EDITORIAL

Calcium-Phosphate Complex in Chronic Kidney Disease

In Chronic kidney disease several biochemical and hormonal abnormalities occurs. Minerals and bone disorders that includes abnormalities in phosphate retention, hypocalcaemia, hyperparathyroidism, increased alkaline phosphatase level and abnormalities in bone turnover. Secondary hyperparathyroidism is the first and most important complication because parathyroid hormone (PTH) secretion is a compensatory mechanism to maintain calcium and phosphate concentration within physiological ranges[1]. Hyperparathyroidism and hypoparathyroidism both interfere calcium and phosphate metabolism[2].

Mineral metabolism has emerged as an important predictor of morbidity and mortality in dialysis patients. Kidney Disease Outcomes Quality Initiative (K/DOQI) clinical practice guidelines for bone metabolism and disease in chronic kidney disease (CKD)[3]. It is recommend that, in Stage 5 CKD, the target levels for calcium (Ca) (corrected for serum albumin), phosphate (P), calcium x phosphate (Ca-P complex) product and PTH levels should be maintained[4].

With regard to the adjusted baseline analysis for total serum calcium, many reports has been published to evaluate the high serum calcium level, the patients with high serum calcium levels are in higher risk of death than those who were within target range (2.10–2.37 mmol/L)[5]. On the other hand several study showed that the adjusted baseline analysis for low serum calcium levels showed no effect on the risk of death[6]. High phosphate level inhibits calcitriol production which in turn causes hypocalcaemia; furthermore uremia may directly affect the function of calcitriol and parathyroid receptors. High phosphate also impairs the calcaemic action of PTH, which is another cause of hypocalcaemia in renal patients. Several research workers suggested that the beneficial effect of dietary phosphorus restriction for the control of secondary hyperparathyroidism in CKD patients[2]. Furthermore clinical observations have suggested that high phosphate may have a direct effect on parathyroid glands. The dietary phosphorus modulates serum PTH independent of the severity of renal failure and this effect is observed even with no changes in serum calcitriol[7].

Chronic kidney disease (CKD) is a powerful risk factor for all-cause mortality and its most common aetiology, cardiovascular mortality[8]. Mineral metabolism disturbances occur very early during the course of CKD but their control has been poor. A number of studies have assessed the relationship between all-cause mortality, CV mortality and events with mineral disturbances in CKD patients, but with considerable discrepancy and heterogeneity in results[9].

So, in chronic kidney disease it is to be consider to maintain the serum calcium and phosphate level and to keep the Ca- Phosphate within < 55 mg^2/dl.

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