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Whole blood viscosity as a predictor of left ventricular dysfunction in patients with Non-Alcoholic Fatty Liver Disease (NAFLD)

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Abstract
Background: NAFLD is the commonest liver pathology globally which is distinguished by hepatic steatosis without evidence of secondary injurious hepatic agents.
The spectrum of liver affection in NAFLD is variable starting from mild steatosis to evident fibrosis and may extend to extra hepatic morbidities.
CVD is one of the highly serious systemic comorbidities of NAFLD including various degrees of cardiovascular dysfunction especially at the level of left ventricle.
Whole blood viscosity (WBV) is an important rheological parameter that affects blood flow in the circulation with subsequent affection of tissue perfusion, so investigators try to elucidate the role of WBV as a crucial player in the etiopathogenesis of LVD in NAFLD cases and hence its use as an early preditictor marker.
Patients and Methods: This is a cross sectional study that was carried out on 50 NAFLD cases grouped into 2 groups based on the existence presence of LVD: Group 1 included 26 patients with LVD, group 2 included 24 patients without LVD. The patients' demographic, clinical, laboratory and radiological data were recorded on a special observation sheet. Whole blood viscosity and ECHO cardiography assessment of the left ventricular function of patients were also recorded. Statistical analysis was done for all collected data using the IBM, SPSS version 23. Significance of obtained results was considered at p-value of less than 0.05.
Results: WBV was significantly increased in group 1 compared to group 2. WBV had appeared to be an excellent predictor of LVD in patients with NAFLD; it was positively correlated with hemoglobin, hematocrit, fatty liver status and fibrosis and all LVD parameters except for ejection fraction and relative wall thickness. By logistic regression analysis hemoglobin, whole blood viscosity, and left ventricular end-diastolic diameter are the only predictors in NAFLD patients with LVD. WBV at a cut-off value of 4.38; the AUC was 0.756, the sensitivity was 96.15%, the specificity was 83.33%, the PPV was 86.20%, and the NPV was 95.23%.
Conclusion: WBV is a good, easily obtained and affordable marker for the determination of LVD in cases with NAFLD.

Keywords: Whole blood viscosity, predictor, left ventricular dysfunction, NAFLD

1. Introduction

1.1 The poor scientific foundation of medical interventions: A threat to Evidence-Based Medicine

Nonalcoholic fatty liver disease (NAFLD) refers to the pathological condition having the same characteristics as alcohol-induced liver lesion, yet without alcohol abuse. It comprises a set of hepatic disorders that ranged from simple steatosis, steatohepatitis, advanced fibrosis to cirrhosis, and it becomes a growing public health problem all over the world [1].
The prevalence of NAFLD was estimated to reach twenty-five percent in adult individuals, and approximately 70–80% in obese and patients with diabetes. If no management is applied, NAFLD, in particular the severe types, could progress to cirrhosis, hepatic failure, and even HCC [2]. The issue of NAFLD isn't limited to its ability to produce fatal hepatic related morbidity and mortality. It usually appears with manifestations of the metabolic syndrome that includes obesity, type 2 DM, dyslipidemia, and elevated ABP [3, 4].
Indeed, the metabolic syndrome is an important predictor of NAFLD, and it is a well-determined precursor of CVD [5].

Currently, growing evidence postulates that NAFLD is related to elevated risk of cardiovascular disease (CVD) events [6]. It was demonstrated that cases suffering NAFLD have ECHO criteria of early left ventricular diastolic dysfunction (LVDD) as evaluated by tissue Doppler ECHO [7, 8]. Blood viscosity refers to the intrinsic resistance of bulk blood to flow via a wide-bore vessel. Its essential determinants include packed cell volume, plasma fibrinogen level and plasma viscosity [9]. Accumulating evidence demonstrated that abnormal whole blood viscosity (WBV) is incorporated in insulin resistance that could result in NAFLD which is linked to cardiovascular disease events including left ventricular dysfunction (LVD). Hence, for evaluation of WBV as a predictor of LVD in cases with NAFLD, this work was designed [10].

