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ORIGINAL RESEARCH

HEART FAILURE AND CARDIOMYOPATHIES

Empagliflozin in Heart Failure and Previous Coronary Revascularization



Insights From EMPEROR-Pooled

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ABSTRACT

BACKGROUND Sodium glucose co-transporter-2 inhibitors are a foundational therapy for the management of chronic heart failure (HF).

OBJECTIVES The purpose of this study was to investigate whether the efficacy of sodium glucose co-transporter-2 inhibitors in patients with HF is maintained in patients with a history of coronary revascularization.

METHODS The EMPagliflozin outcomE tRial in Patients With chrOnic heaRt Failure (EMPEROR)-Pooled database was a prospectively designed pooled analysis of the EMPEROR-Reduced (NCT03057977) and EMPEROR-Preserved (NCT03057951) trials, which randomized patients with HF with a left ventricular ejection fraction below and above 40%, respectively, to empagliflozin or placebo. The primary outcome was time to first HF hospitalization or cardiovascular death. We studied the baseline risk of revascularized HF patients and the impact of previous revascularization on the efficacy and safety of empagliflozin.

RESULTS Overall, 3,437/9,718 (35.4%) had a history of coronary revascularization with either percutaneous coronary intervention or coronary artery bypass grafting (REVASC), and 6,281/9,718 (64.6%) did not (no REVASC). The median follow-up time was 21.23 months (IQR: 14.93-28.97 months). Of those randomized to placebo, a history of revascularization was not associated with an elevated risk of clinical outcomes, including the primary outcome and 3-point major adverse cardiac and cerebrovascular event. The efficacy and safety of empagliflozin were consistent irrespective of revascularization history for all major outcomes (primary outcome: REVASC: 0.76 [0.66-0.89], no REVASC: 0.77 [0.68-0.87], $P_{\text{interaction}}$: 0.94). Empagliflozin had neutral effect on a 3-point major adverse cardiac and cerebrovascular event in both groups.

CONCLUSIONS In this large HF trial program, revascularized patients with HF exhibited similar rates of HF and ischemic events as those without prior revascularization. The efficacy and safety of empagliflozin in HF were consistent in those with and without a revascularization history. (JACC Adv. 2026;5:102419) © 2026 Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

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**ABBREVIATIONS
AND ACRONYMS****AE** = adverse event**CABG** = coronary artery
bypass grafting**CAD** = coronary artery disease**CV** = cardiovascular**eGFR** = estimated glomerular
filtration rate**HF** = heart failure**HHF** = hospitalization for heart
failure**KCCQ-CSS** = Kansas city
cardiomyopathy questionnaire
clinical summary score**LVEF** = left ventricular
ejection fraction**MACE** = major adverse cardiac
and cerebrovascular event**PCI** = percutaneous coronary
intervention**SGLT2i** = sodium-glucose
cotransporter 2 inhibitor

Ischemic heart disease (IHD) is a central mechanistic underpinning for the development of heart failure (HF).¹ Current guidelines provide a Class I recommendation for revascularization with percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) for all patients with stable IHD and refractory symptoms, as well as for CABG in patients with ischemic cardiomyopathy and multivessel disease.²

Nevertheless, significant residual risk of major adverse cardiovascular (CV) events exists following coronary revascularization, underscoring the need for concerted secondary prevention strategies.³ Empagliflozin has emerged as a first-line treatment option for the management of chronic HF based on findings from the landmark EMPagliflozin outcome tRial in Patients With chrOnic heaRt Failure (EMPEROR)-Reduced⁴ and EMPEROR-Preserved⁵ trials, and the combined analysis EMPEROR-

Pooled.⁶ Although the recently reported EMPACT-MI (Trial to Evaluate the Effect of Empagliflozin on Hospitalization for Heart Failure and Mortality in Patients with Acute Myocardial Infarction) trial showed no reduction in the primary outcome of first HF hospitalization or all-cause mortality with empagliflozin among over 6,000 randomized patients following acute myocardial infarction (MI),⁷ 89.3% of which underwent revascularization prior to randomization,⁸ it suggested beneficial effects on the reduction of first and total HF hospitalizations as well as other HF-related events.⁹ It should be highlighted that EMPACT-MI specifically recruited patients following acute coronary syndromes, and therefore the efficacy of empagliflozin in reducing the risk of HF events among patients with chronic HF on the background of IHD and a revascularization history remains an important unanswered question.

EMPEROR-Pooled enrolled patients with stable, chronic HF syndromes across the spectrum of left ventricular ejection fraction (LVEF) and irrespective of etiology, and randomized them to either empagliflozin 10 mg or matching placebo. In this post hoc analysis of EMPEROR-Pooled, we report outcomes with empagliflozin in HF stratified by a history of coronary revascularization (REVASC vs no REVASC).

