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Cardiac Structural Alterations in Patients with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): A Cross-Sectional Analysis at a Tertiary Care Center

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Abstract

Background: Metabolic dysfunction Associated Steatotic Liver Disease (MASLD) is increasingly recognized as a systemic cardiometabolic disorder. Data on subclinical cardiac alterations from tertiary care centers in South Asia remain limited.

Objective: To evaluate cardiac structural and functional alterations by 2D echocardiography in MASLD patients compared with controls, and to examine their relationship with hepatic fibrosis severity.

Methods: This cross-sectional study enrolled 50 MASLD patients and 20 age- and sex-matched controls. MASLD was diagnosed by liver ultrasonography meeting 2023 consensus criteria. Hepatic fibrosis was assessed by FIB-4 index, APRI, and transient elastography. All participants underwent standard 2D transthoracic echocardiography assessing Left Ventricular (LV) Mass Index (LVMI), wall thickness, ejection fraction, and diastolic parameters.

Results: Mean patient age was 47.2±9.8 years (female-to-male ratio 1.38:1). LV hypertrophy was present in 60% of MASLD patients (as an isolated finding in 40%, and co-existing with diastolic dysfunction in a further 20%), while isolated diastolic dysfunction was present in an additional 20%; overall 80% demonstrated at least one echocardiographic abnormality versus 10% of controls. Ejection fraction was preserved in all participants. Binary logistic regression yielded an odds ratio of 28.5 (95% CI 5.8-139.4) for any echocardiographic abnormality in MASLD versus controls. Mild-to-moderate fibrosis (F0-F2) was present in 88% and moderate-to-severe fibrosis (F3-F4) in 12%, with more pronounced cardiac alterations in the latter subgroup.

Conclusion: MASLD is independently associated with subclinical cardiac structural and functional alterations even at low-to-moderate fibrosis stages, mandating routine echocardiographic surveillance in this population.

Keywords: MASLD; NAFLD; Left Ventricular Hypertrophy; Diastolic Dysfunction; Echocardiography; Hepatic Fibrosis

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Introduction

Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD) is the contemporary nomenclature for the disease spectrum previously designated as Non-Alcoholic Fatty Liver Disease (NAFLD), a terminological revision recommended by a multinational expert consensus in 2023 to better reflect the central role of cardiometabolic dysregulation in its pathogenesis [1]. MASLD encompasses a clinicopathological continuum ranging from isolated hepatic steatosis through Metabolic Dysfunction-Associated Steatohepatitis (MASH), progressive hepatic fibrosis, and ultimately cirrhosis or hepatocellular carcinoma [1, 2]. Population-based estimates place its global prevalence at approximately 25%, with a rising burden in South and Southeast Asia attributable to sedentary lifestyles, dietary transition, and escalating rates of central obesity [1, 2].

While hepatic complications have traditionally dominated the clinical foreground, accumulating evidence has repositioned cardiovascular disease as the primary driver of morbidity and mortality in MASLD [3]. The mechanisms underlying hepato-cardiac cross-talk are multifactorial. Hepatic steatosis amplifies insulin resistance, promotes atherogenic dyslipidaemia, and drives hepatic secretion of pro-inflammatory cytokines - including tumour necrosis factor-alpha, interleukin-6, and plasminogen activator inhibitor-1 - that exert direct injurious effects on myocardial architecture

and electrophysiology [4, 5]. Expansion of epicardial adipose tissue, acting as a paracrine source of adipokines and inflammatory mediators, has been independently linked to hepatic fibrosis severity and adverse cardiac remodeling [5].

Two-dimensional echocardiography remains the most widely accessible, radiation-free, and reproducible imaging modality for assessing cardiac structure and function in resource-constrained settings. Large epidemiological cohorts, including the Framingham Heart Study, have demonstrated that hepatic fat correlates significantly with LV mass, wall thickness, and LV filling pressure even after adjustment for conventional cardiometabolic risk factors [3]. A meta-analysis of 31 observational studies confirmed that MASLD patients have more than twofold higher odds of diastolic dysfunction compared with those without steatotic liver disease, alongside significantly elevated LV mass index and left atrial volume index [6]. Longitudinal data from the Coronary Artery Risk Development in Young Adults (CARDIA) study established that MASLD independently predicts incident LV hypertrophy and abnormal LV geometry over five years [7].

