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Sonographic assessment of portal vein haemodynamics in patients with Type 2 Diabetes

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Abstract

Objectives: The aim of this study was to evaluate portal vein Doppler indices in patients with type 2 diabetes without non-alcoholic fatty liver disease and determine their relationship with demographic, anthropometric, and biochemical parameters.

Methods: This prospective case-control study included 85 patients with T2D without sonographic evidence of NAFLD and 138 healthy controls. Demographic, anthropometric, and biochemical data were obtained. Doppler ultrasonography was performed following 8–12 hours of fasting. Portal vein maximum velocity (Vmax), minimum velocity (Vmin), mean flow velocity (MFV), and pulsatility index (PI) were measured. Group comparisons were performed using independent t-test, while Pearson's correlation assessed relationships between Doppler indices and clinical variables. Statistical significance was set at $p < 0.05$.

Results: The mean age of participants with T2D and controls was 53.76 ± 12.61 years and 43.46 ± 13.78 years, respectively. Among males, PVVmax and PVPI were significantly reduced in T2D compared with controls ($p = 0.022$ and $p = 0.025$, respectively). PVPI demonstrated weak negative correlations with BMI ($r = -0.379$, $p = 0.017$) and body weight ($r = -0.363$, $p = 0.023$), while MFV ($r = 0.343$, $p = 0.033$) and PVVmin ($r = 0.403$, $p = 0.011$) showed positive correlations with BMI.

Conclusion: Type 2 diabetes was associated with reduced PVVmax and PVPI, particularly among males, and significant associations between portal vein Doppler indices and BMI, and weight suggesting early hepatic vascular dysfunction.

Keywords: Type 2 diabetes; Portal vein; Doppler ultrasonography; Portal vein pusaltility index

1. Introduction

Type 2 diabetes is a chronic metabolic disorder characterized by insulin resistance, impaired insulin secretion, and persistent hyperglycaemia [1]. The disease has become a major global health challenge due to its rapidly increasing prevalence and associated complications. In addition to its well-known macrovascular and microvascular complications, T2D has been associated with alterations in visceral organ perfusion and vascular haemodynamics, including changes within the hepatic circulation [2].

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The liver plays a central role in glucose metabolism and receives approximately 75% of its blood supply from the portal vein. The portal venous system plays a critical role in maintaining hepatic perfusion and metabolic homeostasis. Alterations in portal vein haemodynamics may occur in patients with T2D as a result of endothelial dysfunction, increased vascular resistance, hyperinsulinaemia, and chronic inflammatory changes associated with diabetes [3]. These vascular alterations may precede overt structural liver disease and may be detectable using Doppler ultrasonography

Doppler Ultrasonography is a non-invasive, safe, inexpensive, and readily available imaging modality used in the evaluation of vascular flow characteristics within the liver. Portal vein Doppler indices such as portal vein mean flow velocity, and portal vein pulsatility index provide valuable information about hepatic haemodynamics and have been employed in the assessment of several liver diseases [4]. The technique has gained increasing attention because it allows real-time assessment of blood flow without exposure to ionizing radiation or invasive procedures.

Several studies have demonstrated abnormal portal vein Doppler findings in patients with NAFLD [5,6,7,8,9,10]. Since NAFLD commonly coexists with T2D, many haemodynamic studies in diabetic patients have focused primarily on fatty liver disease. However, it remains unclear whether T2D alone, in the absence of NAFLD, can independently influence portal vein parameters.

Although ultrasonography is widely used for detecting fatty liver, conventional B-mode imaging may not identify early functional vascular alterations occurring in diabetic patients without NAFLD. Doppler assessment of the portal vein may therefore provide additional information regarding subtle hepatic circulatory changes associated with T2D before the development of overt liver pathology. Identifying such changes may contribute to earlier detection of hepatic involvement and improve monitoring of diabetic complications.

There is limited literature specifically evaluating portal vein Doppler indices in patients with T2D without NAFLD. Most available studies have concentrated on diabetic patients with fatty liver disease or on NAFLD populations generally, leaving a gap in knowledge regarding the isolated effect of T2D on portal venous haemodynamics [11]. Consequently, further studies are necessary to determine whether portal vein Doppler indices differ in T2D patients without NAFLD when compared with apparently healthy individuals.

