Koristi in pasti zdravljenja s SGLT-2 zaviralci

Balancing the Benefits and Risks of SGLT2 Inhibitors

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Izvleček

Srčnožilne, ledvične in presnovne bolezni so eden izmed vodilnih vzrokov obolevnosti in umrljivosti po svetu. Zaviralci natrij–glukoznega kotransporterja 2 (angl. sodium-glucose cotransporter-2 inhibitors, SGLT2i) so izkazali pomembne koristi, ki presegajo zgolj uravnavanje glikemije. V preglednem članku so obravnavani trenutno dostopni dokazi o ledvičnih, srčnožilnih in presnovnih učinkih SGLT2i s poudarkom na ključnih kliničnih raziskavah o njihovi uporabi.

Z zaviranjem ponovnega privzema natrija in glukoze v proksimalnem ledvičnem tubulu SGLT2i ponovno vzpostavijo tubuloglomerularno povratno zanko, zmanjšajo intraglomerularni tlak in zmanjšajo glomerularno hiperfiltracijo, s čimer upočasnijo napredovanje kronične ledvične bolezni. Večje randomizirane kontrolirane raziskave so pokazale

Abstract

Cardiovascular, renal, and metabolic diseases frequently coexist and are leading contributors to global morbidity and mortality. Sodium–glucose cotransporter 2 inhibitors (SGLT2is), originally developed for the treatment of type 2 diabetes mellitus, have demonstrated substantial benefits extending beyond glycemic control. This review synthesizes current evidence on the renal, cardiovascular, and metabolic effects of SGLT2is and highlights key clinical outcome trials supporting use.

SGLT2is restore tubuloglomerular feedback, reduce intraglomerular pressure, and attenuate glomerular hyperfiltration by inhibiting sodium–glucose reabsorption in the proximal renal tubule, thereby slowing the progression of chronic kidney disease. Large randomized controlled trials have consistently

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zmanjšanje števila hospitalizacij zaradi srčnega popuščanja ter zmanjšanje srčnožilne umrljivosti, in to tudi pri populacijah brez sladkorne bolezni. Poleg tega imajo SGLT2i številne ugodne metabolne sistemske učinke, vključno z zmernim zmanjšanjem telesne teže in krvnega tlaka, izboljšano presnovno učinkovitostjo, ter tudi modulacijo vnetja in oksidativnega stresa.

V obravnavo so prav tako zajeti neželeni učinki, vključno z genitourinarnimi okužbami, hipovolemijo, prehodnim poslabšanjem ledvične funkcije ter redkimi zapleti, kot sta diabetična ketoacidoza in Fournierjeva gangrena.

demonstrated reductions in kidney disease progression, hospitalization for heart failure, and cardiovascular mortality in populations with and without diabetes. In addition, SGLT2is exert systemic effects, including modest reductions in body weight and blood pressure, improved metabolic efficiency, and modulation of inflammatory and oxidative stress pathways.

Adverse effects, including genitourinary infections, volume depletion, transient declines in renal function, and rare events, such as diabetic ketoacidosis and Fournier's gangrene, are also discussed.

In summary, SGLT2is represent a transformative therapeutic strategy with broad, multi-system benefits across cardiovascular, renal, and metabolic diseases.

INTRODUCTION

Chronic kidney disease (CKD), type 2 diabetes (T2D), and cardiovascular disease (CVD) are major contributors to global morbidity and premature mortality. Cardiovascular–renal–metabolic (CRM) syndrome describes the interconnected dysfunction of these systems, leading to multiorgan and cardiovascular complications. CRM syndrome is often driven by excess or dysfunctional visceral adipose tissue, which releases proinflammatory and prooxidative mediators that promote vascular, cardiac, and renal injury and impair insulin sensitivity (1, 2).

The 1999–2020 NHANES data indicated that ~25% of individuals have at least 1 CRM condition with multimorbidity affecting 8% overall and 25% of adults ≥ 65 years. The burden increases with age and is associated with male gender, Black race, adverse socioeconomic factors, and a higher prevalence of T2D, obesity, physical inactivity, and uncontrolled hypertension (2).