Despite this global evidence base, data from tertiary care centers in the Indian subcontinent remain sparse. Distinctive epidemiological features of Indian MASLD patients - including a lower BMI threshold for metabolic risk, a higher proportion of lean MASLD, and prevalent comorbidities such as Type 2 Diabetes Mellitus (T2DM) and hypertension - may modulate the cardiac phenotype in ways not fully captured by Western cohorts [8]. The present cross-sectional study was therefore designed to characterize the spectrum of echocardiographic structural and functional alterations in MASLD patients at a tertiary care center in North India, and to examine their relationship with hepatic fibrosis severity determined by non-invasive indices and transient elastography.

Materials and Methods

Study design and setting

This cross-sectional, observational study was conducted from Jun 2025 to Dec 2025 at the Department of General Medicine of a tertiary care center in North India. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

Participants

Fifty consecutive MASLD patients and 20 age- and sex-matched controls were enrolled. Eligible MASLD patients were adults (≥18 years) with hepatic steatosis on ultrasonography meeting at least one criterion for metabolic dysfunction per the 2023 MASLD nomenclature consensus [1, 2]. Exclusion criteria included significant alcohol consumption (>14 units/week for women, >21 units/week for men), secondary causes of hepatic steatosis (thyroid dysfunction, corticosteroid use, total parenteral nutrition), established heart failure, valvular heart disease, ischemic heart disease, cardiac arrhythmia, chronic kidney disease (eGFR <60 mL/min/1.73 m^2), and pregnancy.

Clinical assessment

Anthropometric measurements (height, weight, BMI, waist circumference), blood pressure, comorbidities, and medication and addiction histories were systematically documented. Hypertension was defined as systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg on two separate occasions, or current antihypertensive use. T2DM was diagnosed per ADA 2025 criteria [9].

Laboratory investigations

Fasting blood glucose, lipid profile, complete blood count, renal and liver function tests (AST, ALT), and platelet count were measured. Hepatic fibrosis risk was estimated using two validated non-invasive indices. The FIB-4 index was calculated as: (Age [years]×AST [U/L]) / (Platelet count [×10^9/L] × √ALT [U/L]), with thresholds of <1.30 (low risk), 1.30-2.67 (intermediate), and >2.67 (high risk) [10]. The APRI was calculated as: (AST / upper limit of normal) / Platelet count (×10^9/L)×100, with <0.5 indicating low risk [11].

Liver elastography

Hepatic stiffness was measured by transient elastography (FibroScan; Echosens, France) and fibrosis staged as: F0-F1 (<7.0 kPa), F2 (7.0-9.4 kPa), F3 (9.5-12.4 kPa), and F4 (≥12.5 kPa). For analytical purposes, patients were dichotomized into mild-to-moderate (F0-F2) and moderate-to-severe (F3-F4) fibrosis groups.

Echocardiographic assessment

Standard 2D transthoracic echocardiography was performed by a trained cardiologist blinded to fibrosis status, following American Society of Echocardiography (ASE) guidelines [12]. Parameters assessed included IVST-d, PWT, LVIDd, LVMI (Devereux formula), Relative Wall Thickness (RWT), ejection fraction (EF, modified Simpson's biplane method), mitral E and A wave velocities, E/A ratio, deceleration time, tissue Doppler-derived mitral annular early diastolic velocity (e'), and E/e' ratio. LV hypertrophy was defined as LVMI >115 g/m^2 in men and >95 g/m^2 in women. Diastolic dysfunction was graded per ASE/EACVI guidelines [12].

Statistical analysis

Analysis was performed using SPSS version 26.0 (IBM Corp., USA). Continuous variables are expressed as mean±SD and compared by independent samples t-test or Mann-Whitney U test as appropriate. Categorical variables are expressed as frequencies and percentages and compared by Chi-square or Fisher's exact test. Binary logistic regression estimated the OR for any echocardiographic abnormality in MASLD *versus* controls, adjusted for age and sex. A *p*-value <0.05 was considered statistically significant.