Therefore, this study aims to sonographically evaluate portal vein Doppler indices in patients with type 2 diabetes mellitus without NAFLD and determine their relationship with demographic, anthropometric, and biochemical parameters, as well as the utility of Doppler ultrasonography in detecting diabetes-related hepatic vascular changes.

2. Materials and Methods

This prospective case-control study was conducted among 85 participants with T2D. Additionally, 138 participants with normal clinical and sonographic abdominal findings served as controls. Participant eligibility was verified through detailed medical history obtained via structured interviews. Purposive sampling was employed, and eligible patients with T2D were recruited from the endocrinology clinic after referral for abdominal ultrasonography.

Ethical approval was obtained from the Health Research Ethics Committee of the National Hospital Abuja (NHA/EC/199/2025). All participants provided written informed consent, and confidentiality was strictly maintained.

Patient group inclusion criteria: clinically confirmed T2D, no ultrasound features of nonalcoholic fatty liver disease, no history of syndromic or endocrine causes of obesity. Exclusion criteria: portal vein anatomical abnormalities, severe comorbidities (e.g., malignancy or life-threatening conditions), hepatitis B or C infection, chronic liver disease and previous cholecystectomy. Control group inclusion criteria: Non-diabetic status with normal fasting blood glucose, normal liver findings on ultrasound, normal liver enzyme levels.

Demographic and anthropometric data (height and weight) were obtained, and body mass index (BMI) was calculated as weight/height^2 (kg/m^2). Laboratory investigations included fasting blood sugar (FBS), aspartate transaminase (AST), and alanine aminotransferase (ALT).

Ultrasound examination was performed using a GE Voluson E8 system with a 2.5–9.1 MHz convex transducer. Participants fasted for 8–12 hours. Doppler assessment was performed with an angle <60°. The portal vein was evaluated in the supine position at the midclavicular line during quiet arrested inspiration. Measurements were obtained midway between the portal vein origin and bifurcation.

Portal vein parameters measured included peak maximum velocity (Vmax), peak minimum velocity (Vmin), and mean flow velocity (MFV, time-averaged maximum velocity). The portal vein pulsatility index (PVPI) was calculated as [12]:

$$PVPI = \frac{V_{max} - V_{min}}{V_{max}}$$

Three to five optimal spectral Doppler waveforms were recorded.

Analysis of the data involved calculation of the descriptive statistics (mean ± standard deviation) for Doppler indices and clinical variables. Comparisons between groups were performed using independent t-test. Pearson's product moment correlation analysis assessed relationships between portal vein parameters and clinical variables (age, weight, height, BMI, FBS, AST, ALT). Data was analyzed using IBM SPSS Statistics for Windows, Version 24.0 (IBM Corp., Armonk, NY, USA). Statistical significance was set at $p < 0.05$.

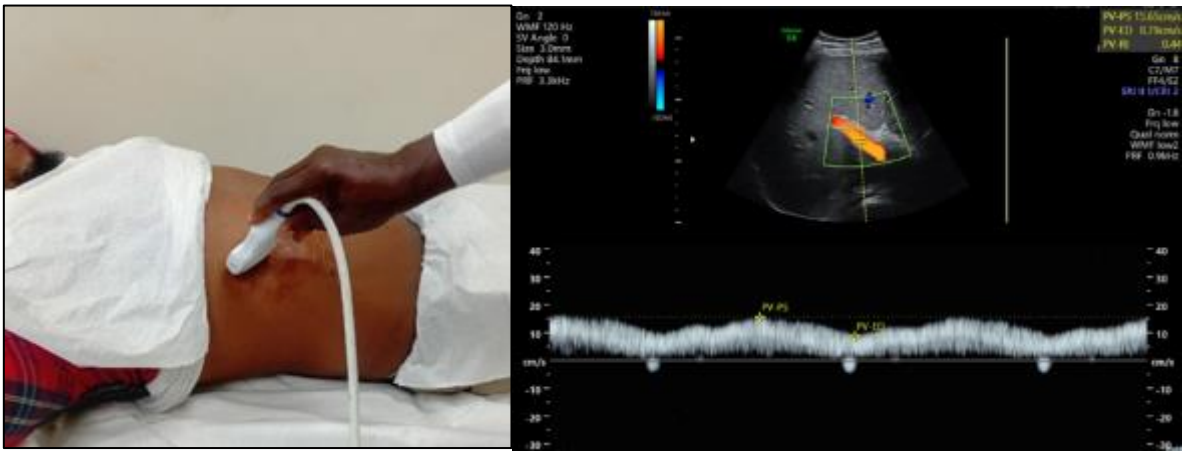

Figure 1 Ultrasound probe position and portal vein Doppler measurement

3. Results

The participants who met the inclusion criteria and participated in this study were 223, comprising 85 individuals with type 2 diabetes (T2D) and 138 healthy controls. Females predominated slightly in both groups, with female-to-male ratios of approximately 8:7 among participants with T2D and 5:4 among controls. The overall mean age of participants with T2D and control participants were 53.76 ± 12.61 years and 43.46 ± 13.78 years, respectively (Table 1 & 2).

