



## Original Research Article

# EFFECT OF SGLT-2 INHIBITORS ON ECHOCARDIOGRAPHIC PARAMETERS OF DIASTOLIC DYSFUNCTION IN TYPE 2 DIABETES MELLITUS PATIENTS

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### ABSTRACT

**Background:** Type 2 Diabetes Mellitus (T2DM) is associated with a high risk of cardiovascular complications, including diastolic dysfunction, which often precedes overt heart failure. Early identification and intervention are essential to prevent progression. Sodium-Glucose Cotransporter-2 (SGLT-2) inhibitors have shown promising cardioprotective effects beyond glycemic control. **Aim:** To evaluate the effect of SGLT-2 inhibitors on echocardiographic parameters of diastolic dysfunction in patients with Type 2 Diabetes Mellitus.

**Materials and Methods:** A prospective interventional study was conducted on 90 patients with T2DM. Baseline clinical and echocardiographic parameters including E/A ratio, E/e' ratio, left atrial volume index (LAVI), and left atrial mass (LAM) were recorded. Patients were treated with SGLT-2 inhibitors and followed up at 3 and 6 months. Statistical analysis was performed using paired t-test and ANOVA, with p-value <0.05 considered significant.

**Results:** Significant improvement was observed in E/e' ratio (p=0.005), LAVI (p<0.001), and LAM (p<0.001) after 6 months of therapy. However, E/A ratio showed no significant change (p=0.580).

**Conclusion:** SGLT-2 inhibitors significantly improve echocardiographic parameters of diastolic dysfunction in patients with T2DM, suggesting their role in preventing progression to heart failure.

**Keywords:** Type 2 Diabetes Mellitus; Diastolic Dysfunction; SGLT-2 Inhibitors.

## INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a rapidly growing global health concern characterized by chronic hyperglycemia resulting from insulin resistance and impaired insulin secretion. According to the International Diabetes Federation, the global burden of diabetes continues to rise, with a substantial proportion contributed by developing countries such as India. T2DM is associated with multiple microvascular and macrovascular complications, among which cardiovascular disease remains the leading cause of morbidity and

mortality. Among these, heart failure is increasingly recognized as a major complication, often presenting even in the absence of overt coronary artery disease or hypertension.<sup>[1]</sup>

Diastolic dysfunction is an early manifestation of diabetic cardiomyopathy and frequently precedes systolic dysfunction. Its early identification is crucial as timely intervention can prevent progression to heart failure with preserved ejection fraction (HFpEF). The pathophysiology involves multiple mechanisms including myocardial fibrosis, oxidative stress, endothelial dysfunction, lipotoxicity, and activation of the renin-

angiotensin-aldosterone system. These changes ultimately lead to increased myocardial stiffness and impaired ventricular compliance.<sup>[2]</sup>

Early detection of diastolic dysfunction is crucial, as timely intervention can prevent progression to heart failure with preserved ejection fraction (HFpEF), which constitutes a major proportion of heart failure cases in diabetic individuals. Echocardiography remains the gold standard for evaluating diastolic function, with parameters such as E/A ratio, E/e' ratio, left atrial volume index (LAVI), and left ventricular mass providing valuable insights into cardiac function.<sup>[3]</sup>

In recent years, Sodium-Glucose Cotransporter-2 (SGLT-2) inhibitors have emerged as a breakthrough in the management of T2DM, not only for glycemic control but also for their cardiovascular benefits. Drugs such as dapagliflozin, empagliflozin, and canagliflozin act by inhibiting glucose reabsorption in the proximal renal tubules, thereby promoting glycosuria. Beyond their metabolic effects, SGLT-2 inhibitors have demonstrated significant reductions in heart failure hospitalizations and cardiovascular mortality in multiple large clinical trials.<sup>[4]</sup>

The beneficial effects of SGLT-2 inhibitors on cardiac function are thought to be mediated through various mechanisms including reduction in preload and afterload due to osmotic diuresis, improvement in myocardial energetics, reduction in inflammation and oxidative stress, and favorable effects on cardiac remodeling. Emerging evidence suggests that these agents may improve echocardiographic parameters of diastolic dysfunction, thereby playing a key role in preventing progression of diabetic cardiomyopathy.<sup>[5]</sup>

#### **Aim**

To evaluate the effect of SGLT-2 inhibitors on echocardiographic parameters of diastolic dysfunction in patients with Type 2 Diabetes Mellitus.

