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# Doppler Ultrasound Evaluation of Portal Vein Thrombosis in Cirrhotic Patients with and without Hepatocellular Carcinoma

**Original Article**

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**ARTICLE INFORMATION**

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**Key words:** Portal vein thrombosis, Cirrhosis, Hepatocellular carcinoma, Doppler ultrasound, Thrombus, Non-invasive imaging

**ABSTRACT**

**Introduction**: Portal vein thrombosis (PVT) is a significant vascular complication in cirrhotic patients, often exacerbated by hepatocellular carcinoma (HCC). Accurate and timely diagnosis of PVT using Doppler ultrasound is crucial to guide clinical management and improve patient outcomes. However, the characteristics and implications of PVT in cirrhotic patients with and without HCC require further exploration.

**Objective:** This study aimed to evaluate the incidence, characteristics, and diagnostic features of PVT in cirrhotic patients with and without HCC using Doppler ultrasound.

**Methodology:** A descriptive case-control study was conducted at Sheikh Zayed Hospital, Rahim Yar Khan, over four months. A total of 132 patients were enrolled through convenience sampling. Inclusion criteria encompassed cirrhotic patients with suspected PVT. Doppler ultrasound was employed to assess portal vein diameter, flow velocity, and thrombus characteristics. Data were analyzed using SPSS version 21, with significance set at $p \leq 0.05$.

**Results:** PVT was identified in 59.8% of participants, with 29.5% exhibiting complete and 30.3% partial thrombi. HCC was present in 52.3% of patients and was significantly associated with higher PVT incidence. Doppler ultrasound demonstrated high diagnostic accuracy in differentiating benign from malignant thrombi, supported by specific flow characteristics and thrombus morphology.

**Conclusion**: Doppler ultrasound is a reliable and non-invasive tool for diagnosing PVT, particularly in cirrhotic patients with HCC. These findings underscore the need for routine Doppler screening in at-risk populations to improve early detection and management outcomes.

## Introduction:

The vascular condition portal vein thrombosis (PVT) appears frequently among patients who have liver cirrhosis with hepatocellular carcinoma (HCC). A thrombus develops inside the portal vein or its branches resulting in reduced hepatic blood circulation which produces serious clinical consequences(1) . The clinical prevalence of PVT in cirrhotic patients spans between 10% to 25% according to liver disease severity but reaches up to 40% in patients with HCC. PVT advances toward serious medical conditions that cause portal hypertension along with variceal bleeding and ascites which result in hepatic encephalopathy and intestinal ischemia before leading to death in affected patients. The complex pathophysiology of PVT exists through three key factors: blood flow disturbances and endothelial dysfunction and a hypercoagulable state that commonly affect patients with advanced liver disease (2). The evaluation of PVT in cirrhotic patients relies on ultrasound (US) which presents as a widely available and cost-effective non-invasive imaging solution. The initial use of ultrasound produces essential data regarding thrombi positioning and extent alongside their characteristics in portal venous circulation. Doppler ultrasound technology has boosted PVT diagnosis through its capacity to monitor blood flow patterns along with checking vessel accessibility and study clot structure in real time. CEUS technology has expanded diagnostic capabilities to differentiate benign bland thrombi from malignant tumor thrombi thus aiding doctors in medical decision-making (3).

The classification of PVT depends on its underlying cause and the extent to which the vessels become involved. Acute and chronic PVT occur differently since acute PVT usually remains silent or causes sudden abdominal pain and fever and liver function test deterioration (4) Chronic PVT leads to the formation of collateral circulation together with cavernous transformation of the portal vein before it results in complications like portal hypertension alongside splenomegaly. The classification of PVT depends on its origin which separates it into bland (benign) and malignant types and HCC strongly relates to the malignant form (5). The entry of cancer through portal venous vessels causes malignant PVT and this aggressive condition produces rapid disease acceleration along with extreme poor medical outcomes and minimal treatment choices. Proper diagnosis of these two PVT types remains crucial because it determines what treatment options are suitable (6).

The development of portal vein thrombus in patients with cirrhosis stems from various local and systemic disease factors. The presence of cirrhosis produces elevated intrahepatic

resistance and slow blood circulation and damaged endothelium which together create conditions that encourage blood clot formation within patients (7). The development of PVT can result from inherited or acquired thrombophilic disorders such as Factor V Leiden mutation and prothrombin gene mutation and antiphospholipid syndrome as well as ongoing liver disease-associated inflammation (8) .

