

Are the current guidelines on contrast-induced nephropathy prevention superfluous?

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To the editor,

Moos *et al.* prospectively evaluated the risk of contrast-induced nephropathy (CIN) in 998 patients undergoing intravenous contrast-enhanced computed tomography (CECT).¹ They conclude that 'extensive CIN prevention guidelines seem superfluous'.

Unfortunately, this conclusion is not supported by the data. The authors calculated that only 5.8% of the patients who were referred for CECT were at high risk for CIN using the Dutch guideline criteria. It is questionable if the data, obtained in an Academic Hospital, can be extrapolated to the population in a general hospital. Still, even the 5.8% percentage would implicate that in the Netherlands yearly 5000 patients at risk for CIN are evaluated by CECT. The introduction of a lower value of estimated glomerular filtration rate (eGFR) as threshold for defining risk would decrease the number of patients at risk, but not obviate the need for screening to detect the high-risk patient. As stated in the current clinical practice guidelines, ordering eGFR in all patients undergoing CECT is most efficient and considered cost-effective when compared with selection of patients based on history, drug use etc.

The authors conclude that the incidence of CIN in the studied population is low. This is a remarkable and incorrect conclusion. In our view, a diagnosis of CIN (or exclusion thereof) requires a valid serum creatinine value after contrast administration. In fact, the authors measured a follow-up serum creatinine in only 18 of 58 high-risk patients. CIN was found in two patients, with a calculated incidence of CIN of 11%, higher than reported in studies that followed the guidelines. The incidence of CIN was 9% (1/11) in hydrated patients versus 14% in non-hydrated patients. The low power of the study explains the lack of significance. Moreover, the study was uncontrolled, and thus biased by confounding by indication, with patients at highest risk more likely to receive therapy.

The study is also underpowered to reliably evaluate the need for dialysis. Moreover, long-term effects were not addressed, which are relevant in view of studies showing that episodes of acute kidney injury contribute to a persistent loss of kidney function and a faster subsequent rate of decline in kidney function.²

We agree that the current guidelines for prevention of CIN should be reconsidered. However, the study by Moos *et al.* provides no guidance. When rewriting the guidelines the Hippocratic injunction 'primum non nocere; above all, do no harm' should be kept in mind. Indeed, prophylactic hydration with intravenous saline solution may cause pulmonary oedema; hydration with sodium bicarbonate, in the amount that was introduced by Merten *et al.*,³ has been shown to be at least as effective as the hydration with saline solution and has a substantially lower risk of pulmonary oedema.⁴

We agree with the authors that a randomised control trial, comparing at-risk patients receiving preventive hydration with at-risk patients not receiving preventive hydration, is necessary. This study should take into account both short-term and long-term effects in order to determine which patients benefit from such preventive measures.

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