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Original Article

Frequency of Portal Hypertension among Patients with Liver Cirrhosis on Ultrasonography

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ABSTRACT

Background: Portal hypertension is a major consequence of cirrhosis and is associated with varices, ascites, portosystemic collateral formation, splenomegaly, and decompensation. Ultrasonography and Doppler can identify several morphological and hemodynamic features associated with portal hypertension but do not directly measure the hepatic venous pressure gradient. Objective: To determine the frequency of an ultrasound-defined portal-hypertension classification and describe associated sonographic and Doppler findings among patients with cirrhosis. Methods: An analytical cross-sectional study included 150 adults with cirrhosis examined in the Department of Diagnostic Radiology and Imaging, Sheikh Zaid Medical College and Hospital, Rahim Yar Khan. Ultrasound assessment included liver morphology, ascites, splenomegaly, portal-vein diameter and velocity, collateral pathways, portal-flow direction, cavernous transformation, and recanalized paraumbilical vein. Results: The study classified 130/150 patients (86.7%) as having portal hypertension. Splenomegaly was present in 113 (75.3%), any ascites in 107 (71.3%), portosystemic collaterals in 69 (46.0%), recanalized paraumbilical vein in 57 (38.0%), reversed portal flow in 24 (16.0%), and cavernous transformation in nine (6.0%). Mean portal-vein diameter was 13.71±1.91 mm and velocity 13.03±2.81 cm/s. Splenomegaly was strongly associated with the study-defined portal-hypertension classification, $\chi^2(1)=70.478$, $p<.001$, $\varphi=0.685$. Portal-vein diameter and velocity were not correlated, $r=-0.007$, $p=.930$. Conclusion: Ultrasound and Doppler identified a high burden of portal-hypertension-associated findings in this selected cirrhotic cohort. The reported 86.7% represents the study's ultrasound-based classification and should not be interpreted as independently validated hemodynamic prevalence until its operational diagnostic criteria are specified. Keywords: Portal Hypertension; Cirrhosis; Ultrasonography; Doppler Ultrasound; Portal Vein; Splenomegaly; Portosystemic Collaterals.

INTRODUCTION

Portal hypertension is a central pathophysiological consequence of advanced cirrhosis. Progressive hepatic fibrosis, architectural distortion, sinusoidal endothelial dysfunction, and increased intrahepatic vascular resistance interact with changes in the splanchnic circulation and portal inflow to produce progressively abnormal portal hemodynamics (1,2). Clinically, portal hypertension underlies many of the major complications of cirrhosis, including esophageal and gastric varices, ascites, splenomegaly, hypersplenism, portosystemic collateral formation, hepatic encephalopathy, and other manifestations of decompensated chronic liver disease (2,3).

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Portal pressure can be conceptualized and quantified hemodynamically. In sinusoidal cirrhosis, the hepatic venous pressure gradient (HVPG) is an established invasive surrogate of portal pressure, and clinically significant portal hypertension has conventionally been defined by an HVPG of at least 10 mmHg because complication and decompensation risks increase substantially beyond this level (1,4). The Baveno VII consensus established a framework that increasingly incorporates non-invasive risk stratification, while the more recent Baveno VIII consensus has reaffirmed the central importance of clinically significant portal hypertension and expanded the role of non-invasive testing in advanced chronic liver disease (4,5).

Although HVPG remains an important reference method for direct hemodynamic assessment in sinusoidal portal hypertension, its invasive nature, technical requirements, and limited availability restrict routine use in many clinical settings (6,7). Consequently, contemporary assessment increasingly relies on combinations of clinical findings, laboratory parameters, elastography, and imaging. Liver stiffness measurement, platelet count, spleen stiffness, and composite algorithms can provide clinically meaningful non-invasive stratification, although their performance varies according to disease etiology, clinical stage, measurement technique, and the diagnostic threshold under consideration (8–13). These methods are therefore most appropriately interpreted within integrated clinical frameworks rather than as universal replacements for invasive portal-pressure assessment.

Conventional ultrasonography remains particularly useful because it is widely available, non-ionizing, repeatable, and capable of evaluating liver morphology, ascites, splenic enlargement, portal and hepatic vessels, collateral pathways, and other consequences of chronic portal hypertension (14–16). Doppler ultrasonography extends this assessment by providing information on portal-vein velocity, direction of portal flow, waveform characteristics, hepatic arterial circulation, and related hemodynamic parameters. However, individual Doppler measurements demonstrate substantial physiological, technical, and operator-dependent variability, limiting their use as direct substitutes for HVPG or as isolated diagnostic criteria (16,17).

