Hyperuricemia is highly prevalent in patients with chronic kidney disease (CKD) and has shown to be a risk factor for the progression of CKD. However, this relationship is still controversial. Although a few studies showed that hyperuricemia was associated with progression of CKD, others failed to show this relationship.

From the Modification of Diet in Renal Disease Study cohort, nondiabetic CKD patients showed that hyperuricemia associated with increased risk of cardiovascular disease and all-cause mortality, but not with kidney failure in long-term follow-up.

And also, in a recent randomized, double-blind, placebo-controlled trial, febuxostat, a novel potent nonpurine-selective inhibitor of xanthine oxidase for oral use, did not improve the decline in kidney function among patients with stage 3 CKD and asymptomatic hyperuricemia compared to placebo. It did not show any statistically significant difference in mean eGFR slope between the two groups.

In a double-blind placebo-controlled study of topiroxostat in hyperuricemic patients with stage 3 CKD with or without gout that examined the effects on serum uric acid levels and urinary albumin excretion, no between-group difference in eGFR change or a reduction in eGFR in the placebo group at completion was found, as was the case with our study.

From the previous trials, the current KDIGO guideline clinical practice recommendation that, “there is insufficient evidence to support or refute the use of agents to lower serum uric acid concentrations in people with CKD and either symptomatic or asymptomatic hyperuricemia in order to delay progression of CKD.”