Original Article

Impact of Sodium–Glucose Cotransporter-2 Inhibitors on Patient Outcomes and Left Atrial Mechanics in Atrial Fibrillation Patients: Insights from Speckle Tracking Echocardiography

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Abstract

Background and Aim: Evidence on the direct impact of sodium–glucose cotransporter-2 (SGLT2) inhibitors on outcomes and complications in atrial fibrillation (AF) patients, independent of diabetes status, is still limited. Therefore, this study sought to evaluate the effects of SGLT2 inhibitors in patients with AF, particularly regarding left atrial (LA) function. **Materials and Methods:** This prospective, multicenter observational study included 200 patients with nonvalvular persistent AF. The study population was split into two groups, with Group 1 consisting of 100 patients treated with SGLT2 inhibitors as part of their treatment regimen, whereas Group 2 comprised 100 patients who were not treated with these drugs. All participants underwent a comprehensive assessment, including detailed medical history, thorough general and local physical examination, laboratory investigations, electrocardiography, and echocardiography. **Results:** The present study demonstrated a marked enhancement of all parameters of LA function, including LA dimensions, volumes, emptying fraction, and strain, in the group treated with SGLT2 inhibitors. These positive effects appear to be consistent regardless of gender, comorbid conditions, or various clinical variables. Moreover, factors such as heart rate, blood pressure, hemoglobin, and platelet counts showed no significant impact on outcomes, supporting the idea that SGLT2 inhibitors directly contribute to enhanced cardiac function in AF patients. **Conclusion:** SGLT2 inhibitors demonstrate efficacy in improving LA function among patients with AF, emphasizing their role as a potential treatment option for AF patients with impaired LA performance.

Keywords: Atrial fibrillation, left atrial mechanics, sodium–glucose cotransporter-2 inhibitor, speckle tracking echocardiography

Introduction

The left atrial (LA), which contributes up to one-third of total cardiac output, plays a crucial role in left ventricular (LV) filling by acting as a reservoir during systole, a conduit during diastole, and a pump that improves LV performance. LA remodeling – both structural and functional – develops in response to conditions including tachycardia, volume and pressure overload, diastolic dysfunction, myocardial ischemia, and valvular disease.[1]

As the most frequently encountered cardiac arrhythmia, atrial fibrillation (AF) is a key driver of mortality worldwide. AF has a significant health and socioeconomic burden because of the higher rates of stroke and heart failure (HF).[2]

Sodium–glucose co-transporter 2 inhibitors (SGLT2I), a recently introduced group of oral antidiabetic agents, have been shown in several large placebo-controlled randomized trials and meta-analyses to significantly reduce cardiovascular events, especially HF outcomes and all-cause mortality.[3] The proposed mechanisms likely stem from the direct cardiac effects of SGLT2 inhibitors – particularly their impact on diuresis and myocardial energy metabolism. Furthermore, HF and AF are tightly associated conditions that share major risk factors such as hypertension, diabetes, and obesity.[4] Improvements in HbA1c levels, blood pressure, and body

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weight have been noted with the use of SGLT2 inhibitors. These beneficial systemic effects may play an important role in the improvement of AF outcomes.[5]

There is a lack of robust data addressing the direct influence of SGLT2 inhibitors on AF-related outcomes and complications regardless of diabetic status. Thus, the present study seeks to examine the clinical outcomes with SGLT2 inhibitor therapy in AF patients and to evaluate its effects on LA function using speckle-tracking echocardiography.

Materials and Methods

Study design and population

The study was designed as a multicenter, prospective observational investigation and was carried out at the Cardiology Department of Benha University and 15 May Hospitals during the period from January 2024 to March 2025. It included patients with nonvalvular persistent AF. Persistent AF was defined based on the latest ESC guidelines at 2024 as AF episodes that do not terminate spontaneously. Several interventional trials have adopted a 7-day cutoff to define persistent AF.[6] Exclusion criteria were patients with chronic rheumatic heart diseases, acute rheumatic fever or prosthetic heart valves, patients who are known to be allergic to SGLT2I, hematologic disorders, malignancy, advanced renal dysfunction (estimated glomerular filtration rate < 30 mL/min/1.73 m^2), hepatic disease, active infection or chronic inflammatory conditions, and autoimmune diseases, history of ketoacidosis, hypotensive patients, pregnant woman, women planning to be pregnant, lactating women. Patients were allocated into two groups; Group 1 comprised AF patients using SGLT2 inhibitors added to their treatment. Group 2 included AF patients who did not use SGLT2 inhibitors. All patients underwent assessment of baseline demographics and a comprehensive clinical examination, biochemical analysis, echocardiography, and electrocardiography (electrocardiographic characteristics of AF include: irregularly irregular R–R intervals, absence of clear *P* waves, and disorganized atrial activity).

