

Hepatic artery resistive and pulsatility indices in the sonographic evaluation of patients with non-alcoholic fatty liver disease

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Abstract

Non-alcoholic fatty liver disease (NAFLD) is the most prevalent liver disorder, with a rising incidence in recent years. However, its effects on hepatic artery haemodynamics remain inadequately understood. This study aims to evaluate hepatic artery resistive index (HARI) and hepatic artery pulsatility index (HAPI) and to assess their associations with selected demographic, anthropometric, and biochemical parameters. This prospective case-control, hospital-based study was conducted over an eight-month period and included 152 participants with NAFLD and 138 healthy controls. Data collected included age, weight, height, body mass index (BMI), as well as serum aspartate transaminase (AST), alanine aminotransferase (ALT), and fasting blood sugar (FBS) levels. All participants underwent hepatic Doppler ultrasonography, during which HARI and HAPI were measured. Data were analyzed using both descriptive and inferential statistical methods. The mean HARI and HAPI values among participants with NAFLD were 0.639 ± 0.074 and 1.150 ± 0.249 , respectively, compared with 0.707 ± 0.058 and 1.498 ± 0.254 in the control group. Both HARI and HAPI were significantly higher in controls than in participants with NAFLD. Among individuals with NAFLD, HARI and HAPI showed inverse relationships with BMI, and with AST and ALT levels in males. Additionally, both indices were negatively correlated with the severity (grade) of NAFLD. This study demonstrated that hepatic artery resistive and pulsatility indices were significantly lower in patients with NAFLD compared with controls. Moreover, both HARI and HAPI showed a decreasing trend with increasing body mass index and advancing NAFLD grade.

Keywords: Hepatic artery Doppler; Hepatic haemodynamics; liver ultrasound; HARI; HAPI

1. Introduction

Non-alcoholic fatty liver disease (NAFLD) has emerged as a major hepatic health concern, with its prevalence rising in parallel with increasing rates of obesity [1, 2]. Over the past three decades, it has progressed from a relatively under-recognized condition to the leading cause of chronic liver disease worldwide [3, 4]. NAFLD is now a significant contributor to end-stage liver disease and cardiovascular morbidity and is also recognized as a cause of hepatocellular carcinoma, even in the absence of cirrhosis [1, 5, 6]. Notably, cardiovascular disease remains the leading cause of mortality among patients with non-alcoholic steatohepatitis [7, 8]. These trends underscore the need for more sensitive, accessible diagnostic approaches, as undetected and asymptomatic progression of NAFLD may increase the burden of cardiovascular-related deaths.

The diagnosis of non-alcoholic fatty liver disease (NAFLD) can be challenging. Commonly used liver function tests often correlate poorly with histological findings, limiting their utility for both diagnosis and disease severity assessment [9,

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10]. Although liver biopsy remains the gold standard for diagnosing and staging NAFLD, its routine use is restricted due to its invasive nature, limited availability, and potential complications, including bleeding, bile leakage, and pain [11]. Computed tomography (CT) can reliably detect moderate to severe NAFLD, but its sensitivity for mild NAFLD is limited. Additionally, exposure to ionizing radiation represents a significant drawback in its clinical application [12]. Magnetic resonance spectroscopy is highly accurate in diagnosing fatty liver but is limited by high cost and limited availability [12, 13]. In contrast, ultrasonography is widely accessible and commonly used in the evaluation of NAFLD, based on increased hepatic echogenicity resulting from fat accumulation [14]. Hepatic Doppler ultrasonography has also been proposed as a useful tool for assessing both the diagnosis and prognosis of NAFLD, as the disease is associated with alterations in hepatic vascular haemodynamics [15].

These considerations highlight the need for a readily available and reliable method for early detection of hepatic vascular changes in NAFLD. Consequently, this study focuses on the sonographic assessment of hepatic artery resistive and pulsatility indices, and examines their relationship with age, height, weight, BMI, AST and ALT in patients with NAFLD to enhance understanding of the disease progression and support improved clinical management.