Sodium-glucose cotransporter 2 inhibitors (SGLT2is), also known as gliflozins, are oral antidiabetic drugs that act by selectively blocking the SGLT2 cotransporter in the S1 segment of the proximal renal tubule, thereby preventing glucose and sodium reabsorption. This mechanism results in sustained glucosuria and natriuresis and is associated with clinically relevant reductions in glycated hemoglobin (HbA1c), body weight, blood pressure, and albuminuria (3).

SGLT2is provide cardiovascular and renal protection, even in non-diabetic patients. SGLT2is are now a central therapy for heart failure and CKD. Additional benefits include modest weight loss and blood pressure reduction via natriuresis, although monitoring for adverse effects remains essential (3-6).

This review discusses the effects of SGLT2is and summarizes the key landmark clinical trials evaluating use. The therapeutic benefits and potential adverse effects associated with treatment are highlighted. Crucial steps in prescribing SGLT2is are presented in Figure 1.

BENEFICIAL EFFECTS OF SGLT2IS

Renoprotective effects

SGLT2is protect the kidney by restoring tubuloglomerular feedback, reducing intraglomerular pressure, and reversing hyperfiltration, thereby stabilizing filtration and slowing CKD progression (6-15). An initial glomerular filtration rate (GFR) reduction is later followed by long-term GFR