Females with T2D had a higher mean body weight than males (83.5 ± 15.4 kg vs. 81.1 ± 13.15 kg). Similarly, the mean BMI was higher among females than males (29.61 ± 6.49 kg/m^2 vs. 27.99 ± 4.73 kg/m^2). The overweight category was the most prevalent BMI group in both sexes, comprising 15 of the 39 males and 14 of the 46 females. Females also exhibited a higher mean fasting blood sugar (FBS) level than males (9.32 ± 1.04 mmol/L vs. 8.49 ± 0.95 mmol/L) (Table 3).

Among the control participants, males had a higher mean body weight than females (75.9 ± 11.5 kg vs. 70.09 ± 10.92 kg). Similarly, the mean BMI was greater in males than females (26.01 ± 4.61 kg/m^2 vs. 24.22 ± 3.86 kg/m^2). The normal-weight category was the most prevalent BMI group in both sexes, comprising 29 of the 60 males and 46 of the 78 females. Males also exhibited a slightly higher mean aspartate aminotransferase (AST) level than females (26.9 ± 7.78 U/L vs. 25.68 ± 8.86 U/L) (Table 4).

Among T2D participants, the overall mean portal vein Vmax, Vmin, mean flow velocity (MFV), and pulsatility index (PI) were 17.49 ± 4.28 cm/s, 12.38 ± 2.50 cm/s, 14.08 ± 3.05 cm/s, and 0.283 ± 0.054, respectively. Corresponding values in controls were 19.29 ± 4.08 cm/s, 13.31 ± 2.35 cm/s, 15.30 ± 2.85 cm/s, and 0.302 ± 0.063, respectively.

Comparison of portal vein parameters between participants with T2D and controls showed significant differences in PV Vmax ($p = 0.022$) and PI ($p = 0.025$) among males, as presented in Table 5.

Among males, significant negative correlation was observed between PVPI and BMI (r = −0.379, p = 0.017). A weak negative correlation was also found between PVPI and body weight (r = −0.363, p = 0.023). Mean flow velocity (MFV) showed significant positive correlation with BMI (r = 0.343, p = 0.033), while a moderate positive correlation was observed between portal vein minimum velocity (PVVmin) and BMI (r = 0.403, p = 0.011). The remaining anthropometric and biochemical parameters demonstrated weak correlations with the Doppler indices (Table 6).

Table 1 Age and gender distribution of participants with T2D

Age (years)	Males		Females		Total	
	Freq (%)	Mean±SD	Freq (%)	Mean±SD	Freq (%)	Mean±SD
18-28	2 (2.35)	27.50±0.71	3 (3.53)	27.00±1.00	5 (5.88)	27.20±0.84
29-39	2(2.35)	38.00±1.41	6 (7.06)	35.33±3.08	8(9.41)	36.00±2.93
40-50	10 (11.77)	46.30±2.00	5(5.88)	44.80±4.76	15(17.65)	45.80±3.10
51-61	12(14.12)	55.00±3.52	19(22.35)	54.37±1.95	31(36.47)	54.61±2.63
62-72	9(10.59)	64.89±2.76	11(12.94)	66.09±3.51	20(23.53)	65.55±3.17
73 -83	4(4.71)	77.00±2.94	2(2.35)	73.50±0.71	6(7.06)	75.83±2.93
Total	39(45.88)	55.03±12.49	46(54.12)	52.70±12.76	85(100.00)	53.76±12.61

