Portal Vein Doppler Velocimetry as a Non-Invasive Tool for Assessing Liver Fibrosis in Hepatic Steatosis: A Comparative Study from Nnewi, Nigeria

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ABSTRACT

Background: Hepatic steatosis is a major global health burden, often progressing to fibrosis, cirrhosis, and hepatocellular carcinoma. Liver biopsy remains the gold standard for fibrosis assessment, but its invasiveness limits its widespread use. Portal vein Doppler velocimetry and Serum biomarkers, such as the Aspartate Aminotransferase to Platelet Ratio Index (APRI), which provides biochemical insight, offer a non-invasive alternative for detecting hemodynamic changes associated with fibrosis.

Objective: To evaluate the correlation between portal vein Doppler velocimetry parameters and serum biomarkers of liver fibrosis in patients with hepatic steatosis, compared with apparently healthy controls in Nnewi, Nigeria.

Methods: A comparative cross-sectional study of 180 participants (90 with hepatic steatosis, 90 healthy controls) was conducted at Nnamdi Azikiwe University Teaching Hospital, Nnewi. Doppler parameters, including Peak Systolic Velocity (PSV), End Diastolic Velocity (EDV), and Portal Vein Pulsatility Index (PVPI), were measured. Serum biomarkers (AST, platelet count, APRI score) were analysed.

Results: Hepatic steatosis patients showed increased portal vein diameter (1.7 mm; 95% CI: 1.39–2.01) and PVPI (0.10; 95% CI: 0.081–0.119), with reduced PSV (−4.9 cm/s; 95% CI: −7.07 to −2.73) and EDV (−2.3 cm/s; 95% CI: −3.28 to −1.32) ($p < 0.001$). PSV decreased and PVPI increased with steatosis severity ($p = 0.002$ and $p < 0.001$). AST and APRI were higher, while platelet counts were lower ($p < 0.001$). PSV correlated inversely with APRI ($r = −0.243$, $p = 0.022$).

Conclusion: Portal vein Doppler velocimetry demonstrates significant alterations in hepatic steatosis and correlates with serum biomarkers of fibrosis. It represents a promising, cost-effective, non-invasive tool for fibrosis assessment in resource-limited settings.

Keywords: Hepatic steatosis, Portal vein Doppler, Liver fibrosis, APRI score, Non-invasive diagnostics.

INTRODUCTION

The normal adult liver is a wedge-shaped solid organ with a smooth outline. Sonographically, normal liver parenchymal echotexture is homogeneous, slightly more reflective than adjacent renal cortex and similar to or a bit less than that of the spleen. The portal triads are seen as echogenic foci well out into the periphery of the liver. Fissures and ligaments are usually invested in fat and are highly echogenic.[1]

Hepatic steatosis, fatty liver disease, occurs when abnormal lipid accumulation in hepatocytes exceeding 5% of liver weight. It encompasses a spectrum ranging from simple steatosis to steatohepatitis, fibrosis, cirrhosis, and hepatocellular carcinoma (HCC) [2,3]. Globally, fatty liver disease is the leading cause of chronic liver disease, with prevalence rising due to obesity, diabetes, and metabolic syndrome [4,5].

Alcoholic fatty liver disease (AFLD) remains a major contributor to cirrhosis worldwide [6]. However, non-alcoholic fatty liver disease (NAFLD) plays a significant role in hepatic steatosis, especially in individuals with

diabetes and other features of the metabolic syndrome[7]. NAFLD affects approximately 30% of adults in Western countries and 9–31% in developing nations, including Nigeria [8,9]. A study by Ekhorose estimated the prevalence of NAFLD in Abuja, Nigeria, at 25.5% in non-obese subjects[10]. Chukwurah et al[11] reported a prevalence of NAFLD among suspected liver disease patients in Nnewi as 38.2%. In Africa, data on the prevalence of NAFLD are limited. A meta-analytic study reported a prevalence of 13·5%, ranging from 9% in Nigeria to 20% in Sudan[12,13]. Riazi et al. [14] in recent research done in Canada estimated the global prevalence of NAFLD at 32.4%.

Fibrosis is the most important prognostic determinant in fatty liver disease [15]. Early detection is crucial to prevent progression to cirrhosis and HCC. While liver biopsy remains the gold standard, its invasiveness, cost, and sampling errors limit its utility [12]. Non-invasive alternatives such as transient elastography (Fibro Scan) and serum biomarkers (APRI, FIB-4) have gained prominence [16,17].

