Role of Systemic Inflammatory Markers and Portal Vein Indices by Doppler Ultrasound in Predicting Portal Vein Thrombosis in Patients with Liver Cirrhosis

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Background and study aim: Portal vein thrombosis (PVT) is a serious complication of liver cirrhosis that aggravates portal hypertension and further impairs liver function. Systemic inflammatory markers and Doppler-derived portal vein indices are considered promising non-invasive tools for predicting PVT. This study aimed to evaluate their role in the early detection of PVT in cirrhotic patients.
Patients and Methods: A case-control study was conducted on 309 participants at Menoufia University Hospitals: 103 cirrhotic patients with PVT (Group I), 103 cirrhotic patients without PVT (Group II), and 103 healthy controls (Group III). All participants underwent detailed history taking, clinical examination, laboratory assessment of inflammatory markers as Systemic Immune-Inflammation Index (SII), Neutrophil-to-Lymphocyte Ratio (NLR), Monocyte-to-Lymphocyte Ratio (MLR), Platelet-to-Lymphocyte Ratio (PLR), Erythrocyte Sedimentation Rate (ESR), C-reactive Protein (CRP), and Ferritin, and Doppler ultrasonography for portal and splenic vein diameters and flow velocities.
Results: Group I showed significantly higher SII (291.66 ± 32.21), NLR (3.67 ± 0.73), MLR (0.48 ± 0.10), and PLR (128.03 ± 18.75) compared with Group II ($p < 0.001$). Doppler findings demonstrated markedly reduced portal vein velocity (≤17 cm/sec in 92.2%) and enlarged portal vein diameter (>17 mm in 86.4%) among PVT patients. Portal vein velocity achieved the highest diagnostic accuracy (96.8%), followed by SII (90.5%). Ferritin, ESR, and CRP showed limited predictive value (51–53.6%).
Conclusion: Systemic inflammatory markers (SII, NLR, MLR, PLR) and Doppler-based vascular indices (portal and splenic vein diameters and velocities) represent reliable non-invasive predictors of PVT in cirrhotic patients. Their combined use may improve early detection of PVT and support timely therapeutic decisions.

INTRODUCTION

The last stage regarding long-term liver illness, liver cirrhosis is distinguished by the creation of regenerative nodules, disturbance of normal liver architecture, and persistent hepatic fibrosis. It is the consequence of long-term liver damage brought on by several illnesses, including chronic hepatitis B and C, non-alcoholic steatohepatitis (NASH), infections, and excessive alcohol use. In Egypt, HCV is the most common cause of chronic liver disease and liver cirrhosis [1,2]. Patients may deteriorate from a compensated phase to a decompensated phase as the illness worsens, exhibiting symptoms such varices hemorrhage, hepatic encephalopathy, and ascites [3].

One of significant side effect of liver cirrhosis is thrombosis of the portal vein (PVT), which is the entire or partial obstruction of the portal vein, or its branches through a thrombus. It is linked to decreased survival and increased morbidity rates, and its incidence among cirrhotic patients has been shown to range from 0.6% to 26% [4].

Although PVT is frequently asymptomatic, it can cause stomach discomfort or be unintentionally discovered when imaging for problems associated to portal hypertension, including gastrointestinal hemorrhage [5].

PVT in cirrhosis has a complex etiology. It was once thought that people with cirrhosis were auto-anticoagulated because their coagulation factor production was reduced. Nevertheless, new data points to a delicate, but balanced, hemostatic condition that may lean toward thrombosis or bleeding [6]. Endothelial dysfunction, inflammation, and changes in primary, secondary, and tertiary hemostasis all contribute to hypercoagulability in end-stage liver disease [7]. Particularly important is chronic inflammation, with signs of systemic inflammation providing details regarding the prothrombotic milieu of individuals with cirrhosis [8].

Hematological indicators that show systemic inflammation and immunological activation include Neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and C-reactive protein (CRP). These measurements are easily available and reasonably priced. Due to their ability to predict worse outcomes in chronic liver illness, including the development of PVT, these markers have drawn increasing interest [9].

Doppler ultrasonography, which measures portal vein diameter, flow velocity, and direction, is still the major non-invasive technique for assessing the venous portal system. Because of its accessibility, safety, and reproducibility, it is especially helpful for the early diagnosis of PVT in high-risk cirrhotic patients [10]. In more complicated cases, the size of the thrombus and the differentiation between benign and malignant forms can be ascertained using contrast-enhanced computed tomography (CT) or magnetic resonance imaging (MRI) [11].

Given the clinical significance of PVT and the potential value of non-invasive predictive tools, this study aimed to evaluate the function of indicators of systemic inflammation and portal vein Doppler ultrasound indices in predicting portal vein thrombosis among individuals with liver cirrhosis.

PATIENTS AND METHODS

Study design and participants:

This case-control study was carried out in the Department of Tropical Medicine at Menoufia University Hospitals, in collaboration with the Radiology and Clinical Pathology Departments from October 2023 to January 2025. A total of 309 participants were included, comprising 206 patients with liver cirrhosis (103 diagnosed with portal vein thrombosis and 103 without PVT) alongside 103 healthy individuals a control group .

