DOI:https://doi.org/10.53350/pjmhs2023177160

ORIGINAL ARTICLE

Prevalence and Risk Factors of Asymptomatic Left Ventricular Hypertrophy among Type 2 Diabetic Patients in Urban and Rural Settings

MUHAMMAD ZAHID ALI RAZA[1], AAMIR SIDDIQUE[2], SHAOIB AHMED ZIA[3], TAYYAB MOHYUDDIN[4], ARSLAN ASLAM CHAHUDHARY[5], FAIZA ALTAF[6]

[1]Assistant Professor, Department of Cardiology, Nawaz Shareef Medical College, Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan
[2,3]Assistant Professor, Department of Cardiology, Chaudhary Pervaiz Elahi Institute of Cardiology, Wazirabad, Pakistan
[4]Associate Professor, Department of Cardiology, Central Park Medical College, Lahore, Pakistan
[5]Assistant Professor, Department of Cardiology, King Edward Medical University, Lahore, Pakistan
[6]Medical Officer, Department of Medicine, Hawa Memorial Hospital, Wazirabad, Pakistan
Correspondence to: Muhammad Zahid Ali Raza, Email: Zahid_fcps@yahoo.com

ABSTRACT

Background: Left ventricular hypertrophy (LVH) is a prevalent yet often asymptomatic cardiovascular complication in patients with type 2 diabetes mellitus (T2DM), contributing significantly to increased morbidity and mortality. Early detection of asymptomatic LVH is crucial, particularly in regions with disparities in healthcare access between urban and rural populations.

Objective: This study aimed to determine the prevalence and associated risk factors of asymptomatic LVH among T2DM patients in both urban and rural settings.

Methodology: A cross-sectional study was conducted at the Department of Cardiology, Nawaz Shareef Medical College / Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan. A total of 100 patients with T2DM were enrolled, with 50 each from urban and rural populations. Clinical assessments included demographic data, anthropometric measurements, and medical history. Biochemical investigations comprised fasting blood glucose (FBG), HbA1c, lipid profile, high-sensitivity C-reactive protein (hs-CRP), and serum insulin levels. Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). Transthoracic echocardiography was performed to calculate left ventricular mass index (LVMI), with LVH defined based on standard gender-specific criteria.

Results: The prevalence of asymptomatic LVH was 60% in the urban group and 48% in the rural group (p=0.23). Urban patients had significantly higher BMI (28.6 vs. 26.9 kg/m², p=0.01), LDL (128 vs. 121 mg/dL, p=0.04), hs-CRP (5.2 vs. 4.6 mg/L, p=0.03), and LVMI (118.4 vs. 113.2 g/m², p=0.01), while HDL was significantly lower (38 vs. 41 mg/dL, p=0.02). HOMA-IR values were elevated in both groups, showing borderline significance.

Conclusion: Asymptomatic LVH is common in both urban and rural T2DM patients, with urban individuals exhibiting a more adverse cardiometabolic profile characterized by obesity, dyslipidemia, and subclinical inflammation. These findings highlight the importance of routine cardiac screening and targeted metabolic risk reduction strategies to prevent progression to overt cardiac disease.

Keywords: Diabetes, Hypertrophy, Echocardiography, Insulin, Cholesterol, Inflammation, Screening

INTRODUCTION

Left ventricular hypertrophy (LVH), characterized by an increase in left ventricular mass, is a well-established independent predictor of adverse cardiovascular outcomes, including heart failure, arrhythmias, and sudden cardiac death [1]. Among patients with type 2 diabetes mellitus (T2DM), the development of LVH is particularly concerning, as it often remains clinically silent and undetected until significant structural or functional cardiac deterioration has occurred. The coexistence of diabetes and LVH substantially increases the risk of cardiovascular morbidity and mortality, underscoring the need for early recognition and targeted preventive strategies [2].

Asymptomatic LVH in T2DM patients is frequently subclinical, emerging in the absence of overt cardiovascular symptoms [3]. It is hypothesized to arise through complex and multifactorial mechanisms, including chronic hyperglycemia, insulin resistance, low-grade systemic inflammation, and activation of the renin angiotensin aldosterone system, all of which contribute to myocardial remodeling [4]. Additionally, coexisting hypertension, obesity, dyslipidemia, and microvascular complications further accelerate myocardial structural alterations in diabetic individuals. Despite the growing burden of diabetes worldwide, particularly in low- and middle-income countries, the asymptomatic nature of LVH has led to underdiagnosis and underappreciation of its clinical significance in routine diabetic care [5].