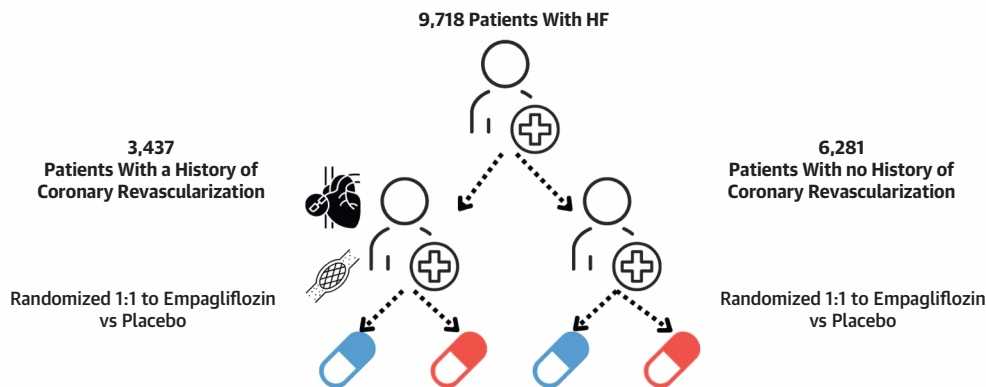
METHODS

STUDY DESIGN. EMPEROR-Pooled was a prospectively designed combined analysis of EMPEROR-Reduced⁴ and EMPEROR-Preserved,⁵ the methods of which have been published previously.¹⁰⁻¹² In brief, the trials included adult patients with chronic NYHA functional class II-IV HF with evidence of natriuretic peptide elevation. Patients with LVEF below 40% were included in EMPEROR-Reduced, while those with LVEF \geq 40% were included in EMPEROR-Preserved. Symptomatic hypotension and/or systolic blood pressure $<$ 100 mm Hg, along with estimated glomerular filtration rate (eGFR) $<$ 20 mL/min/1.73 m² represented some of the key exclusion criteria. Patients with MIs or CABG in the 90 days prior to enrollment were also excluded. Patients included were randomized in a 1:1 fashion to either empagliflozin 10 mg or placebo. In the present study, the recruited population was divided based on any history of coronary revascularization (ie, CABG or PCI) prior to enrollment (REVASC vs. no REVASC) (**Central Illustration**).

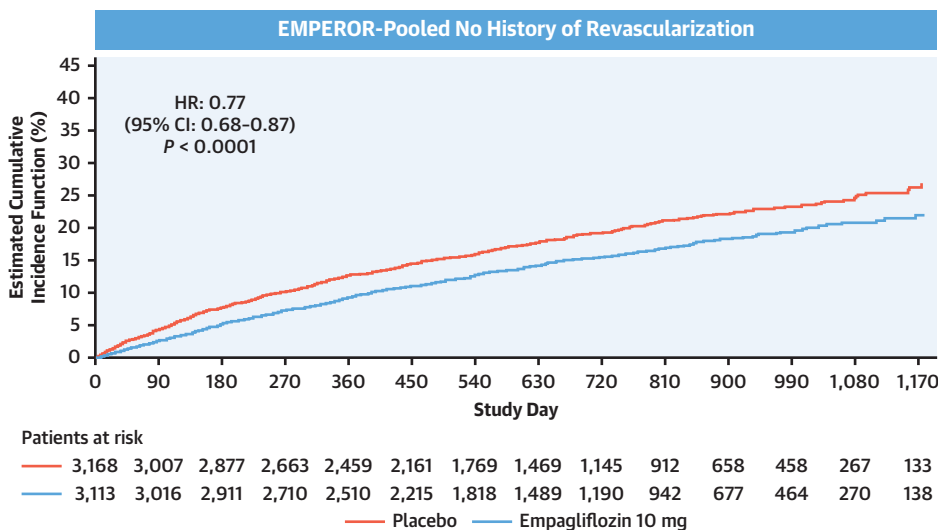
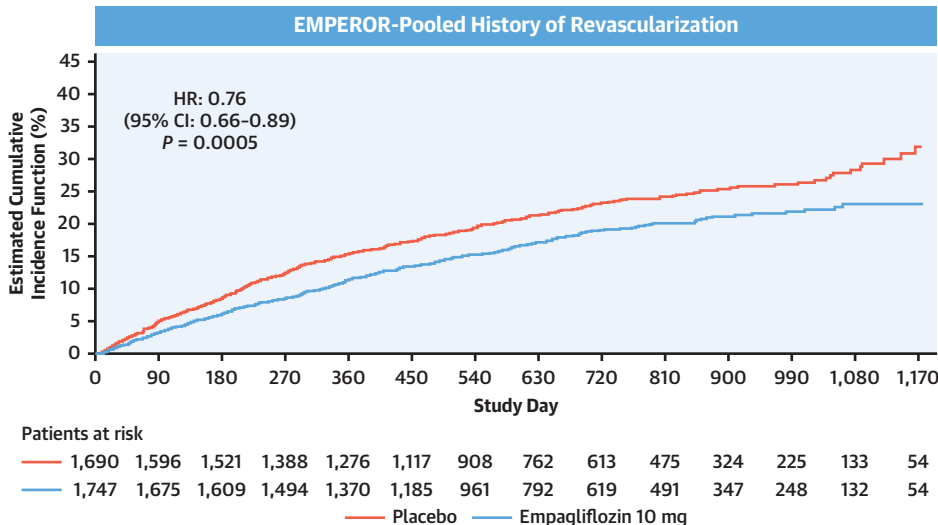
STUDY OUTCOMES. The primary outcome of the index trials was time to first HF hospitalization or CV death; secondary outcomes evaluated included components of the primary outcome, total (first and recurrent) HF hospitalizations, an extended composite outcome consisting of time to first CV death, HF hospitalization equivalent event or visit reporting diuretic intensification, and all-cause mortality. A 3-point major adverse cardiac and cerebrovascular

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CENTRAL ILLUSTRATION Summary of Study Design and Primary Outcome Analysis



Primary Outcome: Time to First HF Hospitalization or CV Death



A history of coronary revascularization did not modify the efficacy of empagliflozin in patients with HF enrolled in EMPEROR-Pooled.