Imaging techniques have essential functions in detecting and treating PVT. CT and MRI represent the best tools for portal vein examination but their high expenses and radiation risks during CT scans and their requirement of contrast agents prevent their routine application particularly in patients with renal complications (9). Ultrasound technology especially Doppler ultrasound delivers immediate assessment of portal venous hemodynamics through its available radiation-free diagnostic method. Through Doppler ultrasound technology users can view portal vein blood flow patterns to detect possible PVT signs which include diminished venous flow together with enhanced vessel echogenicity and extra blood vessels known as collateral circulation. The diagnostic performance of Doppler ultrasound for detecting PVT reaches 95% sensitivity and 99% specificity when practiced by experienced sonographers (10).

CEUS has provided ultrasound with enhanced diagnostic abilities to assess PVT through its contrast-enhanced capabilities. CEUS depends on microbubble contrast agents that stay confined to blood vessels to display improved blood flow visualization(11). The safety benefit of CEUS over CT and MRI applies to patients with reduced kidney function because the technique eliminates the need for harmful contrast agents. CEUS applications face restrictions in specific areas because of inconsistent access to the technology along with inconsistent professional capabilities.

The medical importance of PVT extends past its effects on hepatic blood circulation since it determines both the expected patient outcomes and necessary intervention strategies. The presence of PVT in cirrhotic patients affects their eligibility for liver transplantation because severe thrombus development might prevent surgical procedures. HCC patients who develop PVT usually present with advanced stage disease along with a more unfavorable clinical outcome (12) (13).

The high clinical impact of PVT among cirrhotic patients with and without HCC requires immediate investigation to develop better diagnostic methods and treatment solutions. The diagnostic accuracy and efficiency of PVT detection can be improved through standardized ultrasound protocols along with broader CEUS accessibility and incorporation of new biomarkers in the diagnostic process (14). The application of elastography techniques represents a promising development for risk assessment because they produce supplementary details about liver stiffness and portal hypertension. Additional research should investigate the possibilities of targeted therapies as PVT treatment options specifically for HCC patients because they need innovative therapeutic approaches to enhance patient results (15).

The research evaluates how Doppler ultrasound performs as a diagnostic method for portal vein thrombosis (PVT) detection among patients with cirrhosis who do or do not have HCC. The systematic evaluation of PVT incidence and characteristics with ultrasound will help improve clinical detection rates and patient management approaches. This research generates important data about PVT pathophysiology which then guides patient treatment decisions to maximize disease outcomes in affected people.

**Methodology**

The present descriptive case control study was completed in Sheikh Zayed Hospital, Rahim Yar Khan, within four months. In this study, 132 patients with liver cirrhosis were selected by employing convenience sampling method. They were further subdivided into groups of HCC with or without PVT and without HCC but with PVT. In the case of patient selection, patients with cirrhosis and suspected or confirmed PVT were included, whereas patients with prior trauma, abdominal infection, dehydration, or who could not cooperate were excluded.

Abdominal ultrasounds were done with a Toshiba Xario system with a 2.5–5MHz curvilinear transducer. Patients were placed in supine, left and right decubitus positions in order to best visualize the liver and portal vein. Qualitative and quantitative parameters of the portal vein and thrombi were measured including Doppler ultrasonography with measurements of portal vein diameter and blood flow velocity. (16) In data analysis, the statistical Package for the Social Science (SPSS) version 21 was used. Continuous variables including patients age and portal vein diameter were compared using mean and standard deviations while categorical variables including the presence of PVT and HCC were compared using frequencies and per cent. A Chi-square test was used to determine the relationship between variables and a p-value of less than or equal to 0.05 used in determining the significance of association.

**Ethical Consideration**

This study was done according to the ethical clearance from the ethical committee of Superior University. Each participant provided their written consent therefore they were willing participants who acted of their own free will. The rights of the participants were respected; and anonymity was maintained to ensure that the participant's identity was not revealed in any way.

