European Journal of Therapeutics
pISSN: 2564-7784
eISSN: 2564-7040

Eur J Ther. 2023;29(4):838-845.
https://doi.org/10.58600/eurjther1832

Original Research

Predictive Effects of Platelet Indices in Cirrhotic Patients with or without Portal Vein Thrombosis

Filiz Araz[1], Barış Soydaş[1]

[1] Department of Gastroenterology, Faculty of Medicine, Baskent University Adana Dr. Turgut Noyan Training and Research Hospital, Adana, Turkey.

Received: 2023-08-31 / **Accepted**: 2023-11-06 / **Published Online**: 2023-11-07

Correspondence
Filiz ARAZ, MD
Address: Baskent Üniversitesi Adana Uygulama ve Araştırma Merkezi, Dadaloğlu mah 2591.sk No: 4/A 01250 Yüreğir/ Adana, Turkey
E mail: filizaraz2@hotmail.com

ABSTRACT

Objective: Portal vein thrombosis (PVT) is a common finding in liver cirrhosis. Besides low portal blood flow, thrombophilia, bacterial translocation and endotoxemia, platelets which are considered as important source of prothrombotic agents may play a role in thrombotic events in cirrhosis. Large platelets have been reported to have numerous granules that result in greater thrombotic and proinflammatory activity. We aimed to define the role of platelet indices in PVT among cirrhotic patients.

Method: Cirrhotic patients admitted to Gastroenterology Clinic and having a dynamic radiological examination were assessed retrospectively. Demographic and laboratory findings were recorded including platelet distribution width (PDW) and mean platelet volume (MPV). Severity of cirrhosis was assessed with MELD (Model for End Stage Liver Disease) and Child-Pugh-Turcotte (CPT) scores

Results: Study included 255 patients. Mean age was 60.6±10.2 years. 41.6% of patients were female. 50 (19.6%) patients had PVT. Patients with PVT did not differed from those without PVT in age, gender and presence of diabetes mellitus. Median platelet count was lower in patients with PVT (100 (22-370) vs 79.5 (22-573), p: 0.033). Mean MPV and PDW levels were similar between PVT and non-PVT groups (p >0.05). Although median MELD scores did not differ between groups, median CPT scores were significantly higher in PVT compared to non-PVT group (p:0.027).

Conclusion: Cirrhotic patients with PVT had more prominent thrombocytopenia, but similar MPV and PDW levels compared to those without PVT.

Keywords: Platelet indices; cirrhosis; portal vein thrombosis.

INTRODUCTION

Blockage of main portal vein (PV) or its tributaries by thrombus is named as portal vein thrombosis (PVT). It is a well-known complication of cirrhosis. PVT could also complicate many conditions such as myeloproliferative diseases, malignancies and infection [1]. Cirrhotic patients have different incidence and prevalence rates reported for PVT associated with different target population and disease severity [2]. PVT prevalence in cirrhosis varies between 1% and 25% [3]. 3-year and 5-year incidence of PVT for cirrhotic patients have been reported as 7.6% and 10.7%, respectively [3,4]. Low portal blood flow, inherited or acquired thrombophilia, bacterial translocation and endotoxemia may have a role in occurrence of PVT in cirrhosis [1,5].

Platelets are important source for prothrombotic agents [6]. Platelet activation is initial step of thrombosis. Upon activation, adhesion and aggregation of platelets, secretion of platelet granules and enhancement of thrombin generation take place. With activation, platelets become larger, aggregate, synthesize and produce active mediators [7,8]. Platelets secrete a variety of substances including ADP, histamine, serotonin, fibronectin, fibrinogen, thrombospondin, thromboxane A2, platelet-derived growth factor, von Villebrand factor and so on, from their granules [9]. Large platelets include more granules that result in greater thrombotic and proinflammatory activity [7,10]. The mean platelet volume (MPV) defines the average volume of circulating platelets. Platelet distribution width (PDW) is an index of variation between individual platelet size Thus, MPV and PDW are suggested as indicators of platelet function and activation [11].