#### **Objectives**

1. To assess changes in echocardiographic parameters (E/A ratio, E/e', LAVI, LV mass) following SGLT-2 inhibitor therapy.
2. To evaluate the association between duration of diabetes and severity of diastolic dysfunction.

## **MATERIALS AND METHODS**

#### **Source of Data**

The data were collected from patients diagnosed with Type 2 Diabetes Mellitus attending the Outpatient Department (OPD) and Diabetes Clinic of the Department of General Medicine at a tertiary care teaching hospital. Eligible patients were recruited consecutively after obtaining informed written consent.

#### **Study Design**

This was a prospective interventional study in which all enrolled participants were evaluated at baseline and followed up after initiation of SGLT-2 inhibitor therapy.

#### **Study Location**

The study was conducted at the Department of General Medicine, tertiary care teaching hospital (Diabetes Specialty Clinic).

#### **Study Duration**

The study was conducted over a period of 18 months.

#### **Sample Size**

A total of 90 patients with Type 2 Diabetes Mellitus were included in the study.

#### **Inclusion Criteria**

1. Patients aged 30-70 years diagnosed with Type 2 Diabetes Mellitus.
2. Patients with HbA1c  $\geq 6.5\%$  on standard anti-diabetic therapy.
3. Patients willing to receive SGLT-2 inhibitors.
4. Patients who provided informed consent.

#### **Exclusion Criteria**

1. Type 1 Diabetes Mellitus or other specific types of diabetes.
2. Patients already on SGLT-2 inhibitors or GLP-1 agonists.
3. Severe renal impairment (eGFR  $< 30$  ml/min/1.73 m<sup>2</sup>).
4. Known coronary artery disease, valvular heart disease, or heart failure (NYHA II-IV).
5. Pregnant or lactating women.
6. Patients unwilling for follow-up.

#### **Procedure and Methodology**

All participants underwent detailed clinical evaluation including history, physical examination, and laboratory investigations such as fasting blood glucose, HbA1c, lipid profile, and renal function tests. Baseline echocardiography was performed to assess diastolic function parameters including E/A ratio, E/e' ratio, left atrial volume index (LAVI), and left ventricular mass.

Patients were initiated on SGLT-2 inhibitor therapy (dapagliflozin 10 mg once daily) in addition to their existing anti-diabetic regimen. Follow-up echocardiographic assessments were performed at 3 and 6 months to evaluate changes in diastolic function.

#### **Sample Processing**

Blood samples were collected under aseptic precautions and analyzed using standard laboratory techniques. Echocardiographic evaluation was performed using a standardized protocol by an experienced cardiologist to ensure consistency and reliability.

#### **Statistical Methods**

Data were entered in Microsoft Excel and analyzed using statistical software (SPSS).

- Quantitative variables were expressed as mean  $\pm$  standard deviation.

- Qualitative variables were expressed as frequency and percentage.
- Paired t-test was used to compare pre- and post-treatment parameters.
- Chi-square test was used for categorical variables.

- A p-value <0.05 was considered statistically significant.

#### Data Collection

Data were collected using a pre-structured proforma including demographic details, clinical history, laboratory findings, and echocardiographic parameters. Follow-up data were recorded during scheduled visits at 3 and 6 months.

## RESULTS

**Table 1: Overall effect of SGLT-2 inhibitors on echocardiographic parameters at 6 months (N=90)**

Parameter	Baseline Mean±SD	6 months Mean±SD	Mean change	95% CI of change	Test value	p-value
E/e' ratio	8.9±2.3	7.9±1.8	↓1.0	0.40-1.60	F=5.28	0.005*
E/A ratio	0.69±0.2	0.65±0.3	↓0.04	-0.03-0.11	F=0.53	0.580
LAVI (ml/m <sup>2</sup> )	30.4±9.4	25.3±9.8	↓5.1	2.29-7.91	F=9.354	<0.001*
LAM (g)	36.0±10.3	30.0±9.6	↓6.0	3.09-8.91	F=8.556	<0.001*

The present study demonstrated a significant improvement in key echocardiographic parameters following 6 months of SGLT-2 inhibitor therapy. The mean E/e' ratio decreased significantly, indicating improved left ventricular filling pressures. Similarly, LAVI and LAM showed statistically significant reductions, suggesting

favorable reverse atrial remodeling. However, the E/A ratio showed no statistically significant change. Serial echocardiographic assessment further revealed progressive improvement from baseline to 3 and 6 months, indicating a time-dependent therapeutic benefit of SGLT-2 inhibitors.

