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SGLT2 Inhibitors and Electrolyte Balances

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Sodium-glucose cotransporter-2 (SGLT2) inhibitors have become a cornerstone in the management of type 2 diabetes, chronic kidney disease, and heart failure, offering significant cardioprotective and renoprotective effects beyond their glucose-lowering properties. While much attention has been devoted to their hemodynamic and metabolic benefits, their complex influence on electrolyte balance also warrants careful consideration. The mechanisms underlying changes in key electrolytes, including sodium, potassium, magnesium, calcium, and phosphate, involve natriuresis, osmotic diuresis, alterations in tubular reabsorption, and modulation of hormonal regulators such as aldosterone, parathyroid hormone, and fibroblast growth factor 23. Notably, SGLT2 inhibitors induce mild natriuresis and osmotic diuresis, which can result in volume contraction and compensatory activation of the renin-angiotensin-aldosterone system (RAAS). These adaptations can affect potassium homeostasis, occasionally predisposing patients, particularly those with chronic kidney disease receiving RAAS blockade, to hyperkalemia. Additionally, emerging evidence suggests subtle but clinically relevant effects on magnesium and phosphate levels, with potential implications for vascular calcification and bone metabolism. A thorough understanding of the nuanced effects of SGLT2 inhibitors on electrolyte dynamics is essential for clinicians to anticipate and manage potential complications, especially in vulnerable populations. Further research is necessary to clarify the long-term clinical consequences of these electrolyte alterations and to optimize the use of SGLT2 inhibitors across diverse patient groups, including those with advanced kidney disease, heart failure, or concomitant diuretic therapy.

Keywords: SGLT2, Inhibition, Kidney, Electrolyte, Mineral