Urban-rural disparities in healthcare access, socioeconomic status, lifestyle behaviors, and environmental exposures may influence the prevalence and risk profile of asymptomatic LVH in diabetic populations [6]. Urbanization is often associated with sedentary behavior, dietary transitions, and psychosocial stress, while rural populations may face limited access to diagnostic facilities and suboptimal disease monitoring [7]. However, comparative data evaluating the epidemiological burden and risk determinants of LVH across these geographically and demographically distinct populations remain sparse, particularly in South Asian contexts where diabetes prevalence is rising rapidly [8].

This study aims to determine the prevalence of asymptomatic LVH in type 2 diabetic patients across urban and rural settings and to identify the associated demographic, clinical, and biochemical risk factors. By stratifying risk across diverse environments, the findings may help inform region-specific screening protocols and contribute to the early cardiovascular risk stratification of diabetic individuals, ultimately improving long-term outcomes [9].

Received on 25-02-2022
Accepted on 05-06-2023

MATERIALS AND METHODS

Study Design and Setting: This cross-sectional, observational study was conducted from June 2022 to December 2022 at the Department of Cardiology, Nawaz Shareef Medical College / Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan. The study population included patients recruited from both urban hospital outpatient departments and rural community health centers affiliated with the institution, ensuring balanced geographic representation.

Sampling Method and Sample Size: A stratified purposive sampling technique was employed to enroll a total of 100 adult patients diagnosed with type 2 diabetes mellitus (T2DM), with equal representation from urban (n = 50) and rural (n = 50) settings. Stratification was based on the participants' permanent residential addresses to distinguish urban and rural groups. The sample size was calculated assuming an expected prevalence of asymptomatic left ventricular hypertrophy (LVH) of 50%, a confidence interval of 95%, and a 10% margin of error.

Eligibility Criteria: Inclusion criteria were adults aged between 35 and 70 years, with a confirmed diagnosis of T2DM based on

American Diabetes Association (ADA) guidelines, and residence in either urban or rural areas for a minimum of five years. Only asymptomatic individuals were included. Patients were excluded if they had a known history of ischemic heart disease, symptomatic heart failure, structural cardiac abnormalities, valvular heart disease, congenital heart defects, end-stage renal or liver disease, malignancy, or were pregnant. Individuals who refused to provide informed consent were also excluded.

Clinical and Demographic Evaluation: After obtaining written informed consent, each participant underwent a structured interview and clinical examination. Demographic variables such as age, gender, socioeconomic status, educational background, and area of residence were recorded. Clinical data included duration of diabetes, medication usage, presence of hypertension and dyslipidemia, smoking habits, alcohol consumption, physical activity level, and family history of cardiovascular disease.

Anthropometric and Biochemical Assessment: Anthropometric measurements were performed using standardized methods. Body mass index (BMI) was calculated from weight and height measurements. Waist circumference was measured at the midpoint between the lower rib margin and the iliac crest. Blood pressure was recorded using a calibrated mercury sphygmomanometer after the patient had rested for at least five minutes in a seated position.

Venous blood samples were collected after overnight fasting for the evaluation of fasting blood glucose (FBG), glycated hemoglobin (HbA1c), serum insulin levels, lipid profile (total cholesterol, LDL, HDL, triglycerides), serum creatinine, and high-sensitivity C-reactive protein (hs-CRP). Insulin resistance was determined using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated as:

HOMA-IR = (Fasting insulin in μU/mL × Fasting glucose in mg/dL) / 405.

Cardiac Assessment: All participants initially underwent 12-lead electrocardiography (ECG) to screen for LVH using Sokolow–Lyon and Cornell voltage criteria. Subsequently, transthoracic echocardiography (TTE) was performed by an experienced cardiologist using a standardized protocol. Left ventricular mass (LVM) was calculated and indexed to body surface area (BSA) to obtain the left ventricular mass index (LVMI). LVH was defined according to the American Society of Echocardiography guidelines: LVMI greater than 115 g/m² in men and greater than 95 g/m² in women.