The portal vein is visible on ultrasound as it passes towards the liver posterior to the common bile duct and hepatic artery. It is a tubular, branching anechoic structure traversing the liver with echogenic walls. The portal veins can be differentiated sonographically from the hepatic veins by the bright echogenic walls that surround them. This is due to the thick collagenous tissue in the portal vein walls. The hepatic veins do not exhibit echogenic borders[18]. The diameter of the portal vein is highly variable but usually less than 13mm measured at the broadest point just distal to the union of the splenic and superior mesenteric vein[19]. The portal vein normally exhibits a monophasic, low-velocity Doppler signal, with slight respiratory variation. The normal range of flow velocity is wide but is usually between 20 and 40 cm/sec[18]. The flow is continuous and should demonstrate little or no pulsatility.

Portal vein Doppler velocimetry is a non-invasive ultrasound technique that measures blood flow velocity and pulsatility in the portal vein. Alterations in these parameters reflect vascular compliance changes associated with fibrosis[20,21]. However, few studies have directly compared Doppler findings with serum biomarkers in hepatic steatosis patients.

Liver steatosis is a common finding among patients undergoing abdominal imaging[8]. Overall, it is increasing rapidly with great changes in lifestyle in Nigeria and varies with age, gender, race and aetiology[22] which affects its morbidity and mortality[23].

Ultrasound as an imaging modality is widely available, cheap, non-invasive and safe. It is highly sensitive for detecting steatosis with a specificity of 84–93.6%[24]. Therefore, it is the most frequently used tool in clinical practice and recommended for screening and diagnosing hepatic steatosis in asymptomatic and high-risk individuals[25].

At present, there is a paucity of data on the impact of fatty liver/hepatic fibrosis on portal venous haemodynamics.

Serum biomarkers offer a minimally invasive approach to assessing liver fibrosis by measuring substances in the blood that correlate with fibrosis severity. Alanine transaminase (ALT), aspartate aminotransferase (AST), gamma glutamyl transferase (GGT) and platelet levels have been associated with inflammation and steatosis; however, each marker alone does not correlate with the degree of fibrosis. Hence, several combined serological markers such as APRI (AST to Platelet Ratio Index) score, have been validated for diagnostic and prognostic scores as an alternative to liver biopsy[26]

This study aims to evaluate portal vein Doppler velocimetry as a non-invasive diagnostic tool for liver fibrosis in hepatic steatosis, comparing findings with APRI scores and healthy controls in Nnewi, Nigeria.

METHODOLOGY

This is a comparative cross-sectional study conducted at Nnamdi Azikiwe University Teaching Hospital, Nnewi.

Participants: This study recruited 90 patients with fatty liver disease detected through ultrasound and 90 apparently healthy controls. This will bring the total sample size to 180.

Inclusion Criteria

1. Patients with hepatic steatosis aged 18 years and above.

2. All consenting age and sex matched apparently healthy adults were recruited to serve as controls.

Exclusion Criteria

Subjects with advanced liver pathology such as hepatocellular carcinoma, portal hypertension.

Data Collection and Ultrasonographic Assessment

Participants were recruited consecutively from patients presenting for abdominal ultrasonography at the Radiology Department of Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi, during the study period. The study group comprised individuals diagnosed with hepatic steatosis based on predefined ultrasonographic criteria, while age- and sex-matched controls without sonographic evidence of hepatic steatosis were selected from the same population. Written informed consent was obtained from all participants prior to enrolment.

All ultrasound examinations were performed using a standardized protocol to ensure consistency and reproducibility. Each participant was examined in the supine position with the abdomen exposed. A coupling gel was applied to enhance acoustic transmission. B-mode transabdominal ultrasonography of the liver was carried out using a 3–5 MHz convex array transducer, with scans obtained in sagittal, transverse, and oblique planes.

The diagnosis of hepatic steatosis was established sonographically based on accepted criteria, including increased hepatic echogenicity relative to the renal cortex, and reduced visualization of intrahepatic vascular structures and the diaphragm. To minimize observer variability, all scans were performed by experienced radiologists using the same equipment and imaging settings.

Hepatic steatosis was further graded according to the degree of echogenicity and beam attenuation into mild, moderate, and severe categories, as defined in the study protocol (grading criteria outlined below)[27].

Grade 0 (Normal) - No fatty liver.

Grade 1 (Mild) - Slight diffuse increase in liver echogenicity; liver parenchyma is slightly brighter than the renal cortex.

Grade 2 (Moderate) - Moderate diffuse increase in liver echogenicity; liver appears significantly brighter than the adjacent renal cortex, with poor visualisation of the intrahepatic vessel walls.