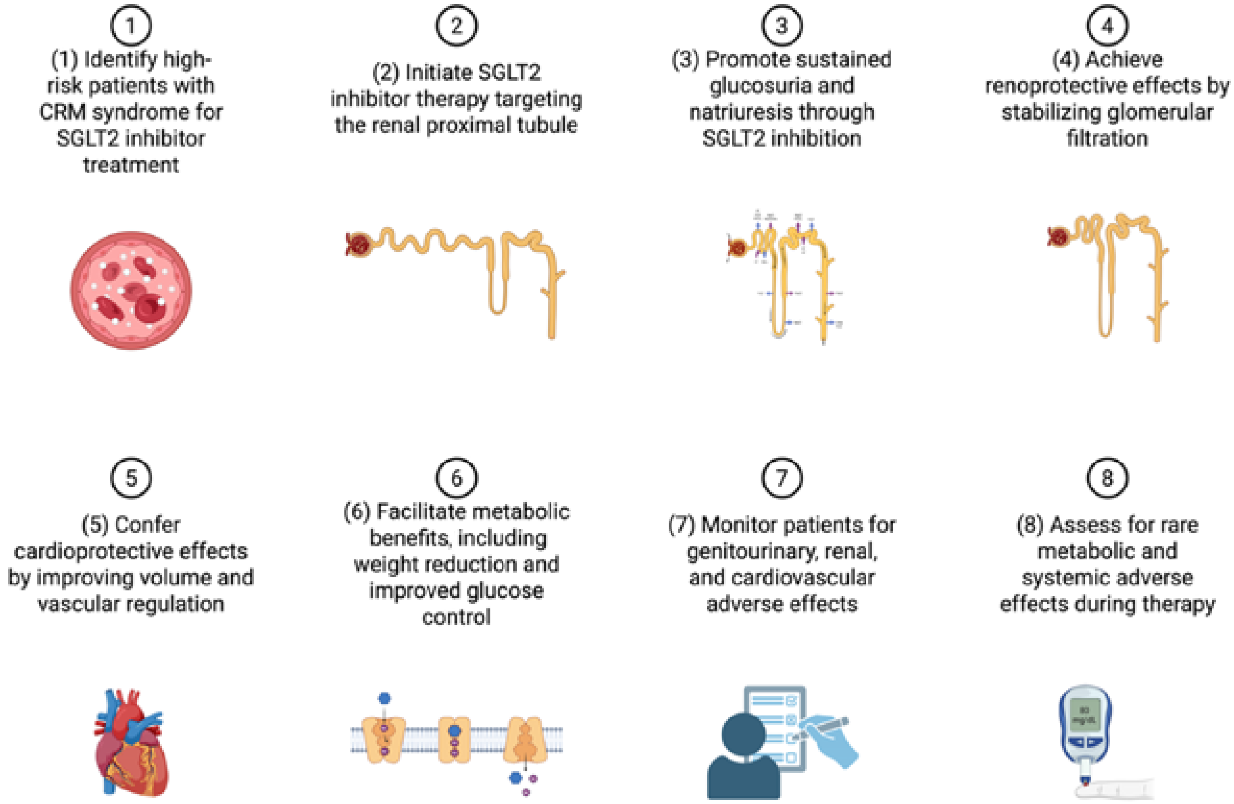

Figure 1. *Crucial steps in prescribing sodium-glucose cotransporters-2.*

Legend: CRM – cardiorenal and metabolic syndrome, SGLT2 – sodium-glucose cotransporter-2

preservation and a reduced risk of developing acute kidney injury (16).

Dapagliflozin reduced the risk of renal composite outcomes compared to placebo (HR = 0.76) in DECLARE–TIMI 58 (10). Empagliflozin significantly lowered the risk of kidney disease progression or cardiovascular death in EMPA-KIDNEY across a broad range of baseline eGFRs, including patients with advanced CKD (12). Canagliflozin reduced the risk of progression to end-stage kidney disease in the CREDENCE trial by 32% compared to placebo [HR = 0.68] (13). These hemodynamic improvements are accompanied by marked reductions in albuminuria and proteinuria, reflecting a strengthened glomerular filtration barrier and reduced tubular stress. Such changes reflect the combined effects of lower intraglomerular pressure, which reduces epithelial sodium channel (EnaC)-mediated sodium retention and consequently limits injury to distal tubular segments (8, 10-13, 17-22). In addition to the hemodynamic effect, SGLT2 inhibition improves renal oxygenation and mitochondrial efficiency, reduces tubular ATP demand, activates autophagy pathways, and attenuates oxidative and inflammatory stress, thereby protecting nephron integrity under metabolic and hypoxic load (6, 7, 9, 19, 21-25). Clinically, these changes manifest as consistently lower rates of acute kidney injury (AKI), including severe episodes requiring hospitalization, and preserved kidney function, even in patients with advanced CKD or a markedly reduced eGFR (3, 10-15, 26, 27).

Cardioprotective effects

SGLT2is confer consistent cardiovascular protection through integrated hemodynamic, vascular, and anti-inflammatory mechanisms. SGLTis lower cardiac preload and afterload, stabilize volume status, and reduce systolic and diastolic blood pressure by inducing natriuresis and osmotic diuresis without compensatory tachycardia (10-15, 17, 23). These blood-pressure–lowering effects were modest but consistent across landmark outcome trials. Dapagliflozin reduced systolic blood pressure by 2.7 mm Hg (95% CI = 2.4–3.0 mmHg) and diastolic blood pressure by 0.7 mmHg (95% CI = 0.6–0.9 mmHg) in DECLARE–TIMI 58 compared to placebo (10). Empagliflozin was associated with reductions of 2.6 ± 0.3 mmHg in systolic and 0.5 ± 0.2 mmHg in diastolic blood pressure in EMPA-KIDNEY (12), while canagliflozin lowered systolic blood pressure by 3.30 mmHg (95% CI = 2.73–3.87) and diastolic blood pressure by 0.95 mm Hg (95% CI = 0.61–1.28) in CREDENCE relative to placebo (13). These hemodynamic effects translated into fewer hospitalizations for heart failure and reductions in cardiovascular mortality across a broad range of patients. Dapagliflozin reduced the composite of cardiovascular death or hospitalization for heart failure (HR = 0.83) but did not significantly reduce cardiovascular death alone (HR = 0.98) in DECLARE–TIMI 58 (10). Empagliflozin significantly reduced death from cardiovascular causes with no significant differences in myocardial infarction or stroke compared to placebo in EMPA-REG OUTCOME (11). Cardiovascular outcomes in EMPA-KIDNEY were consistent with prior evidence. Specifically, pooled analyses showed a 14% reduction in cardiovascular mortality (RR = 0.86) and a 23% reduction in hospitalization for heart failure or cardiovascular death (RR = 0.77) in EMPA-KIDNEY (12). Canagliflozin reduced the risk of cardiovascular death, myocardial infarction, or stroke (HR = 0.80; 95% CI = 0.67–0.95) and substantially lowered the hospitalization rate for heart failure (HR = 0.61; 95% CI = 0.47–0.80) in CREDENCE compared to placebo (13). Improvements in vascular compliance, endothelial function, and arterial stiffness further reduce cardiac workload and enhance circulatory stability (10-15). Additional cardioprotective mechanisms include attenuation of oxidative stress, reduced inflammatory signaling, and improved mitochondrial homeostasis, which together reduce ischemia–reperfusion injury and limit post-MI remodeling (15, 28). Broader clinical benefits include reductions in major adverse cardiovascular events, fewer recurrent heart-failure episodes, decreased need for loop-diuretic therapy, and improved outcomes in non-diabetic populations with pooled analyses demonstrating lower all-cause and cardiovascular mortality (3, 11, 14). Importantly, these cardiovascular benefits occur independent of glycemic control, reflecting the consistency across different SGLT2is (10-15).