The aim of the present study was to assess WBV as a predictor of left ventricular dysfunction in cases with NAFLD.

Patients and Methods

This cross-sectional study included fifty adult cases, both sexes with clinical criteria of NAFLD. The study was carried out following approval from the Research Ethics Committee as a part of the Quality Assurance Unit in the Faculty of Medicine at Tanta University to carry out this study and to utilize the facilities in the hospital from December 2021 to November 2022. Informed consent was attained from all participants following complete explanation of benefits & risks.

Exclusion criteria were pregnant females, previous history of IHD and valvular disease, CHF, known causes of chronic hepatic diseases and cirrhosis, Use of steatogenic drugs and measurement failure or unreliable measurements on transient elastography (TE). Cases were categorized into 2 Groups: Group 1: Includes 26 cases who had ECHO proven LVD. Group 2: Includes 24 patients who had no ECHO proven LVD.

All cases underwent a full history including personal and family history, complete clinical assessment including Body Mass Index (BMI), lab investigations that included (CBC, CRP, HbA1C, blood urea and serum creatinine, liver function tests (Serum Glutamic Oxaloacetic Transaminase (SGOT), bilirubin and albumin), Lipid profile [total cholesterol level, high density lipoprotein-cholesterol level (HDL-C), Low density lipoprotein-cholesterol level(LDL-C) and Triglycerides (TGS)] were assessed), as Patients prepared for the test by asking not to eat or drink anything but water for 12 to 14 hours before this test.

Also, all patients were subjected to special investigation (WBV is calculated using hematocrit and plasma protein concentrations by a validated equation and at 208 seconds1 of shear stress was assessed using a previously approved formula that considers HCT and plasma proteins.

Imaging

Abdominal ultrasonography

NAFLD was diagnosed based on the increase in the parenchymal brightness in comparison with the cortex of the right kidney. The degree of NAFLD was classified into 3 forms Severe NAFLD that refers to the increased liver brightness, visualizing only the main portal vein walls, with all smaller portal vein walls absent. Moderate NAFLD that refers to US findings are between mild and severe NAFLD.

Mild NAFLD that refers to the increased liver brightness with a mild reduction in defining the portal vein walls.

Fibroscan (Transient elastography)

Liver stiffness assessment was carried out using the fibroscan (echosens- France) 502 M probe that was carried out by professional operator according to the instructions of the manufacturer. It was done from the transthoracic window intercostals on the right hepatic lobe with the patient in the upright position and the right arm fully abducted. 10 valid measurements for each patient and the IQR < 30%, and a success rate ≥ 70% were considered reliable. The median value was considered representative of the hepatic elastic status. The software automatically calculated the median value expressing the results in kPa. The measurement was carried out using the Fibro Scan (M) probe or (XL) probe. Elastogram score yielded by the equipment is interpreted by special software to detect the corresponding fibrosis stage in Metavir score. Liver steatosis is evaluated during the same sessions by the M or XL probes to determine CAP. The algorithm is involved in the TE software and data were calculated concurrently with the hepatic stiffness measurement. The CAP score is measured in dB/m, and it ranges between 100- 400 dB/m. The fibrosis result is measured in kPa and it's normally from 2 to 6 kPa. The highest possible result is up to 75 kPa.

Echocardiography

All studies were carried out via (a GE vivid 7 cardiac US phased array system with tissue Doppler imaging via the use of M4S transducer 4 MHz). American Society of ECHO and 3 sequential cycles were averaged for all parameters. Standard ECHO analysis includes 2D, M-mode, Doppler flow, and tissue Doppler flow estimations. Diastolic IVS, diastolic PWT, left atrial (LA) diameter, left ventricle end systolic (LVESD) and LVED dimensions were assessed from parasternal long-axis view.

Assess LV systolic function using Biplane Simpson Method in the apical 4 & apical 2 views also left ventricular volumes were assessed (End diastolic volume and end systolic volume). In the biplane Simpson approach, volume depends upon the 2-chambers view also. Generally, the biplane Simpson approach is more precise and is recommended over the monoplane method.