Calculation of sample size: Based on review of past literature, Han et al., who found that The univariate logistic regression analysis indicated that platelet-to-lymphocyte ratio (PLR) (preoperatively) (odds ratio (OR)=3.963, 95% confidence interval (CI)=2.070–7.587, p<0.000), The sample size is calculated using OpenEpi, Version 3, open source calculator—SSCohort, Fleiss, Statistical Methods for Rates and Proportions, formulas 3.18 &3.19. [12.[

All participants provided written informed consent after receiving a detailed explanation of the study's aims and procedures. Ethical approval was granted by the Research Ethics Committee of the Faculty of Medicine, Menoufia University, Egypt (IRB No: 6/2023 TROP39.(

Inclusion criteria: Adults aged 18 years or older and patients with clinically, biochemically, and radiologically confirmed liver cirrhosis were included.

Exclusion criteria: Patients with recent infections or inflammation such as acute cholangitis, cholecystitis or IBD, patients with congestive heart failure or end-stage renal disease, patients with non-cirrhotic PVT or Budd-Chiari syndrome, and those with hematological diseases and malignancies, including hepatocellular carcinoma were excluded. In addition, patients under 18 years of age or refusal to participate or incomplete data were not included.

Clinical and laboratory assessment:

Every patient with cirrhosis had a thorough clinical assessment, which included full history that focused on demographic information, concomitant conditions such diabetes mellitus or hypertension, and signs of infection, gastrointestinal bleeding, or hepatic decompensation. Body mass index, vital signs, and abdominal abnormalities such as ascites, hepatosplenomegaly, and collateral circulation indicators, a comprehensive physical

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examination was conducted. Complete blood count (CBC), coagulation profile (prothrombin time, INR, activated partial thromboplastin time, D-dimer, protein C, protein S, and antithrombin III), liver function tests (bilirubin, ALT, AST, and albumin), and renal function tests (serum urea and creatinine) were measured.

Inflammatory markers such as CRP, ESR, and serum ferritin were assessed. In addition, systemic inflammatory indices were calculated, including the NLR , PLR , MLR , and SII . Virological screening for hepatitis B surface antigen (HBsAg), anti-HCV antibodies, and anti-HIV antibodies was done using ELISA. Random blood glucose and alpha-fetoprotein (AFP) levels were also measured.

Radiological evaluation:

Using either Toshiba or GE (Logic E9) ultrasound systems with 4.5 MHz curvilinear and 12 MHz linear-array probes, all cirrhotic patients had abdomino-pelvic ultrasonography, including Doppler studies. The ultrasound measured liver size, echotexture, surface nodularity, and the presence of hepatic focal lesions; it also measured portal vein diameter and flow velocity, splenic vein diameter and velocity, spleen size, and the presence of ascites, hernias, masses, or collateral vessels. Triphasic contrast-enhanced computed tomography scans of the abdomen and pelvis were carried out using a Siemens Biograph 128-slice CT scanner, which included pre-contrast and post-contrast arterial, portal venous, and delayed phases to assess liver parenchyma, focal lesions, vascular anatomy, PVT characteristics, spleen size, and extrahepatic findings.

Endoscopic evaluation:

Upper gastrointestinal endoscopy was carried out in 161 cirrhotic patients using an Olympus LUCERA CV-260 system. The procedure was conducted under appropriate fasting and sedation protocols. The presence and grade of esophageal varices were documented, along with other findings such as portal hypertensive gastropathy, gastric varices, and mucosal lesions indicative of recent or active bleeding.

Assessment of liver disease severity :

Using the Child-Turcotte-Pugh (CTP) score and the Model for End-Stage Liver Disease (MELD) score. MELD score was determined using the following formula: MELD = (9.57 × ln [creatinine]) + (3.78 × ln [bilirubin]) + (11.20 × ln [INR]) + 6.4. These scoring systems were used to stratify patients according to the severity of hepatic dysfunction [13].

Statistical analysis:

The data was analyzed with the Statistical Package for the Social Sciences (SPSS) software, version 25.0 (IBM Corp., Armonk, NY, USA). The quantitative data was provided as mean ± standard deviation and compared among groups using either Student's t-test or analysis of variance (ANOVA), depending on the context. Categorical data were presented as frequencies and percentages, with group comparisons made using the Chi-square test. Pearson's correlation coefficient was used to assess the relationships between quantitative variables. Systemic inflammatory indicators and Doppler indices were assessed for their capacity to predict PVT using receiver operating characteristic (ROC) curve analysis. Sensitivity, specificity, PPV, NPV, and total diagnostic accuracy were all determined. A p-value of < 0.05 indicated statistical significance.

RESULTS

The study included two groups of patients with liver cirrhosis (103 with portal vein thrombosis and 103 without PVT) and a group of 103 healthy controls. The mean age was comparable across groups (Group I: 60.4 ± 9.2, Group II: 59.3 ± 7.5, Group III: 59.6 ± 8.5 years; $p = 0.612$). Males constituted 46.6%, 49.5%, and 47.6% of Groups I, II, and III, respectively. Fever, hematemesis, and melena were significantly more frequent causes of admission in Group I compared to Group II ($p = 0.048$, $p = 0.008$, and $p = 0.017$, respectively). Jaundice was significantly more prevalent in Group I ($p < 0.001$ and $p = 0.001$, respectively), while ascites was more marked in Group I with statistically significant differences between the groups ($p < 0.001$). No significant differences were noted regarding other general or local examination findings. (Table 1).