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In this post hoc subanalysis of the EMPEROR-Pooled program, enrolled patients with HF across the LVEF spectrum were stratified based on whether they had a history of coronary revascularization. The efficacy and safety of empagliflozin were demonstrated to be consistent irrespective of revascularization history for all major clinical outcomes. Empagliflozin had neutral effect on a 3-point MACE in both groups. LVEF = left ventricular ejection fraction; other abbreviations as in Figures 1 and 2.

TABLE 1 Baseline Characteristics of Included EMPEROR-Pooled Patients Stratified by History of Coronary Revascularization

	No Revascularization (N = 6,281)	Revascularization (N = 3,437)
Demographics		
Men, n (%)	3,550 (56.5)	2,599 (75.6)
Race, n (%)		
White	4,513 (71.9)	2,658 (77.3)
Black	402 (6.4)	113 (3.3)
Asian	977 (15.6)	519 (15.1)
Other	362 (5.8)	114 (3.3)
Missing	27 (0.4)	33 (1.0)
Age (y), mean (SD)	69.5 (11.0)	70.8 (9.0)
Cardiac risk factors		
Smoking history, n (%)	2,806 (44.7)	2073 (60.3)
Current smoker, n (%)	488 (7.8)	335 (9.7)
Hypertension, n (%)	5,141 (81.9)	2,981 (86.7)
Systolic blood pressure (mm Hg), mean (SD)	128.3 (16.3)	127.7 (16.4)
Hypercholesterolemia, n (%)	3,514 (55.9)	2,780 (80.9)
Diabetic, n (%)	2,870 (45.7)	1924 (56.0)
BMI (kg/m ²), mean (SD)	29.27 (5.95)	28.75 (5.41)
Body weight (kg), mean (SD)	80.38 (19.61)	81.15 (18.23)
Heart failure characteristics, established cardiovascular disease, and laboratory values		
LVEF (%), mean (SD)	45.4 (15.5)	41.5 (14.4)
LVEF ≥50%, n (%)	2,877 (45.8)	1,129 (32.8)
Rates of HHF in previous 12 mo, n (%)	1,644 (26.2)	876 (25.5)
Time since HF diagnosis (y), median (IQR)	2.8 (1.0-6.5)	3.7 (1.3-7.8)
NT-proBNP (pg/mL), median (IQR)	1,334 (687-2,346)	1,206 (643-2,301)
Ischemic cause of HF, n (%)	1,229 (19.6)	2,817 (82.0)
History of atrial fibrillation or flutter ^a , n (%)	3,214 (51.2)	1,362 (39.6)
NYHA functional class, n (%)		
I/II	4,953 (78.9)	2,734 (79.5)
III/IV	1,328 (21.1)	703 (20.5)
KCCQ-CSS, mean (SD)	70.1 (21.5)	71.3 (21.3)
Volume status in the 4 wks before visit 1, n (%)		
Volume overloaded, with evidence of edema or organ congestion	1,083 (17.2)	470 (13.7)
Volume overloaded, without evidence of edema or organ congestion	607 (9.7)	319 (9.3)
Previously volume overloaded, but now euvolemic	957 (15.2)	528 (15.4)
Previously volume depletion, but now euvolemic	115 (1.8)	51 (1.5)
Volume depletion	60 (1.0)	23 (0.7)
Rates of euvolemia in preceding 4 wks	3,455 (55.0)	2043 (59.4)
History of myocardial infarction, n (%)	951 (15.1)	2,452 (71.3)
History of coronary artery disease, n (%)	842 (13.4)	2,962 (86.2)
Acute coronary syndrome in 6 mo prior to enrollment, n (%)	116 (1.8)	288 (8.4)
hs-troponin (ng/L), median (IQR)	18.4 (12.0-28.4)	21.0 (14.0-31.5)
eGFR (mL/min/1.73 m ²), mean (SD)	62.4 (20.9)	58.9 (19.7)
Hemoglobin (g/dL), mean (SD)	13.48 (1.63)	13.45 (1.57)
^a Defined as atrial fibrillation or atrial flutter reported in any ECG before treatment intake or history of atrial fibrillation or atrial flutter reported as medical history. BMI = body mass index; ECG = electrocardiogram; eGFR = estimated glomerular filtration rate; EMPEROR = EMPagliflozin outcome tRial in Patients With chrOnic heart Failure; HF = heart failure; HHF = hospitalization for heart failure; KCCQ-CSS = Kansas city cardiomyopathy questionnaire-clinical summary score; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide.		

event (MACE) endpoint was also evaluated, defined as time to first occurrence of CV death, nonfatal MI, or nonfatal stroke. Renal outcomes including slope of eGFR change and a composite renal endpoint consisting of time to first event of sustained reduction in

eGFR of ≥50%, end-stage renal disease (chronic dialysis/renal transplant or sustained eGFR<15 mL/min/1.73 m² for patients with baseline eGFR ≥30 mL/min/1.73 m² or sustained eGFR<10 mL/min/1.73 m² for patients with baseline eGFR<30 mL/min/1.73 m²)

or renal death, were also evaluated. Finally, patient-reported health status was assessed using the Kansas City Cardiomyopathy Questionnaire clinical summary score (KCCQ-CSS), and adverse event (AE) data were also collected.