EF= (EDV – ESV) / EDV X 100

Tissue Doppler diastolic velocity was calculated from the septal, lateral, inferior, and anterior mitral annuli in the two and four chamber views and we calculated the average. We recorded the following measurements: early diastolic velocity (e′).

The ratio of early diastolic mitral inflow velocity (E) by Pulsed Wave Doppler to e′ (E/e′), that correlates with diastolic filling pressure, was recorded.

LVM was estimated from M-mode ECHO via the use of the American Society of ECHO recommended Cube formula:

LVM (gm) = 0.8 × 1.04 ((IVS + PWT+ LVEDD)3 - LVEDD3) + 0.6 LVM was divided by BSA to yield the LVMI (gm/m2), which cut-off values of 115 gm/m2 for males and 95 gm/m2 for females. BSA (m2) was estimated via the Du Bois formula (weight (kg)0.425 × height (cm)0.725 × 0.007184). Relative wall thickness (relative wall thickness (RWT) = 2 × PWT in end diastole/ LV diastolic diameter in end diastole) was estimated. Normal RWT was defined as value of 0.42 or less and elevated

RWT as greater than 0.42.

Statistical Analysis

Analysis of data was carried out via the IBM SPSS software package version 23.0. (SPSS Inc., Chicago, IL, USA). Qualitative data were described using numbers and %. Quantitative data were described as mean and SD for numerical variables with normal distribution, and median and IQR for numerical variables with abnormal distribution. The Kolmogorov-Smirnov test was used to verify the normality of distribution. The used tests were Chi-square test, Student's t-test, Mann Whitney test, Correlation between variables, Univariate and multivariate logistic regression analysis were used. ROC was used to show diagnostic accuracy. Significance of the obtained results was considered at p-value ≤ 0.05.

Results

Non-significant differences were demonstrated between group 1, and group 2 as regards age, sex distribution, TLC, platelets urea, creatinine, albumin, and total bilirubin. While there were statistically significant differences between group 1 and group 2 as regards BMI, hemoglobin, hematocrit, hemoglobinA1C (HBA1C), CRP, SGOT, SGPT, cholesterol, triglycerides, LDL, and whole blood viscosity. Group 1 showed higher BMI, hemoglobin, hematocrit, cholesterol, triglycerides, LDL, and WBV than group 2. (Table 1)

Table 1: Comparison between the studied groups regarding age, gender, BMI, and risk factors and laboratory tests

Groups Parameters		Group (1) 26 (52%)	Group (2) 24 (48%)	P-Value
Age (Years)		55.15±13.04	53.04±10.62	0.535(a)
Sex	Male	8 (30.8%)	13 (54.2%)	0.094(b)
	Female	18 (69.2%)	11 (45.8%)	
	Male/Female ratio	44.5%	118.18%	
BMI (Kg/m2)		32.40±4.20	28.39±4.22	0.002*(a)
Risk factors	DMT2	2 (7.7%)	5 (20.8%)	0.181(b)
	HTN	2 (7.7%)	1 (4.2%)	0.600(b)
	Smoking	15 (57.7%)	11 (45.8%)	0.402(b)
TLC (10^3/mm3)		10046.15±10613.43	9454.16±3406.76	0.795(a)
Platelets (10^3/mm3)		225.42±69.11	238.04±89.68	0.578(a)
Hemoglobin (g/dL)		11.97±1.53	10.53±1.15	<0.001(a)
Hematocrit (%)		33.58±0.63	30.23±1.17	<0.001(a)
HBA1C (%)		9.76±2.72	6.4±1.96	<0.001**(a)
CRP (mg/L)		30.5 (26.75)	15 (18)	<0.001**(b)
Urea (mg/dL)		73.5 (60.8)	43.5 (34)	0.113(b)
Creatinine (mg/dL)		1.4 (1.2)	1.1 (0.7)	0.302(b)
SGOT (U/L)		36.5 (40)	25.5 (10)	0.045*(b)
SGPT (U/L)		28 (21.8)	19.5 (21)	0.037*(b)
Albumin (g/dL)		3.84±0.43	3.75±0.45	0.463(a)
Total bilirubin (mg/dL)		0.72±0.12	0.67±0.13	0.189(a)
Cholesterol (mg/dL)		198.15±36.28	159.95±31.62	< 0.001**(a)
Triglycerides (mg/dL)		183.76±61.09	134.87±31.5	<0.001**(a)
HDL (mg/dL)		33.83±7.61	41.7±12.06	0.009*(a)
LDL (mg/dL)		111.1±21.18	77.95±32	<0.001**(a)
Whole blood viscosity		5.11±0.48	4.69±0.38	< .001**(a)

