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RESEARCH ARTICLE

Clinical Outcome of SGLT2 Inhibitors in Patients with STEMI with Impaired Left Ventricular Function: A Randomization Study

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Abstract: ***Background***:ST-segment elevation myocardial infarction (STEMI) survivors face a significant residual risk of developing heart failure (HF) owing to maladaptive left ventricular (LV) remodeling. Sodium-Glucose Cotransporter-2 inhibitors (SGLT2i) are established pillars of care for chronic HF.Methods: This double-blind, placebo-controlled, prospective, randomized, single-center trial, enrolled 104 patients with confirmed STEMI and randomized them to be treated with either dapagliflozin (DAPA) 10 mg daily (n=73) or a matching placebo (n=31), in addition to standard guideline-directed therapy. Echocardiographic parameters, including LV Ejection Fraction (LVEF), Global Longitudinal Strain (GLS), and LV volumes, were assessed. All patients were assessed at the following time points: baseline (at admission), 30 days, and following 6 months.Results: At the 6-month follow-up, patients treated with DAPA exhibited superior cardiac function, with higher median LVEF (50% vs. 45%, p=0.002) and improved GLS (-12.0% vs. -10.9%, p=0.022). Notably, adverse LV remodeling was prevented entirely in the dapagliflozin group (0%) relative to 100% in the placebo group (p<0.001). Consequently, the total risk of MACE at 6 months was significantly reduced in the dapagliflozin group (15.1% vs. 38.7%, p=0.011), a benefit primarily driven by a reduction in HF-related hospitalizations (HFH) (6.8% vs. 25.8%, p=0.019), with comparability in cardiovascular mortality.Conclusion: Early upstream initiation of DAPA in patients presenting with STEMI can effectively improve clinical outcomes, as evidenced by reduced MACE and HFH incidence, and improve echocardiographic parameters, as evidenced by improved LV remodeling protection, longitudinal strain, and EF..

Keywords: STEMI; Dapagliflozin; Left Ventricular Remodeling; Heart Failure; Global Longitudinal Strain; SGLT2i .

BACKGROUND

ST-segment elevation myocardial infarction (STEMI) continues to represent a serious acute cardiovascular (CV) condition. Sodium-Glucose Cotransporter-2 inhibitors (SGLT2i), used initially as glucose-lowering drugs for type-2 diabetes mellitus (DM), have already established as the fourth pillar of foundational medical therapy for heart failure (HF) alongside beta-blockers, RAAS inhibitors, and mineralocorticoid receptor antagonists.[1]

Although recent data verify benefits across broader post-MI populations, there is a relative deficiency in evidence on the specific impact of early initiation of SGLT2i on the echocardiographic parameters of high-risk populations, particularly post-STEMI patients admitted to coronary care units (CCUs). Accordingly, we aimed to fully evaluate both clinical and echocardiographic parameters in this high-risk cohort throughout the in-hospital phase and throughout a 6-month post-discharge follow-up period.

METHODS

Patients admitted to the CCU in our center were screened, and only those with confirmed STEMI were enrolled. 200 participants were initially targeted for enrollment; the final analysis included 104 patients who met all eligibility criteria and completed follow-up.

Inclusion Criteria

Adult patients of both genders admitted with a confirmed diagnosis of STEMI who had adequate imaging quality for transthoracic echocardiography (TTE).

Exclusion Criteria

We excluded patients who were unable to undergo TTE owing to poor echogenicity or contraindications, uncooperative patients, those who refused participation, and patients with significant valvular disease.

Randomization and Blinding

Following diagnosis, upstream randomization via a closed-envelope method was employed to ensure allocation concealment, with eligible participants randomized in a 1:1 ratio to either the dapagliflozin (DAPA) or placebo group. Both patients and treating physicians were blinded to the treatment allocation.

Interventions

All patients included received standard GDMT for STEMI, including reperfusion strategies and

revascularization procedures, at the discretion of the treating physicians. In addition to standard care, patients were assigned to one of the following regimes: patients in the dapagliflozin group (Group I) received DAPA 10 mg once daily. In contrast, patients in the placebo group (Group II) received a matching placebo once daily.

