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Chronic heart failure in patients with nonalcoholic fatty liver disease: prevalence, clinical features, and relevance

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Abstract

Objective: This study was performed to assess the prevalence of nonalcoholic fatty liver (NAFL) in patients with symptomatic congestive heart failure (CHF) and compare the clinical features with those of patients without NAFL.

Methods: In total, 102 patients with CHF were divided into NAFL and non-NAFL groups according to their hepatic ultrasonography findings. All patients underwent transthoracic echocardiography and cardiac magnetic resonance examination. Follow-up was performed for major cardiovascular events (MACE) and readmission due to heart failure at 1, 3, 6, and 12 months after the index hospitalization.

Results: NAFL was detected in 37 of 102 patients (36.27%). Compared with the non-NAFL group, patients with NAFL were younger, had a higher body mass index and left ventricular (LV) mass index, and had more severe fibrosis. MACE and readmission occurred in 15 patients in the NAFL group and 29 patients in the non-NAFL group, without a significant difference. Linear regression analysis revealed that after adjusting for confounders, NAFL was independently associated with the LV fibrosis size and the ratio of the LV fibrosis size to the LV mass index.

Conclusions: NAFL is present in more than one-third of patients with CHF and is associated with the severity of LV fibrosis.

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Background

Nonalcoholic fatty liver disease (NAFLD) is the most common chronic liver disorder. It is a clinicopathologic syndrome that is not associated with excessive alcohol consumption or other specific causes of liver damage. The pathological features of NAFLD include diffuse hepatocyte steatosis ranging from steatosis (nonalcoholic fatty liver [NAFL]) to nonalcoholic steatohepatitis, which involves inflammation and liver cell damage and, in some cases, liver cirrhosis. The prevalence of NAFLD in the general population is 14% to 30%,[1] while NAFL affects the majority of these patients. NAFLD is the hepatic manifestation of metabolic syndrome, which is a well-known risk factor for many cardiovascular diseases, including heart failure.

Because heart failure and NAFLD share the same risk factors and a similar pathophysiological process (i.e., organ fibrosis), it is reasonable to hypothesize that these two syndromes might have some potential correlations. Indeed, a few studies have shown that NAFLD is an independent risk factor for subclinical cardiac diastolic or systolic dysfunction.[2] However, no study has focused on the relationship between NAFLD and clinically overt congestive heart failure (CHF). Therefore, this study was performed to determine the prevalence of NAFL, as an example of NAFLD, in patients with CHF and to compare the differences in clinical features in patients with heart failure with versus without NAFL.

Methods

Patients

Consecutive patients with CHF admitted to Beijing Anzhen Hospital from September 2016 to August 2017 were enrolled in this study. The inclusion criteria were an age of $\geq$18 years and a confirmed clinical diagnosis of heart failure with reduced ejection fraction, in agreement with the 2016 European Society of Cardiology Guidelines.[3] Patients with and without typical symptoms and signs of acute decompensation of cardiac function were included in the study.

We excluded the following causes of acute heart failure, pathological states different from those of classic CHF, and other comorbidities that could affect the results: acute myocardial infarction with or without ST-segment elevation; acute myocarditis; pericardial tamponade; valvular heart disease, especially severe aortic stenosis and mitral stenosis; congenital heart disease and perinatal cardiomyopathy; a history of other known causes of chronic liver disease or cirrhosis and excessive alcohol consumption (defined as alcohol intake of $>$20 g/day for women and $>$30 g/day for men); end-stage renal disease; severe lung disease; severe mental illness; a history of malignancy; and pregnancy.

Patients were followed up by means of office visits and telephone interviews at 1, 3, 6, and 12 months after the index hospital admission. The composite study endpoint comprised rehospitalization for heart failure and major adverse cardiac events

(MACE) including cardiovascular death, cardiac transplantation, and cardiac resynchronization therapy due to drug refractory heart failure.

The Ethics Committee of Beijing Anzhen Hospital approved the study protocol, and all participants provided informed written consent.

Clinical and laboratory data

A detailed clinical history was obtained and a physical examination was performed for each patient. The body mass index (BMI) was calculated as body weight in kilograms divided by the square of height in meters when the patient had reached his or her dry body weight after diuretic therapy. The left ventricular mass (LVM) index was calculated by dividing the LVM by the body surface area. The following formula was used to determine the body surface area: $(0.00607 \times \text{height}) + (0.0127 \times \text{weight}) - 0.0698$ for men, and $(0.00586 \times \text{height}) + (0.0126 \times \text{weight}) - 0.0461$ for women. Patients with decompensation of heart failure were stabilized with appropriate treatment before they were further evaluated.