Statistical Analysis: Data were analyzed using IBM SPSS version 25. Continuous variables were expressed as mean ± standard deviation (SD), and group comparisons were performed using independent sample t-tests. Categorical variables were presented as frequencies and percentages, and compared using the chi-square test. Logistic regression analysis was conducted to identify independent predictors of asymptomatic LVH. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations: The study was approved by the Institutional Review Board (IRB) of Nawaz Shareef Medical College, Gujrat, Pakistan. All participants were informed about the purpose of the study, and written informed consent was obtained prior to data collection. All procedures were carried out in accordance with the ethical standards outlined in the Declaration of Helsinki. Patient confidentiality and anonymity were strictly maintained throughout the research process.

RESULTS

A total of 100 patients with type 2 diabetes mellitus were included in the study, with equal representation from urban (n=50) and rural (n=50) settings. Table 1 outlines the demographic and clinical characteristics of both groups. The mean age of urban patients was 56.2 ± 7.5 years, compared to 54.8 ± 8.1 years in the rural cohort (p=0.34), with no statistically significant difference observed. In terms of gender distribution, males constituted 56% (n=28) of the urban population and 60% (n=30) of the rural group (p=0.68). Females represented 44% (n=22) of the urban and 40% (n=20) of the rural participants.

The mean BMI was significantly higher among urban patients (28.6 ± 3.1 kg/m²) compared to their rural counterparts (26.9 ± 2.8 kg/m²), with this difference reaching statistical significance (p=0.01). Hypertension was more prevalent in the urban group (76%) than in the rural group (68%), although this difference was not statistically significant (p=0.43). The mean duration of diabetes was 8.2 ± 3.5 years in the urban group versus 7.6 ± 4.1 years in the rural group (p=0.29) as shown in table 1.

Table 1: Demographic and Clinical Characteristics Including Gender

Parameter	Urban Group (n=50)	Rural Group (n=50)	p-value
Number of Patients	50	50	–
Age (years, mean ± SD)	56.2 ± 7.5	54.8 ± 8.1	0.34
Male Gender (%)	28 (56%)	30 (60%)	0.68
Female Gender (%)	22 (44%)	20 (40%)	0.68
BMI (kg/m², mean ± SD)	28.6 ± 3.1	26.9 ± 2.8	0.01
Hypertension (%)	38 (76%)	34 (68%)	0.43
Duration of Diabetes (Years, mean ± SD)	8.2 ± 3.5	7.6 ± 4.1	0.29
Urban Residents (%)	50 (100%)	–	–
Rural Residents (%)	–	50 (100%)	–

Biochemical markers and echocardiographic findings are presented in Table 2. Fasting blood glucose levels were slightly higher among urban participants (152 ± 25 mg/dL) compared to rural participants (145 ± 27 mg/dL), though the difference was not statistically significant (p=0.16). Similarly, HbA1c levels showed a non-significant difference between urban (7.8 ± 0.6%) and rural (7.6 ± 0.7%) groups (p=0.09). LDL cholesterol levels were significantly higher in the urban group (128 ± 22 mg/dL) than in the rural group (121 ± 20 mg/dL) with a p-value of 0.04, while HDL cholesterol was significantly lower in the urban population (38 ± 5 mg/dL) than in the rural (41 ± 6 mg/dL), showing statistical significance (p=0.02).

Triglyceride levels were slightly higher in the urban group (186 ± 34 mg/dL) compared to the rural group (178 ± 30 mg/dL), but the difference was not statistically significant (p=0.18). Inflammatory marker hs-CRP levels were significantly elevated in the urban group (5.2 ± 1.1 mg/L) compared to the rural group (4.6 ± 1.3 mg/L, p=0.03). Insulin resistance assessed by HOMA-IR was also higher in the urban population (3.9 ± 1.2) than in the rural (3.4 ± 1.0), with borderline significance (p=0.05). Echocardiographic measurement of LVMI revealed significantly higher values in the urban group (118.4 ± 14.2 g/m²) compared to the rural group (113.2 ± 13.6 g/m², p=0.01). The prevalence of asymptomatic LVH was 60% (30 patients) in urban residents versus 48% (24 patients) in rural residents, though the difference was not statistically significant (p=0.23) as shown in table 2.