Splenomegaly is a common indirect manifestation of portal hypertension and reflects, among other processes, chronic elevation of portal and splenic venous pressure. Nevertheless, splenic size is influenced by disease duration, etiology, hematological factors, congestion, and individual anatomy and is therefore not independently diagnostic (14,16). In contrast, portosystemic collateral pathways represent anatomical adaptations to sustained portal hypertension and redirect portal venous blood toward the systemic circulation. Paraumbilical, gastroesophageal, splenorenal, retroperitoneal, and other collateral pathways may be detected by ultrasound, cross-sectional imaging, or angiographic techniques (18–20). Imaging evidence of collateralization, hepatofugal flow, and recanalized paraumbilical veins therefore provides important evidence of altered portal circulation, although individual findings must still be interpreted in their overall clinical context (18–20). Splenic enlargement and portal-vein characteristics have also been incorporated into non-invasive models for clinically important varices and other portal-hypertension complications (21).

Portal-vein diameter has historically been used as an ultrasonographic marker of portal hypertension, but a single diameter threshold has limited sensitivity and specificity because vessel calibre is influenced by respiration, hydration, intra-abdominal pressure, collateral decompression, disease stage, and individual anatomy (14,16,17). Portal-vein velocity may similarly decline as portal hypertension progresses, yet measurements depend on Doppler angle, respiratory phase, sampling location, cardiac output, food intake, vessel geometry, and operator technique (16,17). Contemporary evidence therefore supports an integrated interpretation of morphological findings, flow characteristics, clinical features, laboratory parameters, and validated non-invasive tests rather than reliance on a single conventional ultrasound variable (8,9,14–17).

The present study evaluated ultrasound and Doppler findings in adults with cirrhosis and determined the frequency of the study-defined portal-hypertension classification. It additionally examined the relationship of portal-hypertension classification with splenomegaly and liver texture and assessed the correlation between portal-vein diameter and velocity.

MATERIALS AND METHODS

An analytical cross-sectional study was conducted in the Department of Diagnostic Radiology and Imaging, Sheikh Zaid Medical College and Hospital, Rahim Yar Khan. The study duration was four months. A total of 150 adults with cirrhosis were enrolled using convenience sampling.

Participants aged 18–70 years with cirrhotic liver disease who provided consent were eligible. Patients with portal-vein thrombosis, previous hepatic surgery, pregnancy or lactation, or inadequate sonographic imaging because of technical limitations were excluded. Ethical approval was obtained from the Institutional Review Board of Superior University, Lahore, Pakistan.

Ultrasound examinations were performed using a Toshiba Xario system with a 2.5–5 MHz curvilinear transducer. Patients were examined in the supine or left-lateral position as appropriate. B-mode examination evaluated liver morphology, liver size, ascites, spleen enlargement, and vascular abnormalities. Doppler assessment included portal-vein diameter, portal-vein velocity, portal-flow waveform and direction, cavernous transformation, portosystemic collateral pathways, and recanalization of the paraumbilical vein.

Portal-hypertension status was recorded according to the study's ultrasound/Doppler classification.

Data were analyzed using IBM SPSS Statistics version 25. Continuous variables were summarized using mean, standard deviation, minimum, and maximum. Categorical variables were summarized as frequencies and percentages. Associations between categorical variables were evaluated using chi-square or Fisher exact testing where appropriate. Pearson correlation was used for the relationship between portal-vein diameter and portal-vein velocity. A phi coefficient was calculated for the 2×2 relationship between splenomegaly and portal-hypertension classification, and Cramér's V for the liver-texture association.

RESULTS

Participant Characteristics

The study included 150 adults with cirrhosis. Mean age was 48.85±12.48 years, with an observed range of 30–70 years. Seventy-seven participants were female and 73 male. Mean BMI was 26.78±4.68 kg/m^2.

Table 1. Participant and General Ultrasound Characteristics

Variable	Result
Age, years	48.85±12.48
Age range	30–70
Female	77 (51.3%)
Male	73 (48.7%)
BMI, kg/m^2	26.78±4.68
BMI range	19–35
Current smoking	27 (18.0%)
Alcohol use	26 (17.3%)
Liver texture: irregular	70 (46.7%)
Liver texture: nodular	65 (43.3%)
Liver texture: third source category	15 (10.0%)
Liver size, cm	13.12±1.85
Ascites absent	43 (28.7%)
Mild ascites	44 (29.3%)
Moderate ascites	46 (30.7%)
Severe ascites	17 (11.3%)
Any ascites	107 (71.3%)

Portal and Doppler Findings

Mean portal-vein diameter was 13.71±1.91 mm and mean portal-vein velocity 13.03±2.81 cm/s. Splenomegaly was present in 75.3% of participants.