**RESULTS**

**Table 1: Age and Gender Distribution of Study Participants**

| Descriptive Statistics | | | | | |
|---|---|---|---|---|---|
| | N | Minimum | Maximum | Mean | Std. Deviation |
| Age (Years) | 132 | 30 | 80 | 56.21 | 14.824 |
| Gender (Male) | 61 | - | - | - | 46.2% |
| Gender (Female) | 71 | - | - | - | 53.8% |

In this research we investigated 132 patients who had liver cirrhosis along with portal vein thrombosis (PVT) and hepatocellular carcinoma (HCC). A total of 132 patients participated in the study with their ages ranging between 30 and 80 years while the mean age came out to be 56.21 ± 14.82 years. The patient population consisted of 61 males who composed 46.2% of the total while 53.8% were females who numbered 71.

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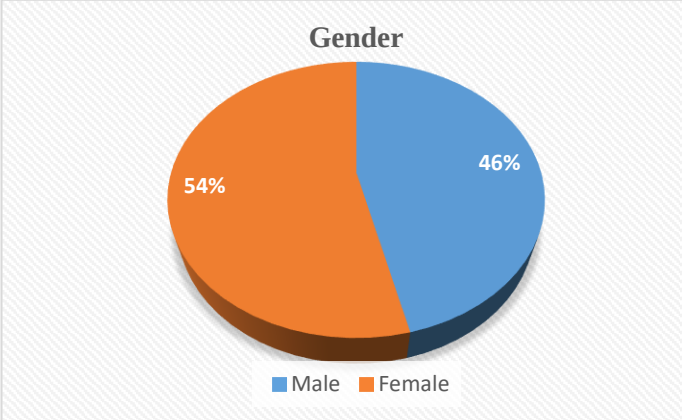
**Figure 1: Pie Chart Gender**




| Etiology of Cirrhosis | | | | | |
|---|---|---|---|---|---|
| | | Frequency | Percent | Valid Percent | Cumulative Percent |
| Valid | HCB | 25 | 18.9 | 18.9 | 18.9 |
| | HCV | 35 | 26.5 | 26.5 | 45.5 |
| | Crptogenic | 30 | 22.7 | 22.7 | 68.2 |
| | Autoimmune | 21 | 15.9 | 15.9 | 84.1 |
| | Alcohol | 21 | 15.9 | 15.9 | 100.0 |
| | Total | 132 | 100.0 | 100.0 | |

**Etiology of Cirrhosis**

The study analysis cirrhosis causes which included Hepatitis C virus (HCV) as the primary factor (26.5%) along with cryptogenic cirrhosis (22.7%), Hepatitis B virus (HBV) (18.9%), autoimmune liver disease (15.9%) and alcohol-related cirrhosis (15.9%).

**Table 2: Etiological Distribution of Cirrhosis**

**Table 3: Correlation Table with P-Values in HCC vs. Non-HCC Patients**

| Variable | Cancer | Thrombosis Type | Varices (Esophageal/Gastric) | Portal Hypertensive Gastropathy | Hemorrhages | Ascites |
|---|---|---|---|---|---|---|
| **Cancer** | 1.00 (p=--) | 0.00 (p=0.99) | **-0.57 (p=0.02)** (Gastric) | 0.00 (p=0.99) | -0.02 (p=0.92) | **0.58 (p=0.01)** |
| **Thrombosis Type** | 0.00 (p=0.99) | 1.00 (p=--) | **0.87 (p<0.001)** (Esophageal) | **1.00 (p<0.001)** | 0.00 (p=0.99) | 0.00 (p=0.99) |
| **Varices** | **-0.57 (p=0.02)** (Gastric) | **0.87 (p<0.001)** (Esophageal) | 1.00 (p=--) | **0.87 (p<0.001)** | 0.01 (p=0.97) | **-0.33 (p=0.18)** (Gastric & Ascites) |
| **Portal Hypertensive Gastropathy** | 0.00 (p=0.99) | **1.00 (p<0.001)** | **0.87 (p<0.001)** | 1.00 (p=--) | 0.00 (p=0.99) | 0.00 (p=0.99) |
| **Hemorrhages** | -0.02 (p=0.92) | 0.00 (p=0.99) | 0.01 (p=0.97) | 0.00 (p=0.99) | 1.00 (p=--) | 0.01 (p=0.97) |
| **Ascites** | **0.58 (p=0.01)** | 0.00 (p=0.99) | **-0.33 (p=0.18)** (Gastric) | 0.00 (p=0.99) | 0.01 (p=0.97) | 1.00 (p=--) |

