Angiology
2014, Vol. 65(3) 225-226

DOI: 10.1177/0003319713483544
ang.sagepub.com
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"Is It possible to Prevent Acute Kidney Injury in the Patients Who Underwent Contrast Medium?"

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We thank Dr Fidanci and colleagues for their comments[1] on our article "Neutrophil gelatinase-associated lipocalin as an early marker of contrast-induced nephropathy after coronary angiography."[2] With increasing coronary contrast medium studies, contrast-induced nephropathy (CIN) has become a major challenge to both cardiologists and nephrologists, because it has longer term implications beyond acute kidney injury such as increased long-term mortality, more rapid progression of underlying chronic kidney disease (CKD), and increased health care costs.[3] Hence, biomarkers that can predict or diagnose CIN earlier than serum creatinine are vital.

We agree that prevention of CIN is as important if not more so than just early diagnosis. Although it was not our objective to study preventive measures of CIN in this article, we wish to clarify the background to the prophylactic protocol adopted by our center since 2008. In 2004, we compared intravenous (IV) normal saline with IV normal saline plus IV N-acetylcysteine (NAC) in patients with CKD undergoing elective coronary angiography and found the frequency of CIN to be 20% (6 of 30) versus 2.9% (1 of 34), respectively ($P = .04$).[4] In a subsequent randomized controlled study (RCT) of oral NAC plus IV normal saline or oral NAC plus IV sodium bicarbonate ($NaHCO_3$) hydration in a similar patient cohort, the frequency of CIN was 14.3% (4 of 28) versus 31% (9 of 29), respectively, albeit not significant ($P = .13$; UKMMC project, unpublished data 2006). In the current study protocol written back in 2009, our patients with CKD received IV saline and oral NAC. The frequency of CIN (11%) was similar to that of the 2006 study.

Although several reviews and meta-analyses, especially more recent ones by Hoste et al,[5] Kunadian et al,[6] and Jang et al,[7] have demonstrated a significant benefit of $NaHCO_3$ hydration, others have shown the contrary.[8,9] Hung et al opined that this issue remained uncertain but recommended that bolus $NaHCO_3$ be used as CIN prophylaxis in emergency cases.[10] Gomes et al in a recent large multicenter RCT ($n = 301$) demonstrated that $NaHCO_3$-based hydration was not superior to NaCl to prevent CIN.[11] At the time when our article was published in *Angiology*,[2] Markota et al also reported that using oral Na/K citrate to alkalinize urine pH may be effective for this purpose provided the urine pH was >6.[12] However, larger, more comprehensive, multicenter, randomized studies are required before this question of best prophylactic regimen can be resolved decisively. In any case, we hope to further explore this issue in an upcoming article.

Of the 3 major races (Malay, Chinese, and Indian) in our study, Indian ethnicity appeared at increased risk of CIN. All these patients were elderly females who, in addition to CKD, also had multiple traditional cardiovascular risks. These in concert with their genetic make up could have predisposed them for CIN. We agree with Fidanci and colleagues that gene polymorphism may play a role in CIN development as explored by Chang et al.[13]

In conclusion, further work is needed to resolve the problem of CIN prevention in these complex patients.

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