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Use of iron-based phosphate binders: A novel solution to an old problem

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Chronic kidney disease (CKD) represents an enormous global public health concern, with an estimated 8-16% of the worldwide population affected by this condition. In the United States, more than 25 million Americans are estimated to have CKD. Hyperphosphatemia, a prevalent disorder of mineral metabolism in CKD patients, is a condition that may predispose patients to an increased risk of cardiovascular mortality related to uncontrolled hyperparathyroidism and vascular calcification. Controlling serum phosphate levels via use of dietary phosphate binders, along with dietary phosphate control, may help to reduce the adverse consequences of uncontrolled hyperphosphatemia. Phosphate binding agents work to reduce serum phosphate levels by forming an insoluble complex with dietary phosphorus in the gastrointestinal (GI) tract and allowing its elimination in the feces. Currently available phosphate binding agents include elemental compounds such as aluminum hydroxide, magnesium and calcium carbonate, calcium acetate, sucroferric oxyhydroxide, and lanthanum carbonate, and the nonelemental agent, sevelamer carbonate. In 2014, a novel iron-based dietary phosphate binder, ferric citrate, received approval from the United States Food and Drug Administration and the Japanese Ministry of Health for the treatment of hyperphosphatemia in patients with CKD on dialysis; consideration for approval by the European Medicines Agency is ongoing. A review of the safety and efficacy of this agent and other iron-based phosphate binders will be presented.

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A study on the effect of cimetidine and L-carnitine on myoglobinuric acute kidney injury in male rats

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Rhabdomyolysis-induced myoglobinuric acute renal failure accounts for about 10-40% of all cases of acute renal failure (ARF). Iron, free radicals, nitric oxide and Cytochrome P 450 are involved in the pathogenesis of myoglobinuric ARF. The aim of this study was comparing the effect of cimetidine, L-carnitine and both agents on myoglobinuric ARF in rats. Forty rats were divided into 5 groups; group 1: Control rats. The remaining rats were injected with 50% glycerol (10 ml/kg, i.m.) and were divided into: Group 2: Myoglobinuric ARF, group 3: Received L-carnitine (200 mg/kg, i.p.), group 4: received cimetidine (150 mg/kg i.p) and group 5: received both agents together. 48 hours later, blood pressure was measured. 24 hours urine collection and blood samples were collected to evaluate GFR, BUN, creatinine, K, sodium, serum creatine kinase, plasma NO and glutathione levels. Kidney specimens were taken to investigate renal cytochrome P450 and for histopathological examination. Cimetidine treatment significantly decreased creatinine, BUN, K, Na, SBP and creatine kinase and increased GFR compared to group 2. L-carnitine exerted similar changes except the effect on K and GFR. NO was significantly decreased while renal glutathione and cytochrome P450 were significantly increased in groups treated with L-carnitine or cimetidine as compared to group 2. Combined treatment further improved renal functions, creatine kinase, oxidative stress parameters and SBP as compared to each therapy alone. The histological changes confirmed biochemical findings. Cimetidine and L-carnitine have protective effect - almost equally- against myoglobinuric renal failure. Using both agents together made the renal injury minimal.

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