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Original Article

Evaluation of Hemodynamic Changes in Portal Hypertension and Their Correlation with Splenomegaly Using Doppler Ultrasonography

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ABSTRACT

Background: Portal hypertension produces structural and haemodynamic alterations in the portal and splenic circulations, frequently resulting in splenomegaly. Doppler ultrasonography enables non-invasive assessment of these changes. Objective: To evaluate associations between Doppler-derived portal haemodynamic parameters and spleen length in adults with portal hypertension and splenomegaly. Methods: This cross-sectional study included 53 adults recruited through convenience sampling from the radiology department of Al Razi Institute, Lahore. Portal vein diameter, portal vein velocity, portal flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, hepatic artery resistance index, and spleen length were assessed. Pearson and Spearman correlations and multiple linear regression were performed using SPSS version 25.0. Results: Mean age was 51.83 ± 8.21 years, and 31 participants (58.5%) were male. Mean spleen length was 14.94 ± 1.37 cm. Spleen length correlated positively with portal vein diameter ($r = 0.981$; 95% CI: 0.967–0.989) and negatively with portal vein velocity ($r = -0.978$; 95% CI: −0.987 to −0.962), splenic vein velocity ($r = -0.976$), and portal flow ($r = -0.975$); all $p < 0.001$. The regression model explained 97.3% of variance in spleen length, although predictors were highly collinear. Portal vein diameter remained independently associated with spleen length ($B = 0.509$; 95% CI: 0.231–0.787; $p = 0.001$). Conclusion: Spleen length was strongly associated with multiple Doppler parameters, particularly portal vein diameter. These findings require cautious interpretation and independent validation because of severe multicollinearity and the small single-centre sample. Keywords: Doppler ultrasonography; portal haemodynamics; portal hypertension; portal vein diameter; portal vein velocity; splenomegaly.

INTRODUCTION

Portal hypertension is a major complication of chronic liver disease and develops when resistance to portal venous blood flow produces a sustained increase in pressure within the portal circulation. Liver cirrhosis is the most frequent underlying cause because fibrosis, regenerative nodules, sinusoidal remodelling, and increased intrahepatic vascular tone progressively obstruct portal blood flow. These changes may lead to portosystemic collateral formation, ascites, gastroesophageal varices, hepatic encephalopathy, and other manifestations of hepatic decompensation (1–3).

The portal venous system is a high-volume, low-pressure vascular network that transports blood from the gastrointestinal tract, spleen, and pancreas to the liver. Under physiological conditions, portal venous flow is continuous, minimally pulsatile, and hepatopetal. Portal hypertension is conventionally defined

by a hepatic venous pressure gradient greater than 5 mmHg, whereas a gradient of at least 10 mmHg indicates clinically significant portal hypertension and is associated with an increased risk of varices and decompensating events (2). Although hepatic venous pressure-gradient measurement is the accepted reference method for quantifying portal pressure, its invasive nature, technical requirements, and limited availability restrict its routine use in many clinical settings.

Splenomegaly is a common manifestation of portal hypertension. Increased resistance to portal blood flow impedes venous drainage from the spleen, producing splenic congestion and progressive enlargement. However, splenic enlargement is not solely a passive consequence of venous congestion. Chronic portal hypertension may also promote splenic tissue hyperplasia, angiogenesis, fibrogenesis, and structural remodelling, suggesting an active interaction between the spleen and portal circulation through the hepatosplenic axis (4–6). Persistent splenic enlargement may contribute to hypersplenism, thrombocytopenia, leukopenia, and anaemia, which can complicate the clinical management of patients with chronic liver disease (6,7).

Doppler ultrasonography provides simultaneous structural and functional evaluation of the portal circulation without ionizing radiation or intravenous contrast. Grey-scale ultrasonography permits assessment of liver morphology, portal vein calibre, splenic dimensions, ascites, and collateral vessels, whereas spectral and colour Doppler enable evaluation of blood-flow direction, velocity, waveform characteristics, and vascular resistance. Commonly assessed parameters include portal vein diameter, portal vein flow velocity, portal flow volume, splenic vein flow velocity, portal venous pulsatility measures, and hepatic artery resistance index (8). Because these measurements reflect different components of portal and splanchnic haemodynamics, their combined evaluation may provide clinically useful information regarding alterations associated with portal hypertension.

