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ORIGINAL ARTICLE

Association between Severity of Nonalcoholic Fatty Liver and Left Ventricular Diastolic Dysfunction Assessed by Tissue Doppler Echocardiography

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ABSTRACT

Background: Numerous metabolic risk factors are shared by a wide range of cardiac problems that may be linked to nonalcoholic fatty liver disease (NAFLD). The purpose of this study was to determine if tissue Doppler echocardiography-measured left ventricular (LV) diastolic dysfunction and nonalcoholic fatty liver disease are related.

Methods: In this prospective cross-sectional investigation, 92 NAFLD patients were involved and were divided according to Transabdominal ultrasound into three groups: Group I (grade I fatty liver) included 27 patients, Group Ⅱ (grade II fatty liver) included 29 patients and group Ⅲ (grade III fatty liver) included 36 patients. All patients underwent transabdominal ultrasound and measuring tissue Doppler transthoracic echocardiography e septal velocity, E/e, E/A ratio, peak E-wave velocity, and peak A-wave velocity.

Results: Regarding echocardiographic functions, there was a statistical significant difference in the NAFLD degrees between the three groups; group III had higher E/A, E/e' ratio, and LA. While A and e' levels were higher among group I when compared with the other groups. E/e' ratio was positively associated with NAFLD grades, while positively associated with HTN. HTN, NAFLD, TC and LA can be used as independent factors for predicting diastolic dysfunction among the studied groups.

Conclusions: In asymptomatic patient there was a significant association between NAFLD and cardiac diastolic dysfunction as NAFLD grades were associated with E/e' ratio (diastolic dysfunction).

Keywords: Non-alcoholic fatty liver disease; Left ventricular Doppler echocardiography.

INTRODUCTION

The most prevalent kind of chronic liver illness, non-alcoholic fatty liver disease (NAFLD), has been found to be strongly associated with liver-related morbidity and death in population-based research [1].

The metabolic syndrome's hepatic manifestation, which includes insulin resistance, obesity, and dyslipidemia is nonalcoholic steatohepatitis (NASH), which varies from basic hepatic steatosis (5% liver fat content) to NASH. Necro-inflammatory damage, either with or without hepatic fibrosis, is its defining feature [2].

When secondary causes of fatty liver disease, such as Wilson disease or hepatitis C, are checked out and there is no major alcohol consumption (>30 g/d for men, >20 g/d for women, or ≥14 weeks for males, ≥7 weeks for women), the abnormal buildup of fat (>5%) in the hepatocytes is referred to as nonalcoholic fatty liver disease (NAFLD) [3].

Non-alcoholic fatty liver disease (NAFLD) is caused by a complex interplay of nutritional, hormonal, genetic, and inflammatory variables. Insulin resistance, hyperlipogenesis, aberrant adipokine levels, raised circulating triglyceride levels, and elevated levels of systemic proinflammatory mediators are the outcomes of these interactions [4].

Abnormalities of the heart's structure and function that are frequently observed in HF patients are linked to NAFLD. NAFLD was found to be independently associated with incident LV hypertrophy, aberrant LV shape, and greater LV

strain in a population-based, longitudinal investigation[5].

Patients with non-acute focal lipodystrophy (NAFLD) who are non-diabetic and normotensive exhibit lower early diastolic relaxation and tissue Doppler echocardiography systolic velocity, which suggests a change in left ventricular diastolic and systolic function[6].

AIM OF THE WORK

The purpose of this research is to determine whether tissue Doppler echocardiography-measured left ventricular diastolic dysfunction and nonalcoholic fatty liver disease are related.

METHODS

In this cross-sectional investigation, 92 NAFLD patients were included, admitted to the Cardiology Department, Zagazig University Hospitals and El Mabara Hospitals in the duration from September 2023 to October 2024. The Ethics Committee of Zagazig University's Faculty of Medicine in Egypt gave the study approval (IRB # 10912). Every patient provided written and informed consent. The study was conducted in compliance with the World Medical Association's (Declaration of Helsinki) ethical criteria for research involving human beings.

