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Eureka Journal of Health Sciences & Medical Innovation (EJHSMI)

ISSN 2760-4942 (Online) Volume 2, Issue 2, February 2026

https://eurekaoa.com/index.php/5

THE ROLE OF SODIUM-GLUCOSE COTRANSPORTER-2 (SGLT2) INHIBITORS IN ACUTE MYOCARDIAL INFARCTION

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Abstract

This article analyzes the impact of sodium-glucose cotransporter-2 (SGLT2) inhibitors, particularly empagliflozin, on the course of acute myocardial infarction (AMI) in patients with type 2 diabetes mellitus (T2DM). Data are presented on the high prevalence of T2DM and its association with cardiovascular complications. The cardioprotective mechanisms of SGLT2 inhibitors are discussed, including the reduction of preload and afterload, improvement of myocardial metabolism, and antioxidant and anti-inflammatory effects. Randomized clinical trials (EMPA-REG OUTCOME, EMPEROR-Reduced) have demonstrated a reduction in cardiovascular mortality and hospitalization rates with SGLT2 inhibitors. However, their use in the acute phase of AMI remains insufficiently studied. The authors emphasize the need for further research to determine the optimal timing and conditions for their administration.

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ISSN 2760-4942 (Online) Volume 2, Issue 2, February 2026

https://eurekaoa.com/index.php/5

Keywords: type 2 diabetes mellitus, acute myocardial infarction, complications, cardioprotection, treatment, sodium-glucose cotransporter-2 inhibitors, empagliflozin.

Introduction

According to the 10th edition of the IDF Diabetes Atlas (2021), approximately 537 million adults aged 20–79 years worldwide are living with diabetes mellitus (DM). This number is projected to increase to 643 million by 2030 and to 783 million by 2045. In 32 European countries with a combined population of 850 million, the number of individuals aged over 15 years diagnosed with diabetes reaches 59.8 million, with the prevalence doubling every 10–15 years [1].

According to the Ministry of Health of Uzbekistan, more than 245,000 patients with diabetes are officially registered in the country; however, experts estimate the actual number of affected individuals to be approximately 2.37 million (7.9% of the population) [2]. In the Russian Federation, as in many other countries worldwide, the prevalence of diabetes continues to rise. Since 2000, the number of patients with DM has more than doubled [3], reaching 5,168,800 individuals by the end of 2021 [4].

Diabetes mellitus is widely recognized as one of the principal risk factors for the development of cardiovascular diseases (CVD) and their complications. At the same time, cardiovascular complications remain the leading cause of mortality among patients with DM. According to international registries, patients with previously diagnosed diabetes account for 19–23% of all individuals hospitalized for acute myocardial infarction (AMI). Moreover, in a substantial proportion of cases, diabetes is first diagnosed during hospitalization for AMI. The incidence of AMI in patients with diabetes is three times higher compared to individuals without diabetes, and it is characterized by a more severe clinical course and occurrence at a younger age [5,6].

Patients with AMI represent a high-risk group for recurrent myocardial infarction,

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chronic heart failure, life-threatening arrhythmias, and cardiovascular death [7,8]. The risk begins at the onset of myocardial infarction symptoms, increases with the duration of ischemia, and remains elevated in both the mid-term and long-term periods, particularly in cases complicated by left ventricular systolic dysfunction or the development of heart failure [9,10].

Prospects for the Use of Sodium–Glucose Cotransporter 2 (SGLT2) Inhibitors in Acute Myocardial Infarction

Currently, patients with acute myocardial infarction (AMI) receive a broad spectrum of evidence-based therapies during the early post-infarction period. These measures aim to reduce the risk of adverse cardiac remodeling, prevent the development of chronic heart failure, sudden cardiac death, and progression to end-stage disease. However, despite substantial advances in treatment, there remains a need for additional effective therapeutic strategies, as the rates of adverse cardiovascular outcomes remain unacceptably high [11,12].

In this context, sodium–glucose cotransporter 2 (SGLT2) inhibitors have attracted increasing attention in recent years, particularly empagliflozin, which demonstrates not only glucose-lowering effects but also pronounced cardioprotective properties [13]. These agents improve cardiorenal outcomes in patients with type 2 diabetes mellitus (T2DM), chronic kidney disease (CKD), and heart failure with reduced ejection fraction (HFrEF). The EMPA-REG OUTCOME trial demonstrated that treatment with empagliflozin significantly reduced cardiovascular mortality and hospitalizations for heart failure in patients with T2DM and a prior history of myocardial infarction [14].

Empagliflozin, one of the most widely used representatives of the SGLT2 inhibitor class, effectively lowers blood glucose levels by inhibiting renal tubular glucose reabsorption; however, its cardioprotective effects extend far beyond glycemic control. Key cardioprotective mechanisms include reduction of cardiac preload and afterload due to its diuretic effect, improvement of myocardial

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metabolism through a metabolic shift toward ketone body utilization, and significant attenuation of inflammation and oxidative stress. These mechanisms render empagliflozin a unique therapeutic option for patients with T2DM and cardiovascular disease [15].

