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Perspective

Glycemic Control and Canagliflozin Medications in Diabetes Mellitus

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DESCRIPTION

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by hyperglycemia and insulin resistance. It affects more than 400 million people worldwide and is associated with increased risk of cardiovascular and renal complications. One of the main goals of diabetes management is to achieve and maintain optimal glycemic control, which can reduce the incidence and progression of microvascular and macrovascular complications. However, many patients with T2DM fail to achieve the recommended glycemic targets with current therapies, which may have limited efficacy, adverse effects, or contraindications.

Therefore, there is a need for new and effective antidiabetic agents that can improve glycemic control and also provide additional benefits on cardiovascular and renal outcomes. Canagliflozin is a novel oral antidiabetic drug that belongs to the class of Sodium-Glucose Co-transporter 2 (SGLT2) inhibitors. It works by inhibiting the reabsorption of glucose in the proximal tubules of the kidneys, resulting in increased urinary glucose excretion and reduced plasma glucose levels. In this article, we will review the pharmacology, efficacy, safety, and clinical implications of canagliflozin for the treatment of T2DM and kidney disease.

Canagliflozin is rapidly absorbed after oral administration and reaches peak plasma concentrations within 1 to 2 hours. It has a high bioavailability of about 65% and a long elimination half-life of about 13 hours. It is mainly metabolized by glucuronidation and excreted in feces (41%) and urine (33%). The dose of canagliflozin should be adjusted according to renal function, as its efficacy and safety are reduced in patients with moderate to severe renal impairment. The recommended doses are 100 mg or 300 mg once daily before the first meal of the day. The efficacy of canagliflozin for glycemic control has been demonstrated in several randomized controlled trials involving patients with T2DM who were either treatment-naive or inadequately controlled with other antidiabetic agents, such as metformin, sulfonylureas, pioglitazone, insulin, or Glucagon-Like Peptide-1 (GLP-1) receptor agonists. The trials showed that canagliflozin significantly reduced Hemoglobin A1c (HbA1c), Fasting Plasma Glucose (FPG), Postprandial Glucose (PPG), and body weight compared with placebo or active comparators. The reductions in HbA1c ranged from 0.5% to 1.2% with canagliflozin 100 mg and from 0.7% to 1.5% with canagliflozin 300 mg over 26 to 104 weeks of treatment. The reductions in FPG ranged from 18 to 31 mg/dL with canagliflozin 100 mg and from 27 to 39 mg/dL with canagliflozin 300 mg over the same period. The reductions in PPG ranged from 36 to 58 mg/dL with canagliflozin 100 mg and from 50 to 78 mg/dL with canagliflozin

The reductions in body weight ranged from 1.9 kg to 3 kg with canagliflozin 100 mg and from 2.8 kg to 4 kg with canagliflozin 300 mg over the same period. The efficacy of canagliflozin for cardiovascular and renal outcomes has been evaluated in two large outcome trials: the CANVAS Program and the CREDENCE trial. The CANVAS Program integrated data from two trials involving a total of 10,142 participants with T2DM and high cardiovascular risk who were randomized to receive canagliflozin or placebo for a mean duration of 188 weeks. The primary outcome was a composite of death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke. The trial showed that canagliflozin significantly reduced the risk of the primary outcome by 14% compared with placebo (occurring in 26.9 *vs*. 31.5 participants per 1000 patient-years; Hazard Ratio (HR), 0.86; 95% Confidence Interval (CI), 0.75 to 0.97; P=0.02). Canagliflozin also significantly reduced the risk of hospitalization for heart failure by 33% and death from any cause by 13%. However, canagliflozin did not significantly reduce the risk of death from cardiovascular causes (occurring in 11 *vs*. 12 participants per 1000 patient-years; HR, 0.87; 95% CI, 0.72 to 1.06; P=0.17).

CONCLUSION

Canagliflozin is a highly selective and potent inhibitor of SGLT2, the main transporter responsible for glucose reabsorption in the kidneys. By blocking SGLT2, canagliflozin reduces the renal threshold for glucose excretion and increases

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the amount of glucose eliminated in the urine. This leads to a reduction in plasma glucose levels and a decrease in insulin requirements. Canagliflozin also has beneficial effects on other metabolic parameters, such as blood pressure, body weight, lipid profile, and uric acid levels. These effects are attributed to the osmotic diuresis, natriuresis, and caloric loss induced by canagliflozin. Moreover, canagliflozin may have direct effects on the cardiovascular system by improving endothelial function, reducing inflammation, and modulating sympathetic activity.