Venous blood samples were drawn in the morning after $\geq$8 hours of overnight fasting. Routine blood tests and biochemistry measurements were performed following a standard protocol of the central laboratory at Beijing Anzhen Hospital. The estimated glomerular filtration rate was determined by the four-variable Modification of Diet in Renal Disease study equation. Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate of <60 mL/minute/1.73 m^2.

Imaging examinations

Transthoracic echocardiography. Conventional transthoracic echocardiography, which was performed for all patients at rest, was used to measure the LV diameter, LV ejection fraction (LVEF), wall thickness, LV end-diastolic volume, LV end-systolic volume, and flow velocity of the cross valve of each valve. The LVEF was determined using the biplane modified Simpson method from LV cavity tracings that included the papillary muscles and were measured in the apical four-chamber and two-chamber views. The LVEF was calculated as follows: [(LV end-diastolic volume – LV end-systolic volume)/LV end-diastolic volume] $\times$ 100.

Cardiac magnetic resonance. All cardiac magnetic resonance examinations were performed using a 3.0-T magnetic resonance imaging (MRI) system (Verio; Siemens Medical Solutions, Erlangen, Germany) with a 32-channel phased-array coil. True fast imaging to obtain steady-state free precession breath-hold cine images was performed in the short-axis view and the two-, three-, and four-chamber views encompassing the entire LV volume from the apex to the base. Diastolic-phase myocardial delayed-enhancement images in the same orientations as the cine images were acquired 10 minutes after intravenous infusion with gadolinium chelate contrast agent (0.2 mmol/kg, Magnevist; Bayer Schering, Berlin, Germany) with a prospectively electrocardiographic-gated gradient echo sequence with an inversion prepulse. Inversion times were optimized for null normal myocardium. The imaging parameters were as follows: repetition time/echo time, 4.1/1.6 ms; flip angle, 20°; image matrix, 256×130; and section thickness, 8 mm (contiguous short-axis images) or 5 mm (long-axis images) with no intersection gap.

Image processing. The QMASS commercial software package (Medis, Leiden, the Netherlands) was used to analyze the cardiac magnetic resonance Digital Imaging and Communications in Medicine images. The cardiac functional indexes, specifically the

LVEF and LVM, were quantified from cine images using standard methods.

Late gadolinium enhancement quantification. Normal myocardium was visually defined as a region of myocardium without any apparent late gadolinium enhancement (LGE) during visual inspection. The mean signal intensity and standard deviation (SD) were determined by drawing a region of interest (ROI) in a portion of the normal myocardium (a sample of $\geq$100 pixels per ROI) on three consecutive midventricular image sections. The mean signal intensity and SD were averaged across the three midventricular sections to yield the average mean signal intensity and SD. Manual planimetry of all highly enhanced pixels on the short-axis stack of LGE images was performed to visually quantify LGE. For comparison, a semi-automated gray-scale threshold technique was performed using 6 SD above the mean signal intensity for the normal null myocardium and 2 SD above noise (i.e., mean signal intensity of a region located outside the body). The quantity of LGE, namely the LV fibrosis size, was expressed in grams and was automatically generated by the software after defining the ROI. To correct the impact of the gross LV mass, we also calculated the ratio of the LV fibrosis size to the gross LV myocardial mass: [LV fibrosis size (g)/LV myocardial mass (g) $\times$ 100%], presented as M_{LGE}/M_M%.

Liver ultrasonography. Fatty liver was diagnosed based on characteristic ultrasonographic features; i.e., diffuse hyperechogenicity of the liver relative to the cortex of the right kidney, ultrasound beam attenuation, and poor visualization of both the intrahepatic vessel walls and diaphragm. Semiquantitative ultrasonographic scoring of the degree of fatty liver (mild, moderate, or severe) was performed. Mild fatty liver was defined as enhanced near-field echo, no obvious attenuated far-field echo, and visible intrahepatic tubular structure. Moderate fatty liver was defined as enhanced near-field echo, attenuated far-field echo, and fuzzy intrahepatic tubular structure. Severe fatty liver was defined as significantly enhanced near-field echo, obviously attenuated far-field echo, and no clear liver tubular structure.