Association between MPV and arterial thrombosis has been reported in cases of ischemic cerebrovascular or cardiac events [12-15]. Increased MPV was also reported to be associated with pulmonary thromboembolism [16]. The relationship between PVT and platelet indices remains uncertain. We aimed to define the role of platelet indices in cirrhotic patients with non-malignant PVT.

MATERIALS AND METHODS

Patients admitted to Gastroenterology Clinic in Başkent University Adana Dr. Turgut Noyan Training and Research Hospital with liver cirrhosis from 2015 to 2020 were analyzed retrospectively. This study was approved by the Institutional Review Board of Başkent University (KA 22/258-31.05.2022-E-94603339-604.01.02-131417).

Cirrhosis was diagnosed by histological examination or by radiology when compatible radiological findings, varices and/or splenomegaly were present. MELD (Model for End Stage Liver Disease) and Child-Pugh-Turcotte (CPT) scores were applied to assess the severity of cirrhosis. Cirrhotic patients having a dynamic examination of computed tomography or magnetic resonance imaging were included into the study. Patients with known malignancy, sepsis, thrombophilia, chronic inflammatory diseases, renal insufficiency, acute coronary or cerebrovascular event, on anticoagulation or anti-aggregation due to any condition and age lower than 18 years were excluded.

Main Points;

- It has been found that cirrhotic patients with PVT had higher median CPT score and lower median platelet count, but similar mean MPV and PDW levels compared to those without PVT.

Age, gender, comorbid diseases, etiology of cirrhosis, presence of portal vein thrombosis in dynamic radiological examination were recorded for all patients, Laboratory tests comprising liver function tests and complete blood count, including white blood cell (WBC), PDW, MPV and platelet count were recorded.

Statistical Analysis

Statistical analysis was performed using SPSS software (version 23.0, IBM Corp., Armonk, NY, USA). Normally distributed continuous variables were described as means ± standard deviations. Non-normally distributed continuous variables were described as medians. Comparison of categoric variables were performed using $\chi 2$ test and Ficher test. Comparison of groups for normally distributed data was made by using Student's t-test. The Mann–Whitney U test was used for non-normally distributed data. For Spearman's coefficient, $p < 0.05$ was accepted as significant.

RESULTS

Cirrhotic patients admitted to our clinic between 2015 and 2020 were screened. Initial screening revealed 450 patients. A total of 195 patient were excluded, due to sepsis (n:30), thrombophilia (n:5), acute coronary or cerebrovascular event (n:20), malignancy (n:105) and not having appropriate radiologic imaging of CT or MRI (n:45). Final analysis included 255 patients. Mean age of patients was 60.6±10.2 (20-83) years. 106 (41.6%) of all patients were female. Demographic characteristics and laboratory parameters of patients were shown in table 1.

Hepatitis B and hepatitis C were present in 60 (23.5%) and 42 (16.5%) patients, respectively. Alcoholic liver disease, PBC/Autoimmune hepatitis and NASH were present in 12, 12 and 20 patients, respectively. Hemochromatosis and hepatitis B/D were present in the remaining 6 patients. No underlying etiology was defined in 103 (40.4 %) patients.

Fifty (19.6%) patients with cirrhosis had PVT. Patients with PVT did not differ from those without PVT in regards to age, gender and presence of diabetes mellitus (Table 2).

Table 1. Demographic characteristics and laboratory parameters of patients

Characteristic		**All patients (n: 255)**
Male /Female (n)		149/106
Age *		60.6±10.2
Etiology n (%)	HBV	60 (23.5)
	HCV	42 (16.5)
	Alcohol	12 (4.7)
	Cryptogenic	103 (40.4)
	NASH	20 (7.8)
	Autoimmune /PBC	12 (4.7)
	Others	6 (2.4)
PVT n (%)	Present	50 (19.6)
	Absent	205 (80.4)
Radiology n (%)	CT	214 (83.9)
	MRI	41 (16.1)
DM n (%)		99 (38.8)
Hb*		11.8±2.3
WBC*		5406.6±2391.1
Neutrophil*		62.4±10.2
Lymphocyte*		24.1±8.9
Platelet*		109.8±65.1
MPV*		9.4±4.8
PDW*		39.1±17.1
Albumin*		3.37±0.7
Total bilirubin*		2.39±2.9
INR*		1.36±0.3
AST*		48.2±43.4
ALT*		45.5±63.5
Creatinine *		0.86±0.5
Na*		137.4±4.6
MELD*		13.1±5.97
CHILD*		7.32±2.1
Ascites n (%)	No	116 (45.5)
	Low-moderate	96 (37.6)
	Tense-refractory	43 (16.9)

