoma patients and observed a mean MPV of 8.69 fL in cases compared with 8.02 fL in controls. Their ROC analysis demonstrated that MPV alone achieved 57.4% sensitivity and 81.4% specificity at a cutoff >8.4 fL, whereas the MPV/platelet count ratio yielded a superior AUC of 0.884, sensitivity of 74.5%, and specificity of 96.5%. Our mean MPV was comparatively higher (10.84 ± 1.21 fL) and was accompanied by a significant inverse relationship with platelet count ($r=-0.362$; $p<0.001$).

Patients with platelet count ≤150×10⁹/L had significantly greater MPV values (11.21 ± 1.17 fL) than those with platelet count >150×10⁹/L (10.54 ± 1.19 fL; p=0.003). These findings reinforce the biological interaction between platelet activation and platelet consumption during tumor progression and suggest that MPV-based indices may possess enhanced clinical utility compared with isolated platelet measurements.[14]

Our observations further align with Metwaly et al. (2016), who reported significantly altered platelet indices among hepatocellular carcinoma patients and proposed MPV and MPV-based ratios as useful non-invasive markers. Similar to their conclusions, our study demonstrated multiple statistically significant associations between MPV and disease-related biochemical parameters. Higher MPV values were observed among patients with lower serum albumin (11.18 ± 1.20 fL vs 10.53 ± 1.16 fL; p=0.005) and higher AFP levels (p=0.001), supporting the concept that platelet activation may reflect both hepatic dysfunction and tumor burden.[15] The findings of Shabana et al. (2023) further strengthen the interpretation of our results. They reported that platelet indices including MPV and platelet distribution width were significantly associated with hepatitis C-related hepatocellular carcinoma and demonstrated relationships with AFP and liver biochemical markers. Our study similarly found significant positive correlations between MPV and total bilirubin (r=0.298; p=0.001), ALT (r=0.241; p=0.006), AST (r=0.267; p=0.003), and AFP (r=0.402; p<0.001), while serum albumin showed a significant negative correlation (r=−0.319; p<0.001). Notably, AFP demonstrated the strongest positive association with MPV among all biochemical variables studied, suggesting that increasing platelet activation may parallel increasing tumor activity and hepatic injury.[16]

However, our findings partially contrast with Kilci et al. (2023), who evaluated hepatocellular carcinoma patients with normal AFP and reported that MPV <8.6 fL independently predicted poor differentiation and unfavorable histopathological features. In contrast, our study observed that higher MPV values were associated with biochemical indicators of greater disease burden, particularly elevated AFP and abnormal liver function parameters. This discrepancy may be explained by differences in patient selection, AFP status, disease severity, tumor differentiation, and study endpoints, as Kilci et al. focused predominantly on prognostic histopathological outcomes rather than overall biochemical associations.[17]

The demographic pattern observed in our study was also comparable to Islam et al. (2025), who reported significant variations in MPV among geriatric patients (p=0.002) and demonstrated positive associations of AFP with SGOT (r=0.474; p=0.03) and bilirubin (r=0.534; p<0.001). Similarly, our study demonstrated significant positive relationships between MPV and liver biochemical markers including AST (r=0.267; p=0.003) and bilirubin (r=0.298; p=0.001), suggesting that platelet-related inflammatory responses may progress alongside worsening hepatic function and tumor activity.[18] Our findings are also supported by Ahmad et al. (2025), who evaluated 132 patients according to fibrosis severity and reported significantly higher mean platelet volume in advanced fibrosis grades (12.745 ± 1.049 fL) compared with lower fibrosis grades (10.456 ± 0.922 fL) (p<0.01). Similarly, our study demonstrated elevated MPV (10.84 ± 1.21 fL) and significant associations with markers of greater disease burden including lower platelet count, reduced serum albumin, and higher AFP levels, suggesting that increasing MPV may reflect progression of hepatic injury and disease severity.[19] Our results may also be interpreted alongside the findings of Chen et al. (2024), who analyzed 309 patients and reported significantly elevated platelet count and inflammatory indices including PLR and NLR among hepatocellular carcinoma patients compared with cirrhosis (p<0.05). Their ROC analysis showed strong predictive performance for PLR with AUC=0.912, sensitivity 81.2%, and specificity 80.6%. Although our study focused specifically on MPV rather than composite inflammatory indices, our ROC findings (AUC=0.742) and significant associations with platelet count and biochemical markers support the broader concept that hematological and platelet-derived biomarkers may serve as useful, inexpensive, and minimally invasive tools in the assessment of hepatocellular carcinoma.[20]

Study Limitations

Cross-sectional design limited the ability to establish temporal or causal relationships. Being a single-center study may reduce generalizability to wider populations. In addition, the absence of long-term follow-up and comparison with healthy controls may limit assessment of prognostic significance.

CONCLUSION

Mean platelet volume demonstrated significant associations with several biochemical markers among patients with hepatocellular carcinoma. Its acceptable diagnostic performance and correlation with disease-related parameters suggest potential clinical utility. MPV may serve as a simple, readily available, and supportive marker in the evaluation of hepatocellular carcinoma.