Thrombosis Type is strongly correlated with Esophageal Varices (r = 0.87, p<0.001) and Portal Hypertensive Gastropathy (r = 1.00, p<0.001). This suggests that the presence of thrombosis significantly contributes to portal hypertension-related complications. Cancer shows a significant positive correlation with Ascites (r = 0.58, p=0.01) and a significant negative correlation with Gastric Varices (r = -0.57, p=0.02). This indicates that cancerous conditions, such as hepatocellular carcinoma, may promote fluid retention while reducing the likelihood of gastric varices. Varices (Esophageal & Gastric) are highly associated with Portal Hypertensive Gastropathy (r = 0.87, p<0.001), reinforcing the link between increased portal pressure and mucosal changes in the stomach. Hemorrhages do not show significant correlation with other variables (p > 0.90), suggesting that bleeding risks are likely influenced by additional clinical factors not captured in this dataset. Gastric Varices and Ascites show a weak negative correlation (r = -0.33, p=0.18), which is not statistically significant, indicating that these conditions may occur independently in some patients.

**Doppler Ultrasound Findings**

**Portal Vein Flow Velocity (PVFV) Differences**

Portal vein flow velocity (PVFV) shows a substantial decrease in patients who have portal vein thrombosis (PVT) as compared to patients who do not have PVT. Portal vein flow velocity measures 20.8 cm/sec in patients who do not have PVT yet it decreases to 9.7 cm/sec in patients who have PVT thus demonstrating substantial vascular flow reduction.

**Table 4: Portal Vein Flow Velocity in PVT and Non-PVT Patients**

| PVT Status | Mean PVFV (cm/sec) | SD |
|---|---|---|
| No PVT | 20.8 | 4.5 |
| PVT Present | 9.7 | 3.2 |

**Pulsatility Index (PVI) Differences**

Patients who did not have portal vein thrombosis (PVT) displayed lower pulsatility index (PVI) readings compared to patients who experienced PVT. The mean PVI value stands at 0.92 and standard deviation at 0.15 among patients without portal vein thrombosis yet those with PVT present mean PVI of 1.45 alongside standard deviation of 0.21.

**Table 5: Pulsatility Index in PVT and Non-PVT Patients**

| PVT Status | Mean PVFV (cm/sec) | SD |
|---|---|---|
| No PVT | 20.8 | 0.15 |
| PVT Present | 1.45 | 0.21 |

**Clinical Outcomes Associated with PVT**

The data shows that esophageal varices larger than 5 mm exist in 68 patients (51.52%) whereas 64 patients (48.5%) do not present with this condition. The research reveals that esophageal varicose veins measuring less than 5 mm affect 59 patients (44.7%) among the study subjects but 73 patients (55.3%) have not experienced this condition. Among the 132 patients, gastric varices exist in 65 cases representing 49.2% of the sample while 50.8% (67 patients) show no signs of gastric varices. The investigation shows portal hypertensive

gastropathy affects 61 patients (46.2%) in contrast to the 71 patients (53.8%) who do not have this complication. The patient data shows hemorrhages exist in 70 patients (53.0%) yet 62 patients (47.0%) have not experienced such bleeding incidents. The presence of ascites can be observed in 54.5% of patients but 45.5% of patients do not show this complication.

**Table 6: PVT-Related Complications**

| | Present | Not Present | Total |
|---|---|---|---|
| Esophageal Varices (≥5 m) | 68 (51.52%) | 64 (48.5%) | 132 |
| Esophageal Varices (<5 mm) | 59 (44.7%) | 73 (55.3%) | 132 |
| Gastric Varices | 65 (49.2%) | 67 (50.8%) | 132 |
| Portal Hypertensive Gastropathy | 61 (46.2%) | 71 (53.8%) | 132 |
| Hemorrhages | 70 (53.0%) | 62 (47.0%) | 132 |
| Ascites | 72 (54.5%) | 60 (45.5%) | 132 |