Results

Demographic and clinical characteristics

Fifty MASLD patients (mean age 47.2±9.8 years; 29 women, 21 men) and 20 controls (mean age 46.1±10.3 years; 13 women, 7 men) were enrolled. Age and sex distribution did not differ significantly between groups (p=0.64 and p=0.81, respectively). BMI (27.4±3.6 *vs.* 23.8±2.9 kg/m^2; p = 0.001), waist circumference (91.3±9.1 *vs.* 80.6±7.4 cm; p <0.001), systolic BP (128.4±14.2 *vs.* 118.6±11.3 mmHg; p=0.004), and diastolic BP (82.1±9.6 *vs.* 76.3±8.2 mmHg; p=0.009) were all significantly higher in MASLD patients. T2DM was present in 22% of patients versus none of the controls (p=0.02). Overall, 44% of MASLD patients had at least one cardiometabolic comorbidity versus 10% of controls (p=0.003). Serum ALT (52.4±28.6 *vs.* 24.2±9.8 U/L; p<0.001), AST (44.6±22.3 *vs.* 22.8±8.4 U/L; p<0.001), triglycerides (168.2±52.6 *vs.* 124.4±38.7 mg/dL; p<0.001), and LDL-cholesterol (128.3±34.2 *vs.* 108.6±26.1 mg/dL; p=0.01) were higher, while HDL-cholesterol was lower (41.8±9.2 *vs.* 48.6±8.7 mg/dL; p=0.002) in the MASLD group (Table 1).

Hepatic fibrosis assessment

FIB-4 classified 94% of patients as low risk, 4% intermediate, and 2% high risk; all controls were low risk. Mean FIB-4 was

Table 1: Baseline demographic, anthropometric, and biochemical characteristics of MASLD patients and controls.

Variable	MASLD (n=50)	Controls (n=20)	*p*-value
Age (years), mean±SD	47.2±9.8	46.1±10.3	0.64
Female, n (%)	29 (58.0)	13 (65.0)	0.81
BMI (kg/m²), mean±SD	27.4±3.6	23.8±2.9	0.001
Waist circumference (cm), mean±SD	91.3±9.1	80.6±7.4	<0.001
Systolic BP (mmHg), mean±SD	128.4±14.2	118.6±11.3	0.004
Diastolic BP (mmHg), mean±SD	82.1±9.6	76.3±8.2	0.009
Hypertension, n (%)	14 (28.0)	2 (10.0)	0.09
Type 2 diabetes mellitus, n (%)	11 (22.0)	0 (0.0)	0.02
Any cardiometabolic comorbidity, n (%)	22 (44.0)	2 (10.0)	0.003
Fasting blood glucose (mg/dL), mean±SD	104.6±28.3	87.2±10.4	0.001
Total cholesterol (mg/dL), mean±SD	196.4±38.1	172.8±29.3	0.007
Triglycerides (mg/dL), mean±SD	168.2±52.6	124.4±38.7	<0.001
LDL-cholesterol (mg/dL), mean±SD	128.3±34.2	108.6±26.1	0.01
HDL-cholesterol (mg/dL), mean±SD	41.8±9.2	48.6±8.7	0.002
ALT (U/L), mean±SD	52.4±28.6	24.2±9.8	<0.001
AST (U/L), mean±SD	44.6±22.3	22.8±8.4	<0.001

BP = Blood Pressure; BMI = Body Mass Index; ALT = Alanine Aminotransferase; AST = Aspartate Aminotransferase; LDL = Low-Density Lipoprotein; HDL = High-Density Lipoprotein

Table 2: Echocardiographic parameters in MASLD patients and controls.