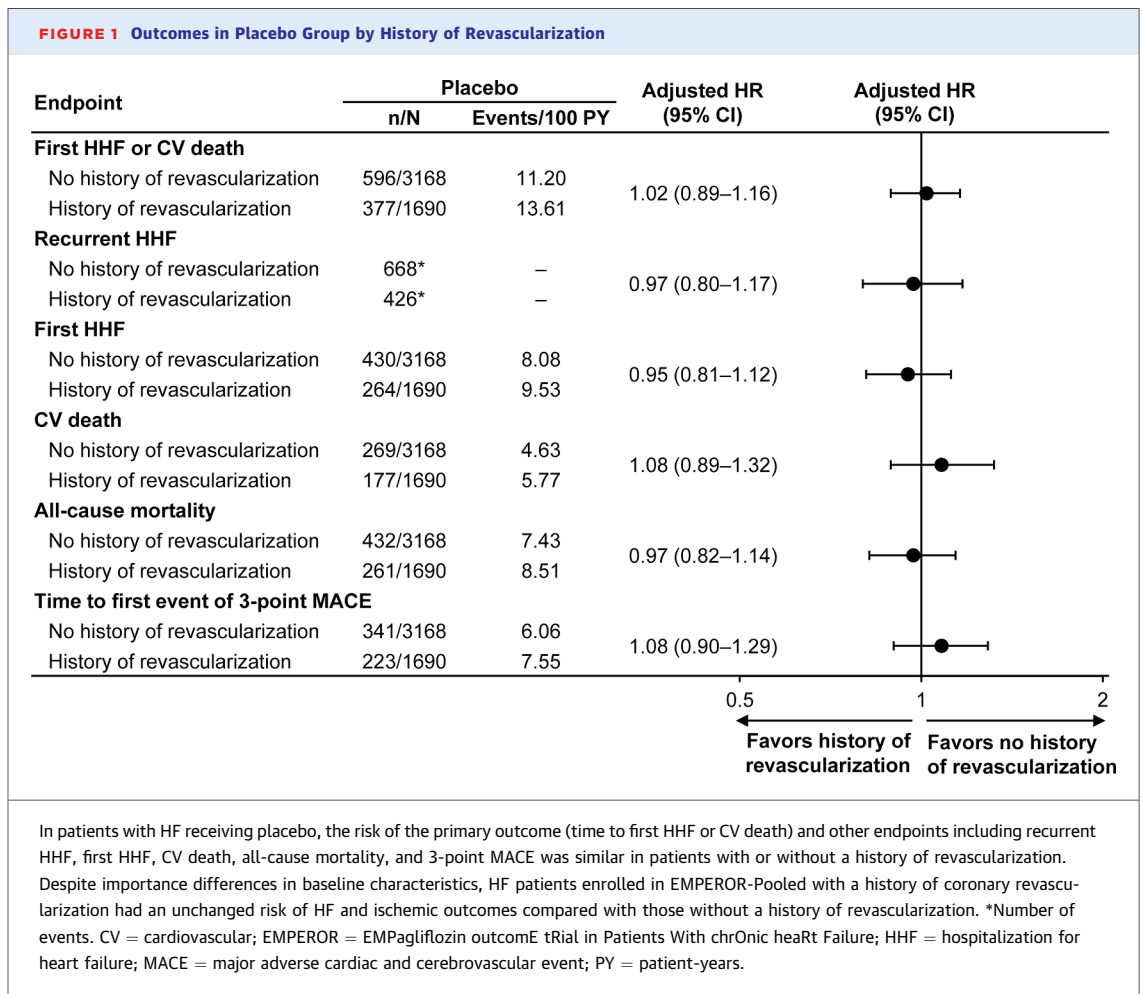
STATISTICAL ANALYSIS. As a prerequisite for pooling, the consistency of the treatment effects by REVASC status has been shown for the efficacy endpoints across EMPEROR-Reduced and EMPEROR-Preserved. A Cox proportional hazards model, adjusted for the prespecified covariates age, sex, region (North America, Latin America, Europe, Asia, and other), diabetes status (diabetes, prediabetes, no diabetes), eGFR at baseline, LVEF at baseline, and study for the pooled analysis, was employed for the time-to-first event analyses. These analyses were conducted using the intention-to-treat principle for all randomized patients and included data till the end of the planned treatment period. Adjusted HRs and event rates per 100 patient-years are reported. Total (first and recurrent) hospitalization for heart failure (HHF) were evaluated accordingly with a joint frailty model that accounted for CV death, adjusted for the same covariates as the Cox model. Continuous endpoints were analyzed with the same covariates as the Cox model, along with visit by treatment by REVASC status interaction and baseline value by visit interaction in a mixed model with repeated measures. The conventional Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation, which utilizes serum creatinine and corrects for age, sex, and race, was used to derive the eGFR values presented. Subgroup-by-treatment interaction terms were added to the models to evaluate the consistency of the treatment effects across REVASC and no REVASC subgroups. AEs occurring during the on-treatment period (including 7 days after last drug consumption) were analyzed descriptively.

