FREQUENCY OF LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN TYPE 2 DIABETES MELLITUS PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

OBJECTIVES

The study aimed to determine the frequency of left ventricular diastolic dysfunction (LVDD) in type 2 diabetes mellitus (DM) patients with non-alcoholic fatty liver disease (NAFLD).

METHODOLOGY

This cross-sectional study was conducted in the Department of Medicine at Hayatabad Medical Complex, Peshawar, Pakistan, between 01.03.2025 and 10.09.2025. The study participants were patients with type 2 DM and concomitant NAFLD. Patients with a history of hypertension, ischemic heart disease, and chronic kidney disease were excluded. All patients underwent echocardiography, and LVDD was diagnosed based on the E/A ratio reversal. The association between LVDD and its risk factors (age, gender, body mass index, smoking status) was determined using univariate and multivariable analyses.

RESULTS

Of the 252 participants, 51 patients (20.2%) were diagnosed with LVDD. It was more frequent in males (22.4% vs 18.4%), individuals over 60 years of age (24.6% vs 16.4%), obese patients (21.9% vs 19%), and smokers (30.1% vs 15.4%). Nevertheless, univariate and multivariable regression analyses indicated that age, gender, and obesity were not statistically significant or independent predictors of LVDD. Conversely, smoking was identified as a significant independent risk factor in both univariate ($p = 0.006$) and multivariable regression analyses (AOR: 3.865; 95% CI: 1.384–10.793; $p = 0.010$).

CONCLUSION

A significant number of patients with type 2 DM and NAFLD showed LVDD. Smoking is independently linked to LVDD, highlighting the importance of quitting smoking and performing early cardiovascular screenings to prevent progression to overt heart failure in this high-risk group.

KEYWORDS: *Diabetes Mellitus, Non-alcoholic Fatty Liver Disease, Metabolically Dysfunctional Liver Disease, Left Ventricular Diastolic Dysfunction, Pakistan*

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD), recently redefined as metabolic dysfunction-associated steatotic liver disease (MASLD), is one of the most common chronic liver disorders worldwide, with a global prevalence of approximately 30.05%, and an even higher burden in South Asia (33.83%).[1] In Pakistan, NAFLD prevalence varies by province, being highest in Punjab (34.03%), followed by Sindh (30.27%) and Khyber Pakhtunkhwa (25.38%).[2] Non-alcoholic fatty liver disease encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis, which may progress to advanced fibrosis, cirrhosis, and hepatocellular carcinoma.[3] Left Ventricular diastolic dysfunction (LVDD) is an important, yet often underrecognized, cardiovascular complication associated with NAFLD. Left Ventricular diastolic dysfunction, a known precursor of heart failure with preserved ejection fraction, results from impaired ventricular relaxation and increased left atrial pressure, leading to reduced cardiac efficiency. Zahra et al. from Pakistan have reported that 71.42% of NAFLD patients exhibited LVDD compared to 14.28% of healthy controls, highlighting a substantial impact of hepatic steatosis on early cardiac impairment.[4] Emerging evidence increasingly supports the association between NAFLD and LVDD among individuals with type 2 diabetes mellitus (T2DM). Bonapace et al. (2012) from Italy showed that 64% of patients with T2DM and NAFLD had LVDD, compared with 36% in patients with T2DM without NAFLD, and that NAFLD independently predicted LVDD.[5] In 2025, Faramarzpour et al. from Iran showed that 81.1% with

T2DM and NAFLD were found to have LVDD.[6] In a Korean cohort, Lee et al. (2019) examined 606 T2DM patients aged ≥50 years and reported a higher prevalence of LVDD in those with NAFLD (59.7% vs. 49.0%).[7] Similarly, Mantovani et al. (2012) found NAFLD as an independent predictor of left ventricular hypertrophy in hypertensive T2DM patients.[8] Qureshi et al. (2016) from Bahawalpur (Pakistan) reported that LVDD was present in 38.16% of patients with T2DM with NAFLD, highlighting a substantial burden of subclinical cardiac dysfunction in the Pakistani diabetic population.[9] In Pakistan, the prevalence of both T2DM and NAFLD is on the rise due to sedentary lifestyles and dietary habits.[10,11] Although Qureshi et al. have evaluated the association between NAFLD and LVDD among diabetic patients in the Punjab province of Pakistan, data on the prevalence of LVDD in T2DM patients with NAFLD in the Khyber Pakhtunkhwa province are lacking.[9] Considering the distinct genetic background, unique dietary, cultural, and lifestyle practices, and different body composition of patients from Khyber Pakhtunkhwa, this study seeks to assess the prevalence of LVDD among local diabetic patients with NAFLD. The study will also have implications for managing NAFLD in patients with T2DM. Pioglitazone is widely used in patients with T2DM and NAFLD due to its beneficial effects on hepatic steatosis and insulin sensitivity.[12] But its use has been associated with fluid retention, which may prove detrimental in the presence of unrecognized LVDD.[13] Therefore, identifying the burden of LVDD and its risk factors in local diabetic patients with NAFLD will help clinicians make evidence-based decisions to screen them early and use alternative agents for managing NAFLD in patients with concomitant LVDD. In addition, the study results will ensure that patients from this region of Pakistan are represented in the pooled data used for systematic reviews and meta-analyses on the subject.

