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Editorial

Contrast-induced Acute Kidney Injury and Inflammation

Kyeong Ho Yun, MD, PhD

Department of Cardiovascular Medicine, Regional Cardiovascular Center, Wonkwang University Hospital, Iksan, Korea

▸ See the article "Association of Syntax Score II with Contrast-induced Nephropathy and Hemodialysis Requirement in Patients with ST Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention" in volume 48 on page 59.

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Correspondence to
Kyeong Ho Yun, MD, PhD
Department of Cardiovascular Medicine, Regional Cardiovascular Center, Wonkwang University Hospital, 895, Muwang-ro, Iksan 54538, Korea.
E-mail: dryunkh@gmail.com

ORCID iDs
Kyeong Ho Yun
https://orcid.org/0000-0003-4911-8854

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Contrast-induced acute kidney injury (CI-AKI) is one of important complication after coronary angiography and percutaneous coronary intervention (PCI). CI-AKI has been found to be strongly associated with morbidity and mortality of the patients. Furthermore, CI-AKI may not be always reversible, and may be associated with development of chronic kidney disease in long term.[1] Therefore, risk scoring and prevention for development of CI-AKI is important during invasive procedures.

In the current issue of the *Korean Circulation Journal*, Rencuzogullari et al.[2] reported the result of multi-center, retrospective study about relationship between the development of CI-AKI and Syntax score II in patients with ST elevation myocardial infarction who treated with primary PCI. They found that both Syntax score and Syntax score II were significantly higher in patients with CI-AKI. Moreover, every Syntax score II increases by 1 unit, the risk of CI-AKI increases by 3% in multivariate analysis. Although, they performed retrospective study, they suggested that Syntax score II would be comprehensive risk score including anatomical and clinical risk factors.

Exact pathophysiological mechanism of CI-AKI is not clear. However, the most important elements of mechanism of CI-AKI seems to be vasoconstriction and oxidative stress followed by renal medullary hypoxia and direct tubular toxicity by contrast media.[3] All of these processes caused to inflammation. To prevent hypoxia and direct toxicity, fluid administration with or without antioxidants or sodium bicarbonate is important work. Moreover, to prevent inflammatory process, statin therapy would be important. Recent meta-analysis reported that pretreatment with high-dose statin had 55% lower risk of CI-AKI compared with placebo.[4] In present study, use of statin was significantly higher in patients without CI-AKI then those with CI-AKI (17.8% vs. 10.8%, p=0.026). In previous my study, the benefit of statin on CI-AKI prevention was distinct in patients with evidence of high inflammatory status such as myocardial infarction or high baseline C-reactive protein (CRP) levels.[5] The association with inflammation and CI-AKI could explain why anatomical lesion extent, severity and complexity, and baseline white blood cell count and CRP levels were correlated with development of CI-AKI. Moreover, if the authors should use statin more frequently, they can reduce the development of CI-AKI.

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Finally, I would like to congratulate the authors for this study. I also comment further work about the association between inflammatory status and CI-AKI. Statin could have reno-protective effect in patients with PCI, and may reduce CI-AKI in patients with high Syntax score or Syntax score II.

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