Parameter	MASLD (n=50)	Controls (n=20)	*p*-value
Structural parameters			
IVST-d (mm), mean±SD	11.2±1.8	8.9±1.3	<0.001
PWT (mm), mean±SD	10.8±1.6	8.7±1.2	<0.001
LVIDd (mm), mean±SD	49.4±4.3	46.2±3.8	0.003
LV mass index (g/m²), mean±SD	117.6±24.8	86.4±16.2	<0.001
Relative wall thickness, mean±SD	0.46±0.09	0.38±0.07	<0.001
LV hypertrophy, n (%)	30 (60.0)	1 (5.0)	<0.001
Systolic parameters			
Ejection fraction (%), mean±SD	61.4±5.2	63.1±4.8	0.17
EF <50%, n (%)	0 (0.0)	0 (0.0)	—
Diastolic parameters			
Mitral E velocity (cm/s), mean±SD	71.3±14.6	79.8±12.2	0.01
Mitral A velocity (cm/s), mean±SD	74.2±16.4	62.4±11.8	0.001
E/A ratio, mean±SD	0.98±0.26	1.29±0.22	<0.001
Deceleration time (ms), mean±SD	226.8±38.4	189.4±28.6	<0.001
e' velocity (cm/s), mean±SD	8.2±2.4	11.6±2.1	<0.001
E/e' ratio, mean±SD	9.1±2.8	7.0±1.9	<0.001
Diastolic dysfunction, n (%)	20 (40.0)	1 (5.0)	0.002
Overall			
Any echocardiographic abnormality, n (%)	40 (80.0)	2 (10.0)	<0.001

IVST-d = Inter-Ventricular Septal Thickness at end-diastole; PWT = Posterior Wall Thickness; LVIDd = LV Internal Diameter at end-diastole; EF = Ejection Fraction; E = early diastolic mitral inflow velocity; A = late diastolic mitral inflow velocity; e' = mitral annular early diastolic velocity by tissue Doppler imaging. LV hypertrophy defined as LVMI >115 g/m² in men and >95 g/m² in women. Diastolic dysfunction graded per ASE/EACVI guidelines.

Table 3: Echocardiographic parameters stratified by hepatic fibrosis severity in MASLD patients.

Parameter	F0-F2 (n=44)	F3-F4 (n=6)	*p*-value
Structural parameters			
IVST-d (mm), mean±SD	11.0±1.8	12.6±1.4	0.04
PWT (mm), mean±SD	10.6±1.6	11.8±1.3	0.08
LV mass index (g/m²), mean±SD	114.8±23.9	138.4±22.6	0.03
Relative wall thickness, mean±SD	0.45±0.09	0.52±0.08	0.048
LV hypertrophy, n (%)	16 (36.4)	4 (66.7)	0.19
Systolic parameters			
Ejection fraction (%), mean±SD	61.5±5.1	60.8±5.6	0.73
Diastolic parameters			
E/A ratio, mean±SD	1.00±0.27	0.82±0.18	0.04
Deceleration time (ms), mean±SD	222.4±37.1	252.6±34.8	0.049
e' velocity (cm/s), mean±SD	8.5±2.3	6.4±1.8	0.04
E/e' ratio, mean±SD	8.8±2.7	11.4±2.6	0.03
Diastolic dysfunction, n (%)	16 (36.4)	4 (66.7)	0.19
Overall			
Any echocardiographic abnormality, n (%)	32 (72.7)	6 (100.0)	0.18

IVST-d = Inter-Ventricular Septal Thickness at end-diastole; PWT = Posterior Wall Thickness; E/A = early-to-late diastolic mitral inflow velocity ratio; e' = mitral annular early diastolic velocity; E/e' = ratio of mitral inflow to annular velocity. Continuous variables compared by independent samples t-test; categorical variables by Fisher's exact test throughout.

higher in patients (1.02±0.44 *vs.* 0.81±0.29; p=0.02). APRI placed all patients (100%) and 90% of controls in the low-risk category. On transient elastography, 62% had F0-F1 fibrosis, 26% F2, 8% F3, and 4% F4; overall, 88% had mild-to-moderate fibrosis (F0-F2) and 12% moderate-to-severe fibrosis (F3-F4). Mean liver stiffness was 6.84±3.12 kPa.

Echocardiographic findings

Structural parameters were significantly deranged in MASLD patients. IVST-d (11.2±1.8 *vs.* 8.9±1.3 mm; p<0.001), PWT (10.8±1.6 *vs.* 8.7±1.2 mm; p<0.001), LVMI (117.6±24.8 *vs.* 86.4±16.2 g/m²; p<0.001), and RWT (0.46±0.09 *vs.* 0.38±0.07; p<0.001) were all significantly elevated. EF was preserved in all participants (61.4±5.2 *vs.* 63.1±4.8%; p=0.17). Diastolic parameters were significantly impaired: E/A ratio was lower (0.98±0.26 *vs.* 1.29±0.22; p<0.001), deceleration time prolonged (226.8±38.4 *vs.* 189.4±28.6 ms; p<0.001), e' reduced (8.2±2.4 *vs.* 11.6±2.1 cm/s; p<0.001), and E/e' elevated (9.1±2.8 *vs.* 7.0±1.9; p<0.001) in MASLD patients (Table 2).

