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Editorial

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Iso-osmolar Iodixanol Is Better than Low-osmolar Contrast for CIN Prevention. And Then?

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▸ See the article "Contrast Induced Nephropathy and 2-Year Outcomes of Iso-Osmolar Compared with Low-Osmolar Contrast Media after Elective Percutaneous Coronary Intervention" in volume 51 on page 174.

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Contrast induced nephropathy (CIN) occurs sometimes and is presumed to influence on the clinical outcomes. Accordingly, lots of interventional methods with medication or device have been tried.[1)2)] Among them, peri-procedural hydration, contrast dose reduction and selection of contrast are suggested as cornerstone for preventing of CIN.[3)] Regarding the contrast media, there have been controversies whether iso-osmolar contrast media (IOCM) is superior to low-osmolar contrast media (LOCM) although both are widely used in coronary procedures. IOCM has same osmolality as that of blood (290 mOsm/kg H_2O) and the LOCM has higher one than that of blood (600–900 mOsm/kg H_2O).[4)] "Low-osmolar" means the lowered osmolality as compared to that of previously developed high-osmolar contrast media (1,500–2,000 mOsm/kg H_2O) which are no longer used for their adverse effect including higher CIN rate. In other words, the term of "low" may come from the development history of contrast media, i.e., LOCM comes earlier than IOCM (currently iodixanol is the only one) which has same osmolality as blood.

We can expect the IOCM would be better in CIN prevention than LOCM in view of "osmolality" which could induce osmotic diuresis and hinder renal blood flow, one of the main mechanism of developing CIN.[5)] Some randomized clinical trials and meta-analysis have supported this concept of IOCM's superiority over LOCM in preventing CIN.[6)7)] But the iodixanol (IOCM)'s chemical structure was dimer which is more viscous than monomeric LOCM (e.g., iohexol, iopromide, iomeprol, isovue). More viscosity can provoke more CIN due to renal medullary blood flow limitations.[8)] Thus, comparison of more viscous dimeric IOCM (iodixanol) with higher osmolar LOCM is very practical problem and of importance.

In this issue of *Korean Circulation Journal*, Du et al.[9)] reported results regarding the comparison between IOCM and LOCM in CIN occurrence. They used retrospective data and performed propensity score matching analysis from one center in patients receiving coronary intervention irrespective of baseline renal function. They reported that CIN rate was lower in IOCM users than in LOCM users, 1.5% (15/979) vs. 4.0% (39/979), respectively. IOCM use was an independent protective determinant for CIN occurrence (odds ratio, 0.393; 95% confidence interval, 0.214–0.722; p=0.003). But all-cause mortality did not differ after multivariate Cox regression analysis at 2 years.

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The study result is well accordance to the previous ones in that IOCM can be better than LOCM[6)7)] but it has some limitations. Firstly the researchers enrolled the patients irrespective of their renal function although it is well known that the patients with impaired renal function like estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m^2 are prone to experience CIN. Despite of overall positive results with IOCM in CIN prevention, their subgroup analysis with the cut-off value of eGFR <60 mL/min/1.73 m^2 did not find the benefit with IOCM other than LOCM. This paradoxical data can mainly be attributed to small number of patients with renal dysfunction enrolled.

Secondly, LOCM that they used was mainly iohexol which have data of inferiority to IOCM.[6)] LOCM other than iohexol had better be included more for clarification.

Thirdly, the exact hydration dose ought to be presented in both groups because hydration prophylaxis is the first and most important measures in CIN prevention and could affect the CIN rate.

Fourthly, other clinical outcome measures pursuing renal prognosis like renal replacement therapy, renal function declining, persistent renal impairment as well as cardiovascular outcome measures make the result more concrete and definite.

Recent research trend in the CIN fields are to investigate the true role of CIN in clinical outcomes, i.e., whether the CIN is a disease maker or marker. The underlying condition like acute coronary syndrome, plaque burden, inflammation, volume depletion and renal dysfunction itself directly affect the poor prognosis as well as influencing temporary serum creatinine elevation after contrast procedure. The concept that the transient serum creatinine elevation per se, at best, minimally affect the clinical outcome and the major determinant is underlying risk sharing the CIN risk is rising.[10)]

Thus, future study on the CIN should focus on the CIN's status and the role in clinical outcomes before searching for CIN prophylaxis which could result in futile.

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