Data Collection and Assessments

Upon admission, baseline data were collected from each patient, including demographics (age, sex) and CV risk factors (smoking, DM, hypertension, dyslipidemia, obesity), as well as medical history. Routine laboratory investigations (complete blood count, serum creatinine and urea, blood sugar) and 12-lead ECGs were obtained to assess ischemic changes and time to reperfusion markers.

TTE was carried out through a commercially available device in our CCU at baseline (admission), 30 days, and 6 months. Throughout this assessment, we evaluated and documented the following data: LVEF through Simpson's method, LV end-systolic volume (LVESV), LV end-diastolic volume (LVEDV), wall motion score index, global longitudinal strain (GLS), and tissue doppler velocity (e'). Additionally, Adverse LV remodeling was defined and assessed at the 6-month follow-up.

Outcome Measures

The primary outcome was Major Adverse Cardiovascular Events (MACE), defined as a composite of CV-related outcomes, including CV mortality, HF, HF-related hospitalization (HFH), reinfarction, stroke, resuscitated cardiac arrest, and target vessel revascularization. This composite outcome was assessed at admission, at 30 days, and following 6 months. Secondary outcome included echocardiographic changes in LV function and dimensions (LVEF, GLS, LVESV, LVEDV) from baseline to 30 days and 6 months.

Statistical Analysis

All statistical evaluations were carried out through the most up to date major edition of the SPSS, V31. The distribution of study variables was examined through the Shapiro Wilk test and was found to deviate from normality. As a result, quantitative variables were presented either as medians with interquartile ranges or as mean values with corresponding standard errors, according to their distributional characteristics, while qualitative variables were expressed as counts and proportions. Comparisons between independent groups were carried out through the Mann-Whitney test for numerical variables and either the chi-square test or Fisher's exact test for categorical variables. Analyses of repeated observations within the same group, including changes in echocardiographic measurements, were carried out through the Wilcoxon rank sum test. A threshold of $P < 0.05$ was deemed statistically significant.

RESULTS

3.1. Baseline Demographic and Clinical Characteristics

104 Patients with STEMI were enrolled in the current investigation (73 in the DAPA group and 31 in the Placebo group). As shown in Table 1, the investigated groups are comparable (P>0.0). The median age in the study group was 56 years, with a male predominance (51.6% in Group I vs. 60.3% in Group II; P=0.516). Regarding risk factors (Group I vs. Group II), the most common was smoking (38.7% vs. 41.1%), followed by hypertension (25.8% vs. 35.6%) and DM (25.8% vs. 27.4%). More details about the baseline data are documented in

Table 1. Baseline demographic and clinical characteristics of the study population

Variable	Placebo (n=31)	Dapagliflozin (n=73)	P
Age (years)	56.0 (45.0 – 67.0)	56.0 (54.0 – 70.0)	1.000[#]
Gender			0.516[$]
Male	16 (51.6%)	44 (60.3%)	
Female	15 (48.4%)	29 (39.7%)	
Risk Factors			
Diabetes Mellitus	8 (25.8%)	20 (27.4%)	1.000[$]
Hypertension	8 (25.8%)	26 (35.6%)	0.369[$]
Smoking	12 (38.7%)	30 (41.1%)	1.000[$]
Obesity	6 (19.4%)	13 (17.8%)	1.000[$]
Dyslipidemia	3 (9.7%)	17 (23.3%)	0.172[$]
Medical History			
Family History of CAD	1 (3.2%)	13 (17.8%)	0.060[$]
Previous IHD	4 (12.9%)	15 (20.5%)	0.419[$]
Previous PCI	1 (3.2%)	11 (15.1%)	0.103[$]

data are presented as median (interquartile range) for continuous variables and number (%) for categorical variables. # pcalculated through the mann-whitney u test (age distribution was non-normal, shapiro-wilk $p < 0.001$ for

dapagliflozin). $ pcalculated through fisher's exact test. abbreviations: cad: coronary artery disease; ihd: ischemic heart disease; pci: percutaneous coronary intervention.