On categorical analysis (Figure 1), LVH was present in 60% of MASLD patients overall - as an isolated finding in 40% and co-existing with diastolic dysfunction in a further 20%. Isolated diastolic dysfunction without LVH was identified in an additional 20%. In total, 80% of MASLD patients demonstrated at least one echocardiographic abnormality versus 10% of controls (p<0.001). After adjustment for age and sex, MASLD remained significantly associated with any echocardiographic abnormality (OR 28.5; 95% CI 5.8-139.4; p<0.001). Given the limited sample size and the small number of events in the control group (n=2), adjustment was restricted to matching variables to avoid model overfitting.

Echocardiographic parameters by fibrosis severity

Among patients with F3-F4 fibrosis (n=6), LVMI (138.4±22.6 *vs.*

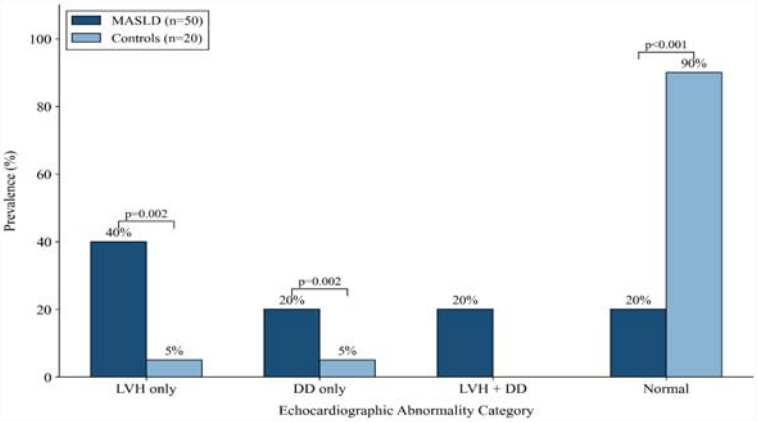

Figure 1: Prevalence of echocardiographic abnormality categories in MASLD patients (n=50) and controls (n=20). LVH = Left Ventricular Hypertrophy; DD = Diastolic Dysfunction. *p*-values by Fisher's exact test.

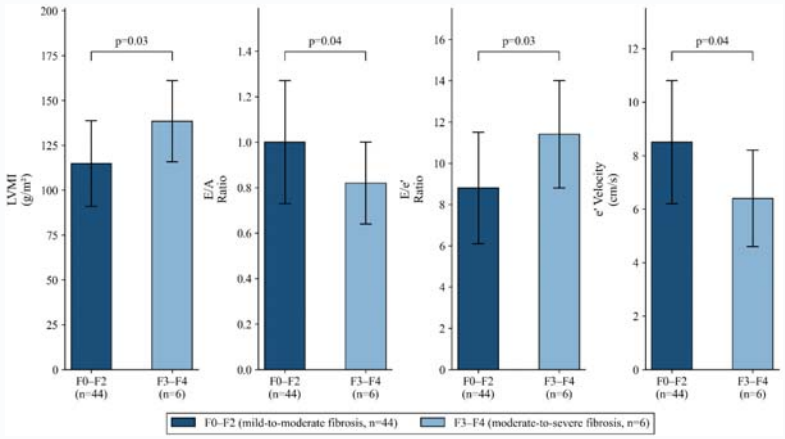

Figure 2: Key echocardiographic parameters stratified by hepatic fibrosis severity in MASLD patients. Data presented as mean±SD. LVMI = Left Ventricular Mass Index; E/A = early-to-late diastolic mitral inflow velocity ratio; E/e' = ratio of mitral inflow to annular early diastolic velocity; e' = mitral annular early diastolic velocity by tissue Doppler imaging. *p*-values by independent samples t-test.