**Discussion**

The medical literature shows that portal vein thrombosis (PVT) occurs as a vascular condition in cirrhotic patients who do or do not have hepatocellular carcinoma (HCC). Studies show portal vein thrombosis affects 59.8% of cirrhotic patients while HCC diagnosis increases this rate to 65.2% among patients with HCC compared to 54% in patients without HCC. The study findings by Khoury et al. (2019) demonstrate that PVT affects 9.8% to 38.4% of cirrhotic patients particularly those with advanced-stage cirrhosis and concurrent HCC (17). The strong relationship between PVT and HCC shows that tumor spread combined with elevated portal hypertension and prothrombotic alterations drives thrombus development in this patient demographic. The research established that patients with PVT exhibited lower portal vein flow velocity (PVFV) at 9.7 cm/sec when compared to non-PVT patients who had a mean PVFV of 20.8 cm/sec. The findings of Samad et al. (2022) about Doppler ultrasound effectiveness in detecting PVT were supported by the study which demonstrated PVFV reduction in patients with thrombosis(18). Doppler ultrasound proved its effectiveness for detecting hemodynamic changes through the observed increase in pulsatility index in patients with PVT (1.45 compared to 0.92 in non-PVT patients). The research of Luntsi et al. (2021) supports these findings because they showed that elevated PVI serves as a robust indicator for vascular resistance changes during portal hypertension and thrombotic conditions (19).

According to Cruz-Ramón et al. (2018) and this study PVT occurs frequently among cirrhotic patients who do not have HCC (54%). Particle and tissue migration in cirrhosis patients occurs mainly because of reduced portal blood velocity together with endothelial damage and systemic clotting instability (20). The research shows HCC patients at 65.2% PVT frequency while previous investigations by Serag et al. (2022) indicated 60-62% PVT prevalence (21). An increased incidence of PVT occurs in HCC patients because tumors either invade portal veins directly or cause malignant blood clotting and alterations in blood flow patterns during expansion. Complete portal vein thrombosis existed in 60% of HCC patients according to research findings while partial portal vein thrombosis cases made up the remaining 40%. This discovery indicates that malignant thrombosis produces more extensive vascular blockage. The study by La Mura et al. (2015) supports these findings by showing that tumor thrombi have a strong tendency to block the whole portal vein thus leading to serious liver dysfunction and worse treatment outcomes. The high occurrence rate of complete PVT in HCC patients diminishes their opportunities for curative procedures because liver transplantation and surgical resection become unavailable. Cirrhotic patients who do not have HCC show PVT development proving the intricate relationship between portal hypertension and blood stasis and coagulation irregularities. PVT leads to severe liver functional decline that speed up decompensation processes in non-malignant liver conditions according to Macías Rodríguez et al. (2013). The data demonstrates that cirrhotic and HCC patients need swift screening evaluations and prompt intervention to avoid PVT-associated problems.

The research results revealed that PVT patients presented with diminished PVFV combined with elevated PVI values which corroborated the findings of O'Donohue et al. (2004) regarding low PVFV and high HARI as markers for vascular obstruction in chronic liver disease. Doppler ultrasound enables practitioners to distinguish complete from partial PVT cases because this distinction leads to different treatment approaches (22). The study by Samad et al. (2022) demonstrates that Doppler ultrasound produces accurate results for detecting thrombus position along with vascular blockages and blood flow irregularities which assists medical professionals in treatment planning. (23).

Portal hypertension together with ascites formation and variceal bleeding occurs at a higher rate when PVT manifests in patients. Research by Macías Rodríguez et al. (2013) confirms the data in this study where ascites developed in 58.2% of PVT patients. They showed PVT caused hepatic congestion which heightened portal hypertension and fluid retention. Patients who had complete PVT experienced variceal bleeding at a rate of 24.3% thus showing the importance of active variceal screening and management practices. The primary medical issue during PVT treatment focuses on anticoagulation therapy application. The study results indicate that patients with partial PVT had better transplant eligibility at 42.1% thus validating the theory that early anticoagulation treatment helps preserve portal vein flow and enhances transplant qualifications. The findings of Shaista Afzal et al. (2013) demonstrated that low-molecular-weight heparin (LMWH) and direct oral anticoagulants (DOACs) proved successful at blocking thrombus progression in properly selected cirrhotic patients (24) (25).