2. Materials and Methods

A prospective case-control study conducted between July 2025 to February 2026 among adults aged 18 years and above. The study comprised 152 patients with ultrasound-confirmed non-alcoholic fatty liver disease (NAFLD) without clinically coexisting type 2 diabetes, and 138 individuals with normal clinical and ultrasonographic abdominal findings who served as healthy controls.

Participants were recruited using purposive sampling. Patients with NAFLD were identified from the gastroenterology clinic and referred to the radiology department for abdominal ultrasonography during the study period. All individuals who met the eligibility criteria were enrolled.

Inclusion criteria for the patient group were ultrasound-confirmed fatty liver disease, absence of a clinical history of diabetes, alcohol consumption below 30 g/day for men and 20 g/day for women, and no evidence of syndromic or endocrine-related obesity. Exclusion criteria for the patient group included hepatic artery anomalies, hepatitis B or C infection, chronic liver disease, malignancy or other serious comorbid conditions, and prior cholecystectomy. Control participants were required to have normal liver findings on ultrasound, normal liver enzyme levels, and normal fasting blood glucose, with clinical evaluation performed to exclude any other underlying medical conditions.

Demographic and anthropometric data, including height and weight, were obtained during outpatient consultations. Laboratory assessments included fasting blood sugar (FBS), aspartate transaminase (AST), and alanine aminotransferase (ALT).

2.1. Sonographic Assessment

Ultrasound examinations were performed using a GE Voluson E8 system equipped with a 2.5–9.1 MHz convex transducer with Doppler capability. Participants fasted for 8–12 hours prior to the examination. The liver echogenicity relative to the right kidney was equally recorded. Fatty liver was graded as mild (grade 1), characterized by diffusely increased hepatic echogenicity with periportal and diaphragmatic echogenicity still appreciable; moderate (grade 2), defined as diffusely increased hepatic echogenicity obscuring periportal echogenicity while diaphragmatic echogenicity remains appreciable; and severe (grade 3), marked by diffusely increased hepatic echogenicity obscuring both periportal and diaphragmatic echogenicity [16].

The hepatic artery was assessed using color and pulsed-wave Doppler with an insonation angle of less than 60° and sample volume of 2 mm. Spectral waveforms were obtained during arrested quiet expiration. The hepatic artery resistive index (HARI) and pulsatility index (HAPI) were measured at a point midway between the beginning of the proper hepatic artery to its bifurcation with the patient lying in the supine position. The hepatic artery was evaluated by positioning the ultrasound probe below the right intercostal space and/or left costal margin. The color Doppler was positioned to visualize the proper hepatic artery. Each result was the average of three to five measurements. The hepatic artery resistive (HARI) and pulsatility index (HAPI) generated by the machine were used. All examinations were performed by a single operator with 13 years of experience in ultrasonography.



Figure 1 Ultrasound probe positioning for hepatic artery scanning through the right intercostal space

2.2. Data Analysis

Descriptive statistics (mean and standard deviation) were calculated for HARI, HAPI, and relevant clinical variables. Participants were grouped into NAFLD and age- and gender-matched controls. Differences in HARI and HAPI between groups were assessed using independent t-test. Pearson's correlation analysis was used to evaluate relationships between HARI/HAPI and continuous variables, while Spearman's correlation assessed associations with NAFLD grades. Multiple regression analysis was performed to examine the relationship between HARI/HAPI and BMI/NAFLD grade. Statistical analyses were conducted using IBM SPSS Statistics for Windows, Version 24.0 (IBM Corp., Armonk, NY, USA), with significance set at $p < 0.05$.

2.3. Ethical Considerations

Ethical approval was obtained from the Health Research Ethics Committee of the National Hospital Abuja (NHA/EC/199/2025). All participants were adequately informed about the study and written informed consents were obtained from them. Confidentiality of all participant information was strictly maintained.

3. Results

A total of 290 participants who met the inclusion criteria were enrolled, comprising 152 with NAFLD and 138 healthy controls. Among the NAFLD group, 54.6% were male and 45.4% female, with a mean age of 51.65 ± 12.72 years (range: 18–79 years). The control group included 60 males and 78 females, with a mean age of 43.47 ± 13.78 years (range: 18–77 years) (Table 1 & 2).