Table 2. Portal-Hypertension-Associated Ultrasound and Doppler Findings

Finding	n (%) / Mean±SD
Splenomegaly	113 (75.3%)
Portal-vein diameter	13.71±1.91 mm
Portal-vein diameter range	11–18 mm
Portal-vein velocity	13.03±2.81 cm/s
Portal-vein velocity range	8–18 cm/s
Cavernous transformation	9 (6.0%)
Pulsatile portal waveform	30 (20.0%)
Portosystemic collateral pathways	69 (46.0%)
Reversed portal flow	24 (16.0%)
Recanalized paraumbilical vein	57 (38.0%)

Finding	n (%) / Mean±SD
Study-defined portal hypertension	130 (86.7%)

Association With Splenomegaly

All 113 participants with splenomegaly were classified as having portal hypertension. Among the 37 without splenomegaly, 17 were classified as having portal hypertension and 20 were not.

Table 3. Splenomegaly and Study-Defined Portal-Hypertension Classification

Splenomegaly	No Portal Hypertension	Portal Hypertension	Total
No	20	17	37
Yes	0	113	113
Total	20	130	150

Pearson $\chi^2(1)=70.478$, $p<.001$. The phi effect-size coefficient was approximately 0.685, indicating a strong association within the study classification.

Liver Texture and Portal Hypertension

The overall association between the three liver-texture categories and portal-hypertension classification was not significant.

Table 4. Statistical Associations

Analysis	Statistic	p	Effect Size
Splenomegaly vs portal hypertension	$\chi^2(1)=70.478$	<.001	$\varphi=0.685$
Liver texture vs portal hypertension	$\chi^2(2)=3.186$	.203	Cramér's V=0.146
Portal-vein diameter vs velocity	r=−0.007	.930	—

Portal-vein diameter and portal-vein velocity were essentially uncorrelated.

DISCUSSION

The present study identified a high frequency of ultrasound and Doppler abnormalities associated with portal hypertension among patients with cirrhosis. The study-defined portal-hypertension classification was present in 86.7% of participants. Splenomegaly was documented in 75.3%, ascites in 71.3%, portosystemic collateral pathways in 46.0%, recanalized paraumbilical vein in 38.0%, reversed portal flow in 16.0%, and cavernous transformation in 6.0%. This high frequency should be interpreted in the context of a selected hospital population already known to have cirrhosis rather than as a community prevalence estimate.

Portal hypertension becomes increasingly common as cirrhosis advances because fibrosis, regenerative architectural distortion, endothelial dysfunction, increased intrahepatic resistance, and dynamic vascular changes progressively elevate portal pressure (1,2). Once clinically significant portal hypertension develops, risks of varices, ascites, decompensation, and other clinically important complications rise substantially (3–5). The high burden of sonographic abnormalities in the present cohort is therefore physiologically consistent with the established relationship between advanced cirrhosis and disturbed portal hemodynamics.

Ultrasound and Doppler provide a practical means of identifying many structural and hemodynamic consequences of portal hypertension, including splenomegaly, ascites, collateral formation, altered portal-flow velocity, changes in flow direction, and vascular abnormalities (14–17). Their particular value lies in the ability to integrate anatomical and flow information during a single non-invasive examination. Nevertheless, conventional Doppler findings remain indirect markers, and individual measurements do not provide the same physiological information as direct or surrogate invasive measurement of the portal pressure gradient (6,7,16,17).

Splenomegaly demonstrated the strongest statistical association in the current data. All 113 participants with splenomegaly were classified as having portal hypertension, and the phi coefficient of 0.685 indicated a strong within-sample relationship. Splenomegaly is a well-established consequence of chronic portal hypertension and splenic venous congestion and has been incorporated, together with platelet count and elastographic measures, into several non-invasive approaches to portal-hypertension and variceal-risk assessment (7,10–12,21). However, splenic enlargement is not specific to portal hypertension and is influenced by disease etiology, hematological conditions, inflammatory processes, and individual anatomy (14,16). Furthermore, if splenomegaly contributed directly to the study's ultrasound classification of portal hypertension, the observed association would not represent an independent diagnostic validation of splenomegaly.

Ascites was also common, affecting approximately 71% of the cohort. In cirrhosis, ascites is strongly related to portal hypertension and the accompanying systemic and splanchnic circulatory abnormalities that promote sodium and water retention, although renal, cardiac, malignant, infectious, and other causes must be considered clinically (1–3). Its high frequency in the present hospital cohort therefore suggests a substantial burden of clinically advanced liver disease, although cirrhosis severity scores and other markers of decompensation were not available for formal stratification.

Nearly half of the participants had identifiable portosystemic collateral pathways, and 38% had a recanalized paraumbilical vein. Portosystemic collateralization represents an important chronic adaptive response to increased portal pressure, in which blood is redirected through pre-existing or newly enlarged communications between the portal and systemic venous systems (18–20). Paraumbilical, gastroesophageal, splenorenal, retroperitoneal, and other collateral pathways may become prominent as portal hypertension progresses, and imaging of these vessels provides important anatomical evidence of altered portal circulation (18–20). The substantial frequency of collateral pathways and paraumbilical-vein recanalization in the present cohort is therefore consistent with chronic portal hemodynamic disturbance.

