Hyperuricemia may be related to contrast-induced nephropathy after percutaneous coronary intervention

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Dear Editor,

We have read with great interest the recently published article titled, ''The relationship between hyperuricemia and the risk of contrast-induced nephropathy after percutaneous coronary intervention in patients with relatively normal serum creatinine'', by Yong Liu et al. (1). In that well-presented paper, the authors investigated the value of hyperuricemia in predicting the risk of contrast-induced nephropathy (CIN) in patients with relatively normal serum creatinine who are undergoing CIN. They demonstrated that CIN might develop in patients with normal creatinine levels. Hyperuricemia is a significant and independent predictor of CIN after percutaneous coronary intervention (PCI) and results in significantly increased in-hospital mortality and CIN incidence, which requires renal replacement therapy after PCI. Thus, despite having normal baseline creatinine levels, patients with hyperuricemia, an intra-aortic balloon pump, emergent PCI, or older age should receive more comprehensive renal prophylaxis to reduce the occurrence of CIN.

CIN has been recognized as a serious complication of PCI and may cause increased morbidity and mortality. CIN is one of the most important reasons for hospital-acquired renal failure and can cause prolonged hospitalization, increased cost and incidence of renal and cardiovascular events, and mortality. Elderly patients have a higher risk of CIN because of decreased renal reserve. In addition, some factors, such as an estimated glomerular filtration rate (eGFR) of <60 mL/min/1.73 m^2, a left ventricular ejection fraction $<45\%$, diabetes mellitus, hypotension, anemia, emergency PCI, myocardial infarction history, and a contrast agent dose >200 mL have been identified as risk factors for CIN after PCI (2). Additionally, alcohol consumption and the arterial blood pressure level before contrast exposure may be associated with CIN. Hypertriglyceridemia, metabolic syndrome, an impaired fasting glucose level, and multivessel disease may also be associated with a higher incidence of CIN (3). Some medications, including renin-angiotensin-aldosterone system medications, may also be related to CIN (4). It would have been interesting for the authors to provide information about these factors in this study.

Finally, the authors used the Cockcroft-Gault equation to measure eGFR. The Cockcroft-Gault equation may underestimate eGFR in younger age groups and may overestimate eGFR in older individuals compared with the Modification of Diet in Renal Disease (MDRD) formula (5). Although the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) recently published an equation for eGFR using the same variables (serum creatinine level, age, sex, and race) as the MDRD formula, the CKD-EPI equation more accurately categorized individuals with respect to long-term clinical risk (6). In addition, even mild chronic kidney dysfunction, defined as an eGFR <90 mL/min, and dehydration were risk factors for CIN. For this reason, it may be useful to include the results using the CKD-EPI equation and accounting for mild chronic kidney dysfunction and dehydration as risk factors for CIN.

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