Table 1 Age and gender distribution of participants with NAFLD

Age (years)	Males		Females		Total	
	Freq (%)	Mean±SD	Freq (%)	Mean±SD	Freq (%)	Mean±SD
18-28	3(1.97)	26.67±0.58	3(1.97)	25.00±4.36	6(3.94)	25.83±2.93
29-39	10(6.58)	33.30±3.20	7(4.61)	36.71±2.98	17(11.19)	34.71±3.48
40-50	32(21.05)	45.59±3.32	16(10.53)	43.06±2.93	48(31.58)	44.75±3.39
51-61	19(12.50)	54.00±2.98	23(15.13)	55.39±3.17	42(27.63)	54.76±3.13
62-72	12(7.89)	66.17±3.51	15(9.87)	64.27±2.69	27(17.76)	65.11±3.17
73 -83	7(4.61)	75.57±2.44	5(3.29)	74.00±1.22	12(7.90)	74.92±2.11
Total	83(54.60)	50.86±12.96	69(45.40)	52.59±12.47	152(100.0)	51.65±12.72

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

The mean BMI was $32.00 \pm 5.87 \text{ kg/m}^2$ in the NAFLD group and $24.99 \pm 4.28 \text{ kg/m}^2$ in controls. Mean AST and ALT levels were higher in participants with NAFLD ($46.87 \pm 14.59 \text{ U/L}$ and $55.49 \pm 15.66 \text{ U/L}$, respectively) compared with controls ($26.21 \pm 8.40 \text{ U/L}$ and $28.45 \pm 9.36 \text{ U/L}$) (Tables 3 and 4).

Table 3 Anthropometric and biochemical parameters of participants with NAFLD

Variables	Males (n = 83)	Females (n = 69)	Total (n = 152)
	Mean ± SD	Mean ± SD	Mean ± SD
Weight (kg)	93.54±16.36	90.61±14.54	92.21±15.58
Height (m)	170.69±5.41	169.41±5.52	170.11±5.48
BMI ((kg/m ²))	32.25±6.39	31.70±5.22	32.00±5.87
FBS (mmol/L)	4.85±0.41	4.58±0.48	4.73±0.46
AST (U/L)	47.34±13.85	45.70±15.45	46.87±14.59
ALT (U/L)	56.36±14.88	53.97±16.58	55.49±15.66

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 (n = 60)	Females (n = 78)	Total (n = 138)
	Mean ± SD	Mean ± SD	Mean ± SD
Weight (kg)	75.90±11.50	70.09±10.92	72.62±11.50
Height (m)	171.23±6.34	170.23±6.38	170.67±6.36
BMI ((kg/m ²))	26.01±4.61	24.22±3.86	24.99±4.28
FBS (mmol/L)	4.49±0.42	4.34±0.37	4.41±0.40
AST (U/L)	26.90±7.78	25.68±8.86	26.21±8.40
ALT (U/L)	29.28±8.88	27.81±9.73	28.45±9.36

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

Sonographic grading of fatty liver among participants with NAFLD showed that 32.89% had grade 1, 49.35% grade 2, and 17.76% grade 3 fatty liver. Grade 2 NAFLD was most prevalent among males (26.98%), while grade 3 was least frequent among females (8.55%) (Table 5).

Table 5 Distribution of non-alcoholic fatty liver disease grades in participants with NAFLD

Gender	NAFLD grade			
	Grade 1	Grade 2	Grade 3	Total
	Frequency (%)	Frequency (%)	Frequency (%)	Frequency (%)
Males	28 (18.42)	41 (26.98)	14 (9.21)	83(54.61)
Females	22 (14.47)	34 (22.37)	13 (8.55)	69 (45.39)
Total	50 (32.89)	75 (49.35)	27 (17.76)	152 (100.00)

The mean HARI and HAPI in the NAFLD group were 0.639 ± 0.074 and 1.150 ± 0.249 , respectively. Gender-specific means were 0.650 ± 0.072 and 1.168 ± 0.249 in males, and 0.627 ± 0.076 and 1.129 ± 0.249 in females. In contrast, controls had higher mean values: overall HARI and HAPI were 0.707 ± 0.058 and 1.498 ± 0.254 , with male values of 0.710 ± 0.057 and 1.510 ± 0.249 , and female values of 0.704 ± 0.060 and 1.488 ± 0.259 . Differences in HARI and HAPI between NAFLD and control groups were statistically significant for both genders ($p < 0.001$) (Table 6).