**Table 3: Changes in echocardiographic parameters after SGLT-2 inhibitor therapy (N=90)**

Parameter	Baseline Mean±SD	3 months Mean±SD	6 months Mean±SD	95% CI at 6 months	Test value	p-value
E/e' ratio	8.9±2.3	8.5±2.1	7.9±1.8	7.53-8.27	F=5.28	0.005*
E/A ratio	0.69±0.2	0.68±0.3	0.65±0.3	0.59-0.71	F=0.53	0.580
LAVI (ml/m <sup>2</sup> )	30.4±9.4	28.8±10.8	25.3±9.8	23.28-27.32	F=9.354	<0.001*
LAM (g)	36.0±10.3	34.0±9.8	30.0±9.6	28.02-31.98	F=8.556	<0.001*

Serial echocardiographic assessment revealed a progressive improvement in diastolic function parameters over time. The E/e' ratio decreased significantly from 8.9±2.3 at baseline to 8.5±2.1 at 3 months and further to 7.9±1.8 at 6 months (95% CI: 7.53-8.27; p=0.005), indicating improved left ventricular filling pressures. Similarly, LAVI showed a consistent decline from 30.4±9.4 ml/m<sup>2</sup> to 28.8±10.8 ml/m<sup>2</sup> at 3 months and 25.3±9.8

ml/m<sup>2</sup> at 6 months (95% CI: 23.28-27.32; p<0.001). Left atrial mass also reduced significantly over time from 36.0±10.3 g to 34.0±9.8 g at 3 months and 30.0±9.6 g at 6 months (95% CI: 28.02-31.98; p<0.001). In contrast, the E/A ratio showed only a marginal and statistically non-significant decrease across follow-up periods (p=0.580).

**Table 4: Association between duration of diabetes and diastolic dysfunction severity/status (N=90)**

Group	Duration of diabetes Mean±SD	95% CI	Test value	p-value
Diabetes patients with DD (n=67)	9.51±4.5 years	8.43-10.59		
Diabetes patients without DD (n=23)	5.63±4.3 years	3.87-7.39		
<b>Mean difference</b>	<b>3.88 years</b>	<b>1.82-5.94</b>	<b>t=3.60</b>	<b>0.0005*</b>

\*Statistically significant.

A significant association was observed between the duration of diabetes and the presence of diastolic dysfunction. Patients with diastolic dysfunction had a longer duration of diabetes (9.51±4.5 years) compared to those without diastolic dysfunction (5.63±4.3 years). The mean difference of 3.88 years was statistically significant (95% CI: 1.82-5.94; t=3.60, p=0.0005).

## DISCUSSION

In the present study, SGLT-2 inhibitor therapy showed significant improvement in

echocardiographic parameters of diastolic dysfunction over 6 months. The E/e' ratio significantly decreased, suggesting improvement in left ventricular filling pressure. Similarly, LAVI and LAM demonstrated significant reductions, indicating reverse cardiac remodeling. However, the E/A ratio did not show significant change, likely due to its load-dependent nature. These findings are comparable with Savcilioglu et al. (2024),<sup>[1]</sup> who reported improvement in diastolic parameters following SGLT-2 therapy. Song et al. (2023)<sup>[2]</sup> and Fu et al. (2023),<sup>[3]</sup> also demonstrated favorable cardiac remodeling effects. El-Saied et al. (2024),<sup>[4]</sup> reported improvement in left atrial

function, supporting the findings of this study. Serial improvements observed at 3 and 6 months suggest that SGLT-2 inhibitors exert gradual cardioprotective effects through mechanisms such as reduction in preload, improved myocardial energetics, and anti-inflammatory actions. These findings align with Biter et al. (2024),<sup>[11]</sup> and Duman et al. (2025).<sup>[10]</sup>

