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Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

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CKD-MBD management: about the new phosphate binder

Seon Deok Hwang
Inha University Hospital, Republic of Korea

CKD-MBD is a common complication seen in patients with chronic kidney disease. It leads to vascular calcification, hyperphosphatemia, and secondary hyperparathyroidism, representing one of the major complications that mutually influence each other. Efforts are being made to reduce phosphate, known as the starting point of CKD-MBD, and recently medications such as ferric citrate and Sucroferric oxyhydroxide have been used. Ferric citrate acts as a phosphate binder, breaking down into ferric iron (Fe3+) in the gastrointestinal tract after oral administration. It then combines with phosphate from food to form insoluble compounds like ferric phosphate (FePO4), reducing blood phosphate levels, which is effective in reducing hyperphosphatemia in chronic kidney disease patients undergoing hemodialysis. Additionally, acting as a phosphate binder, some ferric iron (Fe3+) is absorbed through duodenal cytochrome B (DcytB) into divalent metal transporter 1 (DMT1) and moves into duodenal epithelial cells. Once inside the cells, iron is stored as ferritin or released into the blood as ferrous iron (Fe2+) via the iron transporter ferroportin. The released Fe2+ is oxidized to Fe3+ by hephaestin, forming transferrin, which is then absorbed into the bloodstream, thus having the additional effect of correcting anemia. Furthermore, the structural feature of Sucroferric oxyhydroxide involves 3-valent iron ions encapsulated by sucrose and starch. This structural characteristic stabilizes the 3-valent iron ions with sucrose, minimizing the bioavailability and reducing intestinal absorption of iron. Sucroferric oxyhydroxide binds to phosphate within the digestive tract (mainly in the stomach and small intestine), with 99.8% of the administered dose being excreted unchanged in the feces, thereby reducing phosphate levels.

Keywords: CKD-MBD, Hyperphosphatemia, Phosphate binder