Patients in Group I had statistically significant lower platelet counts (mean: 75.7 ± 20.6 vs. $94.5 \pm 17.7 \times 10^9$/L; $p < 0.001$) than Group II. Group I had considerably greater total and direct

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bilirubin levels ($p < 0.001$), but lower albumin levels ($p < 0.001$). In Group I, coagulation measures such as PT, INR, and APTT were considerably higher ($p < 0.05$). D-dimer levels were significantly higher (664.3 ± 28.9 vs. 407.3 ± 62.1 ng/mL; $p < 0.001$), while Protein C and Protein S levels were significantly lower in Group I ($p < 0.001$). There were no statistically significant variations in other laboratory parameters. The etiology of cirrhosis, mostly HCV, was similar in both groups ($p = 0.651$). (Table 2(

Systemic inflammatory markers were significantly higher in Group I compared to Groups II and III. Group I showed elevated mean SII (291.7×10^9/L), NLR (3.67), MLR (0.48), PLR (128.0), and CRP (12.9 mg/L), all with $p < 0.001$. Ferritin and ESR were also higher in Groups I and II than in Group III ($p < 0.001$). On ultrasound and CT, 39 patients had main PVT, 36 patients had right PVT and 28 patients had left PVT. Cavernous transformation of the portal vein was present in 32% (US) and 38% (CT) of Group I. Group I also had significantly larger PV and SV diameters and lower velocities ($p < 0.001$), more frequent moderate-to-marked ascites (95.1%), and a higher rate of abdominal wall collaterals (24.3% vs. 9.7%, $p = 0.005$). (Table 3(

Among systemic inflammatory markers, PLR >97 had the highest accuracy (92.3%) in predicting PVT, followed by NLR >2.99 (92.6%) and SII >270 (90.5%). MLR >0.4 had an accuracy of 90.1%, while ferritin ≤370 ng/ml, ESR ≤17 mm/h, and CRP ≤10 mg/L had significantly lower accuracies (52.9%, 53.6%, and 51%, respectively). Doppler ultrasonography indices showed the highest accuracy for main portal vein velocity ≤17 cm/sec (96.8%), followed by PV diameter >17 mm (94.7%), SV diameter >10 mm (94.3%), and SV velocity ≤15 cm/sec (92.7%). (Table 4(

In group I, statistically significant positive correlations were found between SII and NLR (r=0.253, p=0.010), MLR (r=0.230, p=0.019), and PLR (r=0.202, p=0.041). Additionally, MLR correlated significantly with PLR (r=0.211, p=0.033). Among Doppler parameters, NLR showed a significant negative correlation with splenic vein (SV) velocity (r=–0.256, p=0.009). However, no significant correlations were observed between the inflammatory markers and portal vein diameter, main PV velocity, or MELD and Child scores (p>0.05). (Table 5(

There were statistically difference between studied patient groups as Regards Endoscopic findings and therapeutic maneuvers, where , frequency of upper endoscopy was statistically higher in group I (p value < 0.001), but , there were no statistically significant difference as regards Endoscopic finding and therapeutic maneuvers (OV band ligation , GFV injection sclerotherapy, ABC for PHG , mild PHG and OV non risky). (Table 6).

Table 1: **Socio-Demographic and clinical data among studied groups**

		Groups						**ANOVA**	
		Group I		**Group II**		**Group III**		**F**	**P-value**
Socio-Demographic data									
Age (Years)	Range	38 - 76		42 - 73		41 - 76		0.491	0.612
	Mean ±SD	60.427 ± 9.203		59.311 ± 7.473		59.592 ± 8.459			
Chi-Square		**N**	**%**	**N**	**%**	**N**	**%**	**X^2**	**P-value**
Sex	Male	48	46.60	51	49.51	49	47.57	0.182	0.913
	Female	55	53.40	52	50.49	54	52.43		
Etiology of hospitalization									
DCL		9	8.74	10	9.71	-	-	0.058	0.810
Fever		20	19.42	10	9.71	-	-	3.902	0.048*
Abdominal pain		67	65.05	55	53.40	--	-	2.895	0.089
Hematemesis		37	35.92	20	19.42	-	-	7.010	0.008*
Melena		29	28.16	15	14.56	-	-	5.664	0.017*

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General examination									
Flapping tremors		9	8.74	10	9.71			0.058	0.810
Jaundice		87	84.47	63	61.17			14.126	<0.001*
Pallor		65	63.11	55	53.40			1.996	0.158
Local examination									
Liver palpation	Palpable	0	0.00	0	0.00			-	-
	Not Palpable	103	100.00	103	100.00				
Spleen palpation	Palpable	83	80.58	86	83.50			0.296	0.586
	Not Palpable	20	19.42	17	16.50				
Ascites	No	4	3.88	12	11.65			32.634	<0.001*
	Mild	8	7.77	35	33.98				
	Moderate	44	42.72	36	34.95				
	Marked	47	45.63	20	19.42				