Additionally, the study demonstrated that longer duration of diabetes was significantly associated with diastolic dysfunction, indicating chronic hyperglycemia as a key contributor to myocardial remodeling. Similar findings were reported by Dzhun et al. (2023),<sup>[8]</sup> and Novo et al. (2023).<sup>[5]</sup>

Serial assessment in the present study showed progressive improvement in E/e', LAVI, and LAM from baseline to 3 months and further at 6 months, suggesting that the cardiac benefits of SGLT-2 inhibitors may be gradual and related to improvement in hemodynamic load, myocardial energetics, inflammation, and cardiac remodeling. The non-significant change in E/A ratio may be due to its load-dependent nature and limited sensitivity in detecting early therapeutic response. These findings are consistent with Biter et al. (2024),<sup>[11]</sup> who reported improvement in myocardial strain and biomarkers after 6 months of SGLT-2 inhibitor therapy. Duman et al. (2025),<sup>[10]</sup> also demonstrated significant improvement in left atrial function with SGLT-2 inhibitors, further supporting the role of these drugs in cardiac remodeling. Additionally, Berezin et al. (2022),<sup>[7]</sup> highlighted that SGLT-2 inhibitors exert cardioprotective effects through modulation of neurohormonal pathways and biomarkers such as apelin.

The present study also found that patients with diastolic dysfunction had a significantly longer duration of diabetes compared with those without diastolic dysfunction (9.51±4.5 years vs. 5.63±4.3 years; p=0.0005). This suggests that prolonged exposure to hyperglycemia contributes to progressive myocardial fibrosis, endothelial dysfunction, and impaired ventricular relaxation. Similar observations were reported by Dzhun et al. (2023),<sup>[8]</sup> who found that chronic glycemic variability is strongly associated with diastolic dysfunction. Furthermore, Novo et al. (2023)<sup>[5]</sup> and Karaduman et al. (2025),<sup>[9]</sup> reported that longer duration of diabetes and associated metabolic disturbances significantly increase the risk of cardiac dysfunction.

## CONCLUSION

The present study concludes that diastolic dysfunction is highly prevalent among patients with Type 2 Diabetes Mellitus, with nearly three-fourths of the study population demonstrating some degree of impaired diastolic function. The most common form observed was Grade I diastolic

dysfunction, indicating early subclinical myocardial involvement. The study also establishes a significant association between longer duration of diabetes and the presence of diastolic dysfunction, highlighting the progressive nature of diabetic cardiomyopathy.

SGLT-2 inhibitor therapy demonstrated a significant beneficial effect on echocardiographic parameters of diastolic dysfunction. There was a statistically significant reduction in E/e' ratio, left atrial volume index (LAVI), and left atrial mass (LAM), indicating improvement in left ventricular filling pressures and reversal of structural remodeling. These findings suggest that SGLT-2 inhibitors not only provide glycemic control but also exert cardioprotective effects by improving myocardial function and reducing cardiac workload. However, parameters such as E/A ratio did not show significant change, possibly due to its limited sensitivity in early therapeutic assessment.

Overall, the study supports the role of SGLT-2 inhibitors as an effective therapeutic option in improving diastolic function and preventing progression to overt heart failure in patients with Type 2 Diabetes Mellitus. Early screening using echocardiography and timely initiation of SGLT-2 inhibitors can significantly improve cardiovascular outcomes in this high-risk population.

### Limitations of the study

1. The sample size was relatively small (N=90), limiting generalizability.
2. The study was conducted at a single tertiary care center, which may introduce selection bias.
3. Short duration of follow-up (6 months) may not reflect long-term effects.
4. Lack of a control group not receiving SGLT-2 inhibitors.
5. Echocardiographic assessment is operator-dependent and subject to variability.
6. Other confounding factors such as hypertension, obesity, and dyslipidemia were not fully controlled.
7. Only one SGLT-2 inhibitor (dapagliflozin) was evaluated.
8. Advanced imaging modalities like strain imaging or cardiac MRI were not used.
9. Medication adherence and lifestyle factors were not strictly monitored.
10. Biochemical markers such as BNP or inflammatory markers were not assessed.

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