* Mean ± Standard Deviation

Abbreviations: **HBV**: Hepatitis B Virus, **HCV**: Hepatitis C Virus, **NASH**: Non-alcoholic steatohepatitis, **PBC**: Primary biliary cholangitis, **PVT:** Portal vein thrombosis, **CT**: Computed tomography, **MRI**: Magnetic resonance imaging, **DM**: Diabetes Mellitus, **Hb**: Hemoglobin, **WBC**: White blood cell count, **MPV:** Mean platelet volume, **PDW**: Platelet distribution width, **INR**: International normalization ratio, **AST**: Aspartate aminotransferase, **ALT**: Alanine aminotransferase, **Na**: Sodium, **CPT**: Child-Pugh Turcotte score, **MELD**: The Model for End Stage Liver Disease

Table 2. Characteristics and laboratory parameters of patients according to presence of portal vein thrombosis

		No PVT (n:50)	PVT present (n:205)	p
Female n (%)		89 (43.4)	17 (34.0)	0.226
Age**		62 (25-83)	61,5 (20-83)	0.841
DM n (%)		77 (37.6)	22 (44.0)	0.402
Ascites n (%)	No	102 (49.8)	14 (28.0)	**0.022**
	Low-moderate	71 (34.6)	25 (50.0)	
	Tense-refractory	32 (15.6)	11 (22.0)	
Hb*		12.1±2.2	10.5±2.2	**<0.001**
WBC**		5050 (1580-16400)	4495 (1010-10100)	0.233
Neutrophil*		61.6±10.4	65.6±10.4	**0.022**
Lymphocyte**		24.6 (2.9-53.9)	20.8 (6.88-36.4)	**0.002**
Platelet**		100 (22-370)	79.5 (22-573)	**0.033**
MPV**		8.94 (6.09-19.2)	8.6 (6.07-81.0)	0.134
PDW**		42.5 (14.5-75.5)	46.5 (16.9-81.8)	0.259
Albumin**		3.5 (1.8-4.89)	3.09 (1.57-4.21)	**0.005**
Total bilirubin*		1.55 (0.4-23.5)	1.7 (0.46-12.5)	0.441
INR**		1.27 (1-4.18)	1.38 (1-2.63)	**0.050**
AST**		47 (13-474)	36.5 (14-175)	**0.001**
ALT**		33 (9-880)	23 (10-250)	**0.001**
Creatinine **		0.76 (0.3-5.89)	0.78 (0.49-2.92)	0.319
Na**		139 (122-145)	138 (114-144)	0.153
MELD**		11 (5-39)	12.5 (6-29)	0.121
CPT**		7 (5-15)	7 (5-16)	**0.027**

*****Mean ± Standard Deviation, ******Median (Upper Limit-Normal Limit).

Abbreviations: PVT: Portal vein thrombosis, **DM**: Diabetes Mellitus, **Hb**: Hemoglobin, **WBC**: White blood cell count, **MPV:** Mean platelet volume, **PDW**: Platelet distribution width, **INR**: International normalization ratio, **AST**: Aspartate aminotransferase, **ALT**: Alanine aminotransferase, **Na**: Sodium, **CPT**: Child-Pugh Turcotte score, **MELD**: The Model for End Stage Liver Disease

Ascites in any grade was observed more frequently in PVT group compared to non- PVT (p: 0.022). Patients with PVT had lower median platelet count (100 (22-370) vs 79.5 (22-573), p: 0.033), but mean MPV and PDW levels were similar between groups (p >0.05). Although mean WBC count was similar, mean neutrophil count was significantly higher among those with PVT (61.6±10.4 vs 65.6±10,4, p: 0.022). Mean albumin level was lower for those with PVT (p: 0.005). Although mean total bilirubin level was similar, INR was higher in those with PVT (p:0.050). Patients with PVT had higher median MELD score, but this trend was not statistically significant (p: 0.121). Median CPT score were significantly different and higher in PVT group (p:0.027).