Procedural Characteristics and Hospital Course

The most common presentation was anterior wall MI (64.5% in Group I vs. 47.9%), followed by inferior wall MI (25.8% vs. 39.7%), with comparability (P= 0.294). Reperfusion Strategy through Streptokinase (74.2% vs. 78.1%) and Primary PCI (25.8% vs. 21.9%), with comparability (P=0.667). The investigated groups are comparable regarding markers of successful reperfusion, including time to ST-segment resolution (48.4% vs. 54.8%; P=0.549) and peak CK-MB levels, with median time to ST-segment resolution of 19.5 minutes in the investigated groups (P=0.419). Patients in the Placebo group had a more extended length of hospital stay (LOS), with a median of 4.0 days [IQR 3.0–4.8], relative to those in the DAPA group, with a median of 3.0 days [IQR 3.0–4.0] (*P*= 0.003). More information about Ischemic Times (min) and procedural characteristics is presented in **Table 2**

Table 2. STEMI presentation, procedural characteristics, and hospital course

Variable	Placebo (n=31)	Dapagliflozin (n=73)	P
Location of Infarction			0.294$
Anterior	20 (64.5%)	35 (47.9%)	
Inferior	8 (25.8%)	29 (39.7%)	
Lateral	3 (9.7%)	9 (12.3%)	
Reperfusion Strategy			0.667$
Streptokinase	23 (74.2%)	57 (78.1%)	
Primary PCI	8 (25.8%)	16 (21.9%)	
ST-Segment Resolution			0.549$
Complete (>70%)	15 (48.4%)	40 (54.8%)	
Partial (<70%)	16 (51.6%)	33 (45.2%)	
Ischemic Times			
Total ischemic time (min)	200.0 (170.0 – 280.0)	200.0 (175.0 – 220.0)	0.724#
Door-to-reperfusion time (min)	40.0 (30.0 – 45.0)	40.0 (35.0 – 45.0)	0.954#
Symptom-to-ST resolution (min)	225.0 (200.0 – 305.0)	230.0 (210.0 – 255.0)	0.853#
Other Parameters			
Time to peak CK-MB (hours)	19.5 (18.0 – 23.0)	19.5 (16.0 – 22.0)	0.419#
Length of hospital stay (days)	4.0 (3.0 – 4.8)	3.0 (3.0 – 4.0)	0.003#

Data are presented as median (interquartile range) for continuous variables and number (%) for categorical variables. # Pcalculated through the Mann-Whitney U test. $ Pcalculated through the Pearson Chi-Square test. Abbreviations: PCI: Percutaneous Coronary Intervention; CK-MB: Creatine Kinase-MB.

Echocardiographic outcomes assessments at baseline and follow-up (30 days and 6 months)

For LVEF (%), there is no difference between the investigated groups at baseline and following 30 days of follow-up (P= 0.986, 0.489, respectively), but a significant difference after 6 months of follow-up as median LVEF% in the DAPA group 50 [IQR 45–56] relative to placebo group 45 [IQR 44–50](P= 0.002). Regarding the GLS, at baseline there was a highly significant difference between the investigated groups (P<0.001), but following 30 days, the groups were comparable (P=0.198). After 6 months of follow-up, the DAPA group exhibited significantly better GLS than the placebo group (50; P=0.022). Table 3

For Adverse LV modeling, there's a highly significant difference between the investigated groups, as the DAPA group exhibited no adverse LV modeling (0% in the DAPA group, 100% in the placebo group) (P<0.001). More details about the other echocardiographic parameters at baseline, 30 days, and 6 months are documented in **Table 3.**