DCL: Disturbed conscious level

Table2: laboratory investigations between studied patients groups

		Groups		T-Test	
		Group I	**Group II**	**T**	**P-value**
Complete blood count, renal function tests and electrolytes					
HB (g/L) N:(M: 13 -18) N:(F: 12-16)	Range	5.5 - 12	6.5 - 11.8	-1.271	0.205
	Mean ±SD	8.664 ± 1.374	8.910 ± 1.400		
TLC ($x10^9$ /L) N:(adults:4.5-11)	Range	1.5 - 11	1.8 - 10	-0.641	0.522
	Mean ±SD	2.776 ± 1.660	2.917 ± 1.512		
Platelets ($x10^9$ /L) (N:150-400)	Range	45 - 102	70 - 125	-7.059	<0.001*
	Mean ±SD	75.650 ± 20.552	94.524 ± 17.720		
Liver function tests, alpha feto protein					
Total bilirubin (mg/dl) (N: 0.1-1.2)	Range	0.5 - 9.1	1.5 - 6	13.421	<0.001*
	Mean ±SD	5.769 ± 1.808	2.683 ± 1.476		
Direct bilirubin (mg/dl) (N: less than 0.3)	Range	0.9 - 5.4	0.6 - 3.5	15.129	<0.001*
	Mean ±SD	3.631 ± 1.148	1.477 ± 0.878		
Albumin (gm/dl) (N: 3.5-5.2)	Range	0.9 - 2.9	1.1 - 3.1	-5.588	<0.001*
	Mean ±SD	2.025 ± 0.570	2.421 ± 0.439		
Alpha feta protein (ng/ml) (N: less than 10)	Range	5 - 25	3 - 25	-0.848	0.398
	Mean ±SD	12.534 ± 5.058	13.165 ± 5.614		
Coagulation profile					
PT (Sec) (N: 11-13.5)	Range	11 - 27	10 - 21	6.968	<0.001*
	Mean ±SD	20.233 ± 4.388	16.641 ± 2.849		
INR (N:0.8-1.2)	Range	1.2 - 2.6	1.1 - 2.2	2.905	0.004*
	Mean ±SD	1.787 ± 0.369	1.660 ± 0.247		
APTT(sec) (N:25-35)	Range	40.5 - 65.5	35 - 57	2.568	0.011*
	Mean ±SD	48.976 ± 6.115	47.012 ± 4.779		
Anti-thrombin III activity (%) (N:80-120)	Range	28 - 59	33 - 62	-1.320	0.188
	Mean ±SD	48.087 ± 7.667	49.456 ± 7.205		
D Dimmer (ng /ml) (N: less thn 500)	Range	600 - 720	314 - 520	38.085	<0.001*
	Mean ±SD	664.272 ± 28.922	407.262 ± 62.081		
Protein C (ug/ml) (N:3.5-5.5)	Range	0.4 - 3.5	4 - 5.2	- 24.754	<0.001*
	Mean ±SD	2.805 ± 0.682	4.699 ± 0.371		
Protein S (ug/ml) (N:	Range	3.5 - 15.2	16.5 - 21.9	-	<0.001*

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20-30)				35.804	

Hb : hemoglobin, TLC: total leucocytic count, PT : prothromnin time , INR : international normalized ratio , APTT : activated partial thromboplastin time

Table 3: systemic Inflammatory markers and imaging data among studied groups

		Groups			ANOVA		TUKEY'S Test		
		Group I	Group II	Group III	F	P-value	I&II	I&III	II&III
systemic Inflammatory markers									
SII (10^9/L) (N:300-600)	Range	180-342	158 - 300	265 - 440	324.590	<0.001*	<0.001*	<0.001*	<0.001*
	Mean ±SD	291.660 ± 32.213	216.942 ± 32.746	354.845 ± 49.260					
NLR (N:1-3)	Range	1.93-5.34	1.58-3.95	1.87 - 3.12	182.351	<0.001*	<0.001*	<0.001*	0.016*
	Mean ±SD	3.665 ± 0.731	2.323 ± 0.491	2.533 ± 0.329					
MLR (N:0.15-0.35)	Range	0.12-0.67	0.02-0.49	0.24-0.36	172.830	<0.001*	<0.001*	<0.001*	0.003*
	Mean ±SD	0.480 ± 0.099	0.255 ± 0.120	0.298 ± 0.036					
PLR (N:100-200)	Range	81 - 165	20 - 130	75 - 108	215.027	<0.001*	<0.001*	<0.001*	0.001*
	Mean ±SD	128.029 ± 18.751	87.291 ± 16.440	95.049 ± 7.109					
Ferritin (ng/ml) N:(M:30-300) N:(F:15-200)	Range	150- 550	100 - 565	23 - 180	281.899	<0.001*	0.991	<0.001*	<0.001*
	Mean ±SD	381.563 ± 104.757	383.223 ± 115.505	116.223 ± 39.637					
ESR (mm/h) N:(M:0-20) N:(F:0-30)	Range	8 - 32	5 - 32	4 - 20	51.579	<0.001*	0.767	<0.001*	<0.001*
	Mean ±SD	18.068 ± 5.536	17.621 ± 4.816	12.204 ± 3.160					
CRP (mg/L) (N:less than 5)	Range	5 - 25	4 - 26	3 - 8	115.919	<0.001*	0.982	<0.001*	<0.001*
	Mean ±SD	12.893 ± 4.935	12.786 ± 5.282	5.058 ± 1.251					