Freq: Frequency, SD: standard deviation

Table 2 Age and gender distribution of control participants

Age (years)	Males		Females		Total	
	Freq (%)	Mean±SD	Freq (%)	Mean±SD	Freq (%)	Mean±SD
18-28	5(3.62)	26.20±2.49	11(7.97)	23.09±3.75	16(11.59)	24.06±3.64
29-39	20(14.49)	32.85±3.27	24(17.39)	34.25±2.95	44(31.88)	33.61±3.14
40-50	15(10.87)	43.87±2.62	21(15.22)	44.57±3.54	36(26.09)	44.28±3.17
51-61	10(7.25)	53.90±2.33	14(10.14)	54.93±3.45	24(17.39)	54.50±3.02
62-72	7(5.07)	66.43±2.44	6(4.35)	65.67±3.14	13(9.42)	66.08±2.69
73 -83	3(2.17)	72.67±1.53	2(1.45)	77.50±0.71	5(3.62)	74.60±2.88
Total	60(43.48)	44.53±14.06	78(56.52)	42.64±13.59	138(100.0)	43.46±13.78

Freq: Frequency, SD: standard deviation

Table 3 Anthropometric and biochemical parameters of participants with T2D

Variables		Males		Females		Total	
		Freq	Mean±SD	Freq	Mean±SD	Freq	Mean±SD
Weight (kg)		39	81.10±13.15	46	83.50±15.40	85	82.40±14.38
Height (m)		39	170.59±6.95	46	168.59±6.29	85	169.51±6.64
BMI (kg/m^2)	Normal	10	22.43±1.66	7	19.91±0.65	17	21.39±1.83
	Overweight	15	26.87±1.29	14	25.58±0.65	29	26.25±1.21
	Obese class 1	11	32.06±1.49	12	31.11±1.37	23	31.56±1.48
	Obese class 2	2	35.90±1.13	9	36.29±1.14	11	36.22±1.10
	Obese class 3	1	40.00±0.00	4	41.20±1.33	5	40.96±1.27
	Total	39	27.99±4.73	46	29.61±6.49	85	28.87±5.78

FBS (mmol/L)	39	8.49±0.95	46	9.32±1.04	85	8.93±1.08
AST (U/L)	39	29.21±7.72	46	28.93±6.65	85	29.06±7.11
ALT (U/L)	39	34.38±8.93	46	36.50±8.02	85	35.53±8.47

Freq: Frequency, SD: standard deviation, BMI: body mass index, FBS: fasting blood sugar, AST: aspartate transaminase, ALT: alanine aminotransferase

Table 4 Anthropometric and biochemical parameters of control participants

Variables		**Males**		**Females**		**Total**	
		Freq	**Mean±SD**	**Freq**	**Mean±SD**	**Freq**	**Mean±SD**
Weight (kg)		60	75.90±11.50	78	70.09±10.92	138	72.62±11.50
Height (m)		60	171.23±6.34	78	170.23±6.38	138	170.67±6.36
BMI (kg/m^2)	Normal	29	22.66±1.35	46	21.86±1.68	75	22.17±1.60
	Overweight	21	26.63±1.36	27	26.28±0.99	48	26.43±1.16
	Obese class 1	5	31.40±0.77	2	30.37±0.52	7	31.10±0.83
	Obese class 2	3	35.32±0.07	2	36.29±0.65	5	35.71±0.62
	Obese class 3	2	40.58±0.25	1	40.30±0.00	3	40.48±0.24
	Total	60	26.01±4.61	78	24.22**±**3.86	138	24.99**±**4.28
FBS (mmol/L)		60	4.49±0.42	78	4.34±0.37	138	4.41±0.40
AST (U/L)		60	26.90±7.78	78	25.68±8.86	138	26.21±8.40
ALT (U/L)		60	29.28±8.88	78	27.81±9.73	138	28.45±9.36

Freq: Frequency, SD: standard deviation, BMI: body mass index, FBS: fasting blood sugar, AST: aspartate transaminase, ALT: alanine aminotransferase

Table 5 Difference between portal vein Doppler measurements of T2D and control participants

Portal vein measurements	**Males**			**Females**		
	T2D	**Control**	***p***	**T2D**	**Control**	***p***
Vmax (cm/s)	17.69±3.85	19.77±4.21	0.022	17.32±4.66	18.91±3.97	0.111
Vmin (cm/s)	12.68±2.52	13.45±2.30	0.317	12.13±2.48	13.20±2.40	0.058
MFV (cm/s)	14.35±2.92	15.56±2.86	0.108	13.92±3.17	15.10±2.84	0.094
PI	0.279±0.048	0.311±0.062	0.025	0.287±0.059	0.296±0.063	0.848