Table 2: Biochemical Markers and Echocardiographic Findings

Biomarker	Urban Group (n=50)	Rural Group (n=50)	p-value
Fasting Blood Glucose (mg/dL)	152 ± 25	145 ± 27	0.16
HbA1c (%)	7.8 ± 0.6	7.6 ± 0.7	0.09
LDL Cholesterol (mg/dL)	128 ± 22	121 ± 20	0.04
HDL Cholesterol (mg/dL)	38 ± 5	41 ± 6	0.02
Triglycerides (mg/dL)	186 ± 34	178 ± 30	0.18
hs-CRP (mg/L)	5.2 ± 1.1	4.6 ± 1.3	0.03
HOMA-IR	3.9 ± 1.2	3.4 ± 1.0	0.05
LVMI (g/m²)	118.4 ± 14.2	113.2 ± 13.6	0.01
Prevalence of LVH (%)	30 (60%)	24 (48%)	0.23

The pie chart titled "Urban group & Rural group" illustrates the proportional distribution of four key biochemical markers: fasting blood glucose (FBG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and HbA1c among the combined study population. The largest segment of the chart is occupied by FBG, constituting 47% of the overall biochemical profile, indicating its dominant role in the glycemic evaluation of diabetic patients. LDL follows closely, representing 39%, which underscores the significant burden of dyslipidemia in the cohort**.** HDL accounts for 12%, reflecting the relatively lower contribution of protective lipoproteins, while HbA1c, an important marker of long-term glycemic control, comprises only **2%**, suggesting it had the smallest share in this comparative visualization. This distribution highlights that glucose and lipid abnormalities are the predominant biochemical concerns in the studied diabetic population across both urban and rural settings as shown in fig 1.

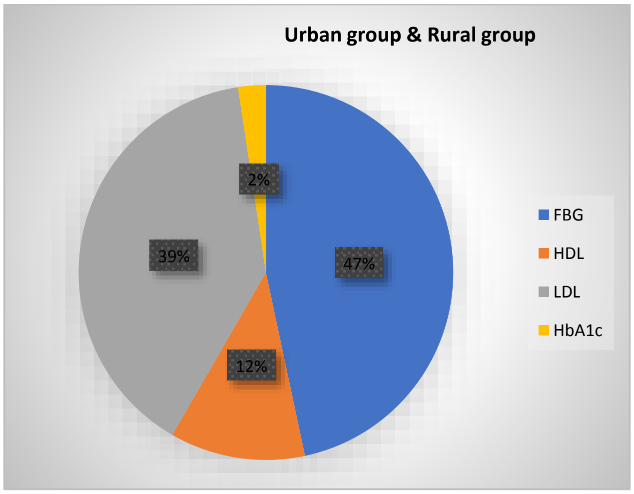

Fig-1: Distribution of Key Biochemical Parameters Among the Study Population.

The study included 100 type 2 diabetic patients, equally divided between urban and rural settings, with a focus on evaluating the prevalence and risk factors of asymptomatic left ventricular hypertrophy (LVH). The overall prevalence of asymptomatic LVH was found to be higher among urban participants (60%) compared to rural participants (48%), though this difference was not statistically significant (p=0.23). Urban patients exhibited significantly higher mean BMI (28.6 vs. 26.9 kg/m², p=0.01), LDL cholesterol (128 vs. 121 mg/dL, p=0.04), hs-CRP (5.2 vs. 4.6 mg/L, p=0.03), and left ventricular mass index (LVMI) (118.4 vs. 113.2 g/m², p=0.01), while HDL levels were significantly lower (38 vs. 41 mg/dL, p=0.02), indicating a more adverse cardiometabolic profile. Although fasting blood glucose and HbA1c levels did not show significant differences, insulin resistance measured by HOMA-IR was borderline higher in urban patients (3.9 vs. 3.4, p=0.05). These findings suggest that urban diabetic patients are more likely to exhibit subclinical cardiac remodeling driven by obesity, dyslipidemia, low-grade inflammation, and insulin resistance. The results underscore the importance of routine echocardiographic screening and aggressive risk factor management in diabetic populations, particularly in urban settings where lifestyle-related metabolic disturbances appear to be more pronounced.

DISCUSSION

This study investigated the prevalence and associated risk factors of asymptomatic left ventricular hypertrophy (LVH) among patients with type 2 diabetes mellitus (T2DM) in urban and rural settings. The findings reveal a high prevalence of asymptomatic LVH in both populations, with a slightly higher, though not statistically significant, rate in the urban group (60%) compared to the rural group (48%) [10]. This aligns with previous evidence that diabetic cardiomyopathy, particularly in its early and asymptomatic stages, is a frequent yet underrecognized complication of T2DM. The increased LVH burden in the urban population could reflect greater exposure to modifiable cardiovascular risk factors such as obesity, sedentary lifestyle, and subclinical inflammation [11].