RESULTS

A total of 9,718 patients were included in the present analysis, 3,437/9,718 (35.4%) of which had a history of revascularization, and 6,281/9,718 (64.6%) of which did not. Of the patients with a history of revascularization, 1,523/3,437 (44.3%) were enrolled in EMPEROR-Reduced, while 1,914/3,437 (55.7%) were enrolled in EMPEROR-Preserved. Of those without a history of revascularization, 2,207/6,281 (35.1%) were enrolled in EMPEROR-Reduced, and 4,074/6,281 (64.9%) were enrolled in EMPEROR-Preserved. The median follow-up time for the Pooled population was 21.23 months (IQR: 14.93-28.97 months).

In EMPEROR-Pooled, patients with a history of coronary revascularization were more likely to be men (2,599/3,437 [75.6%] vs 3,550/6,281 [56.5%]), White (2,658/3,437 [77.3%] vs 4,513/6,281 [71.9%]), and marginally older (70.8 ± 9.0 vs 69.5 ± 11.0 years). As expected, REVASC patients also more commonly had a documented history of coronary artery disease (CAD), smoking, hypertension, hypercholesterolemia, diabetes, and rates of acute coronary syndrome in the 6 months prior to enrollment, along with higher baseline hs-troponin levels. With respect to HF characteristics, REVASC patients had a longer time since HF diagnosis (median [IQR]: 3.7 [1.3-7.8] vs 2.8 [1.0-6.5] years), lower LVEF at baseline ($41.5\% \pm 14.4\%$ vs $45.4\% \pm 15.5\%$), and lower N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels (median 1,206.0 vs 1,334.0 pg/mL), with similar rates of HHF in the previous 12 months. Of interest, while 2,817/3,437 (82.0%) of revascularized patients had an ischemic etiology for HF, there was also nearly 20% (1,229/6,281 [19.6%]) of the nonrevascularized group that also had an ischemic source of their HF syndrome. REVASC patients also had a lower baseline eGFR (58.9 ± 19.7 vs 62.4 ± 20.9 mL/min/1.73 m²), as well as lower rates of atrial fibrillation or flutter (1,362/3,437 [39.6%] vs 3,214/6,281 [51.2%]). Baseline KCCQ-CSS scores (71.3 ± 21.3 vs 70.1 ± 21.5) and rates of euolemia in the preceding 4 weeks (2,043/3,437 [59.4%] vs 3,455/6,281 [55.0%]) were higher among REVASC patients, although NYHA functional class distributions were similar regardless of revascularization history (Table 1). Finally, despite similar rates of background triple therapy (1,410/3,437 [41.0%] vs 2,522/6,281 [40.2%]), patients with a history of revascularization had higher rates of treatment with implantable cardioverter-defibrillator or cardiac resynchronization therapy-defibrillator (746/3,437 [21.7%] vs 657/6,281 [10.5%]) (Supplemental Table 1).

Patients with HF and a history of revascularization were not at an elevated risk of clinical outcomes compared to those without a history of revascularization in EMPEROR-Pooled. Indeed, among patients randomized to placebo, similar risks between subgroups were documented for the primary outcome (REVASC vs no REVASC, HR [95% CI]: 1.02 [0.89-1.16], $P = 0.8101$), total HHF (0.97 [0.80-1.17], $P = 0.7509$), and other secondary endpoints. Similarly, time to first event of the 3-point MACE was not significantly different between REVASC and no REVASC patients randomized to placebo (HR: 1.08 [0.90-1.29], $P = 0.4114$) (Figure 1).