Inclusion criteria were patients both sexes, Patients with NAFLD proved by ultrasonography: Because fat droplets within hepatocytes produce more interfaces, fatty liver is more echogenic (or "bright liver"), meaning that more echoes return to the transducer. Excluded criteria were valvular abnormalities (stenosis or regurge). Patient known to have CAD or cardiac muscle disease , patients with end organ liver cell failure, other liner disorder and alcoholic liver disease , patients with end organ renal failure , patients with significant rhythm disturbance , poor ECHO window TTE (poor myocardial delineation), signs of LV systolic dysfunction and heart failure.

Three primary categories for the patients were established; Group I (n=27): grade I fatty liver by Transabdominal Ultrasound enrolled as a case group. Group II (n=29): grade II fatty liver by Transabdominal Ultrasound enrolled as a case group. Group III (n=36): grade III fatty liver by Transabdominal Ultrasound enrolled as a case group.

All patients were subjected to complete history taking , general examinations including pulse, blood pressure, chest examination, abdominal examination and lower limb oedema, Evaluation of BMI, Local cardiac exam for signs of heart failure e.g : S3 gallop , S4 and lung rales. Routine laboratory investigations including Lipid profile which include total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) were measured after at least 12 h of fasting and AST, ALT with LVT to exclude liver cell failure. Renal function test to exclude renal failure.

Regarding those who may have non-alcoholic fatty liver disease (NAFLD), transabdominal ultrasonography was the main imaging modality. The primary indicator of steatosis is the liver parenchyma's higher echogenicity as compared to the right kidney's cortex due to intracellular fat vacuole accumulation reflecting the ultrasound beam. The following is how steatosis was often categorized: Grade 0: The echogenicity of the right liver lobe in relation to the right kidney cortex is normal. Grade 1: There is a slight, generalized increase in fine echoes in the liver parenchyma, but the diaphragm and intrahepatic artery borders are still visible. Grade 2: a slight, diffuse increase in fine echoes and a somewhat reduced ability to see the diaphragm and intrahepatic arteries. Grade 3: markedly elevated fine echoes combined with little to no visualization of the diaphragm, the liver's posterior right lobe, and the intrahepatic vascular borders [7].

Every subject received conventional 2D transthoracic echocardiography performed by a single, skilled physician echocardiographer utilizing a Vivid E9 machine (GE). The patients were scanned while in the left lateral decubitus position. Standard parasternal (long and short axis) pictures were acquired and apical (four, two, and long axis) perspectives using a 3.5 MHz transducer. The obtained image frame rates ranged from 82 to 95 frames per second [8].

Diastolic function:

Peak E-wave velocity (cm/sec); an apical four-chamber system with color flow imaging is used to achieve the best PW Doppler blood flow alignment.PW Doppler sample volume between the mitral leaflet tips (1-3 mm axial dimension). The early diastole peak velocity, or E wave velocity (after the T wave of the ECG), is measured.

Peak A-wave velocity (cm/sec): The A wave velocity is the peak velocity of the leading edge of the spectral waveform in the late diastole, which comes after the ECG P wave.

MV E/A ratio: calculated by dividing the velocity of MV E by the velocity of A waves.

e` velocity (cm/sec): by Pulsed-wave TDI

Chamber image from the apical four: PW Doppler sample volume, typically with an axial size of 5–10 mm, in the septal basal areas in order to calculate the average eV.

***Mitral E/e`:*** derived from dividing MV E velocity on e` velocity.

Left Atrium (Linear Dimensions and Area Measurements): LA anteroposterior (AP) using M-mode echocardiography in the parasternal long-axis perspective [9–11].

