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REVIEW ARTICLE

Cardiovascular effects of GLP-1 receptor agonists in patients with type 2 diabetes mellitus

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ABSTRACT

Introduction: type 2 diabetes mellitus is associated with high cardiovascular morbidity and mortality, which drives the search for therapies that go beyond traditional glycemic control.
Objective: to evaluate the scientific evidence on the cardiovascular effects of glucagon-like peptide-1 receptor agonists in patients with type 2 diabetes mellitus.
Methods: a systematic review of the scientific literature was conducted across multiple databases. The search was performed using an algorithm with keywords and Boolean operators to identify relevant sources. After applying predefined inclusion and exclusion criteria, the selected studies were critically analyzed in terms of currency, methodological quality, and thematic relevance, and subsequently integrated into the final synthesis of the review.
Development: the findings show that glucagon-like peptide-1 receptor agonists reduce major adverse cardiovascular events, cardiovascular mortality, and hospitalizations for heart failure. Liraglutide and semaglutide stand out for their significant benefits, while other agents exhibit variable effects. Pleiotropic mechanisms were identified, including improvement of endothelial function, reduction of oxidative stress, and protection of cardiomyocytes. However, efficacy varies according to the specific drug and the population studied, highlighting the need for individualized therapy.
Conclusions: glucagon-like peptide-1 receptor agonists represent a promising strategy for patients with type 2 diabetes and high cardiovascular risk. Although some agents demonstrate consistent benefits, the heterogeneity of outcomes underscores the need for further comparative studies and a personalized approach that integrates metabolic control with cardiovascular protection.

Keywords: Glucagon-Like Peptide-1 Receptor Agonists; Diabetes Mellitus, Type 2; Cardiovascular Diseases; Cardiovascular Health.

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INTRODUCTION

Diabetes is a globally prevalent disease affecting millions of people, with significant consequences for health and well-being. Recent data indicate that approximately 8,5 % of the world's adult population lives with diabetes. In the Americas, an estimated 62 million people have diabetes, with a marked concentration in low- and middle-income countries. Additionally, diabetes contributes to a considerable number of deaths annually, with over 244,000 cases reported in the Americas alone.[1]

Diabetes mellitus (DM) encompasses a range of metabolic disorders resulting in chronic hyperglycemia and dysfunction in carbohydrate, fat, and protein metabolism. These disturbances arise from defects in insulin secretion, insulin action, or both. Specifically, type 2 diabetes (T2DM) is one of the most impactful chronic diseases on global quality of life and a major cause of physical disability due to multiorgan complications.[2]

Among its most common forms, T2DM involves either insulin resistance or insufficient production of this vital hormone. The alarming rise in T2DM prevalence has been observed across all income levels, reflecting a growing challenge for global health systems.[3] The primary goal in managing T2DM is to reduce hyperglycemia to prevent both acute and chronic complications, including microvascular damage (nephropathy and retinopathy) and macrovascular disease (coronary artery disease, cerebrovascular disease, and peripheral vascular disease).[4]

Individuals with T2DM face a significantly higher risk of developing severe cardiovascular diseases such as myocardial infarction (MI), heart failure (HF), peripheral arterial disease, stroke, and cardiovascular-related death. Despite intensive efforts to lower glucose levels, no conclusive reduction in cardiovascular disease incidence has been observed in diabetic patients at high cardiovascular risk.[5,6]

Multiple antidiabetic agents are available for T2DM management; however, intensive strategies focused solely on glycemic reduction have not consistently demonstrated reductions in cardiovascular morbidity and mortality in high-risk patients. For years, strict blood pressure control and LDL cholesterol reduction were considered the only effective measures to mitigate cardiovascular risk in T2DM. Nevertheless, over the past decade, several clinical trials have evaluated new hypoglycemic drugs—including sodium-glucose cotransporter-2 inhibitors (SGLT2i) and GLP-1 receptor agonists. Available evidence shows these agents achieve substantial and statistically significant reductions in major adverse cardiovascular events, as well as other relevant outcomes—including hospitalizations for heart failure. This progress has greatly expanded therapeutic options, enabling not only optimal glycemic control but also improved cardiovascular outcomes in T2DM patients.[7]

Given this context, the present study was conducted with the objective of evaluating the scientific evidence on the cardiovascular effects of GLP-1 receptor agonists in patients with type 2 diabetes mellitus.