Table 2: Comparison between all the studied groups regarding pelvic abdominal ultrasound

Groups Parameters		Group (1) 26 (52%)	Group (2) 24 (48%)	P-Value
Ultrasound	Mild fatty liver	7 (26.9%)	13 (54.2%)	0.051(b)
	Moderate fatty liver	12 (46.2%)	4 (16.7%)	0.026*(b)
	Severe fatty liver	7 (26.9%)	1 (4.2%)	0.028*(b)
Fibrosis	F0	1 (3.8%)	3 (12.5%)	0.270(b)
	F1	3 (11.5%)	7 (29.2%)	0.191(b)
	F2	4 (15.4%)	10 (41.7%)	0.039*(b)
	F3	8 (30.8%)	2 (8.3%)	0.048*(b)
	F4	10 (38.5%)	2 (8.3%)	0.013*(b)
Steatosis	S0	2 (7.7%)	6 (25%)	0.095(b)
	S1	3 (11.5%)	11 (45.8%)	0.007*(b)
	S2	13 (50%)	5 (20.8%)	0.032*(b)
	S3	8 (30.8%)	2 (8.3%)	0.048*(b)
Fibrosis		8 (9)	4.2 (5)	0.008*(b)
Steatosis		301.19±49.61	269.71±34.66	0.009*(a)

Data are presented as number of (%) and mean± SD or median, BMI: Body Mass Index, T2DM: Type II Diabetes Mellitus, HTN: Hypertension, HBA1C: hemoglobin A1C, CRP: C-reactive protein, SGOT: Serum Glutamate Oxaloacetic Transaminase, SGPT: Serum Glutamate Pyruvic Transaminase, HDL: high-density lipoprotein-c, LDL-c: low-density lipoprotein (a): Independent-Sample T Test, (b): Mann-Whitney U, *: Statistically significant at $p<$ 0.05, **: Statistically significant at $p<$ 0.001.

The comparison between both groups showed insignificant

differences regarding mild fatty liver, F0, F1, and S0. While The comparison between both groups showed significant differences regarding moderate and severe fatty liver, F2, F3, F4, S1, S2, and S3. A statistically significant increase was demonstrated between group 1 and group 2 regarding fibrosis, and steatosis. Group 1 showed higher fibrosis and steatosis than group 2. (Table 2).

Data are presented as number of (%) and mean± SD or median, (b): Mann-Whitney U, *: Statistically significant at $p< 0.05$, **: Statistically significant at $p< 0.001$.

A statistically significant increase between group 1 and group 2 was determined regarding LVEDD, LVESD, LAD, E/e, IVS, PWT, RWT, and LVMI. Group 1 showed higher LVEDD, LVESD, LAD, E/e, IVS, PWT, RWT, and LVMI than group 2. While there was insignificant difference between group 1, and group 2 regarding EF. (Table 3)

Table 3: Comparison between all the studied groups as regards ECHO parameters

Parameters	Group (1) 26 (52%)	Group (2) 24 (48%)	P-Value
LVEDD (mm)	55.69±9.11	47.29±6.06	< 0.001**(a)
LVESD (mm)	35.84±8.37	30.45±5.57	0.011*(a)
EF (%)	64.65±6.67	62.91±6.5	0.357(a)
LAD (mm)	39.34±4.4	33.54±7.06	<0.001**(a)
E/e	9.05±2.1	7.43±1.53	0.003*(a)
IVS (mm)	11.73±2.3	10.2±1.69	0.011*(a)
PWT (mm)	11.73±1.75	9.91±1.47	< 0.001**(a)
RWT (mm)	0.45±0.06	0.41±0.08	0.02*(a)
LVMI	142.48±28.86	86.04±17.31	< 0.001**(a)