Statistical analysis

A computer was used to code, enter, and analyze the gathered data using IBM SPSS 23.0 for Windows, a database management system (SPSS Inc., Chicago, IL, USA). Fisher's exact test (f) is used when the assumption that "less than 20% of cells have expected count less than 5" is not met. To compare the continuous variables between the two groups, the independent samples t test was employed. Whitney-Mamann The U test was utilized in order to compare the outcomes between two separate groups. In order to investigate the differences in independent variable means between more than two groups, the One Way Analysis of Variance (ANOVA) test was utilized. Two or more independent, non-normally distributed samples with equal or different sample sizes were compared using the Kruskal-Wallis test. A quantitative variable's dependency on a group of independent factors was tested and estimated using multiple linear regression. A significant p-value was one that was less than 0.05.

RESULTS

Transabdominal ultrasonography was used to divide the 92 NAFLD patients in this study into three groups. Group I consisted of 27 participants. With a mean ± SD of 44.6 ± 8.18, their ages varied from 23 to 65. Of them, 66.7 percent were men and 33.3% were women. Their BMI was 23.9 ± 1.41 kg/m2 with a range of 21.3 to 26.4 kg/m2. Group II comprised twenty-nine patients. They were between the ages of 31 years and 75 years, with a mean ± SD of 52.4 ± 11.97. Among the patients with NAFLD, 58.6% were male and 41.4% were female. With a mean ± SD of 25.2 ± 1.47, their BMI varied from 23.1 to 28.1 kg/m2. There were 36 patients in Group III. Their ages, with a mean ± SD is 53.3 ± 10.49, varied from 40 to 78 years. There were (30.6%) females and (69.4%) males. Their mean ± SD was 26.8 ± 2.28 kg/m2, and their BMI varied from 23.4 to 31.2 kg/m2.

Table I revealed a statistically significant difference in age and BMI between the NAFLD degrees; patients in group III were older than those in the other groups (P<0.001). Furthermore, BMI was higher in group III patients compared to other groups (P<0.001).

Table 2 demonstrated a statistically significant difference in the lipid profile between the NAFLD degrees, with group III having higher levels of total cholesterol, triglycerides, and LDL when compared to the other groups (P<0.001).

Table 3 demonstrated a statistically significant difference in the diastolic indices between the NAFLD degrees, with group I having greater A and e' levels than the other groups (P<0.001). Group III had greater E/A ratio, E/e' ratio, and LA levels in comparison to the other groups (P<0.001).

Table 4 demonstrated that HTN, NAFLD, TC, and LA can be utilized as independent factors for predicting diastolic dysfunction among the study groups after performing logistic regression analysis for diastolic dysfunction predictors.

Table 1: Demographic data among the studied groups

Variables		Group I (n=27)	Group II (n=29)	Group III (n=36)	*P Value	Post-Hoc
Age (years)	Mean ± SD	44.6 ± 8.18	52.9 ± 11.97	53.3 ± 10.49	**<0.001**[1]	P1=**0.01** P2=**0.004** P3=**0.99**
Sex (n. %)	Male	18 (66.7%)	17 (58.6%)	25 (69.4%)	0.65[2]	-
	Female	9 (33.3%)	12 (41.4%)	11 (30.6%)		
BMI (kg/m^2)	Mean ± SD	23.9 ± 1.41	25.2 ± 1.47	26.8 ± 2.28	**<0.001**[1]	P1=**0.03** P2=**<0.001** P3=**0.001**

**[1]One way ANOVA test, [2]Chi-square test, Non-significant: P >0.05, Significant: P ≤0.05*

**P value=Comparison between the three groups, P1=Comparison between Group I & Group II, P2=Comparison between Group I & Group III, P3= Comparison between Group II & Group III*

*****BMI****=Body mass index*

Table 2: Lipid profile among the studied groups

Variables		Group I (n=27)	Group II (n=29)	Group III (n=36)	*P Value	Post-Hoc
TC (mg/dl)	Mean ± SD	181 ± 25.21	219 ± 7.84	247 ± 9.01	<**0.001**	P1<**0.001** P2<**0.001** P3<**0.001**
TG (mg/dl)	Mean ± SD	132 ± 23.18	170 ± 7.96	210 ± 15.88	<**0.001**	P1<**0.001** P2<**0.001** P3<**0.001**
LDL (mg/dl)	Mean ± SD	119 ± 18.46	140 ± 6.57	162 ± 7.35	<**0.001**	P1<**0.001** P2<**0.001** P3<**0.001**