Grade 3 (Severe) - Markedly increased liver echogenicity, with non-visualisation of the hepatic vessels and diaphragm.

Technique Of Portal Vein Velocimetry

With the patient in that same supine position or turned left lateral, the gain, frequency, and depth of the ultrasound machine were optimised. The portal vein velocimetry was evaluated using the low-frequency convex probe. All portal vein measurements were taken midway between the confluence of the splenic and superior mesenteric veins and the bifurcation of the portal vein during suspended inspiration. At Doppler angle < 60°, PSV, EDV, mean flow (MFV) velocities (cm/ s) and PVPI were recorded for each patient.

To minimize operator-dependent variability, all Doppler measurements were performed using a standardized protocol and averaging of three consecutives waveforms by experienced radiologists ensure consistency. Observer bias was reduced through the use of objective Doppler parameters and independent re-evaluation of stored images by a second radiologist blinded to clinical information and study grouping. Inter- and intra-

observer reliability were assessed using the intraclass correlation coefficient (ICC), with values ≥0.75 considered indicative of good agreement.

Blood sample collection: The patient sits in a comfortable position. A suitable vein, typically in the antecubital fossa or dorsal hand veins, was selected. The selected site was cleaned with an alcohol swab after proper application of a tourniquet. 5ml of blood was collected into a serum separating tube. Gentle pressure was applied on the puncture site, tourniquet was released and removed. Each sample container was properly labelled to ensure proper identification and handling. The sample collected was sent to the NAUTH laboratory for evaluation of AST level and platelet count

Using the AST and platelet count obtained from the laboratory, APRI score was calculated using the formula:

$$\text{APRI} = \frac{\text{AST level}}{\text{AST (upper limit of normal)}} \div \text{Platelet } 10^9/\text{L} \times 100$$

APRI SCORE	DEGREE OF FIBROSIS
<0.5	Minimal to no fibrosis
0.5 – 1.5	Significant fibrosis
> 1.5	Advanced fibrosis or cirrhosis

Data Analysis

Data obtained from this study were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA).

Continuous variables were summarised as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Independent sample t-tests were used to compare means between cases and controls.

To provide more informative and reliable estimates 95% confidence intervals (CI) for mean differences and effect sizes (Cohen's d) were calculated. Effect sizes were interpreted as small (0.2), moderate (0.5), and large (≥0.8).

One-way analysis of variance (ANOVA) was used to compare Doppler parameters across grades of hepatic steatosis.

Pearson's correlation coefficient was used to assess relationships between portal vein Doppler parameters and APRI score.

To control for confounding variables, multivariate linear regression analysis was performed adjusting for age and body mass index (BMI) to determine independent predictors of APRI score and Doppler parameters.

Ethical Consideration

Approval for this study was obtained from the Research Ethics Committee of the Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi.

RESULTS

A total of 180 participants (90 cases and 90 controls) were used for this study. The mean age of the study group is 46.29 years ± 17.32, similar to the controls, 46.51 years ± 17.29. In each group, there were 41.1% female and 58.9% male. Age ($X^2 = 0.000$, $p = 1.000$) and gender ($X^2 = 0.000$, $p = 1.000$), which implies a well-matched age and sex distribution between the case and control groups. Table 1.

Body mass index (BMI) was higher among patients with hepatic steatosis compared to controls (Table 2) supporting its role as a metabolic risk factor

Portal vein Doppler parameters differed significantly between participants with hepatic steatosis and healthy controls (Table 3).

Portal vein diameter was significantly higher in cases compared to controls (mean difference = 1.7 mm, 95%CI: 1.3–2.1, $p < 0.001$), with a large effect size (Cohen's d = 1.60).

Peak systolic velocity (PSV) was significantly lower in cases (mean difference = −4.9 cm/s, 95% CI: −7.2 to −2.6, $p < 0.001$), with a moderate effect size (d = 0.66).

End-diastolic velocity (EDV) was also reduced (mean difference = −2.3 cm/s, 95% CI: −3.4 to −1.2, $p < 0.001$), demonstrating a moderate effect size.

Portal vein pulsatility index (PVPI) was significantly higher in cases (mean difference = 0.1, 95% CI: 0.08–0.12, f=21.46, $p < 0.001$), indicating increased vascular resistance.

There were no significant changes observed in the portal vein diameter across mild, moderate, and severe steatosis groups (f=0.19, p = 0.825), though PSV showed a remarkable decline from mild to severe steatosis (f=6.64, p = 0.002) Table 4.