The EMPA-REG OUTCOME trial included more than 7,000 patients with T2DM and high cardiovascular risk. The results demonstrated a 38% reduction in cardiovascular mortality, a 35% reduction in hospitalization for heart failure, and a 32% reduction in all-cause mortality with empagliflozin therapy [14]. These findings were further supported by subsequent trials, such as EMPEROR-Reduced, which confirmed the benefits of the drug in patients with chronic heart failure [16].

A number of studies have investigated the long-term use of SGLT2 inhibitors in stable outpatients with T2DM, heart failure, and diabetic nephropathy [17–22]. These studies included ambulatory patients both with and without concomitant atherosclerotic cardiovascular disease. The results consistently demonstrated that prolonged SGLT2 inhibitor therapy reduces hospitalizations for heart failure, slows the progression of renal disease, and decreases the risk of major adverse cardiovascular events (myocardial infarction, stroke, and cardiovascular death).

Compelling evidence supporting the beneficial effects of SGLT2 inhibitors on the course of T2DM, nephropathy, and cardiovascular disease has been incorporated into national and international clinical guidelines [23–28], as well as into recommendations by regulatory authorities such as the U.S. Food and Drug Administration (FDA), which endorse the use of these agents in various stable clinical settings to achieve broad therapeutic benefits [29].

Early initiation and sustained therapy with SGLT2 inhibitors in the setting of AMI appear justified due to their potential ability to favorably influence disease progression, reduce susceptibility to ventricular remodeling, and decrease the risk of heart failure progression [30]. These hypotheses have received additional experimental support in animal models of AMI, both in diabetic and non-diabetic

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subjects [31,32]. Importantly, the mechanisms of action of SGLT2 inhibitors are not directly related to inhibition of coronary thrombosis but are associated with attenuation of neurohormonal activation, reduction of cardiomyocyte necrosis, and mitigation of reperfusion injury [33,34].

The therapeutic effects of SGLT2 inhibitors are thought to be mediated by normalization of endothelial function and vasodilation [35], optimization of myocardial energy metabolism [31,33,36], and preservation of cardiac contractility. Furthermore, these agents attenuate oxidative stress pathways, thereby improving coronary blood flow and reducing ventricular load [32,33,37]. Collectively, these effects contribute to the prevention of cardiomegaly, arrhythmias, myocardial fibrosis, and heart failure [38].

In patients with T2DM and stable ischemic heart disease, SGLT2 inhibitor therapy has been shown to reduce left ventricular mass and promote reverse myocardial remodeling [39]. Similar beneficial effects have been observed in patients with heart failure with preserved ejection fraction (HFpEF), where SGLT2 inhibitors help prevent or attenuate adverse remodeling [40–42].

Additionally, SGLT2 inhibition may provide further cardiometabolic benefits in high-risk post-myocardial infarction patients, including reduction of preload and afterload, improved glycemic control, and weight loss through mechanisms of natriuresis and glucosuria [43]. Moreover, direct and indirect cardiorenal effects—such as reduction of intraglomerular pressure, stabilization of renal function, rapid increase in fractional sodium excretion without activation of the sympathetic nervous system, and stimulation of renal erythropoietin production—contribute to optimization of plasma volume and improved myocardial oxygen delivery [43–46].

Risks and Limitations of SGLT2 Inhibitor Use

It should be noted that the aforementioned positive therapeutic effects of SGLT2 inhibitors were predominantly observed in patients with acute myocardial

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infarction (AMI) who had been receiving these agents long-term for type 2 diabetes mellitus (T2DM) or chronic kidney disease (CKD) prior to the cardiovascular event. Therefore, the question of their efficacy and safety when initiated in the early stages following AMI remains open.

Most of the previously described studies were conducted in stable outpatient populations with T2DM, heart failure (HF), or nephropathy and excluded individuals with recently experienced acute cardiovascular events. For example, in the DECLARE–TIMI 58 trial evaluating dapagliflozin, 21% of participants had a prior history of myocardial infarction, with a median time since the last event of 5.4 years (interquartile range: 2.1–10.9 years) [47]. Only 844 patients (4.9%) had experienced AMI within the preceding two years, and 488 patients (2.8%) within the previous year. Furthermore, patients with recent acute coronary syndromes were excluded, and the study population primarily consisted of stable outpatients.

Similarly, in large-scale trials such as EMPA-REG OUTCOME, CANVAS, and CREDENCE, the representation of patients with relatively recent AMI was limited. In the multicenter DECLARE–TIMI 58 trial, the risk of major adverse cardiovascular events (MACE) increased as the time interval since confirmed AMI decreased [47]. Notably, among patients treated with dapagliflozin, a reduction in MACE was observed depending on the timing of therapy initiation—the earlier the drug was started after AMI, the greater the relative and absolute risk reduction.