114.8±23.9 g/m^2; p=0.03), IVST-d (12.6±1.4 *vs.* 11.0±1.8 mm; p=0.04), and RWT (0.52±0.08 *vs.* 0.45±0.09; p=0.048) were significantly higher, while E/A ratio (0.82±0.18 *vs.* 1.00±0.27; p=0.04), e' (6.4±1.8 *vs.* 8.5±2.3 cm/s; p=0.04), and E/e' (11.4±2.6 *vs.* 8.8±2.7; p=0.03) reflected more pronounced diastolic impairment compared with F0-F2 patients. All 6 patients with F3-F4 fibrosis had at least one echocardiographic abnormality, compared with 72.7% of F0-F2 patients (Table 3; Figure 2). The difference in overall abnormality prevalence between fibrosis strata did not reach statistical significance (p=0.18), likely reflecting the limited power conferred by the small F3-F4 subgroup (n=6); this comparison should therefore be interpreted as exploratory. EF did not differ across fibrosis strata (p=0.73).

Discussion

The principal finding of the present study is that MASLD patients carry a substantially elevated burden of subclinical cardiac structural and functional alterations detectable by 2D echocardiography, independent of established cardiometabolic risk factors. An OR of 28.5 (95% CI 5.8–139.4) for any echocardiographic abnormality in MASLD versus controls underscores the magnitude of this association, and the observation that 80% of patients demonstrated LV hypertrophy (60%), diastolic dysfunction (40%), or both contextualizes MASLD as a systemic cardiometabolic disorder rather than a purely hepatic condition.

LV hypertrophy, identified in 60% of our patients, is among the most consistently reported echocardiographic findings in MASLD. Hallsworth et al., using cardiac MRI in adults with NAFLD matched for age, sex, and BMI, demonstrated significantly increased LV wall thickness at both systole and diastole, with elevated eccentricity ratio consistent with concentric remodeling, independent of metabolic confounders [13, 14]. A large population-based analysis from the Study of Health in Pomerania (SHIP), utilizing cardiac MRI in 1,096 adults, confirmed that MASLD was independently associated with greater LVMI, LV concentricity, and end-diastolic wall thickness after multivariable adjustment, with a dose-response relationship between liver fat content and LVMI [15]. The Framingham Heart Study reported significant associations between hepatic fat and LV mass and wall thickness, though these attenuated after additional BMI adjustment except for the LV filling pressure association [3]. In contrast to this attenuation, MASLD remained an independent predictor of echocardiographic abnormalities after multivariable adjustment including BMI in the present study, possibly reflecting the metabolic phenotype of Indian MASLD patients in whom cardiometabolic risk is not fully captured by BMI alone.

The pathophysiological substrate for LV hypertrophy in

MASLD involves overlapping mechanisms. Insulin resistance activates the renin-angiotensin-aldosterone system and promotes cardiomyocyte hypertrophy via angiotensin II-mediated signaling [4, 5]. Hepatic secretion of TNF-α and IL-6 generates systemic low-grade inflammation contributing to oxidative stress and myocardial fibrosis [5]. Expansion of epicardial adipose tissue acts as a paracrine source of free fatty acids and adipokines that directly impair myocardial energetics [16]. Petta *et al*., in 147 biopsy-proven NAFLD patients, demonstrated that epicardial fat thickness was independently associated with severe hepatic fibrosis and correlated with LV mass, PWT, and RWT after controlling for cardiometabolic confounders [16], a fibrosis-severity gradient consistent with our F3-F4 subgroup findings.

Diastolic dysfunction - present in 20% of patients as an isolated finding and a further 20% in combination with LV hypertrophy - represents the functional corollary of structural remodeling and a recognized precursor of Heart Failure with preserved Ejection Fraction (HFpEF) [6, 17]. In the present study, E/A ratio, e' velocity, E/e' ratio, and deceleration time were all significantly deranged, a diastolic dysfunction phenotype consistent with pooled findings from prior meta-analyses [6, 18].