DISCUSSION

PVT was present in 19.6% of our patients. Prevalence of PVT among patients with cirrhosis ranges between 0.6% and 22% [17]. Rate of PVT depends on both severity of cirrhosis and the method used for diagnosis. Similar with our results, Abdel-Razik et al. reported 17.9% of PVT rate in cirrhotic patients [11]. Prevalence of PVT assessed by ultrasound, was reported to be 8–25% in candidates for liver transplantation [18]. A 12-months prospective study of cirrhotic patients by Zocco et al reported a PVT incidence rate of 16% [19]. Ultrasound is sensitive method for diagnosis of PVT in cirrhosis. Ultrasound has 92% specificity and 89% sensitivity in detection of PVT [20]. Sensitivity further declines in the case of partial PVT. It is also

operator dependent and presence of ascites, gas and obesity may also affect the diagnosis. Dynamic BT that we used is a specific tool for diagnosis of PVT in cirrhosis [20,21].

Chen et al. reported age and sex as risk factors for PVT, however age and sex were similar between PVT and non-PVT patients in our study [22]. Diabetes mellitus was defined as an independent risk factor for PVT by Abdel-Razik et al [11]. Similarly, DM was reported as a risk factor for thromboembolic events for cirrhotic patients in a report by Gîrleanu et al. [23]. The rationale for association between diabetes mellitus and PVT comes from endothelial abnormalities that frequently occur among diabetic patients. Endothelial abnormalities may enhance platelet activation. In our cohort, we did not find any association between DM and PVT. We did not assess endothelial abnormalities in our study.

Patients with PVT had lower platelet levels in our study. Two studies by Ushitora et al. and Abdel-Razik et al. reported similar results [11,24]. Platelets are considered as an important source of prothrombotic agents associated with inflammatory markers [6]. Etiology of thrombocytopenia in chronic liver disease is multifactorial. Sequestration of platelets within spleen, suppression of bone marrow due to various causes, including antiviral therapy, alcohol, myelosuppressive effects of hepatitis-C (HCV) infection, or decreased activity of thrombopoietin (TPO), and platelet destruction through anti-platelet antibodies can cause or contribute to cirrhotic thrombocytopenia [25]. Gîrleanu et al. and Abdel-Razik et al. reported higher platelet indices of MPV and PDW for cirrhotic patients with PVT [11,23,26]. Larger platelets and increased platelet indices of MPV and PDW were related to pre-thrombotic state of patients with cirrhosis, despite thrombocytopenia observed in such patients [23]. Other studies reported that platelets with large size have more granules that result in greater thrombotic and proinflammatory activity [7,10]. Compared to controls, PDW, but not MPV, was higher and platelet count was lower in splanchnic vein thrombosis in non-cirrhotic patients in a study by Sharma et al. [27]. However, we did not find any significant difference for MPV and PDW between two groups in our study. Swelling of platelets in edetic acid (EDTA) may be an explanation for this result. Platelets swell in EDTA in a time dependent manner making the measurements with standard anticoagulant of hematology potentially unreliable [28]. Data is more stable when measurement is applied within two hours of blood sampling [29]. This situation should be taken into account during interpretation of MPV and PDW results.

PVT is reported to be a complication of decompensated liver disease and is associated with late-stage liver cirrhosis [20]. White blood cell count, albumin, bilirubin and prothrombin time were not risk factors for PVT in cirrhotic patients according to findings of Abdel-Razik et al [11]. An association between low albumin level and presence of PVT in cirrhotic patients was reported by another study [23]. In our results low mean albumin and higher INR levels were present in PVT group. Median CPT score was also significantly higher in PVT group, however an incrementally higher MELD score in PVT group did not reach statistical significance compared to patients who did not have PVT.