**Kruskal-Wallis test, Non-significant: P >0.05, Significant: P ≤0.05*

**P value=Comparison between the three groups, P1=Comparison between Group I & Group II, P2=Comparison between Group I & Group III, P3= Comparison between Group II & Group III*

*****TC****=Total cholesterol,* ***TG****=Triglycerides,* ***LDL****=Low density lipoproteins*

Table 3: Echocardiographic findings among the studied groups (Diastolic indices)

Variables		Group I (n=27)	Group II (n=29)	Group III (n=36)	P Value	Post-Hoc
E (m/s)	Mean± SD	0.96 ± 0.12	0.98 ± 0.11	0.91 ± 0.16	0.08[1]	-
A (m/s)	Mean ± SD	1.16 ± 0.12	0.79 ± 0.06	0.46 ± 0.06	<**0.001**[1]	P1<**0.001** P2<**0.001** P3<**0.001**
e' (m/s)	Mean ± SD	0.11 ± 0.02	0.07 ± 0.01	0.05 ± 0.01	<**0.001**[2]	P1<**0.001** P2<**0.001** P3<**0.001**
E/A ratio	Mean ± SD	0.81 ± 0.09	1.24 ± 0.12	1.95 ± 0.22	<**0.001**[1]	P1<**0.001** P2<**0.001** P3<**0.001**
E/e' ratio	Mean ± SD	9.1 ± 1.33	14.72 ± 1.03	19.27 ± 1.51	<**0.001**[2]	P1<**0.001** P2<**0.001** P3<**0.001**
LA (mm)	Mean ± SD	34.93 ± 2.34	39.17 ± 1.83	43.28 ± 1.61	<**0.001**[1]	P1<**0.001** P2<**0.001** P3<**0.001**

**[1]One way ANOVA test, [2]Kruskal-Wallis test, Non-significant: P >0.05, Significant: P ≤0.05*

P value=Comparison between the three groups, P1=Comparison between Group I & Group II, P2=Comparison between Group I & Group III, P3= Comparison between Group II & Group III***E****=Early diastolic mitral inflow velocity,* ***A****=Late diastolic mitral inflow velocity,* ***e'****=Early diastolic myocardial velocity,* ***LA****=Left atrium*

Table 4: Logistic regression analysis for predictors of E/e' ratio (Diastolic indices)

Variables	Univariate analysis		Multivariate analysis	
	P value	**Odds (CI 95%)**	**P value**	**Odds (CI 95%)**
Age	**0.02**	1.06 (1.007 – 1.12)	0.12	3.23 (1.97 – 5.29)
Sex	0.67	1.23 (0.48 – 3.16)	-	-
BMI	**<0.001**	3.3 (2.01 – 5.42)	0.07	3.29 (0.76 – 14.35)
Smoking status	0.67	0.79 (0.28 – 2.26)	-	-
DM	0.34	1.67 (0.59 – 4.74)	-	-
HTN	**0.003**	4.1 (1.11 – 15.13)	**0.006**	4.21 (1.51 – 11.74)
NAFLD grades Mild Moderate Severe	**<0.001** **<0.001** **<0.001**	2.56 (1.74 – 2.63) 2.71 (1.82 – 2.87) 2.77 (1.84 – 2.91)	**0.008** **<0.001** **<0.001**	2.16 (1.61 – 2.51) 2.63 (1.84 – 2.89) 2.74 (1.92 – 2.89)
TC	**<0.001**	1.1 (1.06 – 1.15)	0.008	1.07 (1.02 – 1.13)
TG	**<0.001**	1.06 (1.03 – 1.08)	0.25	1.03 (0.97 – 1.11)
LDL	**<0.001**	1.06 (1.04 – 1.09)	0.16	1.04 (0.99 – 1.09)
E	0.15	16.87 (0.35 – 813.6)	-	-
A	**0.03**	0.12 (0.02 – 0.79)	0.33	0.41 (0.02 – 7.16)
e'	**0.007**	0.11 (0.02 – 0.65)	0.81	1.07 (0.63 – 1.79)
E/A ratio	**0.002**	19.25 (2.95 – 125.72)	0.27	1.89 (0.02 – 2.19)
LA	**<0.001**	1.66 (1.34 – 2.06)	**0.02**	3.55 (1.19 – 2.59)