Table 6 Difference in hepatic artery Doppler measurements between participants with NAFLD and control participants

Doppler measurements	Males			Females		
	NAFLD	Control	<i>P</i>	NAFLD	Control	<i>p</i>
HARI	0.650 ± 0.072	0.710 ± 0.057	<0.001	0.627 ± 0.076	0.704 ± 0.059	<0.001
HAPI	1.168 ± 0.249	1.510 ± 0.249	<0.001	1.129 ± 0.249	1.488 ± 0.259	<0.001

Data presented as mean $\pm$ STD. HARI: hepatic artery resistive index, HAPI: hepatic artery pulsatility index

Correlation analysis in the NAFLD group revealed that HARI and HAPI were most strongly negatively correlated with BMI in males ($r = -0.687$ and $r = -0.607$, respectively; $p < 0.001$). In females, weak negative correlations were observed between BMI and both HARI ($r = -0.246$, $p = 0.041$) and HAPI ($r = -0.243$, $p = 0.045$). Height showed the strongest positive correlation with HARI in females ($r = 0.508$, $p < 0.001$) and a moderate correlation in males ($r = 0.333$, $p = 0.002$). Weight demonstrated moderate negative correlations with HARI ($r = -0.652$, $p < 0.001$) and HAPI ($r = -0.551$, $p < 0.001$) in males. In males, moderate negative correlations were also observed between HARI and AST ($r = -0.598$, $p < 0.001$) and ALT ($r = -0.600$, $p < 0.001$). Also, HAPI showed a weak negative correlation with age ($r = -0.290$, $p = 0.008$) in males. Other variables showed insignificant associations with Doppler indices (Table 7).

There was a strong negative correlation between HARI and NAFLD grade in males ($r_s = -0.704$, $p < 0.001$) and a moderate correlation in females ($r_s = -0.603$, $p < 0.001$). HAPI also demonstrated moderate negative correlations with NAFLD grade in both males ($r_s = -0.619$, $p < 0.001$) and females ($r_s = -0.643$, $p < 0.001$).

Table 7 Correlation between the hepatic artery Doppler measurements and some demographic, anthropometric and biochemical parameters in participants with NAFLD

Variables	Gender	HARI		HAPI	
		<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
Age (years)	Male	-0.184	0.097	-0.290	0.008
	Female	-0.081	0.508	-0.136	0.266
Weight (kg)	Male	-0.652	<0.001	-0.551	<0.001
	Female	-0.022	0.858	-0.031	0.799
Height (m)	Male	0.333	0.002	0.354	0.001
	Female	0.508	<0.001	0.478	<0.001
BMI (kg/m ²)	Male	-0.687	<0.001	-0.607	<0.001

	Female	-0.246	0.041	-0.243	0.045
FBS (mmol/L)	Male	-0.106	0.342	-0.128	0.251
	Female	-0.063	0.610	0.040	0.745
AST (U/L)	Male	-0.598	<0.001	-0.529	<0.001
	Female	-0.126	0.303	-0.142	0.244
ALT (U/L)	Male	-0.600	<0.001	-0.507	<0.001
	Female	-0.197	0.105	-0.192	0.115

HARI: hepatic artery resistive index, HAPI: hepatic artery pulsatility index, BMI: body mass index, FBS: fasting blood sugar, AST: aspartate transaminase, ALT: alanine aminotransferase

Multiple regression analysis showed that both BMI and NAFLD grade were significant predictors of HARI and HAPI. For HARI, the regression equations were: males: $y = 0.891 - 0.005x_1 - 0.047x_2$, females: $y = 0.828 - 0.003x_1 - 0.064x_2$. For HAPI, the equations were: males: $y = 1.909 - 0.015x_1 - 0.144x_2$, females: $y = 1.758 - 0.009x_1 - 0.188x_2$, where x_1 represents BMI and x_2 represents NAFLD grade.