Ethical approval and consent to participate

This research was approved by the university ethical committee with approval number (MS 36-11-2024). All participants had written informed consent.

Echocardiographic measurements

Echocardiographic evaluation was done by EPIQ equipment (Philips, Erlangen, Germany). All echocardiographic measurements were performed in the supine and left lateral position. Echocardiographic examination was done at the beginning of the study and after 6 months. Three electrocardiogram (ECG)-gated cardiac cycles were recorded at end-expiration, with imaging settings manually optimized for quality. All echocardiographic studies were conducted following American Society of Echocardiography guidelines.[7]

Conventional echocardiography

Global LV systolic function was assessed using the biplane Simpson's method. EF (%) was determined using the standard formula: EF (%) = ([end-diastolic volume (EDV)–end-systolic volume (ESV)]/[EDV]) ×100.[7] LA size was assessed at end-ventricular systole – its point of maximum expansion – by measuring the anteroposterior diameter in the long-axis view and the longitudinal and transverse diameters in the four-chamber view. In AF patients, LA volumes included the minimal volume (V_{min}), obtained just after mitral valve closure in end-diastole, and the maximal volume (V_{max}), measured just prior to mitral valve opening in end-systole. LA reservoir volume (Vmax− Vmin); this volume is used to calculate LA emptying fraction (LAEF) from the following equation: 100× (LAV_{max}–LAV_{min})/LAV max.[8]

Speckle-tracking echocardiography

LA reservoir strain (LASr) was measured using two-dimensional speckle tracking echocardiography. We obtained grey-scale images for the apical 4-chamber and 2-chamber views with a frame rate of 50–80 Hz. We manually traced the LA endocardial border at end-systole, after which the software automatically tracked myocardial motion throughout the cardiac cycle using R-to-R ECG gating. The region of interest was adjusted to match the thinnest atrial wall for optimal tracking. The LA was then segmented into six regions – basal, mid, and apical segments – in both the apical 4-chamber and 2-chamber views. During the reservoir phase, the left atrium expands as it fills with blood, producing a positive strain that peaks in systole at the end of atrial filling and just before the mitral valve opens. This peak value represents the LASr which was assessed at the end of the reservoir phase. Average values of 3 consecutive ECG-gated cardiac cycles were calculated. An LASr value of ≤23% was considered impaired, based on previously published lower reference limits for LASr[9] [Figure 1].

Follow-up

All patients were monitored over a period of 6 months. The primary endpoints are ischemic stroke/transient ischemic attack (TIA), LA function, HF, acute coronary syndrome (ACS), and all-cause mortality.

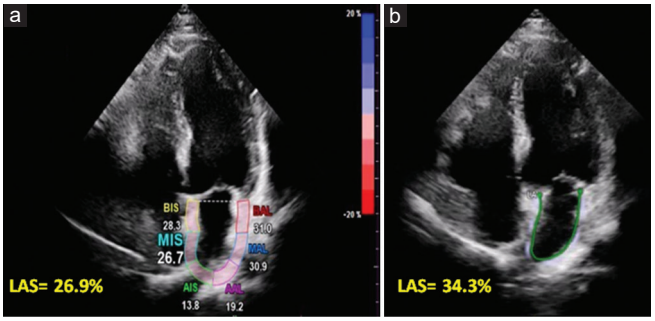

Figure 1: (a) Baseline left atrial strain (LAS) of a patient before sodium–glucose cotransporter 2 (SGLT-2) inhibitor treatment (LAS = 26.9%), (b) Left atrial strain of the same patient after SGLT-2 inhibitor treatment (LAS = 34.3%). LAS: left atrial strain

Statistical analysis

We used SPSS software (version 27, SPSS Inc., Armonk, NY, USA) for statistical analysis. Data normality was assessed using both the Shapiro–Wilk test and the Kolmogorov–Smirnov test. Continuous variables that have a normal distribution were expressed as mean ± standard deviation. The Independent Samples *t*-test was used for comparison between the two groups. Nonnormally distributed continuous variables were presented as median and interquartile range. The Mann–Whitney *U*-test was applied for group comparisons. Categorical variables were expressed as frequencies and percentages. Comparison was done using either the Chi-square test or Fisher's exact test when appropriate. Binary logistic regression analysis was carried out to identify predictors of dichotomous outcomes. $P < 0.05$ was considered statistically significant.