Metabolic and other systemic favorable effects

SGLT2is also exert a broad set of metabolic benefits primarily through insulin-independent glucosuria (10-13). Urinary glucose loss creates a caloric deficit and leads to modest weight reduction. Dapagliflozin reduced body weight by 1.8 kg in DECLARE–TIMI 58 compared to placebo (10). Empagliflozin resulted in a mean decrease in body weight of 0.9 ± 0.1 kg during follow-up in the EMPA-KIDNEY trial (12). Similarly, canagliflozin therapy led to a reduction in body weight of 0.80 kg (95% CI = 0.69–0.92) in the CREDENCE trial relative to placebo (13). Natriuresis and osmotic diuresis represent central systemic mechanisms that improve sodium–water balance and reduce fluid overload, while maintaining stable volume status in patients without pre-existing fluid overload (8, 10-15). Additional systemic effects include reductions in serum uric acid (10-14), a favorable shift in lipid profile (17), and a mild fasting-like metabolic state with enhanced ketone utilization (7, 9). SGLT2 inhibition promotes autophagy via mTOR suppression, preserves mitochondrial structure, reduces oxidative stress through NRF2 activation, and prevents apoptosis by modulating BAX/BCL-2 signaling at the cellular level (6, 21, 22, 24, 25). Improved mitochondrial oxygen efficiency, enhanced erythropoietin production, and increases in hematocrit further support metabolic and oxygen-delivery capacity (7, 24). These mechanisms contribute to reduced all-cause mortality, fewer hospitalizations, and systemic stabilization in high-risk patients (3, 11, 12, 14, 16).

UNDESIRABLE EFFECTS OF SGLT2 INHIBITORS ON THE KIDNEYS

Urogenital tract

Genitourinary infections occur in 10%–15% of patients receiving SGLT2is, mainly due to glucosuria. While the risk of urinary tract infections is not consistently increased in patients receiving SGLT2is, genital infections are more common, particularly in women, patients with T2D, and patients with prior infections or predisposing conditions. These infections are usually mild-to-moderate and respond to standard treatment (3-5, 17, 23, 26, 28-30).

Fournier gangrene (FG) is a very rare but serious complication of SGLT2i treatment, occurring in 1.6 per 100,000 males (3.3 per 100,000 among males 50–79 years of age) and 0.25 per 100,000 in the US (31). FG is more common in males and patients with diabetes and other comorbidities, and is likely driven by bacterial growth in glucose-rich tissues and impaired immunity. Infections are typically polymicrobial and may follow local breaches, such as procedures or trauma (4, 18). Empagliflozin is associated with the highest number of FG reports, followed by canagliflozin and dapagliflozin. Among SGLT2is, the combination of empagliflozin and metformin had the strongest association with FG (1).

As the eGFR declines, the anti-hyperglycemic effect of SGLT2is diminishes, particularly when the eGFR falls below 30 mL/min/1.73 m². This finding may lead to fluctuations in renal function and in some cases to AKI. Risk factors for AKI include older age (≥ 65 years), pre-existing renal impairment, diuretic use, hypovolemia, infection, sepsis, or exposure to nephrotoxic agents (4, 17, 29).