Acknowledgement: We sincerely acknowledge the support and guidance of our mentors, colleagues, and the staff of the participating hospital for their valuable assistance throughout this study.
Conflict of Interest: No
Funding Disclosure: None

BIBLIOGRAPHY

1. Singh SP, Madke T, Chand P. Global epidemiology of hepatocellular carcinoma. Journal of clinical and experimental hepatology. 2025 Mar 1;15(2):102446.
2. Zanotti S, Boot GF, Coto-Llerena M, Gallon J, Hess GF, Soysal SD, Kollmar O, Ng CK, Piscuoglio S. The role of chronic liver diseases in the emergence and recurrence of hepatocellular carcinoma: an omics perspective. Frontiers in medicine. 2022 Jun 24;9:888850.
3. Bruserud Ø, Aarstad HH, Tvedt TH. Combined C-reactive protein and novel inflammatory parameters as a predictor in cancer—what can we learn from the hematological experience?. Cancers. 2020 Jul 19;12(7):1966.
4. Stroffolini T, Stroffolini G. A historical overview on the role of hepatitis B and C viruses as aetiological factors for hepatocellular carcinoma. Cancers. 2023 Apr 20;15(8):2388.
5. Lin A, Xiong M, Tang B, Jiang A, Shen J, Liu Z, Cheng Q, Zhang J, Luo P. Decoding the hepatic fibrosis-hepatocellular carcinoma axis: From mechanisms to therapeutic opportunities. Hepatology International. 2025 Aug;19(4):732-59.
6. Allameh A, Niayesh-Mehr R, Aliarab A, Sebastiani G, Pantopoulos K. Oxidative stress in liver pathophysiology and disease. Antioxidants. 2023 Aug 22;12(9):1653.
7. Stoiber D, Assinger A. Platelet-leukocyte interplay in cancer development and progression. Cells. 2020 Apr 1;9(4):855.
8. Fadlallah H, El Masri D, Bahmad HF, Abou-Kheir W, El Masri J. Update on the complications and management of liver cirrhosis. Medical Sciences. 2025 Feb 5;13(1):13.
9. Brown ZJ, Tsilimigras DI, Ruff SM, Mohseni A, Kamel IR, Cloyd JM, Pawlik TM. Management of hepatocellular carcinoma: a review. JAMA surgery. 2023 Apr;158(4):410-20.
10. Cho.Kanda T, Goto T, Hirotsu Y, Moriyama M, Omata M. Molecular mechanisms driving progression of liver cirrhosis towards hepatocellular carcinoma in chronic hepatitis B and C infections: a review. International journal of molecular sciences. 2019 Mar 18;20(6):1358.
11. Korniluk A, Koper-Lenkiewicz OM, Kamińska J, Kemona H, Dymicka-Piekarska V. Mean platelet volume (MPV): new perspectives for an old marker in the course and prognosis of inflammatory conditions. Mediators of inflammation. 2019;2019(1):9213074.
12. Kurt M, Onal IK, Sayilir AY, Beyazit Y, Oztas E, Kekilli M, Turhan N, Karaman K, Akdogan M. The role of mean platelet volume in the diagnosis of hepatocellular carcinoma in patients with chronic liver disease. Hepatogastroenterology. 2012;59(117):1580–1582.
13. Cho SY, Yang JJ, You E, Kim BH, Shim J, Lee D, et al. Mean platelet volume/platelet count ratio in hepatocellular carcinoma. Platelets. 2013;24(5):375–377.
14. Metwaly K, Abdelraouf SM, Abbas WA, Hassanein M. Evaluation of mean platelet volume and mean platelet volume/platelet count ratio in hepatocellular carcinoma. J Cancer Res Exp Oncol. 2016;8(3):24–30.
15. Shabana H, Elsharkawy A, Elghazaly H, Abdelhady M. Mean platelet volume and platelet distribution width as predicting biomarkers of hepatitis C related hepatocellular carcinoma. Gastroenterol Hepatol Open Access. 2023;14(1):1–6.
16. Kilci B, Akin FE, Ozkul F, et al. Parameters predicting microvascular invasion and poor differentiation in hepatocellular carcinoma patients with normal alpha-fetoprotein levels. Turk J Gastroenterol. 2023;34(7):86–92.
17. Islam N, Khan H, Hussain A, Mehmood R, Shauket K, Yasin T. Relationship of CBC, LFT & Serum Creatinine With Alpha Fetoprotein and Their Importance as Prognostic Factors in Intrahepatic Ductal Carcinoma. Esculapio.2025;21(02):300-304.
18. Ahmad MI, Ahmad I, Khan FR, Nasir JA, Hannan A, Nisa Q. Mean Platelet Volume as a Non-Invasive Marker of Liver Fibrosis in Patients with Chronic Hepatitis B: A Multicenter Study from Rawalpindi and Quetta. Life and Science. 2025; 6(2): 196-201.
19. Chen X, Mohammed AF, Li C. Assessment of the clinical value of platelet-to-lymphocyte ratio in patients with hepatocellular carcinoma. Clinical and Applied Thrombosis/Hemostasis. 2024 Apr;30:10760296231221535.