Results

We evaluated 200 nonvalvular persistent AF patients (100 patients in each group). Baseline characteristics are presented in Table 1. No statistically significant differences were observed between both groups regarding age, gender, hypertension, diabetes mellitus, smoking, dyslipidemia, heart rate (HR), systolic and diastolic blood pressure, laboratory, and baseline echocardiographic parameters.

After 6 months, significant changes in echocardiographic parameters were observed. LV EDV and LV ESV were significantly lower in Group 1. In addition, the LV ejection fraction significantly improved in Group 1. LA diameters and volumes significantly decreased with a significant improvement of LAEF and LA strain in Group 1 [Table 2].

Table 1: Demographic and clinical data of the studied groups

Clinical characteristics	Group I (*n*=100)	Group II (*n*=100)	*P*
Age (years)	56±13.6	55.7±13.7	0.881
Male gender	66 (66)	55 (55)	0.112
Smoking	41 (41)	33 (33)	0.241
Hypertension	48 (48)	49 (49)	0.887
Diabetes mellitus	47 (47)	47 (47)	1.0
Dyslipidemia	47 (47)	38 (38)	0.197
CAD	46 (46)	38 (38)	0.198
HF	46 (46)	50 (50)	0.571
CHA_2DS_2VA	2.5±1.673	2.39±1.53	0.628
NOAC	90 (90)	92 (92)	0.621
HR (beats/min)	90.04±15.25	91.21±11.26	0.95
SBP (mmHg)	124.7±9.33	128.5±8.77	0.36
DBP (mmHg)	79.5±7.82	80.15±7.95	0.52

Values are presented as mean±SD or number (%). CAD=Coronary artery disease, HF=Heart failure, CHA_2DS_2VA=Chronic heart failure, hypertension, age ≥75 years (2 points), diabetes mellitus, prior stroke/transient ischemic attack/arterial thromboembolism (2 points), vascular disease, age 65–74 years, NOAC=Non-Vitamin K oral anticoagulation, HR=Heart rate, SBP=Systolic blood pressure, DBP=Diastolic blood pressure, SD=Standard deviation

The incidence of ischemic stroke and TIAs was significantly higher in Group 1. However, the mortality rate was statistically lower in Group 1. Overall, MACE in Group 1 (52%) was slightly lower compared to Group 2 (56%), but this difference wasn't statistically significant. Out of 100 patients in each group, 30 patients (30.93%) in Group 1 achieved conversion from AF to sinus rhythm, compared to 20 patients (22.47%) in Group 2, with no statistically significant difference in the rates of initial successful cardioversion between the

Table 2: Assessment of echocardiographic parameters at baseline and after 6 months of follow-up

Echocardiographic parameters	Group 1 (*n*=100)	Group 2 (*n*=100)	*P*
LVEDV			
Baseline	101.97±25.67	103.53±29.92	0.693
Follow up	91.62±19.97	105.18±29.22	<0.001
P	<0.001	0.026	
LVESV			
Baseline	53.76±25.66	58.10±26.49	0.241
Follow up	45.51±21.16	59.26±25.21	<0.001
P	<0.001	0.029	
LVEF (%)			
Baseline	49.06±13.92	45.95±13.28	0.114
Follow-up	60.54±15.05	43.19±9.77	<0.001
P	<0.001	0.020	
LA anteroposterior diameter			
Baseline	4.21±0.86	4.04±0.79	0.140
Follow-up	3.99±0.87	4.97±0.86	<0.001
P	<0.001	<0.001	
LA longitudinal diameters			
Baseline	5.20±0.91	4.97±0.88	0.076
Follow-up	4.30±0.77	5.12±0.88	<0.001
P	<0.001	<0.001	
LA transverse diameters			
Baseline	4.29±0.76	4.28±0.83	0.988
Follow-up	3.51±0.75	4.60±1.06	<0.001
P	<0.001	0.008	
LA maximal volume			
Baseline	59.99±19.76	58.12±21.58	0.523
Follow-up	52.34±17.13	61.53±20.35	0.001
P	<0.001	<0.001	
LA minimal volume			
Baseline	37.63±19.08	35.59±14.49	0.397
Follow-up	30.74±17.65	46.72±15.65	<0.001
P	<0.001	<0.001	
LA emptying fraction			
Baseline	47.35±16.82	48.11±14.87	0.736
Follow-up	50.34±15.97	40.43±12.57	<0.001
P	0.008	<0.001	
LA strain (%)			
Baseline	22.93±1.90	23.45±2.03	0.060
Follow-up	28.80±2.4	21.58±1.88	<0.001
P	<0.001	<0.001	