Data presented as mean ± STD. Vmax: maximum velocity, Vmin: minimum velocity, MFV: mean flow velocity, PI: pulsatility index

Table 6 Correlation between the portal vein Doppler measurements and some demographic, anthropometric and biochemical parameters in participants with T2D

Variables	Gender	Portal vein							
		Vmax (cm/s)		Vmin (cm/s)		MFV (cm/s)		PI	
		r	*P*	*R*	*p*	*r*	*p*	*r*	*p*
Age (years)	Male	0.061	0.713	0.060	0.718	0.061	0.710	0.009	0.956
	Female	-0.109	0.471	-0.019	0.898	-0.063	0.675	-0.242	0.106
Weight (kg)	Male	0.111	0.500	0.243	0.136	0.189	0.249	-0.363	0.023
	Female	-0.163	0.279	-0.116	0.441	-0.141	0.351	-0.163	0.280
Height (m)	Male	-0.236	0.148	-0.274	0.092	-0.261	0.108	0.041	0.806
	Female	-0.039	0.795	-0.137	0.364	-0.091	0.549	0.194	0.197
BMI (kg/m^2)	Male	0.252	0.122	0.403	0.011	0.343	0.033	-0.379	0.017
	Female	-0.111	0.463	-0.039	0.796	-0.075	0.621	-0.197	0.189
FBS (mmol/L)	Male	-0.053	0.749	-0.063	0.704	-0.059	0.720	0.133	0.420
	Female	-0.154	0.306	-0.110	0.465	-0.133	0.378	-0.213	0.156
AST (U/L)	Male	-0.089	0.589	-0.062	0.709	-0.075	0.652	-0.086	0.604
	Female	0.058	0.700	0.061	0.686	0.060	0.690	0.055	0.718
ALT (U/L)	Male	-0.136	0.408	-0.092	0.577	-0.113	0.494	-0.143	0.384
	Female	0.130	0.390	0.133	0.380	0.133	0.379	0.115	0.447

Vmax: maximum velocity, Vmin: minimum velocity, MFV: mean flow velocity, PI: pulsatility index, BMI: body mass index, FBS: fasting blood sugar, AST: aspartate transaminase, ALT: alanine aminotransferase

4. Discussion

The present study included 85 patients with T2D and 138 healthy controls. Females slightly predominated in both groups, whereas participants with T2D were approximately ten years older than controls. This finding agrees with previous epidemiological studies demonstrating that the prevalence of T2D increases with advancing age because of progressive β-cell dysfunction, increasing insulin resistance, reduced physical activity, and prolonged exposure to metabolic risk factors [12,13].

Anthropometric assessment showed that females with T2D had higher mean body weight and BMI than males, with overweight being the most prevalent BMI category in both sexes. Excess adiposity, particularly visceral fat accumulation, promotes insulin resistance through increased release of free fatty acids, inflammatory cytokines, and adipokines, resulting in impaired glucose homeostasis and progressive metabolic dysfunction [14,15]. Although women exhibited slightly higher BMI in the present study, previous reports suggest that sex-related differences in adipose tissue distribution and hormonal regulation may partly explain variations in metabolic profiles between males and females with T2D [16]. Premenopausal women generally accumulate more subcutaneous adipose tissue, whereas men develop greater visceral adiposity, which has a stronger association with insulin resistance and vascular dysfunction [17].

In this study, there was a statistically significant difference in portal vein maximum velocity (PVVmax) and portal vein pulsatility index (PVPI) between male patients with type 2 diabetes and male controls. This finding may indicate early haemodynamic changes within the hepatic circulation associated with chronic hyperglycaemia and insulin resistance. Diabetes mellitus is known to cause endothelial dysfunction, vascular stiffness, and microvascular alterations, which may affect portal venous blood flow and vascular compliance [18]. The observed alteration in PVPI among diabetic patients may therefore reflect increased intrahepatic vascular resistance or impaired vascular adaptability secondary to diabetic vascular changes.

The finding of altered portal vein Doppler parameters in diabetic patients agrees with previous studies that reported abnormalities in hepatic and portal venous haemodynamics among individuals with metabolic disorders. Targher et al.

[19] noted that chronic metabolic dysfunction in type 2 diabetes may adversely affect hepatic microcirculation before overt structural liver disease becomes apparent. Similarly, Francque et al. [20] reported that metabolic disturbances may increase intrahepatic vascular resistance through endothelial dysfunction and altered vascular architexture. Although many previous studies focused on patients with NAFLD, the present findings suggest that diabetes itself may contribute to haemodynamic changes independent of fatty liver infiltration.