Mean neutrophil count was significantly higher among those with PVT in our study. Cirrhotic patients with portal hypertension are known to have high levels of bacterial translocation and increased serum levels of inflammatory mediators, such as tumor necrosis factor-α, interleukin-6 and interleukin-8 [30]. Platelets indirectly respond to pathogen invasion through interactions with leukocytes and the endothelium. Following antigen recognition, platelets often become activated. Activated platelets can directly kill pathogens, or facilitate pathogen clearance by activating macrophages and neutrophils, promoting neutrophil extracellular traps (NETs) formation, forming platelet aggregates and microthrombi. Platelet activation may also exacerbate inflammation and promote endothelial damage and thrombosis [31]. A recent study reported neutrophil to lymphocyte ratio (NLR) ≥3.14 was associated with 2.89 times increased risk of PVT [32]. In a study by Nery F et al. as markers of systemic inflammation, IL-6 and lymphopenia were predictive for PVT independently of markers of portal hypertension for cirrhotic patients [33].

A meta-analysis by Lin et al. concluded higher MPV to be associated with the presence of PVT. This meta-analysis of 7 studies which were all case control studies included high degree of heterogeneity. Predictive effect of MPV on PVT was less obvious in the presence of larger diameter of portal vein. Meta-analysis concluded that real effect of MPV was dubious and more studies were needed to explain a possible relationship with PVT [29].

Limitation

Our study is retrospective and data does not include portal vein measurements and could not document the relationship between portal vein diameter and platelet indices in the case of PVT. Also,

time period from sample collection to laboratory measurement is not exactly known which precludes the conclusion of EDTA effect precisely.

CONCLUSION

Cirrhotic patients with PVT had more prominent thrombocytopenia, but similar MPV and PDW levels compared to those without PVT. Further studies are needed to elucidate the effect of these parameters for PVT in cirrhosis.

Acknowledgments: NA

Funding: NA

Informed Consent: NA

Conflict of interest: The author declares no conflict of interest.

Ethical Approval: This study was approved by the Institutional Review Board of Başkent University (KA 22/258-31.05.2022-E-94603339-604.01.02-131417).

Author Contributions: Conception: FA- Design: FA- Supervision: BS -Materials: FA, BS- Data Collection and/or Processing: FA,BS - Analysis and/or Interpretation: FA - Literature: FA, BS- Review: BS- Writing: FA- Critical Review: FA, BS

REFERENCES

[1] Intagliata NM, Caldwell SH, Armando Tripodi A (2019) Diagnosis, Development, and Treatment of Portal Vein Thrombosis in Patients with and without Cirrhosis. Gastroenterology 156:1582-1599. https://doi.org/10.1053/j.gastro.2019.01.265

[2] Pan J, Wang L, Gao F, An Y, Yin Y, Guo X, Nery FG, Yoshida EM, Qi X (2022) Epidemiology of portal vein thrombosis in liver cirrhosis: A systematic review and meta-analysis. Eur J Intern Med 104:21-32. https://doi.org/10.1016/j.ejim.2022.05.032

[3] Ferreira CN, Marinho RT, Cortez-Pinto H, Ferreira P, Dias MS, Vasconcelos M, Alexandrino P, Serejo HF, Pedro AJ, Goncalves A, Palma S, Leite I, Reis D, Damiao F, Valente A, Brita LX, Baldaia C, Fatela N, Ramalho F, Velosa J (2019) Incidence, predictive factors and clinical significance of development of portal vein thrombosis in cirrhosis: a prospective study. Liver Int 39(8):1459-67. https://doi.org/10.1111/liv.14121

4. Nery F, Chevret S, Condat B, de Raucourt E, Boudaoud L, Rautou P-E, Plessier A, Roulot D, Chaffaut C, Bourcier V, Trinchet J-C, Valla D-C (2015) Causes and consequences of portal vein thrombosis in 1,243 patients with cirrhosis: results of a longitudinal study. Hepatology 61(2):660-7. https://doi.org/10.1002/hep.27546

[5] Harding DJ, Perera, Chen F, Olliff S, Tripathi D (2015). Portal vein thrombosis in cirrhosis: Controversies and latest developments. World J Gastroenterol 14,21(22):6769-84. https://dx.doi.org/10.3748/wjg.v21.i22.6769

[6] Abdel-Razik A, Eldars W, Rizk E (2014) Platelet indices and inflammatory markers as diagnostic predictors for ascitic fluid infection. Eur J Gastroenterol Hepatol 26:1342-1347. https://doi.org/10.1097/MEG.0000000000000202