Patients with hepatic steatosis demonstrated significantly altered biochemical parameters compared to healthy controls (Table 5). Aspartate aminotransferase (AST) levels were markedly elevated in cases (45.4 U/L vs 25.9 U/L), with a mean difference of 19.5 U/L (95% CI: 16.5–22.5, $p < 0.001$) and a large effect size (Cohen's d = 1.86).

Platelet count was significantly lower in the steatosis group (243.5 $\times 10^9$/L vs 290.7 $\times 10^9$/L), corresponding to a mean difference of −47.2 $\times 10^9$/L (95% CI: −71.1 to −23.3, $p < 0.001$) and a moderate effect size (Cohen's d = 0.58).

Similarly, APRI scores were significantly higher in patients with hepatic steatosis (0.6 vs 0.2), with a mean difference of 0.40 (95% CI: 0.34–0.47, $p < 0.001$) and a large effect size (Cohen's d = 1.63).

Correlation analysis between portal vein Doppler parameters and APRI score is presented in Table 6.

In patients with hepatic steatosis, a statistically significant negative correlation was observed between peak systolic velocity (PSV) and APRI score ($r = -0.243$, $p = 0.022$), indicating that higher APRI scores are associated with lower PSV values.

No statistically significant correlations were found between APRI score and other Doppler parameters, including portal vein diameter ($r = -0.051$, $p = 0.632$), end-diastolic velocity (EDV) ($r = -0.109$, $p = 0.310$), and portal vein pulsatility index (PVPI) ($r = 0.204$, $p = 0.056$).

In the control group, none of the portal vein Doppler parameters demonstrated significant correlations with APRI score: portal vein diameter ($r = -0.049$, $p = 0.650$), PSV ($r = -0.063$, $p = 0.557$), EDV ($r = 0.026$, $p = 0.810$), and PVPI ($r = -0.106$, $p = 0.326$).

After adjusting for age and body mass index (BMI), multivariate linear regression analysis (table 7) demonstrated that peak systolic velocity (PSV) remained an independent predictor of APRI score ($\beta = -0.22$, $p = 0.015$).

Body mass index (BMI) also showed a significant positive association with APRI score ($\beta = 0.19$, $p = 0.028$), indicating that increasing BMI is independently associated with worsening fibrosis indices.

Portal vein pulsatility index (PVPI) demonstrated a positive but non-significant association after adjustment ($\beta = 0.16$, $p = 0.081$). Portal vein diameter and EDV were not independent predictor

Table 1: Sociodemographic characteristics of the study participants

Variables	Group		X^2	p-value
	Case (n=90)	Control (n=90)		
Age (years)				
19-29	21 (23.3)	21 (23.3)		
30-39	15 (16.7)	15 (16.7)		
40-49	14 (15.6)	14 (15.6)	0.000	1.000
50-59	14 (15.6)	14 (15.6)		
60-69	16 (17.8)	16 (17.8)		
70 and above	10 (11.1)	10 (11.1)		
Mean age (±SD)	46.29± 17.32	46.51± 17.29		
Gender				
Female	37 (41.1)	37 (41.1)	0.000	1.000
Male	53 (58.9)	53 (58.9)		
Total	90 (100.0)	90 (100.0)		

Table 2: BMI and APRI score compared between cases and control

Variables	Group		X^2	p-value
	Case (n=90)	Control (n=90)		
BMI (kg/m^2)				
Underweight	3 (3.3)	13 (14.4)		
Normal weight	34 (37.8)	45 (50.0)	12.976	0.005*
Overweight	31 (34.4)	19 (21.1)		
Obesity	22 (24.7)	13 (14.8)		
APRI score				
Minimal to no fibrosis	42 (46.7)	87 (96.7)		
Significant fibrosis	46 (51.1)	3 (3.3)	55.432	<0.001*
Advanced fibrosis or cirrhosis	2 (2.2)	0 (0.0)		
Total	90 (100.0)	90 (100.0)		

Table 3: Comparison of mean Portal vein Doppler velocimetry parameters between groups with effect sizes and 95% confidence interval

Variables	Case (Mean±SD	Control (Mean±SD	Mean difference	95% CI	Cohen' d	p-value
PV Diameter(mm)	13.3±1.2	11.6±0.9	1.7	1.39 to2.01	1.60(large)	< 0.001*
PSV (cm/s)	24.3±7.7	29.2±7.1	-4.9	-7.07to -2.73	0.66 (moderate)	< 0.001*
EDV (cm/s)	11.3±3.3	13.6±3.4	-2.3	-3.28 to -1.32	0.69 moderate	< 0.001*
Portal vein PI	0.5± 0.07	0.4±0.06	0.10	0.081- 0.119	1.55(large)	< 0.001*

Cohen'd interpreted as small (0.2) moderate (0.5) and large(>0.8).CI=Confidence interval

Table 4: One-way ANOVA showing comparison between portal vein Doppler findings with grades of hepatic steatosis.

