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Research Article:

Cardiovascular Outcomes of Glp-1 Receptor Agonists and Sglt2 Inhibitors in Patients with Coronary Artery Disease: A Study with Emphasis on Overweight and Obesity

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ABSTRACT:
Coronary artery disease often coexists with metabolic disorders, including overweight, obesity, and diabetes mellitus. This study describes cardiovascular outcomes among patients receiving glucagon-like peptide-1 receptor agonists (GLP-1 RAs), sodium–glucose cotransporter-2 inhibitors (SGLT2 inhibitors), or conventional treatment, with particular attention to the burden of overweight and obesity. The clinical summary comprised 147 patients divided into GLP-1 RA (n=40), SGLT2 inhibitor (n=47), and conventional-treatment (n=60) groups, each with a reported 24-month follow-up. In the total cohort, 49 patients were overweight and 78 had obesity, together representing 86.4% of participants. Myocardial infarction occurred in 2, 3, and 5 patients, respectively; stroke in 1, 2, and 3; cardiovascular death in 1, 2, and 4; and hospitalization for heart failure in 2, 4, and 7. Diabetes mellitus was present in 40, 47, and 30 patients, respectively. Observed event proportions were numerically lower in the GLP-1 RA group than in the other groups, while unadjusted group comparisons did not establish statistically significant differences. The findings describe the observed cardiovascular event patterns in a clinical cohort with a substantial burden of excess body weight.
Keyword: Coronary Artery Disease; Overweight; Obesity; GLP-1 Receptor Agonists; SGLT2 Inhibitors; Cardiovascular Outcomes; Diabetes Mellitus.

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INTRODUCTION:

Coronary artery disease (CAD) remains a leading cause of cardiovascular morbidity and mortality worldwide. Despite advances in coronary revascularization, lipid-lowering therapy, anti-platelet treatment, and blood pressure management, patients with established coronary atherosclerosis continue to experience substantial residual cardiovascular risk. This risk is particularly pronounced among individuals with overweight or obesity, who often present with additional metabolic abnormalities, including insulin resistance, type 2 diabetes mellitus, hypertension, and dyslipidemia. Coronary artery disease develops through a complex interaction of lipid accumulation, endothelial dysfunction, vascular inflammation, oxidative stress, and thrombosis. Progressive deposition of atherogenic lipoproteins within coronary arterial walls promotes inflammatory cell recruitment and atherosclerotic plaque formation. Plaque rupture or erosion may subsequently trigger coronary thrombosis, resulting in myocardial ischemia or acute myocardial infarction.

Obesity contributes significantly to the development and progression of coronary atherosclerosis. Excess adipose tissue, particularly visceral fat, acts as an active endocrine organ that produces inflammatory mediators and influences glucose and lipid metabolism. Increased concentrations of interleukin-6, tumor necrosis factor-alpha, and other pro-inflammatory substances promote endothelial dysfunction and chronic vascular inflammation. Obesity is also associated with increased circulating blood volume, elevated cardiac output, and greater myocardial workload. These changes may contribute to left ventricular hypertrophy, impaired ventricular relaxation, and the development of heart failure. Consequently, excess body weight influences

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both atherosclerotic cardiovascular disease and myocardial functional abnormalities. [2,10]

Traditional cardiovascular risk reduction focuses on anti-platelet therapy, lipid-lowering medications, antihypertensive treatment, smoking cessation, dietary modification, and physical activity. Although these interventions remain fundamental, many patients continue to experience cardiovascular events despite receiving guideline-directed medical therapy. This persistent risk has encouraged investigation into additional pharmacological strategies targeting cardiometabolic dysfunction. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) and sodium-glucose cotransporter-2 (SGLT2) inhibitors represent two important therapeutic classes that have demonstrated cardiovascular benefits beyond their original role in diabetes management. [2,10]

GLP-1 receptor agonists reproduce the physiological actions of glucagon-like peptide-1, an incretin hormone involved in glucose regulation and appetite control. These medications stimulate glucose-dependent insulin secretion, suppress inappropriate glucagon secretion, delay gastric emptying, and increase satiety. Selected GLP-1 receptor agonists, particularly semaglutide, produce substantial reductions in body weight. Their cardiovascular benefits may also involve improvements in blood pressure, lipid metabolism, vascular inflammation, and endothelial function. The SELECT trial demonstrated that semaglutide significantly reduced major adverse cardiovascular events among patients with established cardiovascular disease and overweight or obesity without diabetes [1]. This finding provided important evidence that cardiovascular benefits associated with semaglutide are not restricted to glucose-lowering effects.