A key observation in our study was the significantly higher body mass index (BMI) among urban participants. Obesity is a well-established risk factor for LVH due to increased cardiac workload and activation of neurohormonal pathways including the renin–angiotensin–aldosterone system [12]. Furthermore, LDL cholesterol levels were also significantly elevated in urban subjects, accompanied by lower HDL cholesterol levels, which suggests a more atherogenic lipid profile. Dyslipidemia, particularly elevated LDL and low HDL levels, has been implicated in promoting myocardial remodeling and contributing to structural changes in the left ventricle [13]. These lipid abnormalities may exacerbate endothelial dysfunction and accelerate cardiac fibrosis, promoting the development of LVH [14].

Another important finding was the significantly elevated levels of high-sensitivity C-reactive protein (hs-CRP) among urban diabetics [15]. Chronic low-grade inflammation, as reflected by elevated hs-CRP, is increasingly recognized as a contributor to myocardial hypertrophy and diastolic dysfunction in diabetic patients. The significant difference in HOMA-IR values between the groups also highlights the role of insulin resistance in the pathogenesis of LVH. Insulin resistance not only affects glucose metabolism but also promotes sodium retention, sympathetic activation, and smooth muscle proliferation, all of which are linked to cardiac hypertrophy [16].

Although fasting blood glucose and HbA1c levels were not significantly different between the two groups, FBG constituted the largest proportion (47%) in the comparative pie chart of biochemical parameters, emphasizing the clinical prominence of short-term glycemic burden [17]. Interestingly, HbA1c, despite being a critical marker of long-term glycemic control, accounted for only 2% in the pie chart, suggesting that clinicians may still rely more heavily on immediate glucose indices in routine practice. However, HbA1c remains vital for identifying patients at risk of chronic complications like LVH [18].

Echocardiographic findings showed a significantly higher left ventricular mass index (LVMI) in the urban group, which corroborates the biochemical and anthropometric data indicating a greater cardiovascular risk profile in urban diabetics [19]. The lack of statistical significance in LVH prevalence between the groups may reflect the sample size limitation; nevertheless, the trends observed warrant attention and further investigation.

Our results are consistent with prior studies suggesting urbanization contributes to a clustering of cardiovascular risk factors among diabetic patients. However, rural populations are not exempt and may be equally vulnerable due to increasing adoption of urban lifestyles, poor access to healthcare, and delayed diagnosis. The presence of LVH in nearly half of the rural diabetics highlights the need for enhanced screening and risk stratification strategies in both settings. One limitation of this study is its cross-sectional design, which precludes causal inference. The relatively small sample size may have also limited the power to detect significant differences in some variables. Nevertheless, the inclusion of both urban and rural populations enhances the generalizability of findings across diverse geographic and socioeconomic backgrounds [20].

CONCLUSION

This study highlights the high prevalence of asymptomatic left ventricular hypertrophy (LVH) among patients with type 2 diabetes mellitus (T2DM), particularly in urban populations exhibiting a more adverse cardiometabolic profile. The significant associations of LVH with elevated body mass index, dyslipidemia, systemic inflammation, and insulin resistance underscore the importance of early detection through routine cardiac evaluation, including echocardiography, even in the absence of clinical symptoms. These findings call for the implementation of comprehensive, multifactorial intervention strategies aimed at mitigating metabolic and cardiovascular risk factors. Moreover, the results emphasize the necessity for region-specific public health policies that address the rising burden of diabetes-related cardiac complications in both urban and rural settings. Early identification and targeted management may play a pivotal role in preventing progression to symptomatic heart failure and reducing long-term cardiovascular morbidity and mortality in diabetic populations.

Funding: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflicts of Interest: The authors declare no conflicts of interest related to this study.

Authors' Contributions: M.Z.A.R. conceptualized the study and supervised clinical data collection. A.S. and S.A.Z. contributed to echocardiographic evaluations and cardiac data analysis. T.M. and A.A.C. were responsible for biochemical testing and laboratory coordination. F.A. managed data entry and statistical analysis. All authors contributed to manuscript drafting and approved the final version.

Data Availability: The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Acknowledgment: The authors sincerely thank all the patients who participated in this study for their cooperation, time, and trust.

