COMMENTARY

Iron deficiency anemia treatment in CKD and end-stage kidney disease

Benjamin Gronier, Emma Johnston

Gronier B, Johnston E. iron deficiency anemia treatment in CKD and end-stage kidney disease. J Kidney Treat Diagn. 2022; 5(5):61-62.

ABSTRACT

People with chronic renal illness frequently have iron deficiency, which is a primary factor in the emergence of anemia. For patients with chronic renal disease who have an iron deficit, both oral and injectable iron treatments are available. The most common factors influencing the decision of which agent to use are the therapeutic goals, acceptability, practicality, and response to previous therapy. Iron deficiency anemia must be treated with intravenous iron formulations, especially in patients requiring kidney replacement therapy, due to decreased iron absorption in the gastrointestinal system and a high incidence of gastrointestinal side effects. Newer oral medications might help to get around these restrictions and treat iron deficiency in those who don't need renal replacement therapy. According to this research, patients with chronic renal disease who need kidney replacement therapy may benefit from more aggressive iron replacement in terms of managing anemia and important clinical outcomes including cardiovascular disease and survival.

INTRODUCTION

Anemia associated with chronic renal disease is primarily caused by iron deficiency, which is a frequent consequence of Kidney Disease (CKD). Because of this, treating iron deficiency is essential for the effective management of anemia in people with CKD, especially those who require replacement medication for renal failure. Numerous new tools have been developed as a result of the development of innovative iron supplements to treat iron deficiency in CKD patients. With a focus on more recent therapeutics that have been added for the treatment of iron deficiency as well as new studies that have helped inform best practices for how and when to treat iron deficiency across the spectrum of CKD, this review will provide an overview of the current state of iron repletion strategies in patients with CKD. To counteract the necessary iron losses from the skin and digestive tract, 1 to 2 mg of iron is normally absorbed daily from the food. The majority of iron absorption occurs in the proximal small intestine under strict physiologic control. Separate mechanisms allow gastrointestinal epithelial cells to absorb iron from food in either its heme or nonheme forms. Ferroportin, which is expressed in the basolateral membrane of epithelial cells, is necessary for the release of iron into the blood via the basolateral membrane after absorption. Ferroportin is essential for enabling iron efflux over the basolateral membrane of epithelial cells and from macrophages into the blood plasma since it is the only known iron exporter in mammalian cells. The main hormonal regulator of iron management is hepcidin, a 25-amino acid peptide that is largely produced and secreted into the blood by hepatocytes. It does this via acting on ferroportin. Ferroportin is bound to by hepcidin on the basolateral membrane of enterocytes and macrophages, increasing ferroportin breakdown and endocytosis. As a result, less ferroportin is present on cell membranes, thereby reducing the flow of iron from intestinal epithelial cells or from macrophages and hepatocellular cells, which serve as the body's primary iron storage, into the blood. In the pathogenesis of iron insufficiency in CKD, hepcidin is crucial. Absolute iron deficiency, which is indicated by low iron stores and low circulating iron concentrations, and functional iron deficiency, which is indicated by low circulating iron concentrations in the presence of normal iron stores, are the two main categories of iron insufficiency in CKD.

Editorial Office, Journal of Kidney Treatment and Diagnosis, United Kingdom

Correspondence: Benjamin Gronier, Editorial Office, Journal of Kidney Treatment and Diagnosis, United Kingdom, e-mail kidney@eclinicalsci.org

Received: 12-September-2022, Manuscript No. puljktd-22-5720; Editor assigned: 14-September-2022, PreQC No. puljktd-22-5720 (PQ); Reviewed: 21-September-2022, QC No. puljktd-22-5720 (Q); Revised: 24-September-2022, Manuscript No. puljktd-22-5720 (R); Published: 30-September-2022, DOI: 10.37532/puljktd.22.5(5).61-62.

People with CKD frequently have higher hepcidin concentrations, which is probably caused by a combination of increased levels of systemic inflammation, which promotes hepcidin expression, and lower kidney clearance of circulating hepcidin. In CKD, elevated hepcidin levels prevent intestinal iron absorption and iron release from iron storage sites (macrophages, hepatocytes), lowering the amount of iron available for erythropoiesis and causing anemia. The decreased effectiveness of oral iron replacement in patients with CKD is explained by the hepcidin-induced blocking of iron absorption in the gut, which frequently requires iron replacement therapies that bypass the gastrointestinal tract in patients with CKD. This has also sparked interest in the creation of innovative therapeutics that target the hepcidin secretion-inducing and/or ferroportin-stimulating factors; this subject is covered in a number of great reviews and other papers. Additionally, new inhibitors of hypoxia-inducible factor 1's prolyl hydroxylase may target this pathway by lowering hepcidin levels, which would improve gastrointestinal iron absorption and decrease sequestration of iron in reticuloendothelial stores, both of which would increase iron availability for erythropoiesis. There are numerous oral supplements available to address iron deficiency. Iron is a common ingredient in multivitamins, often delivering 18 mg of elemental iron per dose. Ferrous salts typically make up supplements that only contain iron. Ferrous sulfate, which provides 20% elemental iron per tablet, is the one that CKD patients use the most frequently. Ferrous fumarate (33% elemental iron), ferrous succina-

-te (35% elemental iron), ferrous gluconate (12% elemental iron), and iron polymaltose (28% elemental iron) are additional ferrous salts. When administered at doses of less than 45 mg of elemental iron per day, oral iron supplements typically produce gastrointestinal side effects in 35% to 60% of patients, limiting the ability to replenish iron with high doses of oral formulations alone. For oral ironsupplementation, there are numerous ferric salt formulations in addition to ferrous salts. The only oral iron supplement permitted by the US Food and Drug Administration for the treatment of iron deficiency anemia in people with chronic kidney disease is ferric citrate, which has received the most up-to-date research. Each 1000 mg pill of ferric citrate contains about 210 mg of elemental iron. Other ferric salts include sucrosomial iron, which contains ferric polyphosphate wrapped in a phospholipid bilayer with a sucrester matrix to aid in gastrointestinal absorption, and ferric maltol, which contains a complex of ferric iron and maltol in a 3:1 ratio and delivers 30 mg of elemental iron with each capsule. Sucrosomal iron doesn't need a prescription, which is a possible benefit. Ferric maltol has been examined in people with CKD who do not need kidney replacement therapy and is licensed for the treatment of iron deficiency anemia in people with inflammatory bowel disease.