[7] Bath PM, Butterworth RJ (1996) Platelet size: measurement, physiology and vascular disease. Blood Coagul Fibrinolysis 7(2):157-161. https://doi.org/10.1097/00001721-199603000-00011

[8] Colkesen Y, Muderrisoglu H (2012) The role of mean platelet volume in predicting thrombotic events. Clin Chem Lab 50(4):631-634. https://doi.org/10.1515/cclm.2011.806

[9] Kannan M, Ahmad F, Saxena R (2019) Platelet activation markers in evaluation of thrombotic risk factors in various clinical settings. Blood Reviews 37;100583. https://doi.org/10.1016/j.blre.2019.05.007

[10] Braekkan SK, Mathiesen EB, Njolstad I, Wilsgaard T, Stormer J, Hansen JB (2010). Mean platelet volume is a risk factor for venous thromboembolism: the Tromsø study. Journal of Thrombosis and Haemostasis. 8(1):157-162. https://doi.org/10.1111/j.1538-7836.2009.03498.x

[11] Abdel-Razik A, Mousa N, Elhelaly R, Tawfik A (2015) De-novo portal vein thrombosis in liver cirrhosis: risk factors and correlation with the Model for End-stage Liver Disease scoring system. Eur J Gastroenterol Hepatol 27(5):585-92. https://doi.org/10.1097/MEG.0000000000000325.

[12] Greisenegger S, Endler G, Hsieh K, Tentschert S, Mannhalter C, Lalouschek W (2004) Is elevated mean platelet volume

associated with a worse outcome in patients with acute ischemic cerebrovascular events? Stroke. 35 (7): 1688-1691. https://doi.org/10.1161/01.STR.0000130512.81212.a2

[13] Chang HA, Hwang HS, Park HK, Chun MY, Sung JY (2010) The Role of Mean Platelet Volume as a Predicting Factor of Asymptomatic Coronary Artery Disease. Korean J Fam Med. 31;600-606. https://doi.org/10.4082/kjfm.2010.31.8.600

[14] Klovaite J, Benn M, Yazdanyar S, Nordestgaard B.G (2011) High platelet volume and increased risk of myocardial infarction: 39 531 participants from the general population. J Thromb Haemost. 9;49-56. https://doi.org/10.1111/j.1538-7836.2010.04110.x

[15] Sansanayudh N, Anothaisintawee T, Muntham D, McEvoy M, Attia J, Thakkinstian A (2014) Mean platelet volume and coronary artery disease: a systematic review and meta-analysis Int J Cardiol 20;175(3):433-40. https://doi.org/10.1016/j.ijcard.2014.06.028

[16] Lin W, Wu Y, Lu X, Hu Y (2021) Association between mean platelet volume and pulmonary embolism: a systematic review and meta-analysis. Aging (Albany NY) 13(13):17253-17273. https://doi.org/10.18632/aging.203205

[17] Gîrleanu I, Trifan A, Stanciu C, Sfarti C (2018). Portal vein thrombosis in cirrhotic patients - it is always the small pieces that make the big Picture. World J Gastroenterol. 24(39):4419-4427. https://doi.org/10.3748/wjg.v24.i39.4419

[18] Fimognari FL, Violi F (2008) Portal vein thrombosis in liver cirrhosis. Intern Emerg Med. 3:213-218. https://doi.org/10.1007/s11739-008-0128-0

[19] Zocco MA, Di Stasio E, De Cristofaro R, Novi M, Ainora ME, Ponziani F, Riccardi L, Lancellotti S, Santoliquido A, Flore R, Pompili M, Rapaccini GL, Tondi P, Gasbarrini GB, Landolfi R, Gasbarrini A (2009). Thrombotic risk factors in patients with liver cirrhosis: correlation with MELD scoring system and portal vein thrombosis development. J Hepatol 51:682-689. https://doi.org/10.1016/j.jhep.2009.03.013

[20] Senzolo M, Garcia-Tsao G, García-Pagán JC (2021) Current knowledge and management of portal vein thrombosis in cirrhosis. J Hepatol. 75(2):442-453. https://doi.org/10.1016/j.jhep.2021.04.029

[21] Mori H, Hayashi K, Uetani M, Matsuoka Y, Iwao M, Maeda H (1987) High-attenuation recent thrombus of the portal vein: CT demonstration and clinical significance. Radiology 163:353-356. https://doi.org/10.1148/radiology.163.2.3031725

[22] Chen H, Trilok G, Wang F, Qi X, Xiao J, Yang C (2014) A single hospital study on portal vein thrombosis in cirrhotic patients – clinical characteristics & risk factors. Indian J Med Res 139:260-266.