METHODS

This study was designed as a systematic literature review, following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The search period spanned from January 2010 to December 2024 to capture the most recent and relevant evidence on the cardiovascular effects of GLP-1 receptor agonists in T2DM patients.

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Information sources included widely recognized biomedical databases: PubMed/MEDLINE, SciELO, ScienceDirect, Google Scholar, LILACS, and BVSALUD. Additionally, institutional repositories and grey literature (theses, technical reports, and scientific society documents) were consulted to complement the search and reduce publication bias. Reference lists of key articles were also reviewed to identify additional eligible studies.

The search strategy combined keywords and standardized descriptors (MeSH and DeCS) using Boolean operators to optimize information retrieval. The algorithm included terms such as: "Type 2 Diabetes Mellitus" AND "GLP-1 Receptor Agonists" AND ("Cardiovascular Events" OR "Mortality" OR "Heart Failure"). Articles published in Spanish, English, and Portuguese were considered, without geographic restrictions.

Inclusion criteria encompassed original studies (clinical trials, cohort studies, systematic reviews, and meta-analyses) published within the defined timeframe that directly addressed the relationship between GLP-1 receptor agonists and cardiovascular outcomes in T2DM patients. Excluded were duplicates, articles without full-text access, publications outside the search period, editorials, commentaries, consensus statements, and irrelevant studies.

The selection process occurred in two phases: first, title and abstract screening to exclude non-relevant records; second, full-text review of potentially eligible articles. Initially, 1,024 records were identified; after duplicate removal and application of exclusion criteria, 30 high-quality sources were selected based on methodological rigor and thematic relevance.

DEVELOPMENT

GLP-1 receptor agonists (GLP-1 RAs) are notable for their effects on glucose-dependent glucagon suppression, gastric emptying delay, reduced food intake, and glucose regulation, as well as promoting satiety through neural mechanisms. Beyond their hypoglycemic effect, GLP-1 RAs are also associated with significant body weight reduction. In addition to improving glucose metabolism, GLP-1 receptor activation exerts direct protective effects on cardiomyocytes, endothelium, and perivascular adipose tissue. These pleiotropic benefits are particularly important in managing T2DM patients with established cardiovascular disease, who still face residual cardiovascular risk despite adequate control of concurrent factors such as hypertension, dyslipidemia, and smoking cessation. [8,9,10]

GLP-1 is a peptide hormone released by intestinal L-cells after food ingestion, acting through binding to the GLP-1 receptor (GLP-1R). Its effects include glucose-dependent stimulation of pancreatic beta-cell insulin secretion, inhibition of glucagon release from alpha cells, and slowing of gastric emptying. These combined actions lower postprandial blood glucose and improve overall glucose metabolism. Furthermore, by activating hypothalamic neurons expressing GLP-1R, GLP-1 promotes satiety and may aid in weight loss.[11]

GLP-1 is generated from proglucagon cleavage by the enzyme PC1/3, yielding GLP-1(7-36) and GLP-1(9-36). However, GLP-1 has a very short half-life—only a few minutes—due to rapid degradation by dipeptidyl peptidase-4 (DPP-4), an enzyme present in many cell types. To harness GLP-1's potent incretin effects, two therapeutic strategies have emerged: DPP-4 inhibitors (e.g., sitagliptin, vildagliptin, saxagliptin, linagliptin), which typically reduce HbA1c by 0,5–0,8 %; and human GLP-1-based analogs (e.g., liraglutide, semaglutide, dulaglutide), which reduce HbA1c by ~0.8–1,5 % (at current clinical doses) and lead to additional weight loss and reduced fasting plasma glucose.[12,13]

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GLP-1(7-36) and GLP-1(9-36) are further cleaved by neutral endopeptidase (NEP24.11) to produce GLP-1(28–36), which enters human coronary artery endothelial cells via macropinocytosis and acts directly on coronary smooth muscle and endothelial cells. This process increases soluble adenylyl cyclase (sAC)-dependent cAMP levels, activates protein kinase A, and provides cellular protection against oxidative damage.[14]

