Perspective

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Journal of Clinical & Experimental Nephrology
ISSN 2472-5056

2022
Vol.8 No.01:179

Sodium-Glucose Cotransporter-2 Inhibitors and Protection against Stroke

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Received date: December 05, 2022, Manuscript No. IPJCEN-22-15793; **Editor assigned date:** December 07, 2022, Pre-QC No. IPJCEN-22-15793 (PQ); **Reviewed date:** December 16, 2022, QC No. IPJCEN-22-15793; **Revised date:** December 23, 2022, Manuscript No. IPJCEN-22-15793 (R); **Published date:** January 05, 2022, DOI: 10.36648/2472-5056.8.01.179

Citation: Krystal A (2022) Sodium-Glucose Cotransporter-2 Inhibitors and Protection against Stroke. J Clin Exp Nephrol Vol.8 No.01: 179.

Description

Patients with cancer frequently suffer from renal or hepatic impairment, either as a result of the disease itself, the toxic effects of previous anticancer treatments, or other factors that affect organ function, such as getting older. Patients with cancer who have impaired renal or hepatic function may have their pharmacokinetic profile altered, necessitating dose adjustments because renal and hepatic function is one of the main factors that determine drug exposure. The majority of anticancer medications has a narrow therapeutic index and is administered close to their maximum tolerated dose. As a result, it is challenging to select an appropriate dose for patients with either hepatic or renal impairment, or both, and there are few definitive dose adjustments recommendations. The pharmacokinetics of anticancer medications is affected by renal and hepatic impairment, which is the subject of our review. A practical set of recommendations for dose adjustments for 160 anticancer drugs for patients with hepatic and renal impairment was created by combining information from available drug labels and published literature to help clinicians select the appropriate dose adjustments.

Venous Thromboembolism

Reduced-intensity conditioning for allogeneic hematopoietic cell transplant with Melphalan is a common treatment for older patients and those with comorbidities. In patients receiving melphalan conditioning for autologous HCT, renal impairment has been linked to a longer time to engraftment, an increased risk of and severity of mucositis, gastrointestinal toxicities, and renal impairment. However, the influence of renal function on melphalan-based RIC HCT is poorly studied. The purpose of this retrospective study is to determine how renal dysfunction affected the outcomes of melphalan-based RIC HCT as well as regimen-related toxicities and morbidities. An oral direct thrombin inhibitor known as dabigatran etexilate dabigatran is approved for the treatment and prevention of venous thromboembolism and the prevention of stroke in patients with atrial fibrillation. Idarucizumab is a humanized fragment of a monoclonal antibody that has a high affinity for dabigatran. According to the Reversal of Effects of Idarucizumab in Patients on Active Dabigatran study, idarucizumab completely and rapidly reverses the anticoagulant effect of dabigatran in patients who present with uncontrolled or life-threatening bleeding and in those who require urgent surgery or an invasive procedure. The kidneys clear dabigatran and idarucizumab, and many patients who present with severe bleeding or require emergency treatment have either acute or chronic renal impairment. Clinical outcomes and the extent of dabigatran reversal by idarucizumab have not been studied in relation to renal impairment.

The purpose of this analysis was to compare the clinical characteristics, levels of dabigatran, extent of idarucizumab-mediated reversal of dabigatran, and clinical outcomes based on baseline renal function in patients participating in RE-VERSE AD. Diabetes mellitus type 2 is well known to be linked to an increased risk of cardiovascular disease, including ischemic stroke. Only pioglitazone and glucagon-like peptide 1 receptor agonists have been shown to lower the risk of stroke among previous antidiabetic medications. Similarly, it was anticipated that sodium-glucose cotransporter-2 inhibitors would lower the risk of stroke due to their neuroprotective and blood pressure-lowering effects demonstrated by experimental studies. However, human trials have produced contentious outcomes. The first SGLT2i cardiovascular outcome trial, EMPA-REG OUTCOME, demonstrated that empagliflozin significantly decreased the risk of cardiovascular death in T2DM and established cardiovascular disease patients. After the study drug was discontinued, a non-significant increase in the risk of stroke was finally attributed to long-term occurrences. The neutral effect of SGLT2i on stroke risk was confirmed by additional meta-analyses. However, the SCORED trial recently demonstrated that in patients with T2DM and chronic kidney disease, sotagliflozin significantly lowers the risk of total stroke.

Plasma Angiotensin Peptides

Acetazolamide should be used with caution in patients with chronic kidney disease and those undergoing dialysis. Using acetazolamide, we examine the impact of administering aspirin to a CKD patient concurrently. Drug–drug interactions are more likely to occur in elderly patients with multi-morbidity and poly-pharmacy. This patient, who had been taking a lot of aspirin and had poor kidney function, developed severe hyper-ammonemia and hyperchloremic acidosis shortly after taking acetazolamide. Vitamin D levels are lower in CHF-iRF patients than in CHF-nRF patients. The correlations between RAAS and oxidative stress are positive in CHF-iRF but negative in CHF-nRF. Only in CHF-iRF is there a negative correlation between vitamin D and U-Isop. In

CHF-iRF, U-Isop is correlated positively with S-ACE and P-AGT. In CHF-iRF, RAAS blockers, vitamin D, or antioxidants may be helpful. In chronic heart failure patients on optimized medical therapy, stratified by disease severity or renal function, we investigated the association between oxidative stress and RAAS biomarkers. We investigated the connection between vitamin D, RAAS, and oxidative stress in CHF patients with or without renal impairment. Vitamin D has been shown to reduce RAAS activation and oxidative stress.

Sixty CHF outpatients were included and categorized according to renal function or severity of disease. Urinary hydrogen peroxide, plasma and urinary isoprostanes, plasma total antioxidant status, urinary angiotensinogen and plasma angiotensinogen, plasma renin and aldosterone concentration, serum angiotensin-converting enzyme activity, plasma angiotensin peptides, and serum total 25-hydroxyvitamin D were all measured. However, no differences were found for any other redox or RAAS biomarkers in patients with severe CHF Compared to those with normal renal function, patients with impaired renal function had higher urinary angiotensinogen and lower S-total 25(OH) D, but there were no differences in the remaining RAAS and redox parameters. In patients with iRF, there were several positive correlations between oxidative stress and RAAS biomarkers, whereas in patients with nRF, most of these correlations were negative.