METHODOLOGY

This cross-sectional study was conducted in the Department of Medicine, Hayatabad Medical Complex, Peshawar, Pakistan, from 03/01/2025 to 10/09/2025. Patients of both genders aged 25-75 years with T2DM and concomitant NAFLD were eligible for inclusion in the study. Type 2 diabetes mellitus was defined as patients already diagnosed with diabetes mellitus and having glycosylated hemoglobin (HbA1c) greater than 6.5%. Non-alcoholic fatty liver disease was defined as the presence of hepatic steatosis detected on abdominal ultrasonography in diabetic patients who had no history of alcohol consumption and no other known causes of liver disease (viral hepatitis, drug-induced liver injury) as per history and review of record. Obesity was defined as a BMI greater than 30 kg/m^2. Patients with hypertension, chronic kidney disease, and ischemic heart disease were excluded from the study. A total of 252 patients with T2DM and concomitant NAFLD were enrolled in the study through convenience sampling after informed consent, considering a prevalence of LVDD of 38.16% among T2DM with NAFLD, a 95% confidence level, and an absolute precision of 6%.[9] Demographic data, including age, gender, body mass index (BMI), and smoking history, were recorded. All study participants underwent transthoracic echocardiography to assess for LVDD under the supervision of a consultant cardiologist. LVDD was diagnosed based on the reversal of the early diastolic-to-late diastolic velocity ratio (E/A ratio). Statistical Package for the Social Sciences (SPSS) version 21 was used for analysis. Age and BMI were presented as means and standard deviations, while gender, age groups, obesity, smoking, and the presence of LVDD were presented as frequencies and percentages. Based on the presence or absence of LVDD, the two groups were compared for differences in LVDD frequency by gender, age group, obesity status, and smoking status using the chi-square test. To minimize confounding, a multivariable regression analysis including all variables was performed to identify independent risk factors for LVDD. A p-value of less than 0.05 was regarded as significant for all analyses.

RESULTS

The mean age of the study participants (n = 252) was 60.5 ± 11.5 years. Most participants were female (n = 136, 54%), under 60 years of age (n = 134, 53.2%), non-obese (n = 147, 58.3%), and non-smokers (n = 169, 67.1%). The prevalence of LVDD was 20.2% (n = 51). Table 1 summarizes the characteristics of the study participants.

Table 1: Characteristics of the Study Population (n=252)

Variables	Mean ± SD / Freq (%)
Age (Years)	60.5 ± 11.5
BMI (kg/m^2)	30.1 ± 3.9
Gender, No. (%)	
Female	136 (54%)
Male	116 (46%)
Age group, No. (%)	
Up to 60 years	134 (53.2%)
More than 60 years	118 (46.8%)
Obesity, No. (%)	
Non-obese	147 (58.3%)
Obese	105 (41.7%)
Smoking, No. (%)	
No	169 (67.1%)
Yes	83 (32.9%)
LVDD, No. (%)	
No	201 (79.8%)
Yes	51 (20.2%)

SD: Standard Deviation; LVDD: Left Ventricular Diastolic Dysfunction

In univariate analysis, LVDD was more frequent in males (22.4% vs 18.4%), although the difference was not statistically significant (p = 0.427). Similarly, LVDD was more common in patients over 60 years (24.6% vs 16.4%, p = 0.108) and in obese patients (21.9% vs 19%, p = 0.578). Compared to non-smokers, smokers had a significantly higher frequency of LVDD (30.1% vs 15.4%, p = 0.006) (Table 2). After adjusting for other risk factors in multivariable regression analysis, smokers were still more likely than non-smokers to have LVDD (AOR: 3.865; 95% CI: 1.384 – 10.793; p = 0.010) (Table 3).

