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Managing the Patient with Epilepsy and Renal Impairment

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Description

Additionally, it acts on a variety of cell surface plasminogen activator receptors of the urokinase type. uPAR is a membrane-bound glycoprotein that is expressed on a number of different cells, such as activated T-lymphocytes, monocytes, macrophages, megakaryocytes, keratinocytes, fibroblasts, endothelial cells, vascular smooth muscle cells, and podocytes. It is organized in three homologous domains. After uPA cleaves the region between the GPI anchoring molecule and the DIII domain, the soluble form of the urokinase-type plasminogen activator receptor is released from the cell membrane. It is tethered to the cell membrane by a glycosyl-phosphatidyl-inosito. Serum suPAR levels were found to be elevated in patients with type 1 and type 2diabetes, focal segmental glomerulosclerosis, chronic kidney disease, sepsis, cardiovascular diseases, cancer, autoimmune diseases, HIV infection, and smoking. In patients with type 2 diabetes mellitus, it has been demonstrated that serum suPAR increases and decreases in proportion to eGFR, making it a potential biomarker for disease staging.

Renal Impairment

The optimal use of antiepileptic drugs is complicated by kidney disease in epileptic patients. In kidney disease, the disposition of AEDs can change, increasing the likelihood of toxicity or therapy failure. Nephrotoxicity from AEDs is uncommon but unpredictable. It is recommended to monitor. In addition, renal drug interactions and adverse reactions to AEDs must be taken into account. Comorbidities like impaired renal function can complicate the management of epilepsy, which affects over 50 million people worldwide. Clinicians need to know how epilepsy management strategies affect the kidneys and how antiepileptic drugs affect impaired renal function in order to control epilepsy better in kidney disease patients. We discuss the use of AEDs in patients with renal impairment, including those on dialysis, and the nephrotoxic effects of some AEDs in this narrative review and systematic literature search. By achieving the best possible balance between seizure control and adverse effects of antiepileptic drugs one of the primary objectives of treatment is to enhance patient quality of life. Even though AEDs are effective for controlling the majority of patients, it is estimated that as many as 30% of patients do not respond to conventional medical treatment. Given how AEDs are eliminated and how impaired renal function affects them, this can be made more difficult for a patient with comorbid impaired renal function.

The purpose of this review is to discuss the nephrotoxic effects of some AEDs and the use of AEDs in patients with renal impairment, including dialysis patients. Additionally, a practical strategy for utilizing AEDs in renal disease will be presented. Levetiracetam, gabapentin, pregabalin, topiramate, eslicarbazepine, lacosamide, and vigabatrin are eliminated at least in part by the kidney. The parent drug and its metabolites build up in the body and have a longer half-life when renal clearance is reduced. There is a greater chance that these medications will cause side effects if the dose is not adjusted promptly. A classic illustration of AED toxicity in renally impaired patients is gabapentin accumulation, which results in excessive sedation and frequent ER visits. Encephalopathy caused by vigabatrin and levetiracetam are two additional examples. The disposition proportion of drug metabolized, renally eliminated, and protein bound and adult dosing considerations of AEDs in patients with renal disease. The dosage is adjusted according to the degree of renal impairment. Fructose overindulgence causes renal impairment linked to insulin resistance. In a similar vein, the use of estrogen-progestin oral contraceptive therapy has been linked to cardiometabolic syndrome, and there is still no conclusive evidence regarding its non-contraceptive benefits, particularly in metabolic pathologies. Inflammation and oxidative stress are characterized by excessive uric acid production.

Neurodegenerative Diseases

Nitric oxide is a vital vasculature-protective biomolecule that is produced in endothelial cells by nitric oxide synthase from L-arginine. Numerous animal studies show that elevated plasma uric acid causes hypertension and disrupts the macula densa's production of nitric oxide a location on the thick ascending limb's distal end that contains renal-injuring granules packed tightly together. In many diseases, including multiple sclerosis, bipolar disorder, Parkinson's disease and Alzheimer's disease neuropathological alterations associated with CI have been identified through neuroimaging. In addition, it has been shown to be useful in the detection of MCI biomarkers in neurodegenerative diseases. Brain structural disruption is a

hallmark of ESRD patients and is correlated with cognitive decline, according to increasing neuroimaging evidence. For instance, the volume of gray matter consistently decreased in ESRD patients when structural images were analyzed using voxel-based morphometry. In the past, Gu et al. Vertex-wise shape analysis was used to identify subcortical structural volume anomalies in ESRD patients.

Diffusion tensor imaging studies also showed that ESRD patients' entire brains, including the thalamus, corona radiata, amygdala, and prefrontal cortex, had widespread WM microstructural changes. Cognitive dysfunction was thought to be getting worse as these changes occurred. Neuroimaging results have shown that ESRD is associated with CI and that GM and WM are impaired. However, no research has been done on the connection between MCI and abnormal brain microstructures in ESRD patients. Glycopyrronium bromide, a synthetic anticholinergic medication used to treat COPD, is excreted from the body by the kidneys. As a result, patients with declining renal function are more likely to be exposed to it systemically. In clinical studies, no patients with severe renal impairment were included, despite the fact that patients with declining renal function were included to examine the effect of renal impairment on glycopyrronium's pharmacokinetics. Predicting systemic exposure to glycopyrronium in patients with severe renal impairment was made possible by developing a physiologically based pharmacokinetic model in COPD patients with normal renal function.