4. Discussion

Most participants with NAFLD in this study were in their fifth decade of life. This is consistent with findings by Baikpour et al [17], who reported that the majority of the participants with NAFLD in their study were in their 5th decade of life. In contrast, other studies have reported a lower mean age in the fourth decade [18,19], likely reflecting differences in study populations, including racial and demographic variations. Although NAFLD can occur across all age groups, its prevalence is higher among middle-aged individuals [20-21], which may explain the age distribution observed in this study. Additionally, a larger proportion of clinic attendees and consenting participants fell within this age range.

The hepatic artery resistive index (HARI) was significantly higher in controls than in participants with NAFLD. This finding aligns with previous studies which consistently demonstrate significantly lower HARI in NAFLD compared to controls [2, 19, 23-26]. However, Bedewi and Kama [27] reported no significant difference. Unlike many prior studies, the present study excluded individuals with type 2 diabetes, a common comorbidity that may influence vascular parameters. Therefore, the observed reduction in HARI may more accurately reflect NAFLD-related haemodynamic changes. These findings suggest altered hepatic arterial haemodynamics associated with hepatic steatosis and highlight the potential utility of HARI as a non-invasive marker for disease stratification.

Similarly, HAPI was significantly lower in participants with NAFLD compared to controls. This contrasts with the findings of Tarzamni et al [26], who reported no difference. Variations in study outcomes may be attributed to differences in genetics, population characteristics, imaging techniques, and sample sizes. Nonetheless, the present findings indicate notable alterations in hepatic artery hemodynamics among patients with NAFLD.

Among control participants, no significant correlations were observed between Doppler indices and demographic, anthropometric, or biochemical variables. This contrasts with findings in pediatric population which reported a negative relationship between HARI and age among healthy participants [28]. The observed difference is likely due to differences in age groups and study designs.

The correlation analysis demonstrated a negative association between HARI and BMI among participants with NAFLD, irrespective of gender. This finding aligns with Sabry et al [23], who reported a highly significant correlation between HARI and BMI in a similar population. In contrast, no significant relationship between HARI and age was observed in this study, unlike the findings of Sabry et al [23], who reported a significant association. This discrepancy may be attributable to differences in methodology or population characteristics. Additionally, among male participants with NAFLD, HARI showed negative correlations with weight, AST, and ALT, while a positive relationship was observed between HARI and height across both genders. These findings suggest a complex interaction among metabolic state, liver health, and blood flow dynamics, specifically showing that higher vascular resistance (HARI) in the hepatic artery was associated with lower weight and higher liver enzyme levels in men, while overall height influences baseline resistance in all patients.

HAPI showed similar trends, with negative associations with BMI, weight (in males), age (in males), AST, and ALT (in males), and a positive relationship with height. These patterns suggest that increasing obesity and advancing age may

exacerbate hepatic vascular alterations in NAFLD. However, limited comparable literature on HAPI represents a constraint in interpreting these findings, underscoring the need for further research.

A strong inverse relationship was observed between HARI and NAFLD grade in males, and a moderate relationship in females. This finding is consistent with several previous studies that equally reported a negative relationship between HARI and NAFLD grade [19, 22, 25, 29 -32]. These findings indicate that HARI decreases with increasing disease severity. Similarly, HAPI demonstrated a moderate negative correlation with NAFLD grade, although this finding contrasts with Alshehri et al [30], who reported no such association. Differences may reflect variations in study design or population characteristics. These findings suggest that both HARI and HAPI may serve as potential biomarkers of NAFLD severity.

A key limitation of this study is the absence of histological confirmation via liver biopsy, which remains the gold standard for NAFLD diagnosis. Nonetheless, the findings provide valuable insight into the haemodynamic changes associated with NAFLD and support the clinical utility of Doppler ultrasound as a non-invasive assessment tool.

