



ORIGINAL RESEARCH ARTICLE

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# Portal venous and hepatic artery hemodynamic variation in non-alcoholic fatty liver disease

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## Abstract

**Background:** Non-alcoholic fatty liver disease remains asymptomatic until advanced disease, when risk factor modification and available treatment become no longer effective. Studies on hepatic vasculature can be informative about parenchymal injury and disease severity through the study of changes affecting vascular compliance. This study aimed to study portal vein and hepatic artery hemodynamic variation in non-alcoholic fatty liver and to correlate it with disease severity.

**Results:** This case control study included 80 participants; those were further divided into four groups; healthy volunteers and non-alcoholic fatty liver disease patients' grade 1, 2, and 3. We did anthropometric measures, laboratory tests, transient elastography, and Doppler ultrasound for all participants, and then we collected the data and analyzed it using SPSS version 25.

Doppler findings showed that peak maximum velocity, peak minimum velocity, mean flow velocity, portal vein pulsatility index of portal vein, and hepatic artery resistivity index were significantly lower in non-alcoholic fatty liver disease patients than in healthy people. All indices were indirectly proportionate to the grade of the disease except for peak minimum velocity which was significantly lower on comparing grade 3 patients with grades 1 and 2 patients.

**Conclusions:** Reduction of portal flow and increase in hepatic artery flow in fatty liver correlates with disease severity and can help as a non-invasive measure in diagnosis and grading of non-alcoholic fatty liver disease.

**Keywords:** Non-alcoholic fatty liver, Hepatic artery, Portal vein

## Background

Non-alcoholic fatty liver disease (NAFLD) affects about 25% of the population; NAFLD and/or its complications is considered the commonest chronic progressive liver disease especially in developed countries [1].

NAFLD is macrovesicular steatosis in more than 5% of hepatocytes, in the absence of a secondary cause as alcohol or drugs. Histological lesions ranging from non-alcoholic fatty liver to steatohepatitis, fibrosis, and

cirrhosis are included in pathogenesis [2]. Liver biopsy is the sure method to assessment liver fibrosis; yet it is associated with several drawbacks as invasiveness, cost, high sampling errors, and possible related morbidity and mortality [3]. Accordingly, non-invasive methods to assess liver fibrosis have been developed including blood biomarkers and imaging modalities [4]. Transient elastography (TE) is a non-invasive and easy modality that detects the level of fibrosis through measuring liver stiffness using the transmission of mechanical waves. Controlled attenuation parameter (CAP) enables the measurement of stiffness and steatosis simultaneously [5]. However, obesity, ascites, and elevated alanine

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aminotransferase (ALT) value may affect the accurate measurement of liver stiffness [6]. Duplex Doppler ultrasonography (US) is an important non-invasive method in evaluating hepatic vasculature and diagnosing some liver parenchymal diseases [7] as diffuse fatty infiltration in the liver alters hemodynamics in the portal vein as well as hepatic artery resistance [8].

## Methods

A case-control study including 80 participants, after ethical committee approval and informed consent approval, were selected and classified into the following:

Control group: 20 healthy volunteers (with CAP score < 220)

Grade 1: 20 patients with grade 1 NAFLD (CAP score 220–259)

Grade 2: 20 patients with grade 2 NAFLD (CAP score 260–289)

Grade 3: 20 patients with grade 3 NAFLD (CAP score  $\geq$  290) [9]

Subjects with alcohol consumption of more than 20 g/day, other causes of chronic liver disease, morbid obesity, and diabetics were excluded from the study population.

All participants were subjected to clinical examination including anthropometric measures, laboratory investigations including complete blood count (CBC), international normalization ratio (INR), blood urea nitrogen (BUN), serum creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total protein, albumin (Alb.), total bilirubin (T.Bil.), direct bilirubin (D.Bil.), gamma-glutamyl transferase (GGT), fasting blood sugar (FBS), glycosylated hemoglobin (HbA1C), and lipid profile (triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol).