Portal vein dilatation and reduced portal venous velocity have frequently been described in patients with portal hypertension, although the diagnostic performance of an individual Doppler measurement may vary according to disease severity, underlying aetiology, respiratory phase, body habitus, scanning technique, and operator experience (8). Similarly, spleen length is commonly used as an accessible sonographic marker of splenic enlargement, but its relationship with portal venous measurements is not uniform across published studies. Splenic size may be affected by demographic, haematological, infectious, and hepatic factors, and therefore should not be interpreted as an isolated measure of portal pressure (5,9).

Previous investigations have supported an association between portal haemodynamic disturbance and splenic enlargement, but the magnitude and independence of relationships involving portal vein diameter, portal vein velocity, portal flow, splenic vein velocity, portal venous indices, and hepatic arterial resistance remain variable (5,6,8). Some of this variation may arise from differences in patient selection, disease aetiology, ultrasound protocols, measurement definitions, and statistical modelling. Evaluation of multiple Doppler parameters within the same clinical sample may therefore help determine which measurements are most closely associated with spleen dimensions.

Accordingly, this study aimed to evaluate the relationships between Doppler-derived portal haemodynamic parameters and sonographically measured spleen size among adult patients with portal hypertension and splenomegaly. The study specifically examined the associations of portal vein diameter, portal vein velocity, portal flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, and hepatic artery resistance index with spleen size and assessed which variables remained independently associated with splenic enlargement in a multivariable model.

MATERIALS AND METHODS

A cross-sectional observational study was conducted in the diagnostic ultrasound unit and radiology department of Al Razi Institute, Lahore, Pakistan. Data were collected over a two-month period after

approval of the research synopsis. The study population comprised adult patients presenting for radiological evaluation who had portal hypertension and sonographically detectable splenomegaly. The cross-sectional design was selected to examine the relationships between portal haemodynamic measurements and spleen size at a single clinical assessment.

A total of 53 participants were enrolled through convenience sampling during the study period. Patients were eligible if they were aged 18 years or older, had portal hypertension established through available clinical, laboratory, or imaging evidence, and had splenic enlargement detectable on abdominal ultrasonography. Patients were excluded if they had undergone splenectomy or partial splenic embolization, had portal vein thrombosis or hepatic vein obstruction, had a primary haematological disorder associated with splenomegaly such as leukaemia or lymphoma, or were receiving non-selective beta-blockers or other medications known to influence portal pressure. These criteria were applied to reduce the influence of non-portal causes of splenic enlargement and major vascular or therapeutic factors that could alter portal haemodynamic measurements.

All participants underwent abdominal grey-scale and Doppler ultrasonography in the radiology department. Grey-scale assessment was used to evaluate the portal venous system and document spleen size, while Doppler assessment was used to obtain vascular and flow-related measurements. The recorded study variables included portal vein diameter, portal vein velocity, portal vein flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, hepatic artery resistance index, and spleen size. Portal vein diameter represented the measured calibre of the main portal vein. Portal vein velocity and splenic vein flow velocity represented Doppler-derived venous blood-flow velocities, whereas portal vein flow represented the estimated volume of blood passing through the portal vein. Portal vein pulsatility and resistance indices were recorded from the portal venous waveform, and hepatic artery resistance index was derived from hepatic arterial Doppler measurements. Spleen size was documented from the sonographic splenic measurement recorded during the examination. Doppler ultrasonography was used because it permits non-invasive assessment of portal blood-flow direction, velocity, vascular calibre, and resistance-related waveform characteristics (8).

Participant age and gender were recorded as demographic variables. Age was analysed as a continuous variable, while gender was treated as a categorical covariate in the multivariable model. Spleen size was designated as the dependent variable. Portal vein diameter, portal vein velocity, portal vein flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, hepatic artery resistance index, age, and gender were evaluated as explanatory variables. The same set of haemodynamic measurements was used consistently in the descriptive, correlation, and regression analyses.