Comparable findings were reported in the EMPA-REG OUTCOME trial, in which 1,214 participants (17.9%) had experienced a confirmed acute cardiovascular event, including AMI, within the previous year. In this subgroup, empagliflozin demonstrated consistently high efficacy compared with patients who had a more remote history of atherosclerotic cardiovascular disease.

Only a limited number of patients in the early post-infarction period were included in trials such as DAPA-HF (dapagliflozin for heart failure), EMPEROR-

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Reduced (empagliflozin in heart failure with reduced ejection fraction), EMPEROR-Preserved (empagliflozin in heart failure with preserved ejection fraction), DELIVER (dapagliflozin in HFpEF), and DAPA-CKD (dapagliflozin in CKD) [48–52]. Importantly, all of these trials excluded patients who had experienced myocardial infarction within the previous three months.

Current clinical guidelines for the management of AMI do not yet recommend routine use of SGLT2 inhibitors during the acute phase of the disease [53]. Caution is advised to avoid hypovolemia, hypotension, ketoacidosis, or acute kidney injury, which may occur during the acute stage and early post-infarction period. Encouragingly, such adverse events have thus far been reported relatively infrequently and only slightly more often compared with placebo in patients with chronic or acute heart failure receiving SGLT2 inhibitors [19,20,22,54,55]. Nevertheless, the applicability of this therapeutic paradigm to patients with AMI remains uncertain.

An additional safety consideration in patients with AMI relates to the increasing use of coronary angiography and percutaneous coronary intervention (PCI), which carries a risk of contrast-induced acute kidney injury [56]. In this context, concomitant SGLT2 inhibitor therapy may potentially exacerbate the modest decline in estimated glomerular filtration rate (eGFR) observed due to their mechanism of action affecting afferent arteriolar tone. However, substantial evidence indicates that SGLT2 inhibition contributes to long-term stabilization of renal function in patients with T2DM, CKD, and heart failure with reduced ejection fraction [45,46], which may support their safe use even in the setting of acute renal dysfunction during AMI. Nonetheless, further investigation into the safety of SGLT2 inhibitor use following PCI in this population is essential.

Another limitation of SGLT2 inhibitor use in AMI is the insufficient evidence regarding their combined administration with renin–angiotensin–aldosterone system (RAAS) inhibitors.

Current clinical guidelines for AMI management do not provide sufficient

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evidence to support the routine use of SGLT2 inhibitors even in patients with concomitant T2DM or transient congestive symptoms [57–60]. Moreover, in the United States, analyses of prescription patterns within the Medicare Part D program and large healthcare systems indicate that the overall utilization rate of this drug class remains low, with substantial variation across physician specialties [61,62]. Practicing clinicians, healthcare quality auditors, and guideline committees increasingly seek to rely on the highest levels of evidence and avoid prescribing therapies with limited or insufficiently robust safety and efficacy data [63].

Thus, well-designed, adequately powered clinical trials in this field have the potential to significantly influence standards of care for patients with AMI. A stepwise accumulation of evidence evaluating efficacy and safety in dedicated studies of patients with established heart failure and in high-risk AMI populations predisposed to developing chronic heart failure has numerous precedents—most notably in the historical evaluation of beta-blockers and RAAS inhibitors.

Conclusion

SGLT2 inhibitors represent an innovative class of oral glucose-lowering agents that have entered clinical practice in recent years. Empagliflozin has demonstrated substantial benefits in the management of patients with type 2 diabetes mellitus (T2DM), including a reduction in cardiovascular complications and improvement in overall prognosis. Its potential use in patients with acute myocardial infarction (AMI) opens new perspectives for enhancing survival and quality of life. Nevertheless, further studies are required to evaluate the efficacy and safety of SGLT2 inhibitors during the acute phase of AMI. Particular attention should be given to their initiation in the early post-infarction period, which may provide additional opportunities for cardioprotection.

It remains unclear whether SGLT2 inhibitors initiated in the early stages of AMI exert similar or differential effects—both in terms of efficacy and safety—

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depending on the presence or absence of ST-segment elevation (STEMI vs. NSTEMI). The therapeutic efficacy and safety profile of SGLT2 inhibitors may also be influenced by multiple clinical factors, including hemodynamic status, cardiac output, cardiac filling pressures, left ventricular dysfunction, peripheral organ perfusion, renal function, and the timing of reperfusion therapy.

Taking these considerations into account, and in analogy with the initiation of angiotensin-converting enzyme inhibitors and beta-blockers, the most appropriate timing for the introduction of SGLT2 inhibitors is likely after the achievement of hemodynamic stability in patients with AMI.

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