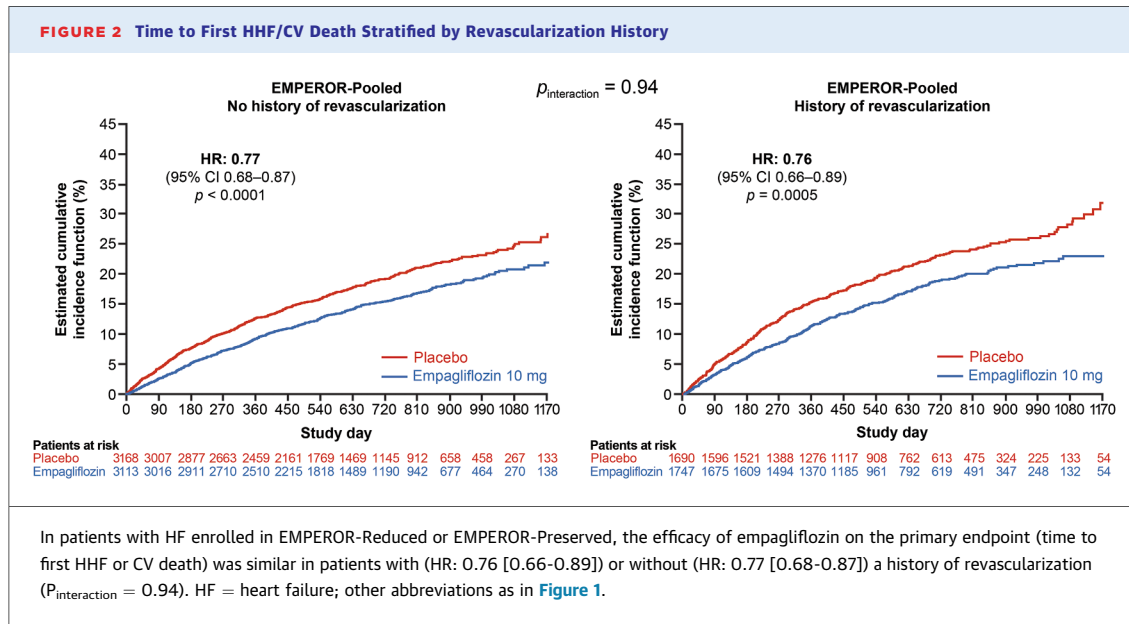


There was an entirely consistent efficacy of empagliflozin in patients with HF irrespective of revascularization history. Indeed, in EMPEROR-Pooled, REVASC and no REVASC patients derived a similar benefit with randomization to empagliflozin with respect to the primary outcome (REVASC: 0.76 [0.66–0.89], no REVASC: 0.77 [0.68–0.87], $P_{\text{interaction}} = 0.9353$) (Figure 2). This efficacy was maintained in both the EMPEROR-Reduced and EMPEROR-Preserved populations, respectively (Supplemental Figure 1).

Similarly, no heterogeneity in the efficacy of empagliflozin on secondary outcomes was introduced by revascularization history in EMPEROR-Pooled including for: total HHF (REVASC: 0.82 [0.66–1.01], no REVASC: 0.66 [0.56–0.78], $P_{\text{interaction}} = 0.1303$), time to CV death (REVASC: 0.90 [0.73–1.12], no REVASC: 0.92 [0.77–1.09], $P_{\text{interaction}} = 0.9091$), time to first HHF (REVASC: 0.74 [0.62–0.89], no REVASC: 0.67 [0.58–0.78], $P_{\text{interaction}} = 0.4438$) as

well as time to all-cause mortality, time to the composite renal endpoint, and time to the extended composite outcome (Figure 3). Empagliflozin had a neutral effect on time to the 3-point MACE in both the REVASC group (HR: 0.95 [0.79–1.15]) and no REVASC group (HR: 0.98 [0.84–1.14]) ($P_{\text{interaction}} = 0.8136$). Data on the efficacy of empagliflozin on HF hospitalizations in REVASC vs no REVASC patients in the EMPEROR-Pooled, -Reduced, and -Preserved populations are presented in Supplemental Figures 2 and 3.

The treatment effect was consistent for REVASC and no REVASC patients as it pertains to slope of eGFR change with randomization to empagliflozin in EMPEROR-Pooled (REVASC: 1.582 [1.122–2.042], no REVASC: 1.384 [1.043–1.726], P for heterogeneity = 0.4991). Patients without a history of revascularization did have a larger improvement in KCCQ-CSS scores at 52 weeks with randomization to empagliflozin compared with REVASC patients in EMPEROR-



Pooled (no REVASC, comparison vs placebo adjusted mean: 2.13 [1.25–3.00], REVASC: 0.54 [–0.63 to 1.72], visit by treatment by revascularization history $P = 0.0350$) (Figure 4). This finding was driven predominantly by subgroup differences seen in the EMPEROR-Reduced population at 52 weeks (visit by treatment by history of revascularization $P = 0.0554$) rather than the EMPEROR-Preserved population (visit by treatment by history of revascularization $P = 0.2567$).

Placebo rates of serious AEs were numerically higher among REVASC patients compared with no REVASC patients in EMPEROR-Pooled; however, the safety profile of empagliflozin was unchanged irrespective of revascularization history (any AEs: [REVASC: empagliflozin: 1,441/1747 (82.5%), placebo: 1,415/1,688 (83.8%); no REVASC: empagliflozin: 2,553/3,112 (82.0%), placebo: 2,633/3,164 (83.2%)], serious AEs: [REVASC: empagliflozin: 846/1747 (48.4%), placebo: 916/1,688 (54.3%); no REVASC: empagliflozin: 1,362/3,112 (43.8%), placebo: 1,523/3,164 (48.1%)]) (Supplemental Table 2).