Data were entered and analysed using IBM SPSS Statistics version 25.0. Continuous variables were summarized using the mean, standard deviation, minimum, and maximum, while categorical variables were presented as frequencies and percentages. The distribution of splenic vein flow-velocity values was additionally summarized using frequency categories. Pearson correlation analysis was used to estimate linear relationships among continuous haemodynamic variables and spleen size. Spearman rank correlation analysis was also performed to examine the direction and magnitude of monotonic relationships and to assess whether the observed associations remained consistent when analysed using a rank-based method. Correlation coefficients were interpreted according to their magnitude and direction, and two-sided p-values were reported.

A multiple linear regression model was constructed with spleen size as the dependent variable. Gender, age, portal vein diameter, portal vein velocity, portal vein flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, and hepatic artery resistance index were entered as explanatory variables. Unstandardized regression coefficients, standard errors, standardized beta coefficients, t statistics, and p-values were calculated. Overall model performance was summarized using the correlation coefficient, coefficient of determination, adjusted coefficient of determination, standard

error of the estimate, and analysis-of-variance F statistic. Statistical significance was defined as a two-sided p-value below 0.05.

Because several Doppler variables represent interrelated components of the same portal haemodynamic system, the correlation structure among explanatory variables was considered when interpreting the multivariable findings. Consequently, a statistically significant coefficient in the adjusted model was interpreted as an independent association within the fitted sample rather than proof of diagnostic superiority, causation, or prospective predictive performance. Complete observations were available for the 53 participants included in the reported analyses, as reflected by the valid sample size across the descriptive, correlation, and regression outputs.

RESULTS

All 53 enrolled participants had complete observations for the variables included in the reported analyses. The mean age was 51.83 ± 8.21 years, with a range of 37–66 years. Of the participants, 31 (58.5%) were male and 22 (41.5%) were female. The mean portal vein diameter was 15.96 ± 1.49 mm, mean portal vein velocity was 14.21 ± 2.15 cm/s, and mean portal vein flow was 942.98 ± 98.06 mL/min. The mean spleen length was 14.94 ± 1.37 cm, ranging from 12 to 17 cm. The remaining Doppler-derived measurements are summarized in Table 1.

Table 1. Demographic and Doppler Ultrasonographic Characteristics of Participants

Variable	n	Mean ± SD or n (%)	Minimum	Maximum
Age, years	53	51.83 ± 8.21	37	66
Male gender	31	31 (58.5)	—	—
Female gender	22	22 (41.5)	—	—
Portal vein diameter, mm	53	15.96 ± 1.49	13	18
Portal vein velocity, cm/s	53	14.21 ± 2.15	11	18
Portal vein flow, mL/min	53	942.98 ± 98.06	792	1135
Portal vein pulsatility index	53	0.508 ± 0.063	0.39	0.61
Portal vein resistance index	53	0.723 ± 0.037	0.66	0.79
Splenic vein flow velocity, cm/s	53	—	9	16
Hepatic artery resistance index	53	0.743 ± 0.028	0.69	0.80
Spleen length, cm	53	14.94 ± 1.37	12	17

SD: standard deviation.

Splenic vein flow velocity ranged from 9 to 16 cm/s. Values of 11 and 12 cm/s were the most frequent, each occurring in 9 participants (17.0%). Values of 9 cm/s were recorded in 7 participants (13.2%), whereas 16 cm/s was observed least frequently, occurring in 4 participants (7.5%) (Table 2).

Table 2. Frequency Distribution of Splenic Vein Flow Velocity

Splenic vein flow velocity, cm/s	n	%
9	7	13.2
10	6	11.3
11	9	17.0
12	9	17.0
13	6	11.3
14	6	11.3
15	6	11.3
16	4	7.5
Total	53	100.0

Pearson correlation analysis demonstrated strong associations between spleen length and all continuous variables examined. Portal vein diameter had the strongest positive correlation with spleen length ($r = 0.981$; 95% CI: 0.967–0.989; $p < 0.001$). Positive correlations were also observed for portal vein pulsatility index, portal vein resistance index, hepatic artery resistance index, and age. Portal vein velocity, splenic vein flow velocity, and portal vein flow were strongly negatively correlated with spleen length (Table 3).