Values are presented as mean±SD. LVEDV=Left ventricular end-diastolic volume, LVESV=Left ventricular end-systolic volume, LVEF=Left ventricular ejection fraction, LA=Left atrium, SD=Standard deviation

two groups. Regarding the continuation of sinus rhythm postcardioversion, 25 patients (25.77%) in Group 1 remained in sinus rhythm, while 10 patients (11.24%) in Group 2 remained in sinus rhythm. The rate continuation of sinus rhythm postcardioversion was higher in Group 1 [Table 3].

After 6 months of follow-up, 82 patients (84.54%) in Group 1 had LA function (LA volume and LA strain) improvement, while only 10 (11.24%) patients in Group 2 had LA function improvement [Table 3]. Multivariate regression analysis was used to detect the predictors of LA function improvement. It demonstrated that the use of SGLT2 inhibitors is an independent predictor for LA function improvement. Patients who received SGLT2 inhibitors as a part of their treatment were approximately 66 times more likely to experience improvement of LA function.

Discussion

AF is independently associated with higher rates of HF, stroke, and mortality. Despite advances in AF management, medical therapies that significantly reduce HF-related hospitalization and mortality in this population remain lacking.[10]

SGLT2 inhibitors, originally developed for glycemic control, significantly reduced HF hospitalization and cardiovascular mortality in patients with DM, renal dysfunction, HF, and cardiovascular disease.[11] They have demonstrated potential in reducing the risk of AF. Emerging evidence suggests that these agents exert beneficial metabolic effects on the circulating metabolites beyond glycemic control, which may contribute to a reduced risk of AF.[12] Data are limited regarding the direct impact of SGLT2 inhibitors on AF-related outcomes and complications, irrespective of diabetes status. Consequently, this study was conducted to assess the outcome of SGLT2 inhibitor use in AF patients and its effect on LA function.

We observed marked improvements across all assessed LA function parameters (LA dimensions, volumes, emptying fraction, and strain) in the SGLT2 inhibitors group. These results are in agreement with Tanaka *et al*.[13] who reported a significant improvement in LA volume index after using of SGLT2 inhibitors. Furthermore, according to El-Saied *et al*.,[14] SGLT-2 inhibitors were associated with improved diastolic function (E/e' ratio) and significantly lower LAV ($P < 0.001$). LV global longitudinal strain showed a significant increase. In addition, LA function and strain parameters improved markedly, with LASr increasing from 17.3 ± 2.0 to 23.8 ± 3.6, LA conduit strain from 11.0 ± 2.2 to 13.7 ± 2.8, and LA contractile strain from 6.5 ± 1.4 to 10.5 ± 2.6. LAEF % increased from 31.6 ± 4.2 to 43.2 ± 10.8. Furthermore, Sehly *et al*.,[15] verified that early use of empagliflozin has a beneficial effect on LA strain parameters in post-ACS patients. Similarly, Thiele *et al*.,[16] found that, after 3 months of treatment, empagliflozin significantly enhanced LA function, demonstrated by increases in LAS reservoir (26.4 ± 8.0% to 29.0 ±7.4%; $P = 0.011$) and LAS contraction phase (10.9 ± 5.7% to 12.5 ± 6.0%; $P = 0.008$) compared with placebo.