The mechanistic basis encompasses myocardial interstitial fibrosis driven by transforming growth factor-β, impaired calcium handling secondary to insulin resistance, and increased myocardial stiffness from subendocardial fat infiltration [4, 5]. The meta-analysis by Goliopoulou *et al*., synthesizing 31 observational studies, quantified a more than twofold higher odds of diastolic dysfunction in MASLD (OR 2.07; 95% CI 1.24-3.44; p=0.01), alongside significantly higher E/e' ratios and LVMI [6]. VanWagner *et al*., in the CARDIA cross-sectional analysis of 2,713 participants, documented lower e', higher E/e', and worse global longitudinal strain in NAFLD versus non-NAFLD participants after adjustment for heart failure risk factors and BMI [7, 19]. The longitudinal CARDIA follow-up further established that NAFLD independently predicted incident LV hypertrophy (OR 1.9) and abnormal LV geometry (OR 1.9) over five years [7]. The meta-analysis by Borges-Canha *et al*., pooling 16 studies, additionally identified significant associations of NAFLD with greater septal and posterior wall thickness, lower E/A ratio, higher E/e', and prolonged deceleration time - a diastolic phenotype closely mirroring our findings [18].

The preservation of EF in all patients mirrors findings from multiple prior studies and is consistent with the recognized phenotype of MASLD-associated cardiomyopathy at early-to-intermediate fibrosis stages. Hallsworth *et al*., confirmed that EF, cardiac output, and stroke volume were unaltered despite significant structural remodeling in NAFLD patients without established cardiac disease [13]. Cassidy et al., comparing cardiac MRI parameters in adults with NAFLD, T2DM, and healthy controls, found concentric remodeling in both NAFLD and T2DM groups, but overt systolic dysfunction was confined to the T2DM group; changes in NAFLD alone were subclinical [20]. Clinically, preserved EF should not provide false reassurance - the subclinical structural changes identified here represent actionable preclinical targets that warrant early intervention.

A key observation, consistent with the literature, is the apparent gradient of cardiac involvement with advancing fibrosis. Among F3-F4 patients (12% of the cohort), LVMI, IVST-d, E/e', and RWT were all significantly worse than in the F0-F2 majority, and all six had at least one echocardiographic abnormality versus 72.7% of F0-F2 patients. Anstee et al. established the biological plausibility of this gradient: advancing fibrosis progressively deranges hepatic capacity to clear pro-inflammatory mediators, amplifying systemic inflammation and its cardiac consequences [21]. Petta *et al*., demonstrated that PWT, LV mass, RWT, and E/A ratio were all linked to severe hepatic fibrosis in biopsy-proven NAFLD [16]. Crucially, the high prevalence of cardiac alterations in our F0-F2 majority underscores that clinically significant cardiac remodeling precedes advanced liver disease. Furthermore, the association between MASLD and arrhythmic risk - mediated through left atrial dilatation and LV hypertrophy - has been well documented; LV hypertrophy itself carries significantly elevated risk of both supraventricular and ventricular arrhythmias [5]. Valvular calcification represents an additional dimension of cardiac risk in MASLD that was not assessed in the present study but has been documented in T2DM patients with NAFLD, with ORs approaching 2.79 for aortic valve sclerosis and 2.70 for bivalvular calcification [22, 23].

Limitations of the Study

The cross-sectional design precludes causal inference. The sample size of 50 patients limits subgroup analyses, and the F3-F4 subgroup (n=6) is small; fibrosis-stratified comparisons should be regarded as exploratory and hypothesis-generating. Non-invasive fibrosis staging carries recognized limitations relative to biopsy. Absence of speckle-tracking echocardiography means subclinical systolic dysfunction may have been under detected. Valvular calcification and left atrial deformation parameters were not systematically assessed. Future studies should employ larger prospective cohorts, advanced echocardiographic modalities, longitudinal follow-up, and parallel cardiac biomarker assessment.

Conclusion

MASLD is significantly and independently associated with subclinical cardiac structural and functional alterations - most prominently LV hypertrophy and diastolic dysfunction - detectable by 2D echocardiography in the majority of affected patients, even at low-to-moderate hepatic fibrosis stages. The odds ratio of 28.5 for any echocardiographic abnormality versus controls reinforces the need to conceptualize MASLD as a systemic cardiometabolic disorder. Routine echocardiographic evaluation and an integrated hepato-cardiac management strategy should be incorporated into standard care for MASLD patients at tertiary centers.

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