Spearman rank correlations showed a similar pattern and direction of association. Portal vein diameter remained positively correlated with spleen length (ρ = 0.986; p < 0.001), whereas portal vein velocity (ρ = −0.983), splenic vein flow velocity (ρ = −0.978), and portal vein flow (ρ = −0.977) remained negatively correlated with spleen length. All Spearman correlations had p-values below 0.001, supporting the consistency of the observed monotonic relationships.

Table 3. Correlations Between Doppler Parameters, Age, and Spleen Length

Variable	Pearson r	95% CI	p-value	Spearman ρ	p-value
Portal vein diameter	0.981	0.967 to 0.989	<0.001	0.986	<0.001
Portal vein velocity	−0.978	−0.987 to −0.962	<0.001	−0.983	<0.001
Portal vein pulsatility index	0.976	0.959 to 0.986	<0.001	0.977	<0.001
Portal vein resistance index	0.976	0.959 to 0.986	<0.001	0.981	<0.001
Splenic vein flow velocity	−0.976	−0.986 to −0.959	<0.001	−0.978	<0.001
Hepatic artery resistance index	0.966	0.942 to 0.980	<0.001	0.972	<0.001
Age	0.972	0.952 to 0.984	<0.001	0.976	<0.001
Portal vein flow	−0.975	−0.986 to −0.957	<0.001	−0.977	<0.001

CI: confidence interval. Pearson confidence intervals were calculated using Fisher's z transformation. All analyses included 53 participants.

A multiple linear regression model was fitted with spleen length as the dependent variable and gender, age, portal vein diameter, portal vein velocity, portal vein flow, portal vein pulsatility index, portal vein resistance index, splenic vein flow velocity, and hepatic artery resistance index as explanatory variables. The overall model was statistically significant, $F(9,43) = 169.79$, $p < 0.001$, and accounted for 97.3% of the observed variance in spleen length ($R^2 = 0.973$; adjusted $R^2 = 0.967$). The standard error of the estimate was 0.248 cm.

Portal vein diameter was the only variable with a statistically significant adjusted coefficient. Each 1-mm increase in portal vein diameter was associated with an estimated 0.509-cm increase in spleen length after adjustment for the other variables in the model (B = 0.509; 95% CI: 0.231–0.787; standardized β = 0.557; p = 0.001). None of the other variables retained a statistically significant independent association with spleen length, and their confidence intervals included the null value (Table 4).

Table 4. Multiple Linear Regression Analysis of Factors Associated With Spleen Length

Variable	B	SE	95% CI for B	Standardized β	t	p-value
Constant	4.239	7.911	−11.715 to 20.193	—	0.536	0.595
Gender	−0.044	0.073	−0.191 to 0.103	−0.016	−0.600	0.551
Age, years	0.001	0.038	−0.076 to 0.078	0.007	0.031	0.975
Portal vein diameter, mm	0.509	0.138	0.231 to 0.787	0.557	3.690	0.001
Portal vein velocity, cm/s	−0.215	0.283	−0.786 to 0.356	−0.340	−0.762	0.450
Portal vein flow, mL/min	−0.004	0.003	−0.010 to 0.002	−0.256	−1.195	0.239
Portal vein pulsatility index	−11.587	8.418	−28.564 to 5.390	−0.536	−1.376	0.176
Portal vein resistance index	11.568	11.888	−12.406 to 35.542	0.312	0.973	0.336
Splenic vein flow velocity, cm/s	0.062	0.262	−0.466 to 0.590	0.098	0.237	0.814
Hepatic artery resistance index	7.772	11.826	−16.077 to 31.621	0.159	0.657	0.515

Dependent variable: spleen length in centimetres. B: unstandardized regression coefficient; CI: confidence interval; SE: standard error. Gender coding and the reference category should be specified from the original dataset.