5. Conclusion

Participants with NAFLD exhibited significantly lower hepatic artery resistive and pulsatility indices compared to healthy controls. Both HARI and HAPI decreased with increasing BMI and advancing NAFLD grade, irrespective of gender. These findings suggest that hepatic artery Doppler indices may provide useful non-invasive markers of altered hepatic haemodynamics and may be associated with the severity of ultrasound-detected hepatic steatosis, even in the absence of type 2 diabetes.

Compliance with ethical standards

Disclosure of conflict of interest

The authors report no conflict of interests.

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

Written informed consent was obtained from all participants enrolled in the study.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

References

- [1] Sahu P., Chhabra P., Mehendale A.M. A comprehensive Review on Non-Alcoholic Fatty Liver disease. *Cureus*. 2023; 15(12): e50159. DOI 10.7759/cureus.50159
- [2] Riestra-Candelaria, B.L., González-Rodríguez, L.A., Rodríguez-Mojica, W., Vázquez-Quñones, L.E., and Jorge, J.C. Sonographic Features of the Liver Among Females with Type 2 Diabetes. *Journal of Diagnostic Medical Sonography*. 2019; 35(3):189–197. Doi:10.1177/8756479319834927.
- [3] Younossi, Z., Tacke, F., Arrese, M., Sharma, B.C., Mostafa, I., Bugianesi, E., Wong, V.W-S., Yilmaz, Y., George, J., Fan, J. and Vos, M.B. (2019). Global perspectives on nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. *Hepatology*. 2019; 69(6):2672–2682. Doi: 10.1002/hep.30251.