[23] Gîrleanu I, Trifan A, Cojocariu C, Sîngeap AM, Sfarti C, Stanciu C (2012) The risk of thrombotic events in patients with liver cirrhosis. Rev Med Chir Soc Med Nat Iasi 116:991-996. https://doi.org/10.3390/diagnostics13061160

[24] Ushitora Y, Tashiro H, Takahashi S, Amano H, Oshita A, Kobayashi T, Chayama K, Ohdan H (2011) Splenectomy in chronic hepatic disorders: portal vein thrombosis and improvement of liver function. Dig Surg 28:9-14. https://doi.org/10.1159/000321886.

[25] Afdhal N, McHutchison J, Brown R, Jacobson I, Manns M, Poordad F, Weksler B, Esteban R (2008) Thrombocytopenia associated with chronic liver disease. J Hepatol 48:1000-1007. https://doi.org/10.1016/j.jhep.2008.03.009

[26] Gîrleanu I, Trifan A, Cojocariu C, Dimache M, Sîngeap AM, Stoica O, Sfarti C, Stanciu C (2013) Platelet indices in patients with de novo portal vein thrombosis and liver cirrhosis. Rev Med Chir Soc Med Nat Iasi 117(3):641-7. https://doi.org/10.1016/s0168-8278(14)60694-2

[27] Sharma S, Kumar N, Bihana I, Uppal V, Ahluwalia J, Naseem S, Varma N, Rana SS (2021) Evaluation of platelet indices in patients with splanchnic vein thrombosis. Indian J Hematol Blood Transfus. 37(4):593-599. https://doi.org/10.1007/s12288-021-01400-5

[28] Bath PM. The routine measurement of platelet size using sodium citrate alone as the anticoagulant. Thromb Haemost, 1993,70(4):687-690

[29] Lin W, Lu X, Fan FJ, Hu Y (2018) Predictive Effect of Mean Platelet Volume in Patients with Portal Vein Thrombosis: A Meta-analysis of Case-control Studies. Current Medical Science. 38(4):575-581. https://doi.org/10.1007/s11596-018-1916-z

[30] Arap JP, Martin-Mateos RM, Shah VT (2018) Gut–liver axis, cirrhosis and portal hypertension: the chicken and

the egg. Hepatology Int. 12,24-33. https://doi.org/10.1007/s12072-017-9798-x

[31] Guo L, Rondia MT (2019) The Era of Thromboinflammation: Platelets Are Dynamic Sensors and Effector Cells During Infectious Diseases. Front Immunol 13:10:2204. https://doi.org/10.3389/fimmu.2019.02204

[32] Xing Y, Tian Z, Jiang Y, Guan G, Niu Q, Sun X, Han R, Jing X (2022) A practical nomogram based on systemic inflammatory markers for predicting portal vein thrombosis in patients with liver cirrhosis. Ann Med. 54(1):302-309. https://doi.org/10.1080/07853890.2022.2028893

[33] Nery F, Carneiro P, Correia S, Macedo C, Gandara J, Lopes V, Valadares D, Ferreira S, Oliveira J, Gomes MT, Miranda HP, Rautau P-E, Valla D (2021) Systemic inflammation as a risk factor for portal vein thrombosis in cirrhosis: a prospective longitudinal study. Eur J Gastroenterol Hepatol. 1;33(1):e108-e113. https://doi.org/10.1097/MEG.0000000000001982

How to Cite;

Araz F, Soydaş B (2023) Predictive Effects of Platelet Indices in Cirrhotic Patients with or without Portal Vein Thrombosis. Eur J Ther. 29(4):838-845. https://doi.org/10.58600/eurjther1832