Fatty liver index was calculated in all subjects [10].

Transient elastography (fibroscan): single experienced operator performed all Fibro Scan examinations as per the manufacturer's recommendations. With the patient lying in the dorsal decubitus position, the tip of the transducer probe was placed on the skin between the ribs over the right lobe of the liver.

## Doppler US

Doppler US was performed by a single radiologist using a Philips HD5 ultrasound machine with a convex arrayed (1 ~ 6 Mhz) transducer and Linear arrayed (3 ~ 12 Mhz) transducer. All subjects fasted overnight

## Portal vein assessment

Portal vein assessment was done with patient in the left lateral decubitus with breath held in inspiration and the

measurements were taken at the level of the main portal vein before the bifurcation. The transducer was oriented along the longitudinal axis of the main portal vein using a para-median or slightly oblique plan. The point of measurement was midway between the confluence of the splenic and superior mesenteric veins and the bifurcation of the portal vein during quiet inspiration. The Doppler angle was always < 60°. The maximum (Vmax), minimum (Vmin), and mean flow (MFV) velocities (cm/s) were recorded in each patient, and the vein pulsatility index (VPI) was calculated using the formula  $VPI = (Vmax - Vmin)/Vmax$  to detect any increase or decrease in portal vein pulsatility in NAFLD

## Hepatic artery assessment

The hepatic artery indices were measured at the level of porta-hepatis with patient lying in the supine position. The main hepatic artery was selected for examination as it supplies both hepatic arteries and is seen at the portal triad, measurements of hepatic artery resistive index (HARI) were obtained with the patient in suspended respiration [peak-systolic velocity (PSV) minus the end-diastolic velocity (EDV) divided by the PSV of the hepatic artery ( $HARI = [PSV - EDV]/PSV$ )].

## Statistics

The collected data was analyzed using Statistical Package for Social Science (SPSS 25). Mean and standard deviation ( $\pm$  SD) was used for parametric numerical data. Student's t test, ANOVA test, and post hoc Bonferroni test to compare between means and correlation analysis (using Pearson's method) to assess the strength of association between two quantitative variables.

*P* value: level of significance: (*P* > 0.05: non-significant (NS), *P* < 0.05: significant (S), *P* < 0.01: highly significant (HS).

## Results

This study included 20 healthy volunteers (control group) and 60 NAFLD patients (case group); case group was further classified into 3 subgroups according to their CAP score (grade 1, grade 2, and grade 3). All patients were sex- and age-matched with average age of 45 years. NAFLD patients had significantly higher BMI and waist circumference, total cholesterol, triglycerides, LDL, FBS, AST, ALT, GGT, BUN, fatty liver index, and lower HDL than control group (*P* < 0.001 for all), yet there were no statistically significant differences between case and control groups regarding creatinine, HbA1C, or bilirubin (direct and indirect).

All examined Doppler indices (Vmax, Vmin, MFV, VPI, HARI) were significantly lower in NAFLD patients than in healthy individuals (Table 1).

**Table 1** Doppler indices comparison between case and control groups

	Control Mean ± SD	Case	Student's t test	
			t	p value
Vmax (cm/s)	32.69 ± 0.95	23.18 ± 3.49	19.090	< 0.001
Vmin (cm/s)	18.88 ± 0.7	17.46 ± 1.45	5.794	< 0.001
MFV (cm/s)	22.95 ± 0.67	12.56 ± 2.56	28.636	< 0.001
VPI (cm/s)	0.65 ± 0.02	0.37 ± 0.12	17.309	< 0.001
HARI	0.82 ± 0.02	0.74 ± 0.04	13.260	< 0.001

Vmax, MFV, VPI, and HARI were significantly indirectly proportionate to NAFLD grade, yet Vmin was significantly lower on comparing grade 3 with grades 1 and 2 (Table 2).

On comparing different grades of NAFLD using ANOVA and post hoc Bonferroni tests, total cholesterol was directly proportionate to the grade of NAFLD, HDL was significantly higher in grade 1 NAFLD than grades 2 and 3, and triglycerides were significantly higher on comparing grade 3 to grade 1 NAFLD patients only (Table 3).