Pelviabdominal Ultrasound findings							
US		Group I		Group II		Chi-Square or T-Test	
		N	%	N	%	X^2 or T	P-value
PV diameter (mm) (N: <13)	Range	14 - 20		13 - 18		17.531	<0.001*
	Mean ±SD	18.825 ± 1.302		15.398 ± 1.497			
Main PV velocity (cm/sec) (N:16-40)	Range	9 - 19		15 - 24		-19.020	<0.001*
	Mean ±SD	14.165 ± 2.030		19.029 ± 1.618			
PV thrombosis		103	100.00	0	0.00	206.000	<0.001*
PV cavernous transformation		33	32.04	0	0.00	39.295	<0.001*
Distribution of PVT	No	0	0.00	103	100.00	206.000	<0.001*
	Right PV	36	34.95	0	0.00		
	Left PV	28	27.18	0	0.00		
	Main PV	39	37.86	0	0.00		

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Hepatic focal lesion		13	12.62	9	8.74	0.814	0.367
SV diameter (mm) (N:5-12)	Range	9 - 14		6 - 12		17.894	<0.001*
	Mean ±SD	11.971 ± 1.279		8.777 ± 1.283			
SV velocity (cm/sec) (N:10-30)	Range	8 - 20		11 – 22		- 18.860	<0.001*
	Mean ±SD	11.728 ± 2.850		18.272 ± 2.068			
Ascites	Mild	5	4.85	47	45.63	45.793	<0.001*
	Moderate	47	45.63	30	29.13		
	Marked	51	49.51	26	25.24		
Anterior abdominal wall collaterals		25	24.27	10	9.71	7.744	0.005*
CT							
Hepatic focal lesion	No	90	87.38	94	91.26	0.963	0.915
	Hemangioma	4	3.88	3	2.91		
	Focal fatty sparing	2	1.94	1	0.97		
	Focal fatty infiltration	1	0.97	1	0.97		
	Regeneration nodule	6	5.83	4	3.88		
PV thrombosis		103	100.00	0	0.00	206.000	<0.001*
PV cavernous transformation		39	37.86	0	0.00	48.108	<0.001*
Distribution of PVT	No	0	0.00	103	100.00	206.000	<0.001*
	Right PV	36	34.95	0	0.00		
	Left PV	28	27.18	0	0.00		
	Main PV	39	37.86	0	0.00		
SV diameter (mm) (N:5-12)	Range	10 - 14		6 - 11		26.504	<0.001*
	Mean ±SD	12.398 ± 0.911		8.583 ± 1.142			
Ascites	Mild	5	4.85	47	45.63	45.793	<0.001*
	Moderate	47	45.63	30	29.13		
	Marked	51	49.51	26	25.24		

SII : systemic immune inflammatory index , NLR : neutrophil lymphocyte ratio , MLR : monocyte lymphocyte ratio , PLR : playlet lymphocyte ratio, ESR : erythrocyte sedmentation rate , CRP : C reactive protein, PV : portal vein , SV : splenic vein , PVT: portal vein thrombosis, CT: computed tomography, US: ultrasound

Table 4: Accuracy of systemic Inflammatory markers and PV indices in prediction of PVT

	Cutoff	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**Accuracy**
systemic Inflammatory markers						
SII	>270	89.32	91.26	91.1	89.5	90.5%
NLR	>2.99	86.41	90.29	89.9	86.9	92.6%
MLR	>0.4	88.35	86.41	86.7	88.1	90.1%
PLR	>97	91.26	88.35	88.7	91.0	92.3%
Ferritin (ng/ml)	≤370	44.66	63.11	54.8	53.3	52.9%

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ESR (mm/h)	≤17	57.28	55.34	56.2	56.4	53.6%
CRP (mg/L)	≤10	41.75	66.99	55.8	53.5	51%
US						
PV diameter (mm)	>17	86.41	89.32	89.0	86.8	94.7%
Main PV velocity (cm/sec)	≤17	92.23	90.29	90.5	92.1	96.8%
SV diameter (mm)	>10	87.38	88.35	88.2	87.5	94.3%
SV velocity (cm/sec)	≤15	89.32	91.26	91.1	89.5	92.7%

PPV: positive predictive value, NPV: negative predictive value

Table 5: correlation between systemic inflammatory markers , portal vein indices ,MELD and Child score

Pearson Correlation								
Group I	**SII**		**NLR**		**MLR**		**PLR**	
	R	**P-value**	**R**	**P-value**	**r**	**P-value**	**r**	**P-value**
NLR	0.253	0.010*						
MLR	0.230	0.019*	0.049	0.623				
PLR	0.202	0.041*	0.038	0.700	0.211	0.033*		
US PV diameter (mm)	0.011	0.910	0.146	0.140	0.065	0.512	0.181	0.068
US Main PV velocity (cm/sec)	-0.056	0.574	-0.132	0.182	-٠,٠٩٢	0.354	-٠,١٢٧	0.202
US SV diameter (mm)	0.156	0.115	0.010	0.918	0.124	0.211	0.049	0.626
US SV velocity (cm/sec)	-0.023	0.816	-0.256	0.009*	-0.032	0.745	-0.086	0.390
MELD score	0.087	0.382	0.009	0.932	0.066	0.508	0.129	0.193