SGLT2 inhibitors act primarily in the proximal renal tubules by inhibiting sodium-glucose cotransport. This mechanism increases urinary glucose excretion and promotes natriuresis and osmotic diuresis. These physiological effects may reduce cardiac filling pressures, improve renal hemodynamics, and decrease congestion in patients with heart failure. Major clinical trials have demonstrated that SGLT2 inhibitors reduce hospitalization for heart failure and slow kidney disease progression, including among patients without diabetes. [5,6,11]

The cardiovascular effects of GLP-1 receptor agonists and SGLT2 inhibitors are therefore complementary but not identical. GLP-1 receptor agonists demonstrate particularly important benefits in obesity management and reduction of atherosclerotic cardiovascular events, whereas SGLT2 inhibitors have strong evidence supporting their role in heart failure and renal protection. The 2024 European Society of Cardiology guidelines for chronic coronary syndromes emphasize comprehensive cardiovascular risk reduction and the importance of appropriate cardiometabolic therapies in selected patients [2].

Nevertheless, comparative clinical evaluation remains important because patients with coronary artery disease often present with several overlapping cardiovascular and metabolic conditions. For example, patients with obesity and poorly controlled cardiometabolic risk factors may require treatment strategies different from those used in patients with coronary artery disease complicated by symptomatic heart failure or chronic kidney disease. Furthermore, the effectiveness of treatment in routine clinical practice may be influenced by medication adherence, baseline disease severity, comorbidities, and access to pharmacological therapy.

The study places particular emphasis on excess body weight because adiposity influences both the prevalence of cardiometabolic risk factors and the clinical priorities of secondary prevention. Body mass index provides a convenient classification of weight status, while waist circumference offers complementary information about abdominal adiposity. Neither measure, taken alone, completely describes metabolic health. In a population with established coronary disease, the coexistence of hypertension, dyslipidemia, smoking, impaired fasting glucose, and insulin resistance can identify overlapping therapeutic needs. For that reason, the evaluation of drug classes with different physiological targets is relevant to routine cardiovascular care. The present investigation examines the clinical characteristics and recorded cardiovascular outcomes of patients receiving GLP-1 receptor agonists, SGLT2 inhibitors, or conventional treatment in a single clinical setting. [4,7,12]

Cardiovascular outcome research must also distinguish the endpoints being studied. Myocardial infarction reflects an acute ischemic event, stroke represents cerebrovascular injury, and cardiovascular death is a mortality outcome. Hospitalization for heart failure reflects clinical deterioration requiring inpatient management and is often evaluated separately in cardiometabolic trials. These outcomes are related but not interchangeable. Reporting their individual frequencies makes it possible to describe the observed

clinical experience without substituting one endpoint for another. The published trials discussed below provide a broader evidence base for the therapeutic classes, whereas the hospital cohort contributes a description of the patients and events recorded in local clinical practice.

MATERIALS AND METHODS:

This study was conducted through clinical observation and review of corresponding clinical and laboratory records of 147 patients with coronary artery disease at the Samarkand Branch of the Republican Scientific Center of Emergency Medical Care, Samarkand, Uzbekistan. The research was carried out under faculty supervision. Patients were classified into three treatment groups: GLP-1 receptor agonists (n = 40), SGLT2 inhibitors (n = 47), and conventional treatment (n = 60). Cardiovascular outcomes were evaluated over a 24-month follow-up period. The exact enrollment dates and detailed eligibility criteria are not specified in the current study report.

The available baseline variables included age, sex, BMI, overweight, obesity, hypertension, dyslipidemia, current smoking, diabetes, heart failure and chronic kidney disease. The outcomes were myocardial infarction, stroke, cardiovascular death and hospitalization for heart failure.

Absolute frequencies and percentages were calculated using the denominator for each treatment group. Continuous baseline variables are presented as means ± standard deviations. Each of the four cardiovascular outcomes was compared across the three groups using a two-sided Fisher–Freeman–Halton exact test for a 3 × 2 contingency table. Exact probabilities were obtained by summing the probabilities of tables with the observed margins that were no more probable than the observed table. Statistical significance was assessed at $p < 0.05$. Analyses were unadjusted; patient-level multivariable adjustment was not performed. Outcomes were evaluated separately because overlap between events in individual patients was not established.

ETHICAL APPROVAL AND DATA CONFIDENTIALITY. The study received formal institutional ethics approval. Patient confidentiality was maintained, and results are presented as de-identified, aggregate data without direct patient identifiers.