Statistical analysis

Data are expressed as mean $\pm$ SD, median, and interquartile range or percentage. The t-test or a Mann–Whitney nonparametric test was used to compare the continuous variables as appropriate, while the chi-square test was used to compare the categorical data. The univariate linear regression analysis was conducted to determine variables that might be associated with the LVM and severity of LV fibrosis. Parameters representing the patient's clinical characteristics, electrocardiography measurements, MRI findings, and the presence of NAFL were tested by univariate linear regression analysis as appropriate. A P value of $\leq$0.1 was considered eligible to enter the multivariate analysis. The multivariate linear stepwise regression analysis results were then estimated. Analyses were performed using SPSS 16.0 statistical software (SPSS Inc., Chicago, IL, USA). A P value of $\leq$0.05 was regarded as statistically significant.

Results

Clinical characteristics and prevalence of NAFL

During the enrollment period, 152 patients were screened according to the inclusion criteria, while 50 patients were excluded because at least one of the exclusion criteria was met. Overall, 102 patients (71 men, 51 women) with heart failure with reduced ejection fraction were entered into the

final analysis. The mean patient age was 53.99 ± 16.97 years. The prevalence of NAFL diagnosed by ultrasonography was 36.27% (mild NAFL, 86.49%; moderate NAFL, 10.81%; severe NAFL, 2.70%). Accordingly, the 102 patients were divided into the NAFL group (37 patients) and non-NAFL group (65 patients). After extensive cardiac examination of all patients at baseline, 36 patients (35.29%) had established ischemic cardiomyopathy (ICM) and 66 patients (64.71%) were diagnosed with non-ischemic dilated cardiomyopathy. Among the entire cohort, 97.06% of patients were prescribed either an angiotensin-converting enzyme inhibitor (ACEI) or an angiotensin II receptor blocker (ARB), and 96.08% were prescribed a beta-blocker.

Compared with patients in the non-NAFL group, patients with NAFL were younger (47.91 ± 16.11 vs. 57.53 ± 16.62 years, $P = 0.006$) and had a higher BMI (26.62 ± 7.01 vs. $24.44 \pm 3.56\,kg/m^2$, $P = 0.036$). Patients with NAFL also had a larger LVM, LVM index, LV fibrosis size, and the proportion for which it account in the gross mass ($M_{LGE}/M_M\%$). Other clinical characteristics and laboratory measurements, including sex, plasma N-terminal pro B-type natriuretic peptide (NT-proBNP) level, plasma triglyceride level, LV end-diastolic diameter, LV end-systolic diameter, LV wall thickness, LVEF, and early diastolic and atrial velocity ratio (E/A ratio), were not significantly different between the two groups (Table 1).

Relationship between NAFL and MRI-defined LV lesions

On univariate linear regression analysis, there was a significant correlation between LV fibrosis size and age (p=0.098), posterior wall thickness (PWT) (p=0.020), and NAFL status (p=0.005). However, no significant correlation was found between LV fibrosis size and sex, weight, BMI, LVEF, interventricular septum (IVS), LV diastolic diameter (LVDd), LV systolic diameter (LVSd), smoking, diabetes mellitus (DM), CKD, ICM, hypertension, use of medications (ACEI/ARB and beta-blocker), NT-proBNP, total cholesterol, triglycerides, or low- or high-density lipoprotein. After adjusting for age and PWT in the multivariate linear regression model, the presence of NAFL became the only factor that was independently associated with LV fibrosis size ($\beta = 0.241$, $P = 0.017$) (Table 2).

We also found a significant correlation between the LVM index and age, LVDd, IVS, PWT, triglycerides, NAFL status, and LV fibrosis size ($P \leq 0.1$). No significant correlation was found between the LVM index and sex, BMI, weight, LVSd, LVEF, ICM, DM, CKD, hypertension, smoking, NT-proBNP, total cholesterol, or low- or high-density lipoprotein. Multivariate stepwise regression analysis revealed that LVDd ($\beta = 0.241$, $P = 0.005$), IVS ($\beta = 0.392$, $P = 0.001$), NAFL ($\beta = 0.139$, $P = 0.039$), and LV fibrosis size ($\beta = 0.170$, $P = 0.044$) were independently associated with the LVM index (Table 3).

Similarly, we found that the presence of NAFL was the only factor that was independently associated with $M_{LGE}/M_M\%$ in the univariate linear regression model ($\beta = 0.203$, $P = 0.040$) (Table 4); therefore, no multivariate analysis was required.

