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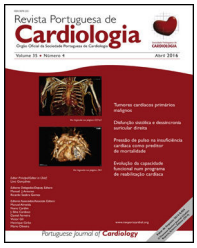

EDITORIAL COMMENT

Contrast-induced nephropathy: Can we better predict and prevent it?

Nefropatia induzida por contraste: poderemos prever e preveni-la melhor?

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Contrast-induced nephropathy (CIN) is a major concern in daily practice for everyone working in interventional cardiology.

The use of contrast agents has been growing due to the increasing number of procedures that use them, such as: structural heart therapies and primary angioplasty for ST-segment elevation myocardial infarction (STEMI). Contrast-induced nephropathy has a major impact on prognosis after primary percutaneous coronary intervention (PCI).[1,2] In this particular setting, it is problematic due to the clinical instability of the patient and the impossibility of prevention in an emerging situation.[3]

Contrast-induced nephropathy is a well-known cause of acute renal failure but the underlying mechanisms are yet to be fully understood.

The evidence is in favor of a combination of direct toxic effects on tubular epithelial cells and renal ischemia as the pathogenetic role. The generation of reactive oxygen species, which in turn scavenge nitric oxide, further increases hypoxia.[4,5]

Identified risk factors for CIN are the baseline renal function itself, diabetes, acute myocardial infarction, shock