Data are presented as mean± SD Group 1: NAFLD patients with LVD, Group 2: NAFLD patients without LVD, No: number, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, EF: ejection fraction, LAD: left atrial dimensions, E/e: E wave divided by e′ velocities, (a): Independent-Sample T Test, EF: ejection fraction, LAD: left atrial dimensions,(b): Chi-Square Test, *: Statistically significant at $p< 0.05$

WBV was positively correlated with hemoglobin, hematocrit, LDL, LAD, PWT, fatty liver status and fibrosis. While, WBV was not correlated with age, sex, BMI, platelets, TLC, CRP, HBA1C, urea, creatinine, SGOT, SGPT, albumin, total bilirubin, cholesterol, triglycerides, HDL, LVEDD, LVESD, EF, E/e, IVS, RWT, LVMI, and steatosis. (Table 4)

Table 4: Correlations between WBV and demographic, NAFLD parameters, and ECHO finding of the patients

Variables	WBV	
	r	p
Age	0.087	0.547
Sex	0.160	0.267
BMI	0.250	0.08
Hemoglobin	0.296	0.037*
Hematocrit	0.383	0.006*
Platelets	-0.024	0.871
TLC	-0.145	0.314
CRP	0.037	0.798
HBA1C	0.224	0.118
Urea	0.125	0.388
Creatinine	0.167	0.247
SGOT	0.138	0.338
SGPT	0.068	0.640
Albumin	-0.162	0.262
Total bilirubin	-0.077	0.597
Cholesterol	0.101	0.485
Triglycerides	0.274	0.054
HDL	-0.190	0.186
LDL	0.399	0.004*
LVEDD	-0.005	0.970
LVESD	-0.061	0.675
EF	0.185	0.198
LAD	0.297	0.036*
E/e	0.172	0.233
IVS	0.267	0.061
PWT	0.278	0.05*
RWT	0.171	0.235
LVMI	0.258	0.071
Fatty liver status	0.389	0.005*
Fibrosis	0.293	0.039*
Steatosis	0.571	<0.001**

*: Statistically significant at $p< 0.05$ **: Statistically significant at $p< 0.001$, r: Pearson correlation, WBV: whole blood viscosity, BMI: body mass index, SGOT: serum glutamate-oxaloacetate transaminase, SGPT: serum glutamate pyruvate transaminase, CRP: C-reactive

protein, TLC: total leucocyte count, HDL-c: high density lipoproteins, LDL-c: low density lipoproteins, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, EF: ejection fraction, LAD: left atrial dimensions, IVS: interventricular septum, PWT: Posterior Wall Thickness, RWT: Relative wall thickness, LVMI: Left ventricular mass index.

By logistic regression analysis hemoglobin, whole blood viscosity, and LVEDD are the only predictors in NAFLD patients with LVD. (Table 5)

Table 5: Regression analysis for predictor factors affecting NAFLD patients with LVH

Independent variables	Odds Ratio (95%) CI	P- value
Age	0.995 (0.920 – 1.076)	0.899
Sex	9.054 (0.601 – 136.455)	0.111
Hemoglobin	0.438 (0.216 – 0.889)	0.022*
Whole blood viscosity	0.005 (0.001 – 0.705)	0.036*
LVEDD	0.736 (0.565 – 0.960)	0.024*
LVESD	0.883 (0.708 – 1.100)	0.265
Fibrosis	0.768 (0.566 – 1.044)	0.092
Steatosis	0.958 (0.914 – 1.003)	0.069

*: Statistically significant at $p \leq 0.05$, **: Statistically significant at $p \leq 0.001$.