Follow-up

After a median follow-up of 9.6 months (range, 3–12 months), nine patients were lost, resulting in a follow-up rate of 91.2%. During the follow-up period, 44 patients (47.3%) developed endpoint events including 3 MACE and 12 readmissions in the NAFL group and 5 MACE and 24 readmissions in the non-NAFL group (44.1% vs. 49.2%, respectively).

Table 1. Patients' characteristics.

	NAFL group (n = 37)	Non-NAFL group (n = 65)	P
Male, %	72.97 (n = 27)	67.69 (n = 44)	0.658
Age, years	47.91 ± 16.11	57.53 ± 16.62	**0.006**
Weight, kg	76.35 ± 20.74	67.91 ± 13.57	**0.031**
BMI, kg/m^2	26.62 ± 7.01	24.44 ± 3.56	**0.036**
Smoking, %	48.65 (n = 18)	46.15 (n = 30)	0.839
AST, U/L	24 (14–29)	26 (20–35)	0.785
ALT, U/L	18 (12–33)	21 (12–32)	0.935
TG, mmol/L	1.56 (0.85–2.11)	1.15 (0.84–1.59)	0.373
TC, mmol/L	4.93 ± 1.15	4.94 ± 1.14	0.971
LDL, mmol/L	3.28 ± 1.30	3.35 ± 1.10	0.107
HDL, mmol/L	1.71 ± 0.38	1.85 ± 0.37	0.819
NT-proBNP, pg/mL	1685 (943–2302)	1876 (1060–3432)	0.133
LVM, g	182.45 ± 69.32	139.98 ± 51.58	**0.001**
LVM index, g/m^2	100.78 ± 52.49	79.89 ± 30.06	**0.012**
LV fibrosis size, g	14.67 ± 9.35	8.80 ± 7.31	**0.025**
M_{LGE}/M_M%	8.81 ± 8.63	6.07 ± 4.70	**0.048**
LA diameter, mm	46.86 ± 11.17	44.51 ± 9.75	0.270
LVDd, mm	66.30 ± 12.40	62.50 ± 10.60	0.073
LVSd, mm	55.34 ± 9.34	53.23 ± 9.81	0.083
IVS, mm	9.00 (7.00–10.25)	9.00 (7.00–10.00)	0.255
PWT, mm	8.50 (7.00–11.00)	8.00 (7.00–10.00)	0.228
LVEF, %	24.11 ± 8.54	25.05 ± 10.58	0.645
E, m/s (n = 87)*	94.32 ± 37.22	104.00 ± 40.6	0.256
A, m/s (n = 87)*	67.35 ± 21.32	66.95 ± 28.70	0.959
E/A (n = 87)*	1.24 ± 0.73	1.71 ± 0.93	0.075
Diastolic dysfunction (E/A < 0.8) (n = 87)	11 (36.71)	16 (24.63)	0.477
DM	6 (16.23)	22 (33.12)	0.067
Hypertension	12 (32.43)	25 (38.46)	0.669
ICM	10 (27.11)	26 (40.12)	0.204
CKD	2 (5.85)	5 (7.64)	0.380
ACEI-ARB	35 (94.59)	64 (98.46)	0.297
Beta-blocker	35 (94.59)	63 (96.92)	0.620

Data are presented as mean ± standard deviation, median (range), or n (%).

*Of 102 patients, 87 had sinus rhythm and the other 15 had persistent atrial fibrillation. Therefore, the A wave was not obtained for those 15 patients.

BMI, body mass index; AST, aspartate aminotransferase; ALT, alanine aminotransferase; TG, triglycerides; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; NT-proBNP, N-terminal pro B-type natriuretic peptide; LVM, left ventricular mass; LV, left ventricular; LA, left atrial; LVDd, left ventricular diastolic diameter; LVSd, left ventricular systolic diameter; IVS, interventricular septum; PWT, posterior wall thickness; LVEF, left ventricular ejection fraction; E, transmitral Doppler early diastolic wave; A, transmitral Doppler atrial diastolic wave; E/A, early diastolic and atrial velocity ratio; DM, diabetes mellitus; ICM, ischemic cardiomyopathy; CKD, chronic kidney disease; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

$P \leq 0.05$ was regarded as statistically significant.

Table 2. Correlations between left ventricular fibrosis size and other factors during linear regression analysis.