Table 3: Six-month outcome

Outcome parameters	Group 1 ($n=97$), *n* (%)	Group 2 ($n=89$), *n* (%)	*P*
Death	3 (3)	11 (11)	0.027
TIA	10 (10)	3 (3)	0.044
Ischemic stroke	7 (7)	1 (1)	0.031
HF	46 (46)	50 (50)	0.571
MACE	52 (52)	56 (56)	0.57
LA function improvement	82 (84.54)	10 (11.24)	<0.001
Sinus rhythm after cardioversion	30 (30.93)	20 (22.47)	0.094
Continuation of sinus rhythm	25 (25.77)	10 (11.24)	0.005

TIA=Transient ischemic attack, HF=Heart failure, MACE=Major adverse cardiovascular events, LA=Left atrial

The current study demonstrated that, while SGLT2 inhibitors may improve LA function, they might additionally be linked to a higher risk of specific cerebrovascular events like ischemic strokes. TIAs were significantly more prevalent in the SGLT2 inhibitors group. Likewise, a higher incidence of ischemic stroke was observed in patients receiving SGLT2 inhibitors compared with those not receiving SGLT2 inhibitors. The observed higher incidence of cerebrovascular events in the SGLT2 group should be interpreted with caution due to the limited number of events and potential residual confounding. No causal inference can be drawn. Similarly, Kimura *et al*.,[17] reported that there is a potential increase in ischemic stroke risk with SGLT2 inhibitors may be explained by volume depletion, orthostatic hypotension, and dehydration secondary to their mild diuretic action. Moreover, a meta-analysis by Wu *et al*.,[18] showed that the incidence of stroke increased significantly with SGLT2I.

Our study revealed that the mortality rate was significantly reduced among patients treated with SGLT2 inhibitors. Similar to our study, Pandey *et al*.,[5] demonstrated that there was a significant decrease in the risk of serious AF events among patients receiving SGLT2 inhibitors (1.1% vs. 1.5%; risk ratio 0.75 [95% confidence interval (CI), 0.66–0.86]; $I^2 = 0\%$). In addition, in diabetic patients with a history of AF, treatment with SGLT2 inhibitors was associated with a lower risk of HF hospitalization or cardiovascular death (HR 0.70 [95% CI, 0.57–0.85]; $I^2 = 0\%$. Li *et al*.,[2] reported that SGLT2 inhibitors were associated with significant reductions in AF/atrial flutter and all-cause mortality.

In our study, the SGLT2 group's higher sinus-rhythm maintenance aligns with multiple analyses showing that SGLT2 inhibitors reduce AF incidence and recurrence which may be due to enhanced LA mechanics with SGLT2I appeared to translate into better rhythm control. A meta-analysis study by Zheng *et al*.,[19] reported a roughly 18%–25% relative reduction in new-onset AF or serious AF events with SGLT2I therapy. In the multivariable logistic regression model, SGLT2 inhibitor therapy was independently associated with improvement in LA function. The estimated odds ratio was

66.345 (95% CI: 26.775–164.396; $P < 0.001$). Although the point estimate suggests a strong association, the relatively wide CI indicates variability in the effect size and warrants cautious interpretation. Given the observational nature of the analysis and the potential for residual confounding, these findings should be considered hypothesis-generating. Nevertheless, the results support a possible favorable impact of SGLT2 inhibitors on LA mechanics in patients with AF, which merits further validation in larger, adequately powered studies. Our results revealed that the beneficial impact of SGLT2 inhibitors on LA function is likely independent of patients' gender, the presence of comorbidities, or numerous clinical factors, reinforcing the hypothesis that the pharmacological action of SGLT2 inhibitors is a primary factor in improving cardiac function in patients with AF. Similarly, Li *et al*.,[2] concluded that SGLT2 inhibitors may reduce AF risk in susceptible individuals with type 2 diabetes, regardless of their baseline characteristics.

Study limitations

Our study has several limitations. The relatively small sample size may limit generalizability. Also, the short duration of the study limited the ability to assess the long-term effects of SGLT2 inhibitors. Moreover, the heterogeneity of patient comorbidities may have influenced the treatment response. As this was a nonrandomized observational study, residual confounding cannot be entirely excluded despite baseline comparability between groups. In addition, the limited number of cerebrovascular events reduces the statistical robustness of these specific outcome analyses and warrants cautious interpretation. Finally, intraobserver and interobserver variability could not be formally assessed. Future adequately powered prospective studies incorporating randomized allocation and advanced confounder-adjustment strategies are warranted to confirm these findings and to establish whether the observed associations reflect a true causal effect of SGLT2 inhibitor therapy on LA remodeling and clinical outcomes.

Conclusion

A substantial proportion of patients on SGLT2 inhibitors exhibited improvements in LA function, underscoring their potential therapeutic benefit. In patients with AF, SGLT2 inhibitors were associated with enhanced LA function and strain parameters, as well as a reduction in AF recurrence and mortality rates after 6 months. However, this therapeutic approach is also correlated with an elevated risk of ischemic stroke and TIA.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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