Portal vein Doppler velocimetry values	Grades of hepatic steatosis (mean±SD)			F-value	p-value
	Mild (n=31)	Moderate (n=34)	Severe (n=25)		
PV Diameter (mm)	13.18± 1.12	13.37± 1.32	13.31± 1.34	0.19	0.825
PSV (cm/s)	27.24± 7.95	24.69± 7.08	20.18± 6.48	6.64	0.002*
EDV (cm/s)	12.03± 3.67	11.13± 3.03	10.92± 3.16	0.93	0.399
Portal Vein PI	0.44±0.05	0.49± 0.05	0.54±0.06	21.46	<0.001*

Table 5: Comparison of mean APRI parameters between groups with Effect sizes and 95% confidence interval

Variables	Case (Mean±SD	Control (Mean±SD	Mean difference	95% CI	Cohen' d	p-value
AST (U/L)	45.4±11.9	25.9±8.6	19.5	16.5 to22.5	1.86(large)	< 0.001*
Platelet(x10^9/L)	243.5±83.4	290.7±79.8	-47.2	-71.1 to -23.3	0.58 (moderate)	< 0.001*
APRI Score	0.6±0.3	0.2±0.1	0.40	0.3.4 to 0.47	1.65 (large)	< 0.001*

Cohen'd interpreted as small (0.2) moderate (0.5) and large (>0.8).CI=Confidence interval

Table 6: Correlation between portal vein Doppler velocimetry values and APRI score in patients with hepatic steatosis and healthy controls

APRI score vs	Case		Control	
Portal vein Doppler velocimetry values	R	p-value	R	p-value
PV Diameter (mm)	-0.051	0.632	-0.049	0.650
PSV (cm/s)	-0.243	0.022*	-0.063	0.557
EDV (cm/s)	-0.109	0.310	0.026	0.810
Portal Vein PI	0.204	0.056	-0.106	0.326

Table 7: Multivariate Linear Regression Analysis Predicting APRI Score

Variable	BETA (β)	95% Confidence Interval	p-value
PSV	-0.22	(-0.39, -0.05)	0.015*
BMI	0.19	(0.02, 0.36)	0.028*
PVPI	0.16	(-0.02, 0.34)	0.081
PV Diameter	-0.04	(-0.20, 0.012)	0.61
EDV	-0.08	(-0.25, 0.09)	0.34
AGE	0.07	(-0.01, 0.15)	0.09*

DISCUSSION

This study demonstrates that patients with hepatic steatosis exhibit significant changes in portal venous hemodynamic, including reduced peak systolic velocity (PSV) and elevated portal vein Pulsatility index (PVPI). These findings are consistent with vascular compliance changes associated with fibrosis, as reported in previous studies.[28,29,30]. The observed reduction in PSV (mean difference = −4.9 cm/s, 95% CI: −7.07 to −2.73) indicates a consistent and clinically meaningful decline in portal venous flow with steatosis. Similarly, the elevation in PVPI (95% CI: 0.081–0.119) reflects increased vascular resistance, supporting its role as a marker of altered hepatic hemodynamics.

Serum biomarkers also showed clear differences between cases and controls. Elevated AST levels (95% CI: 16.5–22.5) and lower platelet counts (95% CI: −71.1 to −23.3) in the steatosis group are consistent with hepatocellular injury and early fibrosis[31,32]. The elevated APRI score (95% CI: 0.34–0.47) further validates its utility as a non-invasive fibrosis marker. Importantly, the inverse correlation between PSV and APRI, although modest (R = -0.243), suggests that as fibrosis severity increases, portal venous flow declines, reinforcing the complementary role of Doppler ultrasound and serum biomarkers and this finding corroborates with previous study. [33,34]

Adjustment for BMI in multivariate analysis highlights the influence of metabolic factors on progression of fibrosis, emphasizing the need to consider obesity-related variables in hepatic disease assessment.

This study is limited by the lack of comparison with established non-invasive fibrosis assessment method such as Fibro Scan and FIB-4 index which could have provided additional validation of the findings.

CONCLUSION

The study highlights the practical relevance of combining Doppler ultrasound with APRI scoring in resource-limited settings. This integrated approach offers a feasible, cost-effective alternative for early detection and monitoring of fibrosis in patients with hepatic steatosis, where access to advanced diagnostic modalities is restricted

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