The peptide GLP-1(28–36) regulates sAC by increasing intracellular ATP levels, leading to cAMP accumulation—a crucial second messenger in cells lacking sAC. Moreover, mitochondrial trifunctional protein α (MTPα) has been shown to interact with GLP-1(28–36), enabling it to shift cellular fuel utilization toward glycolysis and glucose oxidation—thereby conserving oxygen and supporting cardiovascular function. In models of myocardial ischemic injury, myocardial infarction, and heart failure, GLP-1(28–36) prevented cardiac dysfunction, reduced infarct size, and protected coronary vascular cells from oxidative stress-induced damage.[15]

Cardiovascular risk in diabetic patients is classified into three categories: very high, high, and moderate. Very high risk includes patients with established cardiovascular disease, end-organ damage, three or more risk factors (age, hypertension, smoking, dyslipidemia, obesity), or long-standing type 1 diabetes (>20 years). High cardiovascular risk applies to patients with >10 years of diabetes, no organ damage, but at least one risk factor. Moderate risk includes younger individuals (type 1 diabetes <35 years or type 2 <50 years) with <10 years of diabetes and no additional risk factors.[16]

Another stratification categorizes T2DM patients into four cardiovascular risk levels: very high, high, moderate, and low. Very high-risk patients include those with established cardiovascular disease, proteinuria, or glomerular filtration rate <30 mL/min, and those with ≥3 cardiovascular risk factors (age >60 years, hypertension, dyslipidemia, smoking, obesity). High-risk patients do not meet very high- or moderate-risk criteria. Moderate-risk patients must fulfill all three conditions: no additional cardiovascular risk factors, age <50 years, and diabetes duration <10 years. Notably, no T2DM patients are classified as low risk.[17]

Several GLP-1 analogs used in diabetes treatment are compared based on similarity to native GLP-1, half-life, dosing frequency, and glucose effects. Short-acting agents—such as exenatide and lixisenatide—are administered multiple times daily and primarily reduce postprandial glucose by delaying gastric emptying. Long-acting analogs—such as albiglutide, dulaglutide, exenatide once-weekly, liraglutide, and semaglutide—are administered weekly and show strong effects on fasting glycemia by stimulating insulin secretion and reducing hepatic gluconeogenesis, with variable effects on postprandial glucose.[18]

Cardiovascular safety data from outcome trials highlight several antidiabetic medications with proven cardiovascular benefits. Empagliflozin significantly reduces cardiovascular mortality and heart failure hospitalizations. Liraglutide reduces major adverse cardiovascular events and cardiovascular death. Semaglutide lowers major adverse cardiovascular events. Canagliflozina and dapagliflozin also reduce heart failure hospitalizations and, in some cases, cardiovascular mortality. These findings emphasize selecting antidiabetics that not only control glycemia but also provide cardiovascular protection—enabling a more comprehensive approach to diabetes management. Among these, empagliflozin appears to offer the greatest cardiovascular safety due to its marked impact on reducing both cardiovascular mortality and heart failure hospitalizations.[19]

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GLP-1 RA trials show variability in cardiovascular protection among T2DM patients. Liraglutide and semaglutide stand out with 13 % and 26 % relative risk reductions in composite cardiovascular events, respectively, with baseline cardiovascular disease rates of 81 % and 60 %. Albiglutide showed a 22 % risk reduction (100 % baseline CVD). In contrast, lixisenatide and once-weekly exenatide showed no significant effects, while dulaglutide demonstrated a 12 % reduction (31,5 % baseline CVD). Oral semaglutide did not reach statistical significance (84,7 % baseline CVD). These results suggest that certain GLP-1 RAs offer significant cardioprotection in high-risk patients beyond glycemic control.[20]