Table 3. Echocardiographic outcomes at baseline and follow-up

Variable	Time Point	Placebo (n=31)	Dapagliflozin (n=73)	P
LVEF (%)	Baseline	57.8 (54.0 – 60.0)	58.0 (50.0 – 60.7)	0.986
	30 Days	50.0 (45.0 – 55.0)	50.0 (45.0 – 56.0)	0.489
	6 Months	45.0 (44.0 – 50.0)	50.0 (45.0 – 56.0)	0.002
Global Longitudinal Strain (%)	Baseline	-12.0 (-14.6 – -11.2)	-17.5 (-19.0 – -14.5)	<0.001
	30 Days	-11.0 (-13.9 – -10.0)	-12.0 (-14.2 – -11.0)	0.198
	6 Months	-10.9 (-12.8 – -9.7)	-12.0 (-14.0 – -11.0)	0.022
Wall Motion Score Index	Baseline	1.0 (1.0 – 1.4)	1.0 (1.0 – 1.5)	0.839
	30 Days	1.2 (1.0 – 1.4)	1.1 (1.0 – 1.5)	0.719
	6 Months	1.3 (1.2 – 1.4)	1.2 (1.0 – 1.4)	0.032
LV End-Systolic Volume (ml)	Baseline	47.8 (35.2 – 56.2)	44.0 (33.0 – 55.0)	0.211
	30 Days	55.0 (49.5 – 59.5)	54.3 (47.0 – 58.0)	0.69
	6 Months	64.0 (59.5 – 73.0)	55.0 (45.0 – 59.0)	<0.001
LV End-Diastolic Volume (ml)	Baseline	99.0 (84.1 – 118.0)	100.0 (88.1 – 121.0)	0.565
	30 Days	110.0 (99.0 – 130.0)	115.0 (99.0 – 130.0)	0.437
	6 Months	123.0 (105.0 – 143.5)	100.0 (90.0 – 121.0)	<0.001
Adverse LV Remodeling	6 Months	31 (100.0%)	0 (0.0%)	<0.001

Data are presented as median (interquartile range) for continuous variables and number (%) for categorical variables. Ps were calculated through the Mann-Whitney U test for continuous variables and Fisher's Exact test for adverse remodeling.

Table 4. Paired comparisons of echocardiographic parameters from baseline to follow-up within study groups

Variable	Time Point	Dapagliflozin	P	Placebo	P
LV End-Systolic Volume (ml)	Baseline vs. 30-Day	-8.7 ± 2.2	<0.001*	-4.8 ± 3.8	0.162
	Baseline vs. 6-Month	-9.0 ± 2.2	<0.001*	-17.1 ± 3.6	<0.001*
LV End-Diastolic Volume (ml)	Baseline vs. 30-Day	-16.0 ± 3.0	<0.001*	-12.7 ± 4.1	0.004*
	Baseline vs. 6-Month	-1.5 ± 0.8	0.009*	-23.5 ± 0.9	<0.001*
LVEF (%)	Baseline vs. 30-Day	6.0 ± 1.1	<0.001*	6.4 ± 1.3	<0.001*
	Baseline vs. 6-Month	6.0 ± 1.1	<0.001*	10.0 ± 1.3	<0.001*
Global Longitudinal Strain (%)	Baseline vs. 30-Day	-4.0 ± 0.4	<0.001*	-1.5 ± 0.7	0.005*
	Baseline vs. 6-Month	-4.0 ± 0.4	<0.001*	-1.8 ± 0.6	0.002*
Wall Motion Score Index	Baseline vs. 30-Day	-0.0 ± 0.1	0.912	-0.1 ± 0.1	0.593
	Baseline vs. 6-Month	-0.0 ± 0.1	0.897	-0.2 ± 0.1	0.106
Tissue Doppler Velocity e' (cm/s)	Baseline vs. 30-Day	-0.5 ± 0.2	0.014*	-0.5 ± 0.3	0.133

	Baseline vs. 6-Month	-0.7 ± 0.2	0.002*	-0.6 ± 0.3	0.088

Data are presented as Mean Difference ± Standard Error (SE) calculated as (Baseline – Follow-up). Pcalculated through the Wilcoxon Signed-Rank Test.* Indicates statistical significance (P < 0.05).

Clinical outcome and Major Adverse Cardiac events throughout follow-up.