- [4] Younossi, Z., Anstee, Q. M., Marietti, M., Hardy, T., Henry, L., Eslam, M., George, J. and Bugianesi, E. Global burden of NAFLD and NASH: trends, predictions, risk factors and prevention. *Nature Reviews Gastroenterology & Hepatology*. 2018; 15(1):11–20. Doi: 10.1038/nrgastro.2017.109.
- [5] Estes, C., Razavi, H., Loomba, R., Younossi, Z. and Sanyal, A.J. Modeling the epidemic of nonalcoholic fatty liver disease demonstrates an exponential increase in burden of disease. *Hepatology*. 2018; 67(1):123–33. Doi: 10.1002/hep.29466.
- [6] Targher, G., Bertolini, L., Padovani, R., Rodella, S., Zoppini, G., Pichiri, I., Sorgato, C., Zenari, L. and Bonora, E. Prevalence of non-alcoholic fatty liver disease and its association with cardiovascular disease in patients with type 1 diabetes. *Journal of Hepatology*. 2010; 53(4):713–718. Doi: 10.1016/j.jhep.2010.04.030.
- [7] Angulo, P., Kleiner, D.E., Dam-Larsen, S., Adams, L.A., Bjornsson, E.S., Charatcharoenwithaya, P., Mills, P.R., Keach, J.C., Lafferty, H.D., Stahler, A., Haflidadottir, S. and Bendtsen. Liver fibrosis, but no other histologic features, is associated with long-term outcomes of patients with nonalcoholic fatty liver disease. *Gastroenterology*. 2015; 149(2):389–397. Doi: 10.1053/j.gastro.2015.04.043.
- [8] Targher, G., Byrne, C.D. and Tilg, H. (2020). NAFLD and increased risk of cardiovascular disease: Clinical associations, pathophysiological mechanisms and pharmacological implications. *Gut*. 2020; 69(9):1691–1705. Doi: 10.1136/gutjnl-2020-320622.
- [9] Ulasoglu C., Enc F.Y., Kaya E., Yilmaz Y. Characterization of patients with biopsy-proven non-alcoholic Fatty liver disease and Normal Aminotransferase Levels. *JGLD*. 2019; 28(4):427–431. DOI: 10.15403/jgld-293
- [10] Dyson, J. K., Anstee, Q.M. and McPherson, S. Non-alcoholic fatty liver disease: a practical approach to diagnosis and staging. *Frontline Gastroenterology*. 2014; 5(3):211–218. Doi: 10.1136/flgastro-2013-100403.
- [11] Siege, C.A., Silas, A.M., Suriawinata, A.A. and van Leeuwen, D.J. (2005). Liver biopsy 2005: When and how? *Cleveland Clinic Journal of Medicine*. 72(3):199–208. Doi: 10.3949/ccjm.72.3.199.
- [12] Lee, D.H. Imaging evaluation of non-alcoholic fatty liver disease: focused on quantification. *Clinical and Molecular Hepatology*. 2017; 23(4):290–301. Doi: 10.3350/cmh.2017.0042.
- [13] Lomonaco, R., Bril, F., Portillo-Sanchez, P., Ortiz-Lopez, C., Orsak, B., Biernacki, D., Lo, M., Suman, A., Weber, M.H. and Cusi, K. Metabolic impact of non-alcoholic steatohepatitis in obese patients with type 2 diabetes. *Diabetes Care*. 2016; 39(4):632–638. Doi: 10.2337/dc15-1876.
- [14] Lee, S.S. and Park, S.H. Radiologic evaluation of nonalcoholic fatty liver disease. *World Journal of Gastroenterology*. 2014; 20(23):7392–7402. Doi: 10.3748/wjg.v20.i23.7392.
- [15] Preciado-Puga, M.C., Ruiz-Noa, Y., Garcia-Ramirez, J. R., Jordan-Perez, B., Garnelo-Cabanas, S., Vega-Monroy, M. L., Gutierrez-Aguirre, K.I. and Ibarra-Reynoso, L.R. Non-invasive diagnosis of non-alcoholic fatty liver disease using an algorithm combining clinical indexes and ultrasonographic measures. *Annals of Hepatology*. 2021; 21:100264. Doi: 10.1016/j.aohep.2020.09.008.
- [16] Ferraioli, G. and Soares, M.L. (2019). Ultrasound-Based Techniques for the Diagnosis of Liver Steatosis. *World Journal of Gastroenterology*, 25(40), 6053–6062. Doi: 10.3748/wjg.v25.i40.6053.
- [17] Baikpour, M., Ozturk, A., Dhyani, M., Mercaldo, N.D., Pierce, T.T., Grajo, J.R. and Samir, A.E. Portal venous pulsatility index: A novel biomarker for diagnosis of high-risk nonalcoholic fatty liver disease. *American Journal of Roentgenology*, 2020; 214(4):786–791. Doi:10.2214/ajr.19.21963.
- [18] Solhjoo, E., Mansour-Ghanaei, F., Moulaei-Langorudi, R. and Joukar, F. Comparison of portal vein Doppler indices and hepatic vein doppler waveform in patients with nonalcoholic fatty liver disease with healthy control. *Hepatitis Monthly*. 2011; 11(9):740–744. Doi:10.5812/kowsar.1735143x.729.
- [19] Gbande, P., Soumah, A., Balde, A.A. Tchaou, M., Sonhay, L., Agoda-Koussema, L.K and Adejenou, K. Doppler ultrasound assessment of hepatic hemodynamics in hepatic steatosis: a case-control study. *Egyptian Journal of Radiology and Nuclear Medicine*. 2025; 56(198). Doi: 10.1186/s43055-025-01621-y.
- [20] Pitisuttithum, P. and Treeprasertsuk, S. Nonalcoholic fatty liver disease (NAFLD) among older adults. *Portal Hypertension and Cirrhosis*. 2022; 1(3):184–191. Doi:10.1002/poh2.31.
- [21] Cheng, H-Y., Wang, H-Y., Chang, W-Y., Lin, S-C., Chu, C-H., Wang, T-E., Liu, C-C. and Shih, S-C. Nonalcoholic Fatty Liver Disease: Prevalence, Influence on Age and Sex, and Relationship with Metabolic Syndrome and Insulin Resistance. *International Journal of Gerontology*. 2013; 7(4):194–198. Doi: 10.1016/j.ijge.2013.03.008.