In major GLP-1 RA trials, results for heart failure hospitalization (HHF) varied. LEADER (liraglutide) reported a non-significant 13 % HHF reduction (RR: 0,87). SUSTAIN-6 (semaglutide) showed HHF rates of 17,6 vs. 16,1 per 1,000 person-years (RR: 1,11), indicating no HHF benefit despite other cardiovascular advantages. ELIXA (lixisenatide) showed no significant HHF effect (RR: 0,96), nor did HARMONY (albiglutide, RR: 0,85), PIONEER 6 (oral semaglutide, RR: 0,86), or EXSCEL (once-weekly exenatide, RR: 0,94). However, AMPLITUDE-O (efpeglenatide) demonstrated a 39 % HHF reduction (RR: 0,61), highlighting its potential in preventing cardiovascular complications. These findings reflect variability in GLP-1 RA efficacy for HHF reduction, underscoring the need to evaluate each agent and population specifically.[21]

Cardiovascular risk classification in T2DM patients shows both consensus and divergence. While both frameworks categorize risk into very high, high, moderate, and low levels, they differ in specific criteria. One approach defines very high risk based on established CVD, organ damage, ≥3 risk factors, or long-standing T1DM. [22,23,24,25] Another expands this by including proteinuria or eGFR <30 mL/min as very high-risk markers and clearly delineates high, moderate, and low-risk groups using age and diabetes duration. [26,27,28,29]

GLP-1 analogs for T2DM are divided into short- and long-acting based on duration and dosing frequency. Short-acting agents (exenatide, lixisenatide) reduce postprandial glucose via gastric emptying delay. Long-acting agents (albiglutide, dulaglutide, liraglutide, semaglutide) primarily lower fasting glucose through insulin stimulation and reduced hepatic glucose output.[30,31] In cardiovascular safety, empagliflozin excels in reducing CV mortality and HHF; liraglutide in reducing MACE; and semaglutide in lowering major adverse events. These findings reinforce the importance of selecting antidiabetics that integrate glycemic control with cardiovascular protection for optimal T2DM management.[32]

The varied effects of GLP-1 receptor agonists (GLP-1 RAs) on cardiovascular protection, particularly in patients with type 2 diabetes, are evident. Although certain GLP-1 RAs—such as liraglutide and semaglutide—demonstrate significant reductions in the relative risk of composite cardiovascular events (13 % and 26 %, respectively), others—including lixisenatide, once-weekly exenatide, and oral semaglutide—have failed to show statistically significant benefits in these outcomes.[27]

Regarding hospitalization for heart failure (HHF), results also vary considerably across trials. For instance, the LEADER trial with liraglutide reported a 13 % reduction in HHF risk, although it did not reach statistical significance, while SUSTAIN-6 with semaglutide showed no significant difference in HHF outcomes. [28,29,30]

In contrast, the AMPLITUDE-O trial with efpeglenatide demonstrated a statistically significant 39% reduction in HHF, suggesting promising potential in preventing cardiovascular complications among high-risk populations. These outcome variations highlight the complexity of GLP-1 RAs and underscore the importance of considering differences in each drug's pharmacological properties and the characteristics of the studied populations.[31–33]

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Factors such as study duration, patient inclusion criteria, and baseline cardiovascular health status may significantly influence observed results. Moreover, the lack of consistent benefits across all trials emphasizes the ongoing need for research to better understand the specific mechanisms by which GLP-1 RAs affect cardiovascular outcomes in patients with type 2 diabetes.[34,35,36]

CONCLUSIONS

Evidence on GLP-1 receptor agonists (GLP-1 RAs) in type 2 diabetes reveals significant variability in cardiovascular efficacy. While agents such as liraglutide and semaglutide have demonstrated statistically significant reductions in major adverse cardiovascular events, others—including lixisenatide and once-weekly exenatide—show no consistent benefits. Similarly, results for heart failure hospitalization differ: the LEADER trial suggests some potential benefit with liraglutide, SUSTAIN-6 does not confirm advantages with semaglutide, and AMPLITUDE-O reports a 39 % reduction with efpeglenatide, highlighting its utility in high-risk populations. These findings emphasize that therapeutic selection should consider not only glycemic control but also the patient's cardiovascular profile, reinforcing the need for further research to clarify underlying mechanisms and optimize comprehensive diabetes management.

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