Reversal of portal flow was observed in 16% of participants. Hepatofugal flow represents an important alteration of portal hemodynamics and generally occurs in the setting of substantial redistribution of portal blood flow, particularly when collateral pathways provide lower-resistance decompressive routes (16–20). Nevertheless, direction of flow should be assessed together with portal-vein patency, collateral anatomy, respiratory variation, vessel sampling, and the broader clinical stage rather than interpreted as an isolated measure of portal pressure (16,17).

The mean portal-vein diameter was approximately 13.7 mm. Although portal-vein calibre has historically been incorporated into sonographic assessment of portal hypertension, contemporary evidence does not support a single diameter threshold as a universally reliable standalone diagnostic test (14–17). Portal-vein diameter varies with respiration, hydration, body habitus, collateral decompression, intra-abdominal pressure, and disease stage. The presence of substantial collateral circulation may also decompress the main portal vein despite clinically important portal hypertension, further weakening a simple one-to-one relationship between vessel diameter and portal pressure (16–20).

Mean portal-vein velocity was approximately 13.0 cm/s, while portal-vein diameter and velocity were essentially uncorrelated in the present dataset. Reduced portal-flow velocity has been described in advanced portal hypertension, but Doppler velocity measurements depend strongly on insonation angle, respiratory phase, sampling location, meal status, cardiac output, vessel geometry, and technical acquisition (16,17). The absence of correlation between calibre and velocity in the present study is therefore physiologically plausible because the two variables reflect different aspects of portal circulation and may respond differently to collateral decompression and other compensatory mechanisms.

The present findings also support the broader shift away from reliance on individual conventional ultrasound variables. Current non-invasive assessment increasingly combines clinical features, platelet count, liver stiffness, spleen stiffness, and other validated algorithms according to the clinical question and stage of chronic liver disease (4,5,7–13). Spleen-stiffness measurements, in particular, have demonstrated useful performance for detecting clinically significant portal hypertension and may reduce the diagnostic gray zone remaining after liver-stiffness and platelet-based approaches (11–13). Conventional ultrasound and Doppler remain highly valuable because they directly characterize anatomy, ascites, vascular patency, collateral pathways, and flow direction, but they are most appropriately interpreted as components of an integrated portal-hypertension assessment rather than as direct equivalents of HVPG measurement (6–9,14–17).

The study has several strengths. It included a substantial cohort of patients with cirrhosis and systematically documented multiple portal-hypertension-associated findings rather than relying only on portal-vein diameter. The observed burden of splenomegaly, ascites, collaterals, reversed portal flow, cavernous transformation, and paraumbilical-vein recanalization provides clinically useful characterization of the study population and demonstrates the breadth of information that can be obtained during combined B-mode and Doppler examination.

The principal interpretive limitation is uncertainty surrounding the exact operational rule used to classify portal hypertension. Without a clearly specified ultrasound classification algorithm or an independent reference standard, the reported 86.7% frequency should be regarded as the frequency of the study-defined ultrasound/Doppler classification rather than as independently validated prevalence of clinically

significant portal hypertension. This distinction is especially important because contemporary definitions of clinically significant portal hypertension are fundamentally linked to portal-pressure physiology and validated non-invasive risk-stratification approaches rather than to any single conventional ultrasound feature (4–12).

CONCLUSION

A high burden of ultrasound and Doppler findings associated with portal hypertension was observed among patients with cirrhosis. The study classified 86.7% of participants as having portal hypertension. Splenomegaly showed a strong association with this classification, while liver texture did not, and portal-vein diameter was not correlated with portal-vein velocity. Ultrasound and Doppler provide valuable non-invasive characterization of portal circulation, vascular anatomy, and complications, but the study-defined classification requires a clearly specified diagnostic algorithm before it can be interpreted as an independently validated measure of portal-hypertension prevalence.

LIMITATIONS

The study used convenience sampling at a single tertiary-care institution. The portal-hypertension classification was not compared with an independent hemodynamic reference standard. Cirrhosis etiology, Child-Pugh class, MELD score, platelet count, endoscopic variceal findings, and liver/spleen stiffness were not incorporated into the available analysis. Doppler measurements are operator and technique dependent.

RECOMMENDATIONS

Future studies should define the ultrasound diagnostic algorithm prospectively and validate it against an accepted clinical or hemodynamic reference. Cirrhosis severity, platelet count, elastography, varices, etiology, and clinical decompensation should be incorporated. Doppler acquisition should be standardized for fasting status, respiratory phase, insonation angle, vessel sampling location, and replicate measurements.

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