WBV at a cut-off value of 4.38; the area under the curve was 0.756, the sensitivity was 96.15%, the specificity was 83.33%, the PPV was 86.20%, and the NPV was 95.23%. (Figure 1)

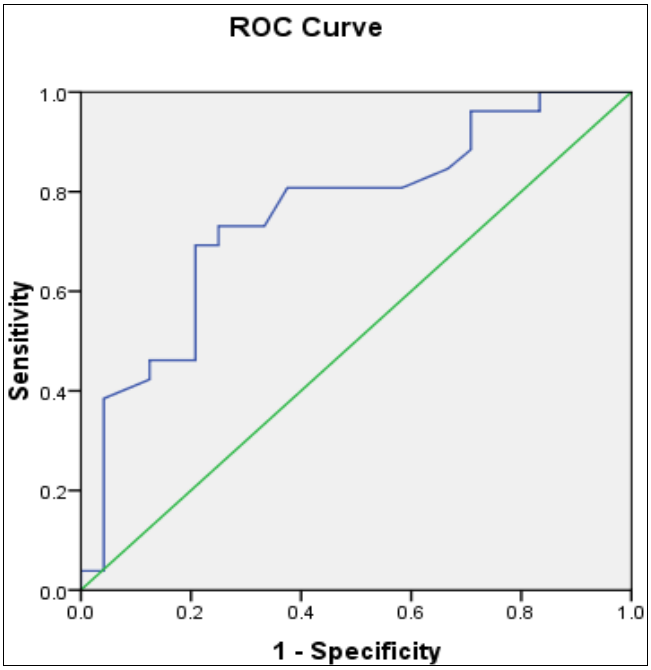

Fig 1: ROC curve of WBV for determination of NAFLD patients with LVD

Discussion

NAFLD is a one of the crucial etiologies of chronic hepatic disorders and currently, the incidence of NAFLD has elevated dramatically all over the world, rendering this disease an essential cause of hepatic-related morbidity and death [11].

NAFLD refers to a multisystem disease that can adversely affect various extra-hepatic organs such as the circulatory system [12]. It causes independently increase in the risk of CVD specially left ventricular dysfunction. This risk is in parallel with the degree of NAFLD (in particular the stage of hepatic fibrosis) [12].

WBV is an important parameter controlling blood flow in the circulation and is affected by multiple variables [12] increased blood viscosity may reduce the flow of blood in the circulator system and subsequently affect tissue blood supply with decrease the delivery of substrate like insulin, glucose and O2 to the skeletal muscle [7]. Blood viscosity has an essential role in the pathological processes of CVD, the elevated WBV in cases with NAFLD may provide a novel aspect to explain the mechanisms underlying the correlation between NAFLD and CVD [7].

Hence, this study was performed to assess whole blood viscosity as a predictor of LVD in cases suffering NAFLD [7]. In the present study, we had compared the two groups on the following scales: patients' characteristics, biochemical parameters whole blood viscosity, US findings, fibroscan findings and ECHO findings.

Regarding demographic data, in this study, there was insignificant difference between both regarding the age, and sex distribution (p-value > 0.05).

This was similar to Walaa Sheba, *et al.*, [13] study who evaluated the association of NAFLD with early LVDD in cases with T2DM and didn't demonstrate significant age differences between LVD and Non-LVD group. But it disagreed with Wei-Chin Hung *et al.* [14] revealed that significant age differences were demonstrated between groups.

Regarding risk factors (DM, HTN and smoking), the present study had reported statistically significant increase in all risk factors in group 1 (with LVD) than group 2 (without LVD) (p-value ≤ 0.05) [14].

Theses results were in contrast with Wei-Chin Hung *et al.* [14] who concluded that current smoking as a risk factor wasn't significantly different in the 2 groups.

Regarding complete blood count, there were insignificant differences between group 1, and group 2 as Regards TLC, platelets, (p-value > 0.05), but there was significant elevation in Hemoglobin and hematocrit values in group 1 compared to group 2 (p-value ≤ 0.05).

This was contradictory to Walaa Sheba, *et al.*, [13] who didn't reveal significant difference as regard TLC, platelets, and hemoglobin in both groups. However, this results were in coherence with Xuekui Liu *et al.*, [15] revealed significantly increased HCT values our study showed higher CRP values in group 1 than group 2 (p-value ≤ 0.05).The same situation was Rahul Kumar, *et al.*, [16] Who was studying the association of high-sensitivity hs-CRP with NAFLD in Asian Indians that showed significant increase in CRP in NAFLD patients.