Table 5. Multiple Linear Regression Model Summary

R	R^2	Adjusted R^2	Standard error of estimate, cm	F	df	p-value
0.986	0.973	0.967	0.248	169.788	9, 43	<0.001

Although the model showed a high coefficient of determination, the explanatory variables were also extremely strongly intercorrelated. Pairwise Pearson coefficients among several predictors exceeded an absolute value of 0.97. Therefore, the adjusted coefficients should be interpreted cautiously because severe multicollinearity may have increased their standard errors, destabilized their direction and

magnitude, and limited the ability of the model to distinguish the independent contribution of each haemodynamic variable. The findings establish strong associations within this sample but do not demonstrate causation, diagnostic accuracy, or prospective predictive performance.

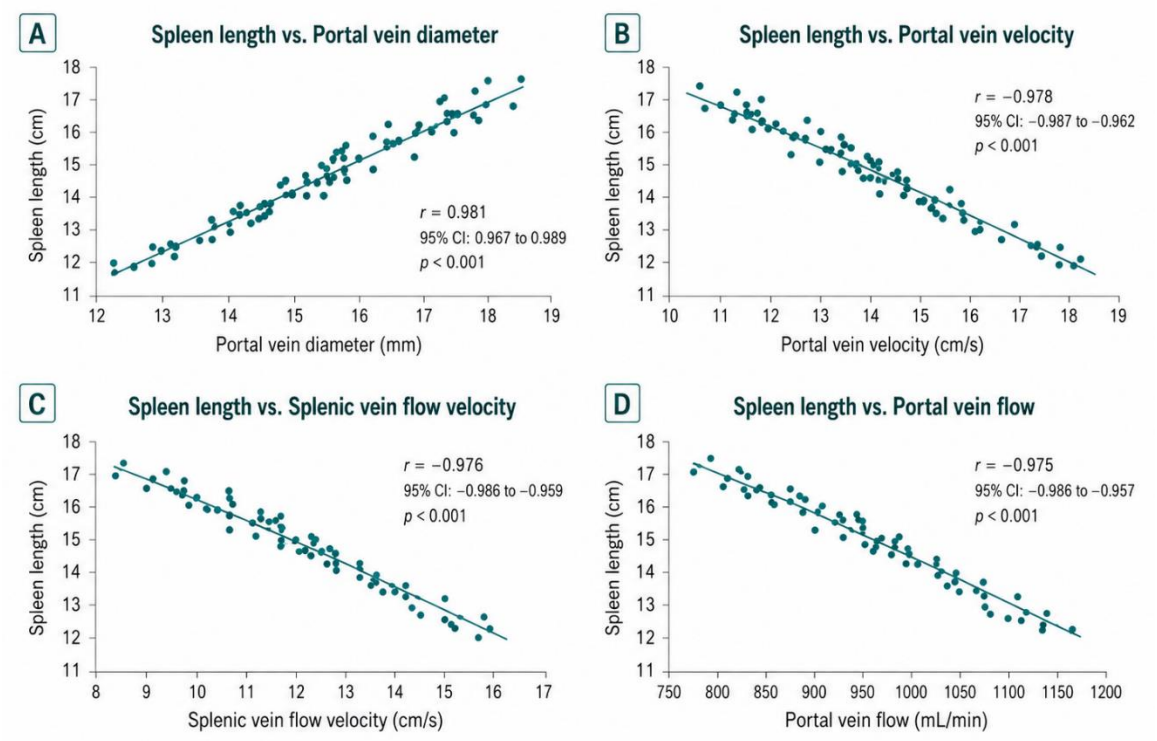

Figure 1 The figure demonstrates strong correlations between spleen length and Doppler parameters in patients with portal hypertension and splenomegaly. Spleen length increased markedly with portal vein diameter (r = 0.981), while it decreased with portal vein velocity (r = −0.978), splenic vein flow velocity (r = −0.976), and portal vein flow (r = −0.975); all associations were statistically significant (p < 0.001).

