KSN 2019
Seoul, Korea · May 23 - 26

Abstract Submission No. : IL-9107

Ca based phosphate binder vs. non Ca base phosphate binder as a first agent for hyperphosphatemia

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In healthy adults, the homeostasis of phosphate is maintained in a delicate balance of absorption in the intestine, renal excretion, and inflow and outflow from the bone. The fibroblast growth factor 23 (FGF-23) and parathyroid hormone (PTH) play a central role in phosphate homeostasis by regulating NPT2a/2c, a phosphate transporter in the proximal tubule of the kidney. Phosphorus is largely reabsorbed in the proximal tubule after glomerular filtration. PTH reduces phosphorus uptake by reducing NPT2a expression, thereby increasing phosphorus excretion. The mechanism by which FGF-23 acts on NPT2a is unclear but is known to downregulate NPT2a in the proximal tubule. FGF-23 binds to FGF receptor only in the presence of Klotho, which is a cofactor, but it is unclear why FGF-23 mediated phosphorus excretion occurs in the proximal tubule, even though Klotho is mainly expressed in distal tubular cells. Increase in FGF-23 and PTH in early and moderate chronic kidney disease (CKD) increases urinary phosphate excretion. When renal function gradually decreases, urinary phosphate excretion is decreased, and hyperphosphatemia and secondary hyperparathyroidism become worse, which leads to increase of bone resorption and further exacerbation of hyperphosphatemia.

Hyperphosphatemia not only causes vascular calcification and endothelial dysfunction, but also increases CKD progression and increases cell stress and apoptosis. In addition, subsequently increased FGF-23 and PTH levels are related with LVH, renal anemia, immune dysfunction, adipose tissue browning, and skeletal muscle atrophy.

The therapeutic agents of hyperphosphatemia are largely divided into Ca-based phosphate binder and non-Ca phosphate binder. Aluminum hydroxide is rarely used recently due to toxicity and concern or aluminum accumulation. Calcium acetate and calcium carbonate have been widely used in Korea. It is known that calcium acetate is slightly more expensive but has less risk of hypercalcemia than calcium carbonate. When prescribing Ca-based phosphate binder, physicians should pay attention to hypercalcemia and extraskeletal calcification. Non-Ca phosphate binders are typically sevelamer and lanthanum. Several studies have reported that non-Ca phosphate binders contribute to lower mortality and vascular calcification in comparison to Ca-based phosphate binders. Although the global guidelines and trend have recommended non-Ca phosphate binders in view of the cardiovascular outcome, Ca-based phosphate binders have been widely used in Korea because of the insurance standards. Recently, the use of Ca-based phosphate binders is expected to decline sharply as insurance standards for non-Ca phosphate binders are relaxed.

Iron-based phosphate binders have recently been studied and are in the process of being introduced in Korea. It showed non-inferiority to conventional non-Ca phosphate binders in terms of efficacy, and one has been shown to reduce pill burden for iron replacement. Those agents are expected to be a good alternative for hyperphosphatemia in CKD patients.