RESULTS

Table 1. Baseline characteristics by treatment group (N = 147)

Characteristic	GLP-1 RA (n=40)	SGLT2i (n=47)	Conventional (n=60)
Mean age, years	58.4 ± 8.2	59.1 ± 7.9	60.2 ± 8.5
Male, n (%)	24 (60.0%)	29 (61.7%)	36 (60.0%)
Female, n (%)	16 (40.0%)	18 (38.3%)	24 (40.0%)
Mean BMI, kg/m²	32.1 ± 4.1	31.4 ± 3.8	29.7 ± 3.6
Overweight, n (%)	12 (30.0%)	19 (40.4%)	18 (30.0%)
Obesity, n (%)	28 (70.0%)	28 (59.6%)	22 (36.7%)
Hypertension, n (%)	34 (85.0%)	39 (83.0%)	48 (80.0%)
Dyslipidemia, n (%)	30 (75.0%)	35 (74.5%)	42 (70.0%)
Current smoking, n (%)	8 (20.0%)	10 (21.3%)	13 (21.7%)
Diabetes mellitus, n (%)	40 (100%)	47 (100%)	30 (50.0%)
Heart failure, n (%)	8 (20.0%)	12 (25.5%)	15 (25.0%)
Chronic kidney disease, n (%)	6 (15.0%)	10 (21.3%)	12 (20.0%)

The confirmed baseline characteristics are presented separately by treatment group in Table 1. Diabetes mellitus affected 40/40 (100%) patients receiving GLP-1 receptor agonists, 47/47 (100%) receiving SGLT2 inhibitors, and 30/60 (50%) receiving conventional treatment. Heart failure was present in 8/40 (20.0%), 12/47 (25.5%), and 15/60 (25.0%), respectively; chronic kidney disease in 6/40 (15.0%), 10/47 (21.3%), and 12/60 (20.0%). The diabetes imbalance and differences in body mass index and obesity substantially limit direct comparisons of unadjusted outcomes.

During the reported 24-month follow-up, myocardial infarction was recorded in 2/40 (5.0%) patients in the

GLP-1 receptor agonist group, 3/47 (6.4%) in the SGLT2 inhibitor group, and 5/60 (8.3%) in the conventional-treatment group. Stroke was recorded in 1/40 (2.5%), 2/47 (4.3%), and 3/60 (5.0%), respectively. Cardiovascular death occurred in 1/40 (2.5%), 2/47 (4.3%), and 4/60 (6.7%), respectively. Hospitalization for heart failure was reported in 2/40 (5.0%), 4/47 (8.5%), and 7/60 (11.7%), respectively.

Table 2. Cardiovascular outcomes during 24-month follow-up

Outcome	**GLP-1 RA (n=40)**	**SGLT2i (n=47)**	**Conventional (n=60)**	Exact p
Myocardial infarction	2 (5.0%)	3 (6.4%)	5 (8.3%)	0.918
Stroke	1 (2.5%)	2 (4.3%)	3 (5.0%)	0.882
Cardiovascular death	1 (2.5%)	2 (4.3%)	4 (6.7%)	0.712
HF hospitalization	2 (5.0%)	4 (8.5%)	7 (11.7%)	0.540

Event frequencies were numerically lowest in the GLP-1 receptor agonist group for all four outcomes, but two-sided Fisher–Freeman–Halton exact tests found no statistically significant differences across groups (myocardial infarction, p = 0.918; stroke, p = 0.882; cardiovascular death, p = 0.712; heart failure hospitalization, p = 0.540). These unadjusted comparisons do not establish a treatment effect, particularly because baseline diabetes prevalence differed markedly across groups.

DISCUSSION:

The principal finding is descriptive: among 147 patients with coronary artery disease, event proportions were numerically different across treatment categories during 24 months, but none of the four prespecified three-group comparisons was statistically significant. Myocardial infarction occurred in 5.0%, 6.4%, and 8.3% of the GLP-1 RA, SGLT2 inhibitor, and conventional-treatment groups; stroke in 2.5%, 4.3%, and 5.0%; cardiovascular death in 2.5%, 4.3%, and 6.7%; and heart-failure hospitalization in 5.0%, 8.5%, and 11.7%. The corresponding exact p-values were 0.918, 0.882, 0.712, and 0.540. None provides evidence of a statistically significant difference at the 0.05 threshold. Small event counts also mean that clinically relevant differences cannot be ruled out merely because a test is nonsignificant.

The most important limitation of treatment-group comparability is the confirmed diabetes distribution. Diabetes was recorded in every patient receiving GLP-1 RA therapy (40/40) and SGLT2 inhibitors (47/47), but in only half of patients receiving conventional treatment (30/60). Diabetes influences baseline cardiovascular risk and may influence clinicians' medication choices. This marked imbalance is compatible with selection by clinical indication, although the actual prescribing and selection criteria were not supplied and therefore cannot be asserted. The treatment categories are not exchangeable groups. A comparison of their crude event proportions cannot separate the effects of therapy from the effects of diabetes status, associated metabolic disease, or differences in patient selection.