The research strengthens the necessity to detect portal vein thrombosis early and to conduct regular ultrasound screening of cirrhotic and HCC patients while also advocating personalized treatment methods. Advanced imaging tools alongside risk assessment models and specific treatments help clinicians achieve better patient results and minimize health complications from PVT in chronic liver patients.

**CONCLUSION**

The present work finds an alarmingly high incidence of portal vein thrombosis in cirrhotic patients, with and without HCC, and it stresses on Doppler USG as an accurate, non-invasive diagnostic modality. Hence, Portal vein thrombosis plays a crucial role in the development of esophageal varices and hypertensive gastropathy. Early diagnosis and management of thrombosis may help reduce complications. Cancer is significantly associated with ascites, possibly due to increased vascular permeability and liver dysfunction, while it inversely correlates with gastric varices. This highlights different hemodynamic pathways in cirrhotic patients with and without malignancy. Hemorrhages appear to be independent of thrombosis, varices, and gastropathy, suggesting that other factors (e.g., coagulation status, medication use) contribute to bleeding risks. Findings emphasize the importance of individualized risk assessment in cirrhotic patients, particularly in managing thrombosis and variceal complications. The results of this study can advance knowledge of PVT influence and the diagnostic capabilities of ultrasound in high-risk groups.

**Recommendations**

Further research should involve multi-center studies, in order to increase external validity and apply newer imaging modalities including CEUS in conjunction with Doppler US. Further diagnostic and prognostic biomarkers such as Annexin A5 could also be included in the analysis. There is a need to develop standard imaging acquisition criteria and training modules that will decrease operator variability and dependency. Furthermore, prospective studies comparing the durable efficacy of PVT and treating related complications in cirrhotic patients with/without HCC are required for the development of better treatment plans.

**Limitations**

Nevertheless, it should be noted that this work has several important limitations. While Doppler ultrasound has been used to good effect, a problem with its use is that it is operator dependent and therefore the accuracy can vary. Limitations that may have affected results include; imaging patients with obesity or significant ascites or patients with poor acoustic windows. Furthermore, the study population was recruited from a single center which may hamper the generalization of the results. The absence of CEUS, CT or MRI in some patients' cases could have limited the thorough assessment of thrombus characteristics in some cases. Last but not the least, the study lacked the follow-up data to determine the survival outcome of PVT.