DISCUSSION

This cross-sectional study evaluated the relationships between spleen length and Doppler-derived portal haemodynamic measurements in 53 adults with portal hypertension and sonographically detectable splenomegaly. Spleen length was strongly positively correlated with portal vein diameter, portal vein pulsatility index, portal vein resistance index, hepatic artery resistance index, and age, while strong inverse correlations were observed with portal vein velocity, splenic vein flow velocity, and portal vein flow. Portal vein diameter remained the only statistically significant variable in the multivariable model; however, the extreme intercorrelations among the explanatory variables require cautious interpretation of their individual adjusted coefficients.

The strongest observed association was between portal vein diameter and spleen length. Enlargement of the portal vein may reflect increased venous pressure, altered vessel compliance, and impaired passage of blood through the hepatic circulation. Increased resistance to portal venous drainage can subsequently produce congestion within the splenic venous bed and contribute to progressive splenic enlargement. Previous studies have similarly described splenic enlargement as an important structural consequence of portal hypertension and have emphasized the interaction between portal venous congestion, splenic blood flow, and remodelling of splenic tissue (4–6). The present findings therefore support a close relationship between portal venous calibre and splenic dimensions, although portal vein diameter should not be interpreted as a direct substitute for invasively measured portal pressure.

Portal vein velocity demonstrated a strong inverse relationship with spleen length. Reduced hepatopetal portal velocity may occur when intrahepatic vascular resistance increases and the forward pressure gradient through the liver becomes impaired. As portal flow slows, venous congestion may increase

within the splanchnic and splenic circulations. Doppler ultrasonography has previously been used to identify reduced portal flow velocity and altered portal waveforms in patients with portal hypertension, although measurements may vary according to disease severity, respiratory phase, body position, equipment settings, and operator technique (8). The current findings are consistent with this physiological pattern but cannot determine whether reduced velocity preceded or resulted from increasing splenic enlargement.

Splenic vein flow velocity was also inversely correlated with spleen length. This finding suggests that greater splenic enlargement was associated with slower measured venous flow within the splenic circulation. Splenic enlargement in portal hypertension is influenced by venous congestion, increased splenic blood pooling, tissue hyperplasia, angiogenesis, and fibrogenic remodelling rather than by a single passive mechanism (4–7). However, the relationship between splenic vein velocity and splenic size requires careful interpretation because velocity is influenced by vessel calibre, Doppler insonation angle, respiratory variation, and the haemodynamic state of the patient. Importantly, splenic vein velocity and portal vein velocity were positively intercorrelated in the reported dataset, indicating that both measurements changed in the same direction across participants.

Portal vein flow showed a strong negative correlation with spleen length. This result indicates that participants with larger spleens generally had lower estimated portal flow volumes. Portal flow estimates combine vessel cross-sectional area and Doppler-derived velocity and may therefore be affected by measurement error in either component. Because portal vein diameter and velocity were themselves strongly intercorrelated with other Doppler variables, the apparent relationship between calculated portal flow and spleen length may partly reflect shared mathematical or physiological information. Accordingly, portal flow should be interpreted as one component of a multiparametric haemodynamic assessment rather than as an isolated marker.

Portal vein pulsatility index, portal vein resistance index, and hepatic artery resistance index were positively associated with spleen length in the bivariate analyses. These findings may reflect increasing vascular resistance and altered waveform characteristics accompanying portal hypertension. The hepatic arterial response may also change when portal perfusion is reduced because hepatic arterial flow can partially compensate for diminished portal venous inflow. Nevertheless, portal venous resistance and pulsatility indices are not uniformly defined or applied across clinical ultrasound protocols. Their clinical interpretation therefore depends on consistent calculation methods, standardised acquisition procedures, and validation against established markers of portal hypertension.

Age was strongly correlated with spleen length and with several haemodynamic measurements in the unadjusted analysis but was not independently associated with spleen length in the multivariable model. This may indicate that the apparent age relationship was largely shared with the Doppler variables included in the model. Gender was also not independently associated with spleen length. These results suggest that haemodynamic characteristics were more closely associated with splenic dimensions than the demographic variables assessed, although the small convenience sample limits firm conclusions regarding demographic differences.