MELD: model for end stage liver disease

Spearman's rho								
Group I	**SII**		**NLR**		**MLR**		**PLR**	
	R	**P-value**	**r**	**P-value**	**R**	**P-value**	**r**	**P-value**
Child score	0.063	0.524	0.034	0.733	0.129	0.193	0.011	0.915

Table 6 : Upper Endoscopic findings and therapeutic maneuvers between studied patientgroups

		Groups				**Chi-Square**	
		Group I		**Group II**			
		N	**%**	**N**	**%**	**X^2**	**P-value**
Upper endoscope	**Done**	90	87.38	71	68.93	10.264	0.001*
	Not Done	13	12.62	32	31.07		
Endoscopic finding	**OV Band ligation**	50	55.56	42	59.15	0.568	0.967
	GFV injection sclerotherapy	12	13.33	8	11.27		
	ABC for PHG	18	20.00	13	18.31		
	Mild PHG	5	5.56	3	4.23		
	OV non risky	5	5.56	5	7.04		

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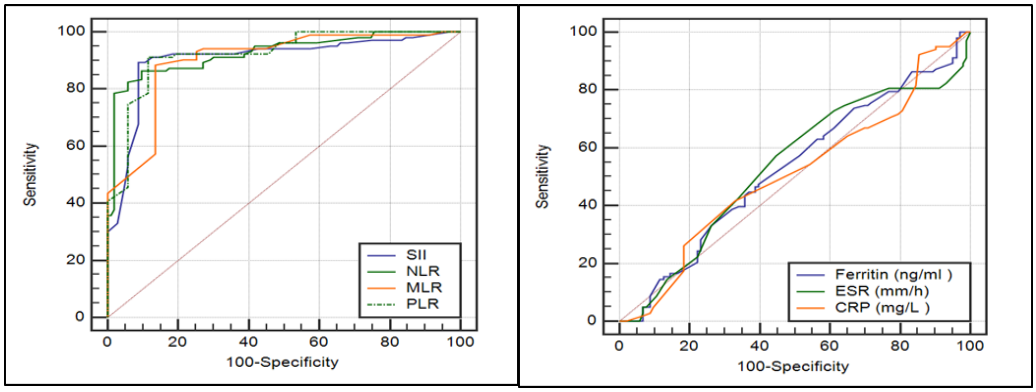

Figure (1): ROC curve of systemic inflammatory markers

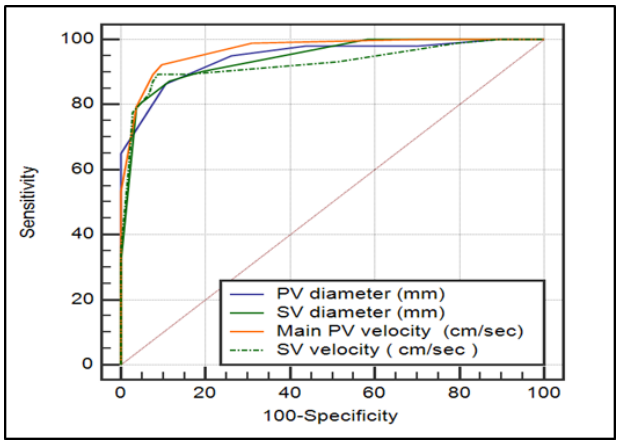

Figure (2): ROC curve of portal vein

DISCUSSION

Portal vein thrombosis occurs frequently as a consequence of cirrhosis of the liver that causes blockage of the portal vein. It worsens portal hypertension and further compromises liver function. Early detection is important, but it remains a clinical issue [4]. Indicators of systemic inflammation (such as the SII, NLR, MLR, and PLR) represent the body's inflammatory response, which influences thrombosis. These indicators are affordable and generally available, making them potentially useful tools for predicting the risk of PVT, albeit their diagnostic reliability requires further clinical confirmation [9].

Doppler ultrasonography is a non-invasive method of measuring portal and splenic vein sizes and blood flow velocities, which can provide insight into hemodynamic changes related with PVT. These assessments could lead to earlier diagnosis and better patient outcomes [10].

This case-control study included 309 participants, patients with PVT who had cirrhosis were in Group I; patients without PVT were in Group II; and healthy controls were in Group III. Inflammatory markers and portal vein characteristics were examined and compared to determine their prognostic value for PVT in individuals with cirrhosis.

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Age and gender did not differ statistically significantly among the groups under investigation. This observation is comparable with the findings of Zhang et al., who likewise found no significant demographic differences between cirrhotic patients with and without portal vein thrombosis [14]. Georgescu et al., found a greater prevalence of PVT among older male patients with cirrhosis. [15].This disparity could be attributed to variances in population characteristics, which could influence the demographic profile of PVT patients.

This study also revealed that symptoms such as fever, hematemesis and melena were substantially more prevalent in Group I compared to Group II. These findings are consistent with those of Harding., who found that gastrointestinal bleeding symptoms were more common in cirrhotic patients with PVT because of elevated portal hypertension and limited venous outflow [16]. The increased prevalence of fever may indicate an underlying systemic inflammatory response, which confirms Raadsen et al., findings that inflammation contributes to thrombosis risk [17].

