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Impaired fasting glucose and development of chronic kidney disease in non-diabetic population: A Mendelian randomization study

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Objectives: Diabetes mellitus is a risk factor of chronic kidney disease (CKD); however, the relationship between fasting glucose and CKD remains controversial in non-diabetic population. Therefore, we aimed to evaluate the relationship between genetically predicted fasting glucose and incident CKD in this population.

Methods: This study included 6,354 participants without diabetes and CKD from the Korean Genome Epidemiology Study. The genetic risk score (GRS_9) was calculated using nine genetic variants associated with fasting glucose in previous genome wide association studies. Incident CKD was defined as estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 or proteinuria (≥1+). The causal relationship between fasting glucose and CKD was evaluated using the Mendelian randomization (MR) approach.

Results: The GRS_9 was strongly associated with fasting glucose (β, 1.01; $P < 0.001$). During a median follow-up of 11.6 years, 531 (8.4%) CKD events occurred. However, GRS_9 was not significantly different between participants with CKD events and those without. After adjusting for confounding factors, fasting glucose was not associated with incident CKD (odds ratio [OR], 0.991; 95% confidence interval [CI], 0.980–1.003; $P = 0.139$). In the MR analysis, GRS_9 was not associated with CKD development (OR per 1 standard deviation increase, 1.179; 95% CI, 0.819–1.696; $P = 0.376$). Further evaluation using various other MR methods and strict CKD criteria (decrease in the eGFR of ≥ 30% to a value of < 60 mL/min/1.73m^2) found no significant relationship between GRS_9 and incident CKD.

Conclusions: Fasting glucose was not causally associated with CKD development in non-diabetic population.