KSN 2017 Abstract

KSN-17-P051

The impact of albuminuria on serum phosphate level via modulating tubular reabsorption in chronic kidney disease : result from the KNOW-CKD study

Hyoungnae KIM[1], Min-uk CHA[1], Misol LEE[1], Ki heon NAM[1], Seong yeong AN[1], Jong hyun JHEE[1], Seohyun PARK[1], Hae-ryong YUN[1], Youn kyung KEE[1], Tae IK[2], Jung tak PARK[1], Seung hyeok HAN[1], Shin-wook KANG[1,3], Kyu hun CHOI[1], *Tae-hyun YOO[1]

[1]Internal Medicine, College of Medicine, Institute of Kidney Disease Research, Yonsei University, Seoul, Korea , Korea,South, [2]Internal Medicine, National Health Insurance Service Medical Center, Ilsan Hospital, Goyang, Gyeonggi-do, Korea, Korea,South, [3]Internal Medicine, College of Medicine, Severance Biomedical Science Institute, Brain Korea 21 PLUS, Yonsei University, Seoul, Korea, Korea,South

Objectives : Both albuminuria and hyperphosphatemia are powerful risk factor of cardiovascular mortality in patients with chronic kidney disease (CKD). A recent experimental study demonstrated that albuminuria increases in serum phosphate level by increasing tubular reabsorption of phosphate via enhancing activity of sodium-phosphate co-transporter in proximal tubule and reduced activity of fibroblast growth factor 23 (FGF-23). The aim of this study was to evaluate the relationship between albuminuria and phosphate reabsorption in adult CKD patients.

Methods : We examined 2207 patients who were enrolled in the KoreaN Cohort Study for Outcome in Patients With Chronic Kidney Disease. Tubular maximum reabsorption of phosphate to glomerular filtration rate (TmP/GFR) was calculated using serum and urine phosphate and creatinine values. Serum intact parathyroid hormone (iPTH), FGF-23 and other parameters of phosphate handling were also measured in study subjects.

Results : The mean age was 52.7±12.3 years, and 1,350 (61.2%) were male. The average estimated glomerular filtration rate (eGFR) was 50.4±30.3 mL/min/1.73m2. When we categorized patients by serum phosphate quartiles, the amount of albuminuria, FGF-23 and iPTH levels were progressively increased according to increasing serum phosphate quartiles. In addition, TmP/GFR was paradoxically increased in high phosphate quartiles. In propensity score matching analysis by age, sex, eGFR and co-morbidities, patients in the macro-albuminuria group had significantly higher levels of phosphate ($P < 0.001$) and TmP/GFR ($P = 0.02$) compared to those in patients without macroalbuminuria, whereas FGF-23 and iPTH level were not different

between the two groups. In multivariate linear regression analysis, FGF-23 was significantly associated with TmP/GFR in the group without macroalbuminuria (β = -0.051, P = 0.001), whereas such relationship was not observed in the macroalbuminuria group. (β= -0.015, P = 0.33).

Conclusions : Present study demonstrated that overt albuminuria affects the serum phosphate level through tubular capacity of phosphate reabsorption in chronic kidney disease. Our study can support the pathophysiologic mechanism of phosphate retention, which urinary protein loss reduces the phosphaturic effect of FGF-23 in patients with macroalbuminuria.

Keywords : albuminuria, phosphate, FGF-23, chronic kidney disease