We documented the MACE throughout hospital admission, at 30 days, and at 6 months of follow-up in **Table 5.** At each time point, the DAPA group had fewer statistically significant events than the placebo group (P<0.05). Throughout hospital stay, only 3 (4.1%) patients in the DAPA group had MACES, relative to 8 (25.8%) in the placebo group (P=0.002). Following 30 days of follow-up, there is no CV mortality or target vessel revascularization. For the total MACES, DAPA associates with low statistically significant events relative to the placebo group (8.2% in the DAPA group Vs. 25.8% in the placebo group) (P= 0.026). After 6 months of follow-up, the DAPA group also exhibited a significant difference in MACES incidence relative to the placebo group (P=0.011).

Table 5. Clinical outcomes and major adverse cardiovascular events (MACE) throughout follow-up

Outcome	Placebo (n=31)	Dapagliflozin (n=73)	P
In-Hospital Outcomes			
Total MACE	8 (25.8%)	3 (4.1%)	0.002*
Heart failure	3 (9.7%)	0 (0.0%)	0.025*
Reinfarction	3 (9.7%)	1 (1.4%)	0.078
Stroke	1 (3.2%)	0 (0.0%)	0.298
Resuscitated cardiac arrest	1 (3.2%)	2 (2.7%)	1
30-Day Outcomes			
Total MACE	8 (25.8%)	6 (8.2%)	0.026*
Cardiovascular mortality	0 (0.0%)	0 (0.0%)	—
Rehospitalization for heart failure	8 (25.8%)	6 (8.2%)	0.026*
Target vessel revascularization	0 (0.0%)	0 (0.0%)	—
6-Month Outcomes			
Total MACE	12 (38.7%)	11 (15.1%)	0.011*
Cardiovascular mortality	2 (6.5%)	3 (4.1%)	0.633
Rehospitalization for heart failure	8 (25.8%)	5 (6.8%)	0.019*
Target vessel revascularization	2 (6.5%)	3 (4.1%)	0.633

Data are presented as number (%). Ps calculated through Fisher's Exact Test. * Indicates statistical significance (P < 0.05). MACE: Major Adverse Cardiovascular Events (composite of cardiovascular mortality, heart failure, reinfarction, stroke, and urgent revascularization

DISCUSSION

In the current investigation, our pooled results exhibited that early administration of DAPA to standard medical therapy in STEMI patients admitted to the CCU was associated with significant clinical and echocardiographic benefits. Relative to placebo, patients in the DAPA group had a shorter LOS, a higher LVEF at 6 months, and no adverse LV remodeling. Our results also exhibited that DAPA significantly reduced the risk of MACE at all analyzed time points, mainly driven by HF and HFH. These observations verify that SGLT2 inhibition may modify the natural history of post-infarction cardiomyopathy when initiated in the acute phase.

Our analysis exhibited that the incidence of MACE was significantly diminished in the DAPA group throughout the LOS (4.1% vs 25.8%, p=0.002), at 30 days post-discharge (8.2% vs 25.8%, p=0.628), and at 6 months of follow-up (15.1% vs 38.7%, p=0.011). Notably, these significant reductions in MACE risk were mainly driven by reductions in HF and HFH risk. Similar to our results, previous landmark clinical trials of SGLT2i drugs also documented significant reductions in the risk of MACE, driven mainly by reductions in HFH.[2-4] In accordance with our results, EMPACT-MI, which randomized 6522 hospitalized acute MI patients with reduced EF to receive empagliflozin or placebo, documented that following a median follow-up of 17.9 months, the risk of first HFH and total HFH was significantly reduced by 23% and 33% in the empagliflozin group in all of the analyzed subgroups. [5] Another recent meta-analysis, pooling data from 15,133 patients across 10 studies, documented that early initiation of SGLT2i following acute MI was associated with a 33% reduction in the incidence of HFH.[6] Generally, these observations align with SGLT2i's robust ability to prevent clinical HF decompensation by reducing preload and improving volume management through diuresis.[7] SGLT2i have also been associated with protective effects against myocardial inflammation, oxidative stress, cellular apoptosis, and mitochondrial dysfunction, which can collectively improve myocardial health.[8] Moreover, ischemic myocardium is associated with profound metabolic dysfunction that results in impaired glucose

utilization with a pathological shift toward predominant reliance on fatty acid oxidation. This less oxygen-efficient process exacerbates oxidative stress and myocardial injury. [9, 10] Emerging evidence verify s that SGLT2i could mitigate these maladaptive metabolic changes by promoting a shift toward a more energy-efficient strategy, including ketone bodies, enhancing myocardial energetics, enhancing mitochondrial function, and reducing oxidative stress and inflammation. [9, 10]