	Univariate linear regression		Multivariate stepwise regression	
	β	P	β	P
Age	−0.147	**0.098**	−0.031	0.761
PWT	0.254	**0.020**	0.187	0.062
NAFL	0.276	**0.005**	0.241	**0.017**
IVS	0.135	0.176	–	
BMI	0.078	0.438		
LVDd	0.027	0.791		
LVSd	0.032	0.748		
LVEF	−0.027	0.786		
Sex	−0.063	0.530		
Weight	0.154	0.122		
CKD	0.101	0.314		
ICM	0.026	0.799		
DM	−0.006	0.953		
Hypertension	0.030	0.767		
Smoking	0.095	0.344		
NT-proBNP	0.013	0.897		
TG, mmol/L	0.053	0.594		
TC, mmol/L	0.011	0.753		
LDL, mmol/L	0.081	0.418		
HDL, mmol/L	0.064	0.522		
ACEI/ARB	0.002	0.986		
Beta-blocker	0.001	0.990		

PWT, posterior wall thickness; NAFL, nonalcoholic fatty liver; IVS, interventricular septum; BMI, body mass index; LVDd, left ventricular diastolic diameter; LVSd, left ventricular systolic diameter; LVEF, left ventricular ejection fraction; DM, diabetes mellitus; ICM, ischemic cardiomyopathy; CKD, chronic kidney disease; NT-proBNP, N-terminal pro B-type natriuretic peptide; TG, triglycerides; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blocker.
$P \leq 0.1$ and $P \leq 0.05$ were regarded as statistically significant during univariate linear regression and multivariate stepwise regression, respectively.

Discussion

To the best of our knowledge, this is the first observational study to explore the probable relationship between chronic CHF and NAFL. We found that in patients with heart failure with reduced ejection fraction of different causes, the prevalence of NAFL diagnosed by ultrasonography was >36%, which is higher than that in the general population.[4] Compared with patients who had normal liver ultrasound findings, patients with both CHF and NAFL were significantly younger and had a higher BMI, larger LVM, larger LVM index, and larger LV myocardial fibrosis size. The presence of NAFL was independently associated with the LV fibrosis size and the proportion for which it accounted in the gross LV mass.

NAFLD has been identified as a common comorbidity in patients with DM, obesity, hypertension, or increased left wall thickness and has been associated

Table 3. Correlations between LVM index and other factors during linear regression analysis.

	Univariate linear regression		Multivariate stepwise regression	
	β	P	β	P
Age	−0.205	**0.039**	−0.016	0.856
LVDd	0.167	**0.093**	0.241	**0.005**
IVS	0.225	**0.025**	0.392	**0.001**
PWT	0.228	**0.005**	0.128	0.248
TG, mmol/L	0.185	**0.063**	0.120	0.138
NAFL	0.248	**0.012**	0.139	**0.039**
LV fibrosis size	0.296	**0.003**	0.170	**0.044**
Sex	−0.155	0.119	–	
BMI	0.107	0.283		
Weight	0.158	0.113		
LVSd	0.137	0.131		
LVEF	−0.120	0.230		
ICM	−0.500	0.538		
DM	0.127	0.203		
CKD	0.113	0.753		
Hypertension	−0.156	0.117		
Smoking	0.047	0.562		
NT-proBNP	0.016	0.871		
TC, mmol/L	0.020	0.842		
LDL, mmol/L	−0.093	0.353		
HDL, mmol/L	−0.107	0.283		

LVM, left ventricular mass; BMI, body mass index; LVDd, left ventricular diastolic diameter; LVSd, left ventricular systolic diameter; LVEF, left ventricular ejection fraction; IVS, interventricular septum; PWT, posterior wall thickness; NAFL, nonalcoholic fatty liver; ICM, ischemic cardiomyopathy; DM, diabetes mellitus; CKD, chronic kidney disease, TG, triglycerides; NT-proBNP, N-terminal pro B-type natriuretic peptide; TC, total cholesterol; LDL, low-density lipoprotein; HDL, high-density lipoprotein.
$P \leq 0.1$ and $P \leq 0.05$ were regarded as statistically significant during univariate linear regression and multivariate stepwise regression, respectively.

with varying degrees of subclinical LV diastolic or systolic dysfunction.[5–12] Even in such patients who do not yet present symptoms of heart failure, it is suggested that early intervention of NAFLD may delay or suppress the progression to heart failure. In the present study, all patients had overt heart failure, indicating that they were sicker than the patients enrolled in previous studies[5–12] and that cardiac remodeling was at a much more advanced stage. Not surprisingly, we found no difference in the cardiac morphological and functional parameters between the NAFL and non-NAFL groups because at this point, the underlying structural heart disease had become the dominant determinant of the pathogenesis of heart failure. However, our patients in the NAFL group were also younger and had a higher BMI, suggesting that they might have had significant metabolic syndrome. This may have resulted in the premature development of decompensated heart failure compared with those without NAFL.