DISCUSSION

Groups II and III in the current study were much older than Group I, although there was no significant difference in sex.

This came in line with Catena et al. [12] study which involved 360 essential hypertensive individuals without significant cardiovascular or renal problems and were not receiving treatment which included in their a cross-sectional study. They discovered that there was a significant difference in age based on whether there were any positive liver steatosis scores or not (1 or ≥ 2).

The lipid profile of NAFLD degrees varied significantly, according to our study, with group Ⅲ having greater levels of total cholesterol, triglycerides, and LDL compared to the other groups. These differences were consistent with an increase in the severity of the illness.

Lai et al. [13] conducted an ultrasound-assisted assessment of 2161 individuals, classifying them into three categories based on the NAFLD Fibrosis Score: (1) non-fatty; (2) fatty with low fibrosis score; and (3) fatty with high fibrosis score. They found that compared to the control group, the groups with low and severe fibrosis had higher levels of LDL, triglycerides, and total cholesterol.

This was in line with findings by Romero et al. [14] and Sert et al. [15], who found that patients with non-alcoholic fatty liver disease (NAFLD) had higher levels of total cholesterol and LDL-C than non-NAFLD patients. Numerous metabolic abnormalities, including IR and the severity of NAFLD, are to blame for this.

The study findings indicate a statistically significant variation in NAFLD degrees with respect to echocardiographic functions. Specifically, group Ⅰ exhibited higher levels of A and e' in comparison to the other groups.

This is supported by Awad et al. [16] they discovered that, in comparison to controls, NAFLD patients had higher mean diastolic blood pressure levels. However, they showed that neither the EF nor the E'/A' ratio differed statistically significantly between controls and obese children and adolescents, with or without NAFLD. This discrepancy from our results could be explained by the differing sample size, age, and BMI.

Zaki et al. [17] conducted a study on 34 healthy normal persons (NL) and 49 patients with varying degrees of hepatic cirrhosis. Using the Child-Pugh score, patients were divided into three groups according to the severity of their conditions: group A included 12 patients, group B included

17, and group C included 21 patients. Group C's tricuspid annular velocity was substantially higher and its T-E/A ratio was significantly lower than that of the NL group. In addition, group A and group C's E/Ea ratios were noticeably larger than those of the NL group.

Our results shown that diastolic dysfunction can be independently predicted by HTN, NAFLD, TC, and LA among the study groups.

This came in line with Armandi et al. [18] who demonstrated the link between diastolic dysfunction and severe liver fibrosis.

Skouloudi et al. [19] found that the only predictor that was independently linked with the outcome was the LA reservoir strain in patients with end-stage liver disease.

Providing evidence for our findings, Catena et al. [12] demonstrated that when the number of steatosis scores increases, diastolic function deteriorates.

Gianotti et al. [20] and Bonapace et al. [21] have shown that non-alcoholic fatty liver disease (NAFLD) is linked to changes in cardiac morphology and left ventricular (LV) diastolic dysfunction, which is in line with our results.

Kim et al. [22] discovered that LV diastolic dysfunction was linked to NAFLD identified by ultrasonography.

Study limitations: The results of this single-center study might differ from those obtained elsewhere. small sample size that could yield unimportant findings. There was no healthy control group in the study.

CONCLUSIONS

Since NAFLD grades were linked to the E/e' ratio, or diastolic dysfunction, in the asymptomatic patient, there was a strong association between cardiac diastolic dysfunction and NAFLD.

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