The regression model accounted for 97.3% of the observed variance in spleen length, and portal vein diameter remained statistically significant after adjustment. Each 1-mm increase in portal vein diameter was associated with an estimated 0.509-cm increase in spleen length. Despite the high coefficient of determination, this result should not be regarded as evidence of diagnostic accuracy or reliable prospective prediction. Most explanatory variables had pairwise correlations exceeding an absolute value of 0.97, indicating severe multicollinearity. Under these conditions, regression coefficients may become unstable, confidence intervals may widen, and the direction or magnitude of individual coefficients may change substantially with small alterations in the dataset. The non-significance of other Doppler variables in the adjusted model therefore does not demonstrate that they lack clinical relevance.

The extreme similarity of the Pearson and Spearman correlation estimates indicates that the relationships were both strongly linear and monotonic within the analysed sample. Although this agreement supports internal consistency between the two analytical approaches, the unusually high magnitude of the correlations warrants verification using the original participant-level dataset and complete statistical output. Future analyses should include variance inflation factors, tolerance statistics, condition indices, residual plots, influential-case assessment, and external validation in an independent sample. Dimension-reduction methods or a prespecified composite haemodynamic index may be more appropriate than simultaneously entering multiple nearly collinear Doppler variables into one regression model.

The findings have potential clinical relevance because Doppler ultrasonography is non-invasive, widely available, repeatable, and capable of assessing both vascular structure and blood-flow characteristics. Evaluation of portal vein diameter, portal velocity, portal flow, splenic vein velocity, hepatic arterial resistance, and spleen length may provide a broader haemodynamic profile than any single measurement. Nevertheless, the current study did not compare Doppler findings with the hepatic venous pressure gradient, elastography, endoscopic findings, clinical decompensation, or another reference standard. It therefore cannot establish diagnostic thresholds, disease staging accuracy, or superiority of one Doppler parameter over another.

This study has several strengths. Multiple portal and hepatic Doppler parameters were examined within the same clinical sample, and both Pearson and Spearman analyses were used to assess the consistency of the associations. The study also evaluated adjusted relationships using a multivariable model. However, the limitations are substantial. The sample was small, obtained through convenience sampling, and recruited from a single centre. The cross-sectional design prevents determination of temporal or causal relationships. All included participants had detectable splenomegaly, so the study did not compare patients with and without splenic enlargement. Portal hypertension was not quantified using an invasive reference standard, and the underlying aetiology and clinical severity of liver disease were not incorporated into the analysis. Doppler ultrasonography is operator-dependent, while respiration, body habitus, vessel measurement, insonation angle, and equipment settings may influence the findings. Inter-observer and intra-observer reliability were not reported. Finally, severe multicollinearity and the exceptionally high correlations among most study variables limit the stability and generalisability of the statistical model.

Future studies should use larger multicentre samples, consecutive recruitment, standardised Doppler acquisition protocols, and blinded repeated measurements. Patients with and without splenomegaly should be compared, and Doppler findings should be evaluated against hepatic venous pressure gradient measurements, elastography, endoscopic indicators, laboratory markers, and clinically relevant outcomes. Longitudinal research would help determine whether changes in portal vein diameter, velocity, flow, and spleen dimensions are associated with disease progression or treatment response.

CONCLUSION

Among adults with portal hypertension and sonographically detectable splenomegaly, spleen length was strongly associated with multiple Doppler-derived haemodynamic parameters. Portal vein diameter showed the strongest positive relationship and remained independently associated with spleen length in the multivariable model, whereas portal vein velocity, splenic vein flow velocity, and portal flow were inversely correlated with spleen length. These findings support the potential value of multiparametric Doppler assessment in characterising portal and splenic haemodynamic changes; however, the extreme multicollinearity, small single-centre sample, and absence of validation against a reference standard require cautious interpretation and independent confirmation.

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