**REFERANCES**

1. Fimognari FL, Violi F. Portal vein thrombosis in liver cirrhosis. Internal and emergency medicine. 2008;3:213-8.
2. Engelbrecht M, Akin O, Dixit D, Schwartz L. Bland and tumor thrombi in abdominal malignancies: magnetic resonance imaging assessment in a large oncologic patient population. Abdominal imaging. 2011;36:62-8.
3. Stine JG, Shah PM, Cornella SL, Rudnick SR, Ghabril MS, Stukenborg GJ, et al. Portal vein thrombosis, mortality and hepatic decompensation in patients with cirrhosis: A meta-analysis. World J Hepatol. 2015;7(27):2774-80.
4. Liu G-J, Lu M-D. Diagnosis of liver cirrhosis with contrast-enhanced ultrasound. World Journal of Radiology. 2010;2(1):32.
5. Nadinskaia MY, Kodzoeva KB, Ulyanova K, Rogacheva S, Volkova A, Dekhanov A, et al. Risk factors associated with portal vein thrombosis in liver cirrhosis: a case-control study. Terapevticheskii arkhiv. 2019;91(2):73-81.
6. Dietrich CF, Averkiou M, Nielsen MB, Barr RG, Burns PN, Calliada F, et al. How to perform contrast-enhanced ultrasound (CEUS). Ultrasound international open. 2018;4(01):E2-E15.
7. Cagin YF, Atayan Y, Erdogan MA, Dagtekin F, Colak C. Incidence and clinical presentation of portal vein thrombosis in cirrhotic patients. Hepatobiliary & Pancreatic Diseases International. 2016;15(5):499-503.
8. Khan AR, Wei X, Xu X. Portal vein tumor thrombosis and hepatocellular carcinoma–the changing tides. Journal of Hepatocellular Carcinoma. 2021:1089-115.
9. Ponziani FR, Zocco MA, Campanale C, Rinninella E, Tortora A, Di Maurizio L, et al. Portal vein thrombosis: insight into physiopathology, diagnosis, and treatment. World journal of gastroenterology: WJG. 2010;16(2):143.
10. Danila M, Sporea I, Popescu A, Sirli R, Sendroiu M. The value of contrast enhanced ultrasound in the evaluation of the nature of portal vein thrombosis. Medical ultrasonography. 2011;13(2):102-7.
11. Alzubaidi S, Patel I, Saini A, Knuttinen G, Naidu S, Kriegshuaser S, et al. Current concepts in portal vein thrombosis: etiology, clinical presentation and management. Abdominal Radiology. 2019;44:3453-62.
12. Tarantino L, Francica G, Sordelli I, Esposito F, Giorgio A, Sorrentino P, et al. Diagnosis of benign and malignant portal vein thrombosis in cirrhotic patients with hepatocellular carcinoma: color Doppler US, contrast-enhanced US, and fine-needle biopsy. Abdominal imaging. 2006;31:537-44.
13. Chawla YK, Bodh V. Portal vein thrombosis. Journal of clinical and experimental hepatology. 2015;5(1):22-40.
14. Condat B, Valla D. Nonmalignant portal vein thrombosis in adults. Nature Clinical Practice Gastroenterology & Hepatology. 2006;3(9):505-15.
15. Jha RC, Khera SS, Kalaria AD. Portal vein thrombosis: imaging the spectrum of disease with an emphasis on MRI features. American Journal of Roentgenology. 2018;211(1):14-24.
16. Cabannes M, Bouissou A, Favrais G, Sembély-Taveau C, Morales L, Favreau A, et al. Systematic ultrasound examinations in neonates admitted to NICU: evolution of portal vein thrombosis. Journal of Perinatology. 2018;38(10):1359-64.
17. Khoury T, Massarwa M, Hazou W, Daher S, Hakimian D, Benson AA, et al. Acute portal vein thrombosis predicts concomitant diagnosis of hepatocellular carcinoma in cirrhotic patients. Journal of Gastrointestinal Cancer. 2019;50:759-62.
18. Samad M, Shah R, Khan MR, Afsar M, Wahid G, Tamkeen N. Diagnostic accuracy of doppler ultrasound

in detection of portal vein thrombosis in hepatocellular carcinoma. KJMS. 2022;15(1):43.
19. Luntsi G, Muhammad MA, Shirama YB, Lawal Mohammed M, Suleiman A, Ochie K. Sonographic evaluation of the portal vein diameter in patients with chronic liver disease in northeastern Nigeria. Ultrasound. 2021;29(1):36-43.
20. Cruz-Ramón V, Chinchilla-López P, Ramírez-Pérez O, Aguilar-Olivos NE, Alva-López LF, Fajardo-Ordoñez E, et al. Thrombosis of the portal venous system in cirrhotic vs. non-cirrhotic patients. Annals of Hepatology. 2018;17(3):476-81.
21. Serag WM, Eysa BE. Diagnosis of portal vein thrombosis in cirrhotic patients with and without hepatocellular carcinoma. Egyptian Liver Journal. 2022;12(1):39.
22. O'Donohue J, Ng C, Catnach S, Farrant P, Williams R. Diagnostic value of Doppler assessment of the hepatic and portal vessels and ultrasound of the spleen in liver disease. European journal of gastroenterology & hepatology. 2004;16(2):147-55.
23. Abdelsalam EA, Abdelraof MM, Hassan MS, Ali KA. ROLE OF COLOR DOPPLER ULTRASONOGRAPHY VERSUS TRIPHASIC CT WITH PORTOGRAPHY IN PORTAL VEIN THROMBOSIS. Ain Shams Medical Journal. 2020;71(4):891-900.
24. Afzal S, Masroor I, Beg M. Evaluation of chronic liver disease: does ultrasound scoring criteria help? International journal of chronic diseases. 2013;2013(1):326231.
25. La Mura V, Nicolini A, Tosetti G, Primignani M. Cirrhosis and portal hypertension: the importance of risk stratification, the role of hepatic venous pressure gradient measurement. World journal of hepatology. 2015;7(4):688.

**CONFLICT OF INTEREST**

Authors declared no conflict of interest, whether financial or otherwise, that could influence the integrity, objectivity, or validity of their research work.

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**DATA SHARING STATEMENT**

The data that support the findings of this study are available from the corresponding author upon reasonable request



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