Furthermore, our results showed that cirrhotic individuals with PVT had considerably greater body temperature, respiration rate and jaundice than those without. These findings are comparable with those of Boccatonda et al., who found that systemic inflammatory symptoms, such as fever and increased respiratory rate, are frequently present in patients with PVT and represent a pro-inflammatory and hypercoagulable condition [18]. Ferrusquía-Acosta et al., linked jaundice and lower limb edema to poor hepatic function and worse venous return due to thrombotic blockage [19], which is consistent with our findings.

However, Zhang et al., found no discernible differences between cirrhotic individuals with and without PVT in terms of the occurrence of edema or jaundice [14]. This inconsistency may be due to differences in liver disease stage .

Our study found that ascites and umbilical hernias were substantially more prevalent than in cirrhotic patients with PVT. These results align with the findings of Tripathi et al., who found that severe ascites was linked to a higher risk of PVT, most likely due to elevated intra-abdominal pressure and decreased portal vein flow velocity [20]. Similarly, the increased prevalence of umbilical hernias in PVT patients is in line with the results of Mustapha et al., who argued that persistent ascites increases abdominal wall tension, particularly in cirrhotic persons with limited venous return [21].

In this study, platelet counts were considerably lower in Group I than in Group II, but there were no discernible intergroup variations in metrics such hemoglobin and total leukocyte count. These results are in line with the findings of Fierro-Angulo et al., who identified thrombocytopenia as a frequent side effect in cirrhotic individuals with PVT, mostly brought on by hypersplenism and splenic sequestration linked to portal hypertension. [22].

In terms of coagulation measures, According to the current study, cirrhotic people with PVT had significantly higher D-dimer readings, prothrombin time (PT), international normalized ratio (INR), and activated partial thromboplastin time (APTT). Antithrombin III activity did not differ significantly between groups, while natural anticoagulants (proteins C and S) were considerably decreased. These results validate those of Hung et al., who related coagulation abnormalities, such as raised PT, INR, D-dimer, and reduced protein C/S, to an increased risk of thrombosis in cirrhotic people [23]. Turon et al., stressed the importance of D-dimer increase and protein C/S shortage as indications of a hypercoagulable state that promotes PVT formation [24]. On the contrary, Xu et al., found no discernible variations in the protein S levels of patients with and without thrombosis, possibly due to disparities in investigative focus and methodology [25].

The current study identified significant variations in systemic inflammatory markers across the three groups examined. Patients in Group I exhibited markedly elevated levels of the SII, NLR, MLR, and PLR in relative to those in Group II (all $p < 0.001$). However, there were no discernible variations in ferritin, ESR, or CRP between the two groups. NLR, MLR, PLR, ferritin, ESR, and CRP were all considerably higher in Group I than in the healthy control group (Group III) ($p < 0.001$), while SII was surprisingly higher in the control group. Similarly, Group II had elevated levels of NLR, MLR, PLR, ferritin, ESR, and CRP relative to Group III, whereas SII remained more elevated

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in the control group (for the majority of comparisons, ($p < 0.001$).

These findings align with those published by Tang et al., who emphasized increased inflammatory markers such NLR, PLR, and SII's diagnostic value in assessing systemic inflammation and liver disease progression [26]. The increased levels of ferritin, ESR, and CRP in more advanced disease stages are also consistent with chronic liver inflammation, as noted by Aslam et al., [27].

This study also discovered that patients reduced main portal vein velocity, increased portal vein diameter, and in Group I had a considerably larger portal vein diameter, lower main portal vein velocity, and a higher incidence of PVT and cavernous transition than those in Group II. These findings are consistent with Marra et al., identification of dilated portal veins and decreased flow velocity as major ultrasonographic indications of PVT [28]. Similarly, Attanasi et al., found a substantial connection between cavernous transformation and persistent PVT [29], which supports the current findings .

In addition, Group I had significantly bigger splenic vein diameter and lower splenic vein velocity, as well as a higher frequency of ascites, umbilical hernia, and anterior abdominal wall collaterals. These outcomes are in line with the observations made by Costache et al., who connected venous congestion due to thrombosis with increased collateral formation and ascitic fluid accumulation [30]. However, they differ from Xiong et al., who discovered no notable variations in splenic vein parameters between PVT and non-PVT patients [31]. Such variation may stem from difference in the degree of portal hypertension.

Further, triphasic pelvic-abdominal CT revealed that PVT, cavernous transformation, increased splenic vein diameter, ascites and abdominal wall collaterals were significantly more common in Group I than in Group II. These features reflect the pathophysiological effects of chronic portal hypertension and thrombosis. These findings are supported by Shukla et al., who also reported more frequent vascular changes (such as splenic vein dilation, ascites, and collateral formation) in cirrhotic patients with PVT [32]. Wei et al., similarly highlighted cavernous transformation as a distinct CT feature of chronic PVT [33]. In contrast, Garg et al., did not observe significant differences in collateral circulation or splenic vein diameter between PVT and non-PVT patients, potentially due to their use of doppler ultrasound, which may be less sensitive than CT in identifying subtle vascular changes [34]. Difference in portal hypertension severity may further explain the divergence.