On correlating Doppler indices in NAFLD patients with variable parameters, there was highly significant correlation between all Doppler indices and age, BMI, waist circumference, lipid profile, fatty liver index, and CAP score, yet there was no significant correlation with fasting blood sugar (Table 4).

## Discussion

In the present study, the mean age of NAFLD patients was 45.95 ± 7.2 years which is consistent with NICE guidelines 2018 [4]

This study showed a significant difference in BMI between case and control groups which agrees with Rui et al. [11]. Ghobadi et al. 2014 [12] as well found a significant correlation between BMI and grades of NAFLD, as our study showed a statistically significant

difference regarding BMI between grade 1 versus grades 2 and 3.

Concerning blood sugar, this study showed statistically significant difference between case and control groups. This agrees with Zhengjun [13], as impaired hepatic lipid and lipoprotein settling and increased oxidative stress in liver cells may increase liver fat accumulation and result in insulin resistance, this leads to increase in hepatic glucose production and elevated blood glucose [14].

Zhengjun [13] postulated that triglyceride, total cholesterol, and LDL cholesterol in NAFLD group were significantly higher than those in control group, which matches our study, as we found statistically significant differences between case and control groups regarding lipid profile. Furthermore, our study showed that HDL in case group was significantly lower than in control group, but this was not consistent with the study of Zhengjun [13] as there was no significant difference regarding HDL.

This study showed that increasing grade of NAFLD was significantly associated with worse lipid profile, where there were differences between three grades as regard total cholesterol and LDL, there was statistically significant difference between grade 1 and grade 3 in triglycerides and HDL, and between grade 1 and grade 2 in HDL. This agrees with Dhumal et al. [15] who found that increasing grades of NAFLD were significantly associated with increasing serum total cholesterol, LDL, and VLDL and decreasing HDL, yet they found no significant association between serum triglyceride.

Regarding portal vein Doppler indices, this study revealed statistically significant differences between case and control groups in all Doppler indices.

Vmax, Vmin, and VPI in case group were significantly lower than in control group. This was compatible with Besir et al. [16]. Balasubramanian et al. [17], although agreed with our finding regarding Vmax, found no significant difference regarding Vmin.

**Table 2** Doppler indices comparison between 3 grades of NAFLD

	Grade 1 Mean ± SD	Grade 2	Grade 3	ANOVA test	
				f	p value
Vmax (cm/s)	27.4 ± 0.8	23.04 ± 0.61	19.1 ± 0.77	645.499	< 0.001 <sup>(A1)</sup>
Vmin (cm/s)	18.52 ± 1.2	18.01 ± 0.59	15.86 ± 0.73	51.440	< 0.001 <sup>(A2)</sup>
MFV (cm/s)	15.78 ± 0.61	12.01 ± 0.57	9.89 ± 0.97	325.229	< 0.001 <sup>(A1)</sup>
VPI (cm/s)	0.52 ± 0.04	0.36 ± 0.04	0.25 ± 0.03	284.708	< 0.001 <sup>(A1)</sup>
HARI	0.79 ± 0.01	0.74 ± 0.01	0.7 ± 0.01	243.388	< 0.001 <sup>(A1)</sup>

<sup>A</sup>ANOVA test.

<sup>B</sup>Post hoc Bonferroni test was significant at: <sup>A1</sup>Between all groups. <sup>A2</sup> G3 group vs. G1 and G2 groups