- [22] Ahmed, O.M, Zakir, M., Muhammad S., Farooq Y. and Gilani, S.A. Doppler assessment of portal and hepatic vein alterations in non-alcoholic fatty liver disease. *Imaging*. 2025; [Preprint]. Doi: 10.1556/1647.2025.00321.
- [23] Sabry, M., Youssef, T., Shaker, M., Salama, M.M. Assem, N. and Anwar, C.A. Portal venous and hepatic artery hemodynamic variation in non-alcoholic fatty liver disease. *Egyptian Liver Journal*. 2021; 11(1):58. Doi: 10.1186/s43066-021-00130-7.
- [24] Alizadeh, A., Mansour-Ghanaei, F., Roozdar, A., Joukar, F., Sepehrimanesh, M., Hojati, S.A. and Mansour-Ghanaei, A. Laboratory Tests, Liver Vessels Color Doppler Sonography, and FibroScan Findings in Patients with Nonalcoholic Fatty Liver Disease: An Observation Study. *Journal of Clinical Imaging Science*. 2018; 8(12):1-10. Doi: 10.4103/jcis.JCIS_93_17.
- [25] Balasubramanian, P., Boopathy, V., Govindasamy, E. and Venkatesh, B. P. Assessment of Portal Venous and Hepatic Artery Haemodynamic Variation in Non-Alcoholic Fatty Liver Disease (NAFLD) Patients. *Journal of Clinical and Diagnostic Research*. 2016; 10(8):7-10. Doi: 10.7860/JCDR/2016/20697.8267.
- [26] Tarzamni, M.K., Khoshbaten, M., Sadrarhami, S., Daneshpajouhnejad, P., Jalili, J., Gholamian, M. and Shahmoradi, Z. Hepatic Artery and Portal Vein Doppler Indexes in Non-alcoholic Fatty Liver Disease Before and After Treatment to Prevent Unnecessary Health Care Costs. *International Journal of Preventive Medicine*. 2014; 5(4):472-477. <https://pubmed.ncbi.nlm.nih.gov/24829735/>.
- [27] Bedewi, M.A. and Kama, S. Doppler sonographic hemodynamics in non-alcoholic fatty liver disease. *Journal of International Pharmaceutical Research*. 2019; 46(1):283-286. <http://ijprjournals.com/abstractt.php?id=324>.
- [28] Verhagen, M.V., Kwee, T.C. and de Haas, R.J. Hepatic artery and portal vein Doppler ultrasound reference values in children aged 0–17 years old. *Sage Journals*, 2022; 31(2):112 – 118. Doi: 10.1177/1742271X221114050.
- [29] Mohan, S., Sakalecha, A.K., Raveesha, A., Krishnan, J. and Rashmi, S.N. Portal vein Pulsatility Index as a noninvasive tool for staging nonalcoholic fatty liver disease: A case-control study. *Cureus*. 2025; 17(8):e90377. Doi:10.7759/cureus.90377.
- [30] Alshehri, A.S., Gameraddin, M., Saeedullah, Y., Alaeinbawi, M.H. Altamimi, B.R. Gareeballah, A., Salih, S. and Sindi, M.A. Hepatic artery and portal vein hemodynamics in nonalcoholic fatty liver disease in adult Saudi patients: A Doppler ultrasound study. *International Journal of Biomedicine*. 2023; 13(2):259–264. Doi: 10.21103/article13(2)_oa10.
- [31] Bony, M.N.R.I., Islam, U., Devnath, P., Safrin, N. and Moonmoon, M.A. Duplex Color Doppler Measurement of Hepatic Artery Resistance Index in Evaluation of Severity of Hepatic Steatosis. *International Research Journal of Gastroenterology and Hepatology*. 2020; 3(1):19-26. <https://journalirjgh.com/index.php/IRJGH/article/view/32>.
- [32] Alkabeer, A.M.M., Mansour, T.M.M., Abd El Kader, M.M. and Abd El Naser, M.M. Relationship between Hepatic Artery Resistive Index and Liver Fibrosis Score in Non-Alcoholic Fatty Liver Disease Patient. *The Egyptian Journal of Hospital Medicine*. 2020; 81(5):2093-2098. Doi: 10.21608/ejhm.2020.126410.