There are reports indicating that among patients with advanced CKD, the anti-hyperglycemic effect of SGLT2is may be reduced, although the renal protective benefits are generally preserved (3, 7, 26, 28).

Early renal hemodynamic effects may be diminished by concurrent loop diuretic use or high dietary salt and protein intake, due to blunting of SGLT2-mediated tubuloglomerular TGF activation (24).

Many of these effects are transient, such as a decline in the eGFR due decreased glomerular hyperfiltration because of reduced intraglomerular pressure, which then typically stabilizes (23, 24, 26, 28). Temporary medullary hypoxia may also occur due to workload shifting to downstream segments until compensatory mechanisms (hypoxia-inducible factor [HIF]-erythropoietin [EPO] pathway) restore balance (7), which can result in AKI. Tubulointerstitial nephritis (TIN), which requires a biopsy for diagnosis, is a rare drug-induced cause of AKI that may occur with SGLT2is. Recognition of TIN is important because TIN may require corticosteroid or immunosuppressive therapy (32, 33).

A possible association between dapagliflozin and bladder cancer has been noted, although SGLT2is are not linked to an overall increased cancer risk compared to other glucose-lowering therapies. Clinical guidance advises caution in patients with hematuria or a history of bladder cancer due to potential tumor-promoting concerns (4).

Cardiovascular adverse effects

SGLT2is can cause mild hypotension and hypovolemia due to volume depletion and diuretic-like effects, particularly in adults ≥ 65 years, patients with a baseline eGFR < 60 mL/min/1.73 m², or patients on diuretics (3, 8, 17, 18, 23, 28-30). This effect may increase fall risk and potentially lead to fractures, especially with canagliflozin. The proposed mechanisms underlying hypotension and hypovolemia include intravascular volume contraction and alterations in calcium, phosphate, and vitamin D homeostasis, which may reduce bone mineral density (4, 6, 24).

Careful monitoring is recommended in patients with ongoing ulcers, peripheral artery disease, neuropathy, a history of diabetic foot ulcers, or prior amputations given the uncertainty regarding the role of SGLT2is in amputation risk and conflicting evidence (4, 18).

Metabolic and other systemic adverse effects

SGLT2is can induce modest changes in lipid metabolism, including slight increases in LDL- and HDL-cholesterol. Canagliflozin has been associated with an elevated risk of hyperkalemia, particularly when combined with angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers. The effect is most pronounced in patients with renal impairment, necessitating close electrolyte monitoring during therapy initiation or adjustment (4).

SGLT2is are associated with an approximately three-fold increased risk of diabetic ketoacidosis (DKA), including euglycemic DKA, due to elevated ketone production, even when blood glucose levels are normal. The highest risk for DKA appears with canagliflozin, followed by empagliflozin and dapagliflozin. Risk is higher during fasting, acute illness, perioperative stress, or in insulin-treated patients and temporary discontinuation 3–4 days before surgery is recommended (4, 23, 29). The incidence of DKA in clinical trials of SGLT2is ranges from 0.2–2.2 events per 1000 patient-years in individuals with T2D, representing a 2.5-fold increase versus placebo. In type 1 diabetes, The risk may be 6–8-fold higher compared to placebo, with real-world incidence rates estimated at 43–71 events per 1000 patient-years (34).

Hypoglycemia is uncommon in patients treated with SGLT2is alone. The weighted incidence rate of hypoglycemia is 2.1 events per 100 person-years with SGLT2is and 0.6 events per 100 person-years for severe hypoglycemia (< 54 mg/dL [3.0 mmol/L]). Hypoglycemia may occur when combined with insulin or sulfonylureas without a dose adjustment (9,3). Moreover, the risk of hypoglycemia is lower with SGLT2is compared to sulfonylureas (35).

CONCLUSION

SGLT2is exert renal, cardiovascular, and metabolic benefits in addition to lowering glucose levels and reducing kidney disease progression, heart failure hospitalization, and cardiovascular mortality. The side effects associated with SGLT2is are generally well-tolerated, although monitoring for adverse effects is required.

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