**Table 3** Comparison between the 3 grades of NAFLD

	Grade 1 Mean ± SD	Grade 2 Mean ± SD	Grade 3 Mean ± SD	ANOVA	
				f	p value
Age ( years )	44.6 ± 7.31	47.8 ± 6.25	45.45 ± 7.91	1.063	0.352
BMI (kg/m <sup>2</sup> )	32.24 ± 2.44	35.23 ± 1.93	36.82 ± 2.77	18.758	< 0.001 <sup>(A3)</sup>
Waist circumference (cm)	103.41 ± 8.58	117.16 ± 8.37	114.77 ± 10.14	13.144	< 0.001 <sup>(A3)</sup>
Total cholesterol (mg/dl)	183.5 ± 8.41	211.25 ± 8.83	234.2 ± 43.89	18.636	< 0.001 <sup>(A1)</sup>
Triglycerides (mg/dl)	162 ± 20.69	163.05 ± 15.58	181.5 ± 34.21	3.920	0.025 <sup>(A2)</sup>
LDL (mg/dl)	102.05 ± 9.97	151.45 ± 11.7	191.4 ± 18.31	210.394	< 0.001 <sup>(A1)</sup>
HDL (mg/dl)	58.05 ± 4.73	44.15 ± 6.1	43.65 ± 5.66	43.766	< 0.001 <sup>(A3)</sup>
FBS (mg/dl)	99.5 ± 4.26	97.7 ± 4.4	102.55 ± 5.65	5.191	0.008 <sup>(A4)</sup>
HBA1C (%)	5.43 ± 0.43	5.37 ± 0.43	5.5 ± 0.56	0.373	0.690
Hb (gm/dl)	12.5 ± 1.25	12.69 ± 1	13.34 ± 1.08	3.129	0.051
TLC	6.12 ± 1.25	5.98 ± 1.18	7.1 ± 1.64	3.991	0.024 <sup>(A4)</sup>
PLT	275.15 ± 36.9	278.75 ± 45.51	286.55 ± 48.47	0.352	0.705
AST (IU/L)	29.75 ± 5.01	41.75 ± 3.23	42.45 ± 4.52	54.580	< 0.001 <sup>(A3)</sup>
ALT (IU/L)	18.55 ± 2.56	28.25 ± 6.75	30.3 ± 8.46	19.102	< 0.001 <sup>(A3)</sup>
T.Bil (mg/dl)	0.97 ± 0.13	0.86 ± 0.14	0.86 ± 0.19	3.337	0.043 <sup>(P1)</sup>
D.Bil (mg/dl)	0.51 ± 0.11	0.41 ± 0.16	0.44 ± 0.19	2.089	0.133
Albumin (gm/dl)	4.07 ± 0.13	4.12 ± 0.15	4.11 ± 0.15	0.776	0.465
INR	1.01 ± 0.03	1.02 ± 0.05	1.02 ± 0.04	0.904	0.411
GGT (IU/L)	40.8 ± 5.4	44.65 ± 3.31	46.2 ± 4.19	8.049	0.001 <sup>(A3)</sup>
Fatty liver index	81.25 ± 13.65	95.2 ± 2.73	93.85 ± 5.79	15.638	< 0.001 <sup>(A3)</sup>

<sup>A</sup>ANOVA test

Post hoc Bonferroni test:

<sup>A1</sup>Between all groups

<sup>A2</sup>Grade 1 vs. grade 3

<sup>A3</sup>Grade 1 vs. grades 2 and 3

<sup>A4</sup>Grade 2 vs. grade 3

<sup>A5</sup>Grade 1 vs. grade 2 group

Post hoc LSD test:

<sup>P1</sup>Grade 1 vs. grades 2 and 3

On comparing 3 grades of NAFLD, this study showed significant decrease in Vmax and VPI with increasing the grade of NAFLD. This agrees with Besir et al. [16] and Balasubramanian [17]. Yet, not with Ehsan et al. [18] who found no significant difference in VPI between fatty liver grades.

Vmin showed significant decrease only on comparing grade 3 versus grades 1 and 2; this agrees with Besir et al. [16] who postulated that Vmin decreased as the degree of hepatosteatosis increased.

MFV in case group was significantly lower than in control group which corresponds with Ehsan et al. [18]. Moreover, MFV was significantly decreasing with increasing NAFLD grade which is consistent with Balasubramanian [17].