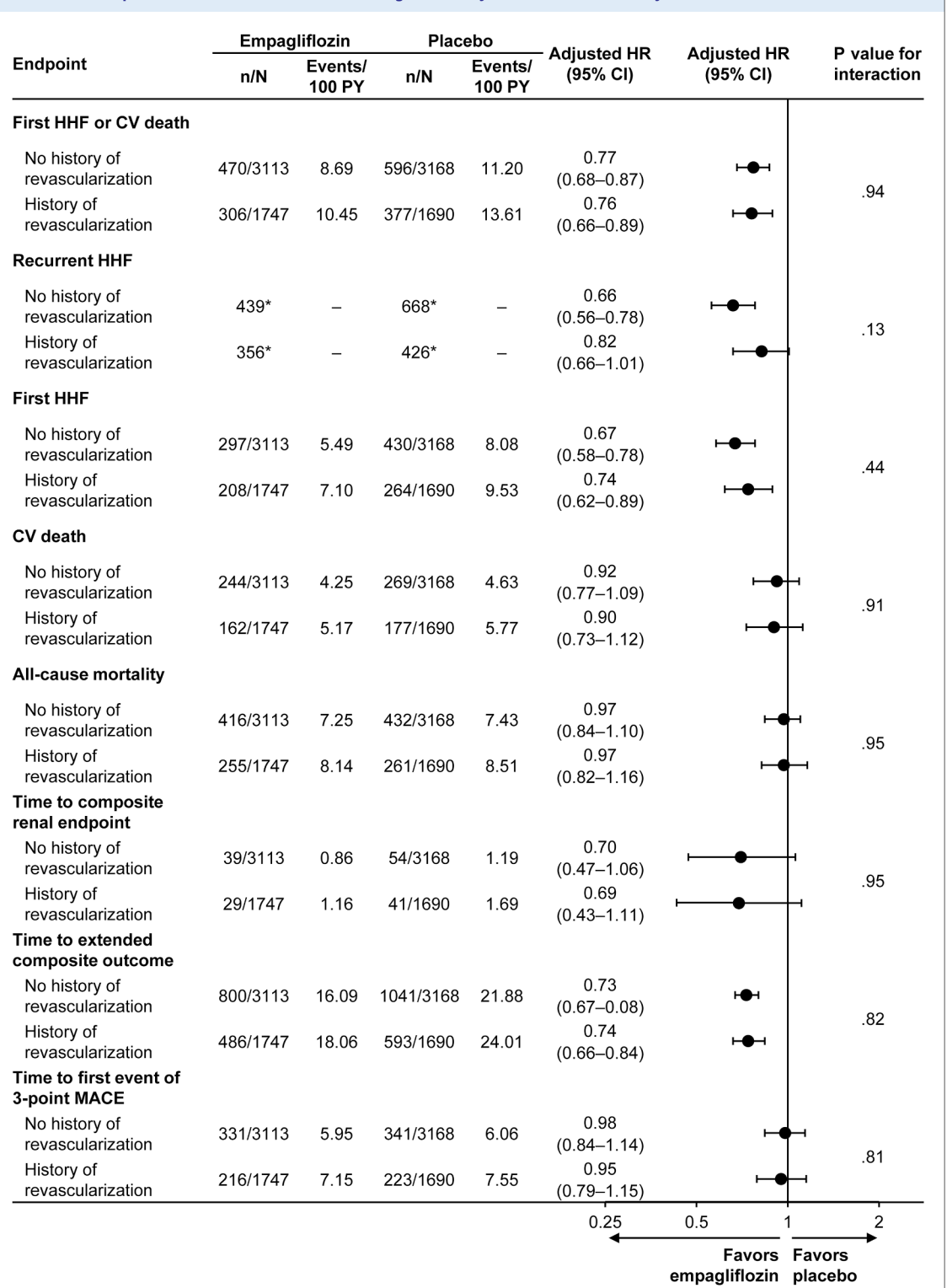
DISCUSSION

This subgroup analysis of the EMPEROR-Program had 3 central observations. First, despite important differences in baseline characteristics, HF patients enrolled in EMPEROR-Pooled with a history of coronary revascularization had an unchanged risk of HF and ischemic outcomes compared with those without a history of revascularization. Second, the efficacy of

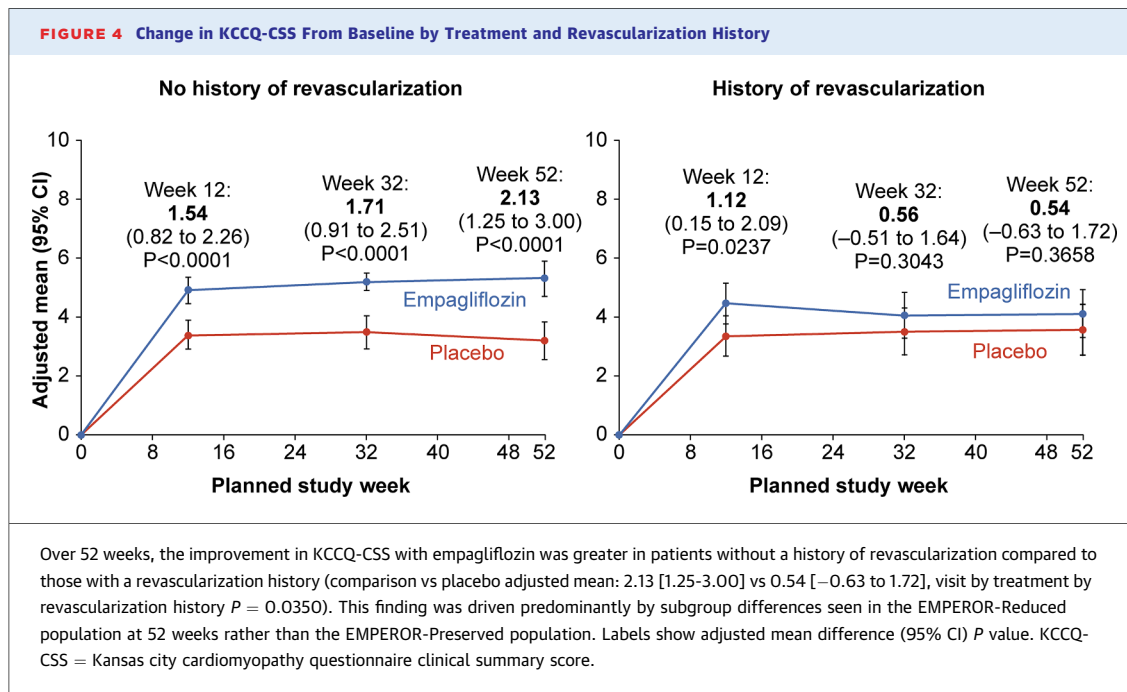
empagliflozin in HF was entirely consistent irrespective of revascularization status. Third, empagliflozin had a neutral effect on a 3-point MACE of CV death, nonfatal MI, or nonfatal stroke irrespective of revascularization status.