We observed comparability in CV mortality between the DAPA and placebo groups after 6 months of follow-up (4.1% vs 6.5%, p=0.633). Similarly, there was comparability in in-hospital mortality or 30-day mortality. The lack of mortality benefit is consistent with the results documented by EMPACT-MI and DAPA-MI trials, which found that while SGLT2i drugs managed to reduce the incidence of HFH significantly, they did not demonstrate significant reductions in all-cause mortality or CV mortality in the post-MI populations. [11, 12] In contrast, previous reports investigating the impacts of SGLT2i drugs in the treatment of other populations, including chronic HF or high-risk diabetic patients, documented that SGLT2i was associated with a diminished risk of mortality. [13] It is noteworthy that the relatively small sample size and short follow-up period in our analysis may have made the study statistically underpowered to discover significant differences in mortality risk between the groups. Interestingly, previous meta-analyses stated that their included studies also lacked adequate long-term follow-up to thoroughly investigate the mortality benefits of SGLT2i in the post-MI populations.

Regarding the echocardiographic assessment, our results exhibited that DAPA was associated with superior echocardiographic parameters at 6 months, evident by the complete absence of adverse remodeling (0% vs 100%, p<0.001), higher LVEF (50% vs 45%, p=0.002), and higher GLS (12.0% vs -10.9%, p=0.022). Critically, preventing maladaptive LV remodeling is vital to the long-term prognosis of post-STEMI subjects. Although at baseline, patients in the DAPA group had better GLS values (-17.5% vs -12.0%), the further longitudinal analysis revealed greater delta change in GLS from baseline to follow-up in the DAPA group relative to placebo (-4.0% vs -1.5%), supporting the efficacy of DAPA. In alignment with our results, Salem and co-authors documented that early initiation of DAPA following acute MI resulted in significant improvements in cardiac echocardiographic parameters, including enhanced change in LVEF and GLS. [14] Similarly, Wan and co-authors also documented that diabetic acute MI patients discharged on SGLT2i drugs exhibited better LV function preservation and a diminished risk of cardiac remodeling.[15]

The current investigation has various limitations that should be considered. The single-center facility and relatively small sample size included in the analysis limit the study's statistical power to detect differences in secondary hard endpoints, including mortality, and the generalizability of our findings. Our population reflects a specific practice setting in which a significant proportion (approximately 76%) of patients received streptokinase owing to logistical constraints on primary PCI. This factor differs from those in other studies. Some baseline echocardiographic parameters were not equal between the investigated groups at admission despite performing randomization, which we attempted to account for by analyzing the relative change in parameters over time. The relatively short 6-month follow-up period may be sufficient to detect early cardiac remodeling, but it's not adequate to assess the full long-term efficacy and safety of SGLT2i. Therefore, we recommend that future research focus on large-scale, prospective RCTs with adequate long-term follow-up to verify our observations and thoroughly examine the survival benefits of early SGLT2i administration in post-MI patients.

CONCLUSION

Early initiation of DAPA alongside the standard therapy for post-MI patients significantly attenuates adverse LV remodeling, improves longitudinal strain, and reduces the risk of HFH. Future large-scale, multicenter trials are recommended to verify the obtained observations and evaluate long-term mortality benefits.