In our study, there was significant difference between the two groups concerning BMI, and HBA1C (p-value < 0.05). This indicates that good control of body weight and DM reduce the incidence of NAFLD.

This agreed with Wei-Chin Hung *et al*. [14] study that reported significantly increased BMI and HBA1C in group 1 than group 2. Regarding renal functions, non-significant differences between both groups were found (p-value greater than 0.05). This was in agreement with Walaa Sheba, *et al*., [13] who reported no significant increase in renal functions.

In the present study, a statistically significant elevation was found in group 1 than group 2 regarding SGOT, SGPT (p-value ≤ 0.05).

The same situation with Wei-Chin Hung *et al*. [14] who also reported significant increase in liver enzymes between group 1 and group 2. However, this was in contrast with Walaa Sheba *et al*., [13] who concluded non- significant increase in liver enzymes.

Dyslipidemia was associated with significant risk of development of hepatic steatosis and fibrosis and their severity in NAFLD patients and hence severity of CVD. This was proved by our present study as there was statistically significant increase between both groups as regards cholesterol, triglycerides, and LDL was demonstrated. Also, statistically significant decrease in HDL in group 1 than group 2.

This was also proved by Wei-Chin Hung *et al*. [14] who reported elevated total cholesterol, triglycerides, LDL and decreased HDL.

Regarding pelviabdominal US, in the present study, significant differences were demonstrated between groups as regards mild, moderate and severe NAFLD diagnosed by US. This was also proved by Wei-Chin Hung *et al*. [14] who revealed this significant difference.

Regarding fibrosis and steatosis diagnosed in the current study by fibroscan, significant differences was exhibited between the 1st and the 2nd groups as regard fibrosis (F0, F1, F2, F3 and F4) (p-value ≤ 0.05) and significant differences were demonstrated between 1st and the 2nd groups as regards steatosis (S1, S2 and S3) (p-value ≤ 0.05). This proved association of degree of NAFLD with the severity of CVD. This association was in accordance with Rosa Lombardi *et al*., [17] who revealed that statistically significant elevation was present in group 1 (with LVD) than group 2 (without LVD) as regard LVEDD, LVESD, E/e′, IVS, RWT, PWT, and LVMI (p-value ≤0.05). On the contrary, there were no significant differences between the 2 groups as regard ejection fraction (p-value ≤0.05).

This was the same as Wei-Chin Hung *et al*. [14] study who reported significant elevation in group 1 (with LVD) than group 2 (without LVD) as regard all cardiac parameters except EF. Interestingly, the present study had reported increase in WBV in group 1 (with LVD) than group 2 (without LVD) and correlation was positive with Severity of NAFLD, LVEDD, LVESD, LAD, IVS, PWT, E/e′, and LVMI.

The results are similar to Hong-yan Zhao *et al*. [7] study who reported significant increase in WBV in group 1 than group 2.Also, the regression analysis revealed that WBV to be an excellent predictor of LVD in cases suffering NAFLD (Sensitivity 96.15% and Specificity 83.33%, with a cut-off value 4.38 PPV was 86.20%, and NPV was 95.23%.The present work had elucidated WBV as s good predictor for LVD in NAFLD patients and by this way, we can have a new insight for early determination of LVD in such widely prevalent group of patients through an easily obtained and affordable test.

Limitations: We had only assessed left ventricular dysfunction despite cardiac morbidities in NAFLD patients can include ischemic heart diseases, arrhythmias, and heart failure. It was a single-center study with a proportionally small sample (n = 50), that might markedly have limited statistical power and its external validity. 3rd, this study is lacking data on other inflammatory markers like TNF-α and IL-6.

Conclusions

WBV was significantly elevated in LVD group of NAFLD cases WBV was positively correlated with degree of NAFLD, LVEDD, LVESD, LAD, IVS, PWT, E/e′, and LVMI, Thus, the present study presents evidence that WBV is a good, easily obtained and affordable marker for detection of LVD in NAFLD cases.

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