Overview of the PRN

The Nephrology Practice and Research Network (PRN) is composed of pharmacists, residents, fellows, and students with an interest in nephrology. Our members practice in a variety of settings such as dialysis units, nephrology units and clinics, transplant units and clinics, intensive care units, and internal medicine units. Our mission is to advocate the scope of nephrology pharmacotherapy through excellence in education, clinical practice, research, and involvement in professional nephrology organizations and to ensure optimal patient outcomes. The Nephrology PRN was founded in 1993 with 73 initial members; Gary Matzke (ACCP President 2007–2008) was the first elected chair. Our members are active in both the National Kidney Foundation (NKF) and the American Society of Nephrology (ASN); many are recognized as fellows of these organizations. The PRN is a member of the ASN – Kidney Health Initiative (KHI). The KHI is a group of organizations supporting the mission "to advance scientific understanding of the kidney health and patient safety implications of new and existing medical products and to foster development of therapies for diseases that affect the kidney by creating a collaborative environment in which FDA [U.S. Food and Drug Administration] and the greater nephrology community can interact to optimize evaluation of drugs, devices, biologics, and food products." One of our members, Wendy St. Peter, was recently elected to the KHI Board and will be representing the PRN in her position.

Opportunities and Resources for Resident and Fellow Members of the PRN

The PRN supports an annual travel grant for either a resident or a fellow. Recipients of the travel award are invited to share their nephrology-related research or project at the Annual Meeting PRN Networking and Business Meeting. Service on all of the PRN committees is open to residents, fellows, and students. There are excellent opportunities for networking through the e-mail list, at our annual business meeting, or at gatherings during the NKF and ASN annual meetings. Our PRN member page on the ACCP website has links to locate members who are interested in research and to find protocols, guidelines, and order sets shared by members practicing in nephrology. The PRN welcomes all residents and fellows with an interest in nephrology, even if it is not their primary area of practice.

Clinical Issue – New Iron-Containing Phosphate Binders

Hyperphosphatemia is a common complication of chronic kidney disease (CKD). As GFR (glomerular filtration rate) declines, phosphorus concentrations begin to increase.[1] Phosphorus retention stimulates an increase in fibroblast growth factor-23 (FGF-23) and parathyroid hormone (PTH). Both of these work to decrease the production of 1,25-dihydroxyvitamin D, which is responsible for the absorption of phosphorus from the small intestine. Parathyroid hormone also works in the proximal tubule to decrease phosphorus reabsorption and decrease excretion. However, in patients with CKD, the kidney loses the ability to regulate phosphorus, and therefore, serum phosphorus concentrations increase in addition to PTH and FGF-23.[2] Elevated phosphorus concentrations have been associated with increased mortality and cardiovascular events.[3,4]

Treatment consists of restricting dietary phosphorus, removing phosphorus by dialysis, and initiating phosphate binders. Commonly used phosphate binders are listed in Table 1. In 2013–2014, the FDA approved two new iron-based phosphate binders, sucroferric oxyhydroxide (Velphoro) and ferric citrate (Auryxia) (Table 1). Unlike the calcium-based binders, calcium carbonate and calcium acetate, these products have not been shown to independently increase vascular calcification. Both products have efficacy comparable with that of sevelamer in lowering phosphorus concentrations.[8,9] Because each product contains iron, it has been hypothesized that they would also help with the treatment of anemia of CKD. However, studies of sucroferric oxyhydroxide have shown no effect on hemoglobin. Although sucroferric oxyhydroxide has a lower pill burden than sevelamer, gastrointestinal (GI) adverse

effects may limit its utility.[8] In contrast, evidence suggests that ferric citrate has positive effects as an iron supplement. The 52-week PERFECTED study found that treatment with ferric citrate increased serum ferritin, increased hemoglobin, decreased the number of patients requiring intravenous iron, and decreased erythropoiesis-stimulating agent doses. However, an increase in the number of patients with ferritin concentrations greater than 1500 ng/mL supports the FDA's warning of risk of iron overload with this product.[10]

Although surrogate end points show improvement in the treatment of hyperphosphatemia with both sucroferric oxyhydroxide and ferric citrate and potential benefits in the treatment of anemia of CKD with ferric citrate, clinical outcomes have not been studied.

Table 1. Phosphate Binders

Product	Dose	Availability	Common Adverse Effects
Calcium carbonate	Max dose: 1500–2000 mg of elemental Ca/day 40% elemental Ca	Several dosage forms available	Hypercalcemia
Calcium acetate[5]	Usual dose: 2001–2668 mg with each meal	667-mg oral capsule or tablet 667 mg/5 mL of oral solution	Hypercalcemia, nausea, vomiting
Lanthanum carbonate[6]	Usual dose: 1500–3000 mg/day in divided doses with meals	500-, 750-, and 1000-mg oral chewable tablet 750 and 1000 mg of oral powder	GI intolerance, poor taste
Sevelamer carbonate, hydrochloride[7]	Usual dose: 1600–2400 mg 3x/day with meals	Carbonate: 800-mg tablet or 0.8 and 2.4 g/packet of oral powder for suspension Hydrochloride: 400- and 800-mg tablet	GI intolerance
Ferric citrate	Initial dose: 2 g (420 mg of ferric iron) 3x/day with meals Max dose: 12 g (2520 mg of ferric iron)/day	1-g oral tablet	Dark stools, diarrhea
Sucroferric oxyhydroxide	Initial dose: 500 mg of iron 3x/day with meals Usual dose: 1500–2000 mg/day	500-mg chewable tablet	Diarrhea

	500 mg of elemental iron equivalent to 2500 mg of sucroferric oxyhydroxide		

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