Importantly, we found that the presence of NAFL is independently associated with the LV fibrosis size and the $M_{LGE}/M_M\%$. Previous studies have shown that hepatic steatosis may lead to insulin resistance,

Table 4. Correlations between M_{LGE}/M_M% and other factors during univariate linear regression analysis.

	β	P
Age	−0.074	0.462
BMI	0.001	0.993
Sex	−0.021	0.837
Weight	0.105	0.294
NAFL	0.203	**0.040**
ICM	0.055	0.584
DM	0.027	0.789
CKD	0.113	0.753
Smoking	0.111	0.267
NT-proBNP	0.030	0.762

BMI, body mass index; NAFL, nonalcoholic fatty liver; ICM, ischemic cardiomyopathy; DM, diabetes mellitus; CKD, chronic kidney disease; NT-proBNP, N-terminal pro B-type natriuretic peptide.
$P \leq 0.1$ was regarded as statistically significant.

which in turn induces excessive lipid metabolites. These metabolites in circulation can cause myocyte lipid toxicity, induce myocardial apoptosis, and activate inflammatory factors.[13–16] These abnormalities aggravate myocardial fibrosis in patients with structural heart disease. The process of excessive deposition of collagen fibers in the extracellular matrix of the myocardium can increase ventricular mass and decrease ventricular compliance, eventually leading to cardiac dysfunction. Because of the increased proportion of fibrotic tissue in the LV (M_{LGE}/M_M% of 8.81), patients with NAFL had a larger LVM and LVM index than patients without NAFL (M_{LGE}/M_M% of 6.07).

Alternatively, abnormal systemic lipid metabolism can cause not only liver steatosis but also lipid deposition in myocardial tissue and myocytes. Excessive triglycerides participate in the circulatory biochemical progress of hydrolysis and re-esterification and produce toxic substances such as ceramides and diacylglycerols in myocytes and interstitial spaces, thus leading to myocardial dysfunction.[17–19] Indeed, a previous study showed that patients with NAFLD had more significant lipid infiltration.[20] This also explains why our patients in the NAFL group had a greater LVM and LVM index.

It is well known that age is an important influential factor for myocardial fibrosis and heart function impairment, and the intensity of the impact grows as age progresses. Patients in the NAFL group of the present study were younger than those in the non-NAFL group, but their LV size, wall thickness, LVEF, and E/A ratio were not significantly different. This suggests that regardless of whether the underlying cause of heart failure was ischemic or non-ischemic, patients with NAFL may have entered the cardiac decompensation stage earlier than those without NAFL. Therefore, we conclude that NAFL might be a contributing factor for the progression of heart failure. The prevalence of NAFL in this group of patients with heart failure was 36.3%; thus, the aggravation and acceleration of heart failure should not be underestimated.

Limitations

The sample size of our study was small and the follow-up time was relatively short; therefore, the study was not powered to investigate the impact of NAFLD on the long-term outcomes of patients with heart failure. Data regarding the fasting serum insulin level or wrist circumference were not available in all patients, so we were not able to assess the correlation of NAFLD with insulin resistance or metabolic syndrome in the present study. Additionally, because of the lack of liver pathology results, we could not stratify or evaluate the relationship between heart failure and NAFLD according to the severity of NAFLD. Finally, quantitative data regarding the myocardial and liver lipid content could not be obtained.

Conclusion

Among patients with chronic CHF, 36.27% had concomitant NAFL. The presence of NAFL was independently associated with the LV fibrosis size and might have accelerated and aggravated the progression of heart failure. Further research focusing on the underlying mechanism and large clinical studies with a long-term follow-up are warranted.

Declaration of conflicting interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr. Rong Bai is an awardee of the Program of Beijing High-caliber Talent from Overseas (BHTO201410007) and the Overseas High-level Talent of the Phoenix Plan of the Chaoyang District, Beijing. The other authors declare that there is no conflict of interest.

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