Table 2: Univariate Analysis of Risk Factors for Left Ventricular Diastolic Dysfunction in Patients with Diabetes Mellitus and Non-Alcoholic Fatty Liver Disease

Variables		LVDD		P-Value
		No (n=201)	Yes (n=51)	
Gender	Female	111 (81.6%)	25 (18.4%)	0.427*
	Male	90 (77.6%)	26 (22.4%)	
Age group	Up to 60 years	112 (83.6%)	22 (16.4%)	0.108*
	More than 60 years	89 (75.4%)	29 (24.6%)	
Obesity	Non-obese	119 (81%)	28 (19%)	0.578*
	Obese	82 (78.1%)	23 (21.9%)	
Smoking	No	143 (84.6%)	26 (15.4%)	0.006*
	Yes	58 (69.9%)	25 (30.1%)	

LVDD: Left ventricular diastolic dysfunction
*Chi-square test

Table 3: Multivariable Regression Analysis of Risk Factors for Left Ventricular Diastolic Dysfunction in Patients with Diabetes Mellitus and Non-Alcoholic Fatty Liver Disease

Variables		AOR	95% CI	P-value
Gender	Female*			0.100
	Male	0.471	0.166 - 1.339	
Age Group	Up to 60 years*			0.309
	More than 60 years	1.437	0.715 - 2.888	
Obesity	Non-obese*			0.391
	Obese	1.344	0.684 - 2.640	
Smoking	No*			0.010
	Yes	3.865	1.384 - 10.793	

*Reference category
AOR: Adjusted odds ratio; CI: Confidence Interval

DISCUSSION

This study explored the burden of LVDD among patients with type 2 DM and NAFLD. This is a high-risk group, often having subclinical cardiovascular involvement. The study aimed to detect this early cardiac dysfunction in these patients. The results will ensure representation of the Pakistani population in the regional and global literature focused on subclinical cardiac dysfunction in patients with type 2 DM and concomitant NAFLD. Clinically, early identification of LVDD and its subsequent management will lead to better outcomes for these patients. This study yielded an overall prevalence of LVDD as 20.2% in T2DM with NAFLD. The LVDD prevalence observed in our study is considerably lower than that reported in most international cohorts. Bonapace et al. from Italy (64%), Lee H et al. from Korea (59.7%), and Faramarzpour et al. from Iran (81.1%) have consistently shown substantially higher diastolic dysfunction rates in T2DM patients with NAFLD.[5,6,14] Mantovani et al. reported the LVDD prevalence of 82% in T2DM patients with NAFLD.[8] A systematic review by Wang et al. analyzing 1,800 T2DM patients across 10 studies reported consistently higher LVDD rates in NAFLD patients compared to non-NAFLD controls.[15] Regionally, Qureshi et al. from Pakistan documented a prevalence of 38.16% of LVDD in T2DM with NAFLD.[9] Similarly, Sahoo et al. from India reported that 55.55% of T2DM patients with NAFLD had LVDD, and NAFLD conferred nearly a two-fold higher risk of diastolic impairment (OR 1.84).[16] Several methodological differences likely account for the lower LVDD prevalence observed in this study. A key factor is the strict exclusion criteria, which systematically removed patients with hypertension, chronic kidney disease, and ischemic heart disease-conditions well-established as independent risk factors for LVDD and frequently comorbid with T2DM and NAFLD. Many prior studies either included hypertensive patients (e.g., Bonapace et al., Mantovani et al., Sahoo et al. focused on hypertensive T2DM) or did not explicitly exclude them, potentially inflating LVDD rates due to the confounding effects of elevated blood pressure on left ventricular filling pressures and myocardial stiffness.[5,8,16] Bonapace et al.5 had a relatively smaller sample size of 50, with only 32 subjects having NAFLD, giving a relatively higher prevalence of LVDD.[5] Inclusion of patients with hypertension and renal impairment in other cohorts may have contributed to higher prevalences, as these conditions exacerbate diastolic impairment through mechanisms such as volume overload, uremic toxins, or subclinical ischemia. Additionally, in this study, the diagnostic criterion for LVDD-reversal of the E/A ratio (E/A<1) represents an earlier and more conservative definition compared to contemporary guidelines, which often incorporate multiple parameters, including E/e', left atrial volume index, and tricuspid regurgitation velocity for graded assessment of diastolic dysfunction. Studies employing tissue Doppler imaging (e.g., Bonapace et al.[5], Lee H et al.) or comprehensive echocardiographic panels (e.g., Wang et al.) detected subtler impairments