Also, our study demonstrated that systemic inflammatory markers (SII, NLR, MLR, and PLR) exhibited high diagnostic performance in predicting PVT among cirrhotic patients. At cutoff values of >270 for SII, >2.99 for NLR, >0.4 for MLR, and >97 for PLR, these markers achieved sensitivities and specificities exceeding 85%, with overall diagnostic accuracy above 90%. These results are consistent with prior research, including the study by Duygulu et al., which identified NLR and PLR as effective indicators of hypercoagulability and PVT [35]. Xue et al., also reinforced the role of SII as a robust inflammatory marker for thrombotic risk in cirrhosis [36]. Nonetheless, some discrepancies exist; for instance, Han et al., reported lower specificity for MLR, which may result from variability in the established cutoff thresholds may also contribute to inconsistent findings across studies [12].

The findings of this study indicated that conventional systemic inflammatory markers including (serum ferritin, ESR, and CRP) had limited diagnostic accuracy for predicting portal vein thrombosis. This outcome aligns with the results of Xing et al., who found that these markers possess restricted diagnostic capability in the context of thrombotic and liver-related conditions [37].

Supporting these observations, Simeon et al., also reported that CRP and ESR had only moderate diagnostic relevance in identifying PVT, reinforcing their limited standalone utility [38]. Additionally, DePalma et al., noted that elevated ferritin levels primarily reflect the intensity of systemic inflammation rather than serving as a direct indicator of thrombotic events [39]. Therefore, our results reinforce the view that while these traditional inflammatory markers are reflective of inflammatory processes, they lack sufficient sensitivity and specificity for effective PVT diagnosis. This highlights the need for the development of more specific markers or

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the use of combined indices to enhance diagnostic precision.

The study also found significant positive associations between systemic inflammatory indicators. Specifically, the SII was positively correlated with NLR, MLR, and PLR, while MLR was favorably associated with PLR. These findings point to a coordinated inflammatory response pattern among PVT patients. Han et al., previously highlighted such interrelationships, describing how these indicators play interwoven roles in vascular inflammation [12]. Similarly, Lin et al., noted that combining inflammatory markers can more effectively capture the systemic inflammatory burden associated with vascular pathology [40].

Interestingly, despite these relationships, no significant associations were found between inflammatory indices and liver disease severity scores, such as MELD and Child-Pugh classifications. This is in line with Hammerich et al., who argued that systemic inflammation does not necessarily parallel liver function or clinical prognosis, indicating that inflammation and hepatic deterioration may progress independently [41].

Furthermore, a strong negative connection was found between NLR and SV velocity, showing that increased inflammation may be associated with lower venous flow. Gao et al., reported a similar adverse connection between inflammatory indicators and portal hemodynamics [42]. Other Doppler metrics, such as main PV velocity and vascular diameters, did not correlate significantly with inflammatory indices or liver severity scores. This echoes the findings of Jagdish et al., who highlighted the multifactorial character of portal hemodynamics in cirrhosis, implying that systemic inflammation alone may not entirely explain for the observed vascular changes [43].

Limitations: The study was conducted at a single center, which may limit the generalizability of the findings to broader populations or different clinical settings and the design does not allow for assessment of causal relationships or progression of portal vein thrombosis over time. Inflammatory markers were measured at a single time point, which may not reflect dynamic changes during the course of liver disease or acute events. Potential confounding factors such as undiagnosed subclinical infections or variations in laboratory techniques may have influenced the accuracy of systemic inflammatory indices.

CONCLUSION

Systemic inflammatory indices such as SII, NLR, MLR, and PLR showed high sensitivity and specificity in identifying PVT, indicating their potential as non-invasive biomarkers in predicting portal vein thrombosis in cirrhotic patients. Doppler ultrasound parameters, especially portal and splenic vein diameters and velocities, offer high diagnostic accuracy in predicting portal vein thrombosis in cirrhotic patients. Conventional inflammatory markers (CRP, ESR, ferritin) exhibited low diagnostic performance, reinforcing the superiority of composite inflammatory indices. Integrating Doppler ultrasound with systemic inflammatory indices can enhance early detection and risk stratification of PVT in cirrhosis, improving patient management and outcomes.

Ethical Approval: Following a detailed presentation of the study's objectives and research questions, all participants have been informed about the nature of the research and have been given the opportunity to give written informed consent prior to participation. The study's methodology, including the sample size determination, received approval from the Research Ethics Committee of the Faculty of Medicine, Menoufia University, Egypt (IRB approval number: 6/2023 TROP39). The research was carried out in line with the ethical standards outlined in the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors assert that they do not have any conflicting interests.

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Authors' contributions

All authors have made substantial contributions to the presented work, encompassing ideation, study design, implementation, gathering data,

analysing it and interpreting it. In addition, Ahmed Abo-Zaid Ahmed Teima, Amany A. Amer , Basam M. Masoud and Mohamed A. Mousa and made substantial contributions to the drafting, revising, and critical evaluation of the article. All authors provided final permission for the published version and selected the journal for submission. Furthermore, they committed to take full responsibility for all parts of the work and agreed to be accountable for the content and conclusions of the article.

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HIGHLIGHTS

- Our findings support that systemic inflammatory markers (SII, NLR, MLR, PLR) provide high sensitivity and specificity for detecting portal vein thrombosis (PVT) in cirrhotic patients, outperforming conventional inflammatory markers (CRP, ESR, ferritin). Doppler ultrasound parameters, particularly portal and splenic vein diameters and velocities, also showed strong diagnostic accuracy.
- The integration of Doppler ultrasound with systemic inflammatory markers may significantly improve early detection, risk stratification, and management of PVT in cirrhosis.

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