The only significant heterogeneity introduced by revascularization status on the efficacy of empagliflozin pertained to quality of life scores. In particular, patients with no revascularization history had a larger improvement in KCCQ-CSS than their revascularized counterparts, which was driven by differences in patients with heart failure with a reduced ejection fraction (HFrEF). This finding could be a result of baseline differences, including a higher baseline KCCQ-CSS among revascularized patients, which potentially left less room for improvement.

The present findings reaffirm that sodium-glucose cotransporter 2 inhibitors (SGLT2is) should be considered as an important part of the optimal medical therapy in patients with evidence of clinical HF and a history of coronary revascularization. Recent advancements in medical therapy for HF, including the approval of SGLT2is for adults with HF, have initiated a paradigm shift in the management of ischemic cardiomyopathy. While a Class I recommendation for CABG still exists for surgical candidates with multivessel CAD in the setting of LV dysfunction (EF <35%),² predominantly based on findings from the STICHES (Surgical Treatment for Ischemic Heart Failure Extension Study STICHES) trial which showed a significant survival advantage

FIGURE 3 Endpoint Data in EMPEROR-Pooled According to Coronary Revascularization History

In patients with HF enrolled in EMPEROR-Reduced or EMPEROR-Preserved, the efficacy of empagliflozin was similar in patients with or without a history of revascularization, for the primary and secondary endpoints. *Number of events. Abbreviations as in [Figure 1](#).



of CABG over medical therapy at 10 years for such patients,¹³ the role of revascularization with PCI for ischemic cardiomyopathy has been less clear. Indeed, the REVIVED (Revascularization for Ischemic Ventricular Dysfunction) trial, which randomized 700 patients with CAD, LVEF <35%, and myocardial viability to either PCI or optimal medical therapy, yielded entirely neutral results for the primary outcome of all-cause mortality or hospitalization for HF (HR: 0.99 [0.78-1.27]; $P = 0.96$) over a median of 41 months.¹⁴ Advancements in medical therapy have more broadly revolutionized the management of stable IHD altogether, with findings from the ISCHEMIA (International Study of Comparative Health Effectiveness with Medical and Invasive Approaches) trial suggesting a more conservative approach to CAD may be as efficacious as invasive strategies.¹⁵

The present investigation documented a neutral effect of empagliflozin on a 3-point MACE of CV death, nonfatal MI, or nonfatal stroke. The culmination of these findings with EMPACT-MI, which reported no benefit of empagliflozin on the primary outcome of first HHF or all-cause mortality in patients with acute MI and either newly reduced LVEF or clinical congestion,⁷ suggest that the efficacy of SGLT2is in HF is mediated through pathways that are independent of ischemic events. The precise mechanisms through which SGLT2is exert their cardioprotective effects in HF are the subject of much

ongoing research and investigation. Prevailing theories regarding its pleiotropic effects include: 1) enhanced erythropoiesis through induction of renal hypoxia leading to increased hematocrit and therefore improved myocardial oxygen delivery, cardiomyocyte mitochondrial function, as well as angiogenesis and cell proliferation;¹⁶⁻¹⁹ 2) activation of nutrient deprivation signaling leading to autophagic clearance of damaged organelles and thus decreased cellular stress and inflammation;²⁰⁻²⁴ and 3) modulation of ventricular loading conditions with subsequent cardiac reverse remodeling via osmotic diuresis (particularly in the short term).²⁵⁻³² These mechanisms are broadly applicable to patients with the syndrome of HF but may not directly modify the occurrence of CV events from atherosclerosis. Importantly, our findings also align with previous analysis from the EMPEROR program, which have shown consistent efficacy of empagliflozin in HFrEF patients with ischemic vs nonischemic causes.³³

STUDY LIMITATIONS. The present findings should be interpreted in the context of important limitations. First, this investigation was a post hoc subgroup analysis with no correction for multiplicity—its findings should be considered exploratory and hypothesis-generating. Second, outcomes in patients that particularly received surgical vs percutaneous revascularization could not be assessed given that such granularity was not collected in the index trials.

Similarly, rates of complete vs incomplete revascularization, and time since revascularization procedure, were not captured. Finally, comparisons made herein between REVASC and no REVASC groups are limited given these were nonrandomized and nonpropensity-matched analyses. Future research including prospective registries or trials should aim to fill these knowledge gaps in order to better clarify whether there are specific postrevascularization clinical scenarios in which empagliflozin may be efficacious.

In summary, despite IHD remaining an important risk factor for the incidence of clinical HF, patients in EMPEROR-Pooled with a history of coronary revascularization were not at an elevated risk of clinical HF or ischemic outcomes. Empagliflozin demonstrated a consistent efficacy among HF patients in EMPEROR-Pooled irrespective of coronary revascularization history.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: In this post hoc analysis of the EMPEROR-Pooled trials, among HF patients randomized to placebo, those who had undergone coronary revascularization showed similar rates of HF and ischemic events as those without prior revascularization. Empagliflozin demonstrated a consistent efficacy in HF patients irrespective of revascularization history.

TRANSLATIONAL OUTLOOK: In the EMPEROR Program, patients with a history of coronary revascularization were not at an elevated risk of clinical HF or ischemic outcomes compared to those without prior revascularization. Based on the findings of this exploratory post hoc analysis, empagliflozin should be considered a foundational therapy for patients with HF irrespective of revascularization history.

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APPENDIX For supplemental tables and figures, please see the online version of this paper.