The corrected baseline data identify additional imbalances. Mean BMI was 32.1 ± 4.1 kg/m² with GLP-1 RA therapy, 31.4 ± 3.8 kg/m² with SGLT2 inhibitors, and 29.7 ± 3.6 kg/m² with conventional treatment. Obesity was present in 70.0%, 59.6%, and 36.7% of the respective groups. Thus, patients receiving the active drug classes carried a higher recorded obesity burden despite having numerically lower event proportions. These findings are clinically interesting but do not imply that either drug class counteracted that baseline risk: the summaries do not link individual BMI, diabetes status, treatment exposure, and outcomes. Age also differed modestly between groups (58.4, 59.1, and 60.2 years), while sex distributions were broadly similar.

Hypertension affected 85.0%, 83.0%, and 80.0% of patients; dyslipidemia 75.0%, 74.5%, and 70.0%; and current smoking 20.0%, 21.3%, and 21.7% in the three groups. Heart failure and chronic kidney disease were also reported in each group. These characteristics can affect both clinical treatment selection and subsequent outcomes. Although the differences in several of these proportions are smaller than the diabetes and obesity differences, the combined baseline profiles reinforce that the groups were not formed by random assignment. Because only group summaries were available for this analysis, conventional covariate adjustment, propensity-score matching, and stratified event comparisons by

diabetes or obesity status could not be carried out reliably.

The results should be distinguished from published randomized evidence. The SELECT trial reported a reduction in major cardiovascular events with semaglutide in a selected population with established cardiovascular disease and overweight or obesity without diabetes [1]. Our GLP-1 RA group, in contrast, consisted entirely of patients with diabetes, and our study did not use the same randomized design or necessarily the same endpoint definition. Therefore, the SELECT treatment effect cannot be applied directly to our crude event counts. Similarly, trials of SGLT2 inhibitors such as EMPEROR-Preserved and DELIVER investigated heart-failure populations under defined protocols [5,6]. Their results support clinical use of specific agents in eligible patients but do not establish comparative superiority of one class over another within our three nonrandomized groups.

Interpretation also depends on the outcome definitions. Myocardial infarction, stroke, cardiovascular death, and hospitalization for heart failure were recorded as separate counts. These outcomes reflect different biological and clinical processes, and an individual patient may potentially contribute to more than one category. Without event-level records and confirmation of overlap, summing these counts would overestimate the number of distinct affected patients. The summary does not document event adjudication, competing events, exposure duration, adherence, dosage, changes in medication during follow-up, or whether both drug classes were used sequentially or together. Accordingly, the report does not claim a validated composite major adverse cardiovascular event rate or estimate treatment-specific hazard ratios.

The baseline characteristics and cardiovascular outcomes reported in this study were established through clinical research and examination of the relevant hospital records at the Samarkand Branch of the Republican Scientific Center of Emergency Medical Care. Data for all 147 patients are traceable to the original hospital records and can be independently verified. The reported group totals, percentages, and statistical comparisons are based on the documented study findings. The present analysis uses group-level results; detailed enrollment criteria, treatment-exposure histories, and patient-level adjustment were not included in the statistical comparisons.

Taken together, the study describes a cohort with a substantial burden of excess body weight (49 overweight and 78 obese patients, 86.4% in total), unequal diabetes prevalence across treatment groups, and low observed numbers of individual cardiovascular outcomes. The observed direction of the event proportions may motivate future investigation but cannot establish that GLP-1 receptor agonists are more effective than SGLT2 inhibitors or conventional treatment. A stronger comparative study would require defined treatment-selection criteria, harmonized outcome definitions, patient-level baseline and event data, documentation of treatment exposure, and an appropriately adjusted or randomized design. Until then, the appropriate interpretation is an exploratory description of clinical experience rather than a comparative effectiveness claim.

CONCLUSION:

Among 147 patients with coronary artery disease followed for 24 months, myocardial infarction, stroke, cardiovascular death, and hospitalization for heart failure were recorded in all three treatment groups. Event proportions were numerically lowest in the GLP-1 receptor agonist group, but the differences across groups were not statistically significant ($p = 0.918$, 0.882, 0.712, and 0.540, respectively). Diabetes prevalence differed substantially between groups (100%, 100%, and 50%), as did BMI and obesity prevalence. These baseline differences must be considered when interpreting the observed outcomes. The study describes cardiovascular outcomes in patients with coronary artery disease and a high prevalence of excess body weight and supports further investigation using appropriately comparable treatment groups.

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