There is significant negative correlation observed among male participants between portal vein pulsatility index and BMI. Also, significant negative correlation was also observed between PVPI and body weight. These findings imply that increasing body size and adiposity may be associated with reduced pulsatility of portal venous flow. Obesity and increased BMI are known to influence vascular resistance and venous return, which may alter portal haemodynamics [21]. Reduced pulsatility may reflect diminished vascular compliance or altered hepatic perfusion associated with metabolic changes occurring in overweight diabetic individuals.

This observation is comparable to reports by Erdogmus et al. [6] who demonstrated alterations in portal vein haemodynamics with increasing metabolic derangement and adiposity. Increased body weight and BMI may contribute to low-grade systemic inflammation and insulin resistance, thereby affecting hepatic vascular regulation. The negative relationship between PVPI and anthropometric indices observed in the present study may therefore indicate progressive haemodynamic adaption associated with worsening metabolic status even in the absence of NAFLD.

Interestingly, portal vein minimum velocity demonstrated a moderate positive correlation with BMI in males. Although this finding may appear paradoxical, it may represent a compensatory haemodynamic response during the early stages of diabetic vascular disease. Experimental study has shown that insulin resistance increases splanchnic blood flow and portal venous inflow before the development of advanced hepatic fibrosis or portal hypertension [22]. As hepatic vascular resistance gradually increases, maintenance of basal portal venous flow may require relatively higher minimum flow velocities throughout the cardiac cycle. Similar compensatory mechanisms have been described in early metabolic liver disease, where total portal blood flow remains relatively preserved despite alterations in vascular compliance [23].

Likewise, significant positive correlation was noted between mean flow velocity and BMI in males, which suggests that obesity influences portal venous haemodynamics through complex adaptive mechanisms. Increased metabolic demand associated with obesity may initially increase hepatic blood flow; however, progressive endothelial dysfunction and hepatic steatosis eventually impair vascular compliance and reduce flow efficiency [22]. This may explain why PVPI decreased while MFV demonstrated a positive association with BMI. Together, these findings indicate that different Doppler indices reflect distinct aspects of portal venous physiology, with PVPI appearing more sensitive to changes in vascular compliance than mean flow velocity.

The predominance of significant findings among male participants may be related to sex-related differences in fat distribution, hormonal influences, and metabolic risk profiles. Males tend to exhibit greater visceral adiposity and are more susceptible to adverse metabolic and vascular effects associated with insulin resistance [24]. This may explain the more pronounced haemodynamic alterations observed among males in the present study.

The findings of this study have important clinical implications. Portal vein Doppler ultrasonography is inexpensive, non-invasive, widely available, and easily incorporated into routine abdominal ultrasound examinations. The observed reductions in PVVmax and PVPI suggest that Doppler ultrasound may detect early hepatic vascular dysfunction before the onset of clinically apparent liver disease or significant biochemical abnormalities. Early identification of such haemodynamic alterations could facilitate timely lifestyle modification, optimisation of glycaemic control, weight reduction, and cardiovascular risk management, thereby reducing the progression of hepatic and systemic vascular complications in patients with T2D.

The limitation of this study was that liver biopsy, transient elastography, or magnetic resonance imaging were not performed to exclude subclinical hepatic steatosis or fibrosis. Consequently, subtle structural liver abnormalities may have contributed to the observed haemodynamic changes. Hence, future multicentre longitudinal studies incorporating elastography, MRI-based liver fat quantification, and histopathological correlation are recommended.

5. Conclusion

This study demonstrated that type 2 diabetes mellitus is associated with alterations in portal vein haemodynamics, characterized by reduced portal vein maximum velocity and pulsatility index, particularly among males, suggesting possible sex-related difference in hepatic vascular effects of T2D. Significant associations between portal vein Doppler

indices and obesity-related anthropometric parameters further suggest that obesity may contribute to early hepatic vascular dysfunction in T2D. These findings support the potential role of Doppler ultrasonography as a non-invasive tool for detecting early hepatic vascular changes before the onset of clinically apparent fatty liver disease.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there are no conflicts of interest.

Statement of ethical approval

Ethical approval for this study was obtained from the Health Research Ethics Committee of the National Hospital Abuja (Approval No. NHA/EC/199/2025).

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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