Introduction

Despite advanced technology and regular and efficient dialysis treatment, the prevalence of hyperphosphatemia still is unacceptably high. Nevertheless, a neutral phosphorus balance level can generally be achieved by optimization of dialysis prescription in combination with individualized dietary and medical strategies. Besides increasing the fraction of inorganic phosphate (iP) removed by convection through the application of hemodiafiltration, extension of daily or weekly treatment time is the most promising way to neutralize phosphorus balance. Dietary phosphate restriction, the second corner stone of phosphate management, bears the risk of development of protein malnutrition. Phosphate binders (PBs) effectively reduce intestinal iP absorption, but are mostly dosed inadequately in relation to meal phosphorus content. Phosphate management may be substantially improved by enabling patients to self-adjust the PB dose to individual meal phosphate content, similar to self-adjusting insulin dose to carbohydrate intake by diabetics. A recently developed Phosphate Education Program (PEP) provides simple training tools to instruct patients to eye-estimate meal phosphorus content based on newly defined phosphorus units instead of milligrams. PEP is the first approach applying the concept of patient empowerment in the management of hyperphosphatemia in dialysis patients ***(Gotch et al;2003).***

Hyperphosphataemia is prevalent among chronic renal failure and dialysis patients. It is known to stimulate parathyroid hormone and suppress vitamin D production, thereby inducing hyperparathyroid bone disease. In addition, it may independently contribute to cardiac causes of death through increased myocardial calcification and enhanced vascular calcification. Hyperphosphataemia is also associated

with cardiac microcirculatory abnormalities. Therefore, phosphate control is of prime importance. It is important to control phosphate levels early in the course of chronic renal failure in order to avoid and treat secondary hyperparathyroidism, and cardiovascular and soft tissue calcifications ***(Ritz , 2005).***

Aggressive dietary phosphate restriction among patients with CKD is impractical and could compromise overall nutrition, particularly protein intake. For preventing malnutrition among patients with CKD, the NKF–K/DOQI guidelines recommend a minimum protein intake of 1.2 g/kg per d (approximately 800 to 1000 mg/d phosphorus). As renal function declines, a net positive balance is inevitable, and thus other therapies are required ***(Colondato et al;2005)***

Hemodialysis does not remove significant amounts of phosphorus, mainly because Phosphate exist mainly in the intracellular compartment. During heamodialysis session, serum phosphorus decrease rapidly, reaching hypophosphatemic nadir at about 120 minutes.There is an immediate postdialysis rebound in which the serum phosphorus level can even exceed predialysis value***(Ayus et al; 2005).***

The use of phosphate binders began in the early 1970s, when the importance of phosphorus control was first emphasized. Use of aluminum-containing phosphate binders was the standard of care among patients with ESKD. Aluminum hydroxide was a very efficient phosphate binder. Unfortunately, long-term use was associated with aluminum accumulation and toxicity, manifesting itself as encephalopathy, osteomalacia, microcytic anemia, and myopathy. Subsequently, the use of aluminum hydroxide has

been limited to salvage, short-term therapy or abandoned entirely ***(Shigematsu et al; 2007).***

Calcium salts then emerged as an alternative to aluminum as a phosphate binder. It was well established that calcium salts bound dietary phosphorus, although less efficiently than aluminum. Later, calcium carbonate has been used extensively worldwide because of its efficacy,tolerability,and affordability ***(Qunibi et al;2004).***

Calcium acetate is an alternative to calcium carbonate as a phosphate binder and contains less elemental calcium (25%) than calcium carbonate. GI washout studies have shown that the amount of phosphorus bound per amount of calcium absorbed was almost twice as great with calcium acetate compared with calcium carbonate. Moreover, Mai et al.showed that phosphorus absorption decreased to 40% of the ingested load with calcium carbonate compared with 21.7% with an equivalent amount of calcium acetate. Thus, among patients with ESKD, calcium acetate binds approximately twice the amount of phosphorus per amount of calcium absorbed. This is believed to be attributable to the increased solubility of calcium acetate in both acid and alkaline solutions in vitro ***(Colondato et al; 2005).***

Calcium acetate is well tolerated and has been shown to significantly reduce and maintain serum phosphorus and calcium x phosphorus product levels during a long-term clinical trial ***(Chertow et al:2002).***