(e.g., reduced e' velocity, elevated E/e'), leading to higher detection rates.[5,14,15] Our reliance on E/A reversal alone may have underestimated preclinical or grade I diastolic dysfunction, capturing primarily more overt reversal. It was further reported that LVDD was more frequent in males as compared to females (22.4% vs 18.4%). Bouthoorn et al. have reported similar LVDD prevalence between men and women with T2DM (46% vs 47%).[17] Similarly, the results of Suresh et al.(28% vs 22%) also support our findings.[18] In this study, LVDD was reported to be more frequent in those aged 60 years or older (24.6% vs 16.4%). However, age did not independently predict LVDD, consistent with findings from Bonapace et al. (OR 0.95, p=0.58) and Sahoo et al., where age either showed no effect or was not reported as significant.[5,16] This contrasts with Lee et al., who observed a modest independent effect of age (OR 1.07 per year, p<0.001), likely due to their larger cohort (n=606), older population (mean 63 years, all ≥50), and broader age variability, which enhanced the detection of subtle cumulative age-related changes.[14] In our cohort, the narrower age range and smaller sample limited variability, reducing the ability to detect a small age effect. Furthermore, LVDD was found to be present in obese patients (21.9% vs 19%). However, obesity did not independently predict LVDD in our study, despite a higher numerical prevalence in obese participants. Using a strict obesity cutoff (BMI >30 kg/m²) may have limited the ability to detect obesity's effect on LVDD, especially if much of the risk was already explained by NAFLD. This supports our findings that NAFLD contributes to additional cardiac risk beyond obesity alone. Bonapace et al. found no significant differences in BMI or waist circumference between patients with and without NAFLD.[5] By matching the groups for obesity measures, NAFLD emerged as an independent factor associated with left ventricular diastolic dysfunction (LVDD), even after adjusting for hypertension and other cardiometabolic risk factors. Similarly, Lee H et al. reported that advanced liver fibrosis, a marker of non-alcoholic steatohepatitis, was independently linked to LVDD (OR 1.58) after accounting for insulin resistance, BMI, and other factors.[14] While BMI predicted LVDD in univariate analysis, it lost significance in multivariable models, indicating that NAFLD progression mediates much of obesity's effect on diastolic function. Sahoo et al. observed higher BMI and waist-hip ratios in NAFLD patients, with strong associations with microvascular complications and LVDD.[16] Yet, NAFLD remained independently associated with LVDD (OR 1.84), suggesting that hepatic steatosis and fibrosis contribute beyond general obesity. Collectively, these findings highlight that NAFLD often mediates the pathway from obesity to LVDD through mechanisms like insulin resistance, inflammation, and lipotoxicity. When analyses adjust for or match on obesity (Bonapace et al.) or focus on NAFLD severity (Lee H et al), the independent effect of obesity on LVDD is reduced.[5,14] However, univariate analysis did not demonstrate that any of these factors reached statistical significance. Additionally, after adjusting for all the other measured variables in multivariate analysis, age, gender, and obesity were not shown to be independent predictors of LVDD. Smoking emerged as the strongest and the only independent predictor of LVDD in this cohort (AOR 3.865) (p = 0.010), as the smokers were seen to exhibit nearly a four-fold increase in the odds of LVDD compared to the non-smokers. This observation thus suggests that smoking may act as a potent and modifiable determinant of early myocardial dysfunction in this metabolically vulnerable population. Although smoking was considered a covariate in several major studies, its impact has been inconsistent across populations. In the Korean study by Lee et al., smoking did not independently predict LVDD, which may be explained by the very low prevalence of active smokers in their older, predominantly female cohort.[14] In contrast, Bonapace et al. from Italy reported a higher prevalence of smoking among NAFLD patients; however, it did not remain significant after adjustment for metabolic risk factors.[5] Similarly, Qureshi et al. from Pakistan noted that smoking was common in their study population but did not evaluate it as an independent predictor.[9]

LIMITATIONS

The diagnosis of LVDD was based only on the reversal of the E/A ratio. Although this is commonly used in clinical practice, it is not the most sensitive marker of early LVDD. Similarly, NAFLD was diagnosed using ultrasound. Recruitment from a single center and the convenience sampling may have contributed to selection bias. Some factors, such as diabetes duration and control, NAFLD severity, medications, and level of physical activity, were not studied and may have served as confounders. The cross-sectional design cannot determine cause-and-effect relationships. Comprehensive, long-term, prospective, and multi-center studies would be needed to clarify causality and disease progression.

CONCLUSIONS

In this study, LVDD was reported in 20.2% of patients with NAFLD and T2DM. This shows that subclinical heart dysfunction is fairly common in this group, even if the prevalence was lower than what some regional and international studies have reported. Among the risk factors, smoking was significantly associated with LVDD, highlighting its role as a modifiable contributor

to LVDD. These results emphasize the importance of encouraging smoking cessation and support the need for early cardiac assessments in patients with T2DM and NAFLD in the smoking population.

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The authors accept responsibility for all aspects of the work and will ensure that any concerns regarding the accuracy or integrity of any part are properly investigated and resolved.

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