These findings regarding velocity of the portal flow and portal vein pulsatility index can be explained by the hypothesis that liver infiltration with fat increases flow resistance in portal vein reducing hepatic portal blood flow [19].

Regarding hepatic artery Doppler, this study revealed that HARI in case group was significantly lower than in control group. This agreed with Claudio et al. [20] and Balasubramanian [17] who agreed also with our finding that HARI was significantly decreasing with increasing NAFLD grade. These findings suggest an increased hepatic artery blood flow which may occur as a compensatory mechanism for reduced portal flow with the progression of hepatic steatosis [21].

### Limitation

Liver biopsy was not carried out to confirm the diagnosis and severity of fatty liver and there was no follow-up for the cases.

### Conclusion

Reduction of portal flow and increase in hepatic artery flow in fatty liver correlates with disease severity and can help as a non-invasive measure for NAFLD diagnosis and grading.

**Table 4** Correlation between Doppler indices and significant variables in NAFLD

		Total cholesterol	Triglycerides	LDL	HDL	Age	BMI	Waist Circumference	Fatty liver index	CAP score
VMax (cm/s)	Pearson correlation	- 0.723	- 0.75	- 0.915	0.704	- 0.83	- 0.64	- 0.64	- 0.779	- 0.979
	<i>p</i> value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
Vmin (cm/s)	Pearson correlation	- 0.583	- 0.535	- 0.71	0.551	- 0.57	- 0.354	- 0.354	- 0.468	- 0.719
	<i>p</i> value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	.001	.001	< 0.001	< 0.001
MFV (cm/s)	Pearson correlation	- 0.696	- 0.79	- 0.857	0.687	- 0.83	- 0.653	- 0.653	- 0.826	- 0.96
	<i>p</i> value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
VPI (cm/s)	Pearson correlation	- 0.748	- 0.745	- 0.927	0.731	- 0.842	- 0.671	- 0.671	- 0.784	- 0.966
	<i>p</i> value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001
HARI	Pearson correlation	- 0.749	- 0.716	- 0.917	0.727	- 0.777	- 0.625	- 0.625	- 0.711	- 0.939
	<i>p</i> value	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001

**Abbreviations**

Alb: Albumin; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; BMI: Body mass index; BUN: Blood urea nitrogen; CAP: Controlled attenuation parameter; CBC: Complete blood count; D.Bil: Direct bilirubin; EDV: End-diastolic velocity; FBS: Fasting blood sugar; GGT: Gamma-glutamyl transferase; HARI: Hepatic artery resistive index; Hb: Hemoglobin; HbA1C: Glucosylated hemoglobin; HDL: High-density lipoproteins; HS: Highly significant; INR: International normalization ratio; LDL: Low-density lipoproteins; MFV: Mean flow velocity; Mhz: Megahertz; NAFLD: Non-alcoholic fatty liver disease; NS: Non-significant; PLT: Platelet; PSV: Peak-systolic velocity; S: Significant; SD: Standard deviation; SPSS: Statistical Package for Social Science; T.Bil: Total bilirubin; TE: Transient elastography; TLC: Total leucocyte count; US: Ultrasonography; VLDL: Very low-density lipoprotein; Vmax: Peak maximum velocity; Vmin: Peak minimum velocity; VPI: Vein pulsatility index

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**Authors' contributions**

T Y designed the study. N A did data analysis and interpretation. C A drafted the manuscript. M S did critical revision of the manuscript for important intellectual content. M S did radiology work-up, and M M supervised the study. All authors read and approved the final manuscript.

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**Availability of data and materials**

Data are available with corresponding author to be presented upon request.

**Declarations****Ethics approval and consent to participate**

This study had been performed in accordance with the ethical standards. Faculty of Medicine, Ain Shams University Ethical Committee approval was

taken before starting the study, and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki. A written consent was obtained from each participant. Committee's reference number is FWA 000017585.

**Consent for publication**

Not applicable.

**Competing interests**

The authors declare that they have no competing interests.

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