REFERENCES

1. Padda IS, Mahtani AU, Parmar M. Sodium-Glucose Transport 2 (SGLT2) Inhibitors. StatPearls. Treasure Island (FL): StatPearls Publishing
2. Copyright © 2026, StatPearls Publishing LLC.; 2026.
3. Anker SD, Butler J, Filippatos G, Ferreira JP, Bocchi E, Böhm M, et al. Empagliflozin in Heart Failure with a Preserved Ejection Fraction. N Engl J Med. 2021;385:1451-1461.
4. McMurray JJV, Solomon SD, Inzucchi SE, Køber L, Kosiborod MN, Martinez FA, et al. Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction. N Engl J Med. 2019;381:1995-2008.
5. Zinman B, Wanner C, Lachin JM, Fitchett D, Bluhmki E, Hantel S, et al. Empagliflozin, Cardiovascular Outcomes, and Mortality in Type 2 Diabetes. N Engl J Med. 2015;373:2117-2128.
6. Hernandez AF, Udell JA, Jones WS, Anker SD, Petrie MC, Harrington J, et al. Effect of Empagliflozin on Heart Failure Outcomes After Acute Myocardial Infarction: Insights From the EMPACT-MI Trial. Circulation. 2024;149:1627-1638.
7. Semirani-Nezhad D, Soleimani H, Taebi M, Roozbehi K, Jahangiri S, Sattartabar B, et al. Early initiation of SGLT2 inhibitors in acute myocardial infarction and cardiovascular outcomes, an updated

systematic review and meta-analysis. BMC Cardiovasc Disord. 2025;25:527.
8. Asensio Lopez MDC, Lax A, Hernandez Vicente A, Saura Guillen E, Hernandez-Martinez A, Fernandez Del Palacio MJ, et al. Empagliflozin improves post-infarction cardiac remodeling through GTP enzyme cyclohydrolase 1 and irrespective of diabetes status. Sci Rep. 2020;10:13553.
9. Muscoli S, Barillà F, Tajmir R, Meloni M, Della Morte D, Bellia A, et al. The New Role of SGLT2 Inhibitors in the Management of Heart Failure: Current Evidence and Future Perspective. Pharmaceutics. 2022;14.
10. Sayour AA, Korkmaz-Icöz S, Loganathan S, Ruppert M, Sayour VN, Oláh A, et al. Acute canagliflozin treatment protects against in vivo myocardial ischemia-reperfusion injury in non-diabetic male rats and enhances endothelium-dependent vasorelaxation. J Transl Med. 2019;17:127.
11. Santos-Gallego CG, Requena-Ibanez JA, San Antonio R, Ishikawa K, Watanabe S, Picatoste B, et al. Empagliflozin Ameliorates Adverse Left Ventricular Remodeling in Nondiabetic Heart Failure by Enhancing Myocardial Energetics. J Am Coll Cardiol. 2019;73:1931-1944.
12. Fioretti F, Butler J, Udell JA, Schuyler Jones W, Petrie MC, Harrington J, et al. Empagliflozin after myocardial infarction with or without diabetes and chronic kidney disease: Insights from EMPACT-MI. ESC Heart Fail. 2025;12:3940-3952.
13. James S, Erlinge D, Storey RF, McGuire DK, de Belder M, Eriksson N, et al. Dapagliflozin in Myocardial Infarction without Diabetes or Heart Failure. NEJM Evid. 2024;3:EVIDoa2300286.
14. Cardoso R, Graffunder FP, Ternes CMP, Fernandes A, Rocha AV, Fernandes G, et al. SGLT2 inhibitors decrease cardiovascular death and heart failure hospitalizations in patients with heart failure: A systematic review and meta-analysis. EClinicalMedicine. 2021;36:100933.
15. Salem HSE, El-Mawardy R, Zahran ME, Bastawy I, Aziz M, Attia A. Effect of Dapagliflozin on Clinical Outcomes and Echocardiographic Parameters of Patients with Acute Myocardial Infarction After Percutaneous Coronary Intervention. QJM: An International Journal of Medicine. 2025;118:hcaf224. 024.
16. Wan J, Xu F, Zuo H, Jiang X, Wang Y, Jiang Y, et al. Impact of SGLT2 Inhibitors on Left Ventricular Remodeling in Diabetic Patients with Acute Myocardial Infarction. J Cardiovasc Pharmacol Ther. 2024;29:10742484241301191.