REFERENCES

1. Dunlay SM, Roger VL, Redfield MM. Epidemiology of heart failure with preserved ejection fraction. Nat Rev Cardiol. 2017;14(10):591–602.
2. van Heerebeek L, Paulus WJ. Understanding heart failure with preserved ejection fraction: where are we today? Neth Heart J. 2016;24(4):227–36.
3. Westermann D, Lindner D, Kasner M, et al. Cardiac inflammation contributes to changes in the extracellular matrix in patients with heart failure and normal ejection fraction. Circ Heart Fail. 2011;4(1):44–52.
4. Paulus WJ, Tschöpe C. A novel paradigm for heart failure with preserved ejection fraction. J Am Coll Cardiol. 2013;62(4):263–71.
5. Wong CY, O'Moore-Sullivan T, Leano R, Byrne N, Beller E, Marwick TH. Alterations of left ventricular myocardial characteristics associated with obesity. Circulation. 2004;110(19):3081–7.
6. López-Sánchez M, Muñoz-Esquerre M, Huertas D, et al. High prevalence of left ventricle diastolic dysfunction in severe COPD associated with a low exercise capacity: a cross-sectional study. PLoS One. 2013;8(6):e68034.
7. Deswal A. Diastolic dysfunction and diastolic heart failure: mechanisms and epidemiology. Curr Cardiol Rep. 2005;7(3):178–83.
8. Upadhya B, Pisani B, Kitzman DW. Evolution of a geriatric syndrome: pathophysiology and treatment of heart failure with preserved ejection fraction. J Am Geriatr Soc. 2017;65(11):2431–40.
9. Meschiari CA, Ero OK, Pan H, Finkel T, Lindsey ML. The impact of aging on cardiac extracellular matrix. GeroScience. 2017;39(1):7–18.
10. Gevaert AB, Shakeri H, Leloup AJ, et al. Endothelial senescence contributes to heart failure with preserved ejection fraction in an aging mouse model. Circ Heart Fail. 2017;10(6).
11. van den Berg MP, Mulder BA, Klaassen SHC, et al. Heart failure with preserved ejection fraction, atrial fibrillation, and the role of senile amyloidosis. Eur Heart J. 2019;40(16):1287–93.
12. Wu CK, Yang CY, Lin JW, et al. The relationship among central obesity, systemic inflammation, and left ventricular diastolic dysfunction as determined by structural equation modeling. Obesity (Silver Spring). 2012;20(4):730–7.
13. Ayalon N, Gopal DM, Mooney DM, et al. Preclinical left ventricular diastolic dysfunction in metabolic syndrome. Am J Cardiol. 2014;114(6):838–42.
14. Mocan M, Anton F, Suciu S, Rahaian R, Blaga SN, Farcas AD. Multimarker assessment of diastolic dysfunction in metabolic syndrome patients. Metab Syndr Relat Disord. 2017;15(10):507–14.
15. Mocan M, Popa T, Blaga SN. The influence of metabolic syndrome components on N-terminal pro B-type natriuretic peptide concentrations: the role of left ventricular diastolic dysfunction. Acta Endocrinol (Buchar). 2013;9(2):229–40.
16. Kuwahara F, Kai H, Tokuda K, et al. Hypertensive myocardial fibrosis and diastolic dysfunction: another model of inflammation? Hypertension. 2004;43(4):739–45.
17. Gu J, Zhao F, Wang Y, et al. The molecular mechanism of diastolic heart failure. Integr Med Int. 2015;2(3-4):143–8.
18. ter Maaten JM, Damman K, Verhaar MC, et al. Connecting heart failure with preserved ejection fraction and renal dysfunction: the role of endothelial dysfunction and inflammation. Eur J Heart Fail. 2016;18(6):588–98.
19. Hu YF, Chen YJ, Lin YJ, Chen SA. Inflammation and the pathogenesis of atrial fibrillation. Nat Rev Cardiol. 2015;12(4):230–43.
20. Drechsler C, Delgado G, Wanner C, et al. Galectin-3, renal function, and clinical outcomes: results from the LURIC and 4D studies. J Am Soc Nephrol. 2015;26(9):2213–21.

This article may be cited as: Raza MZA, Siddique A, Zia SA, Mohyuddin T, Chahudhary AA, Altaf F: Prevalence and Risk Factors of Asymptomatic Left Ventricular Hypertrophy among Type 2 Diabetic Patients in Urban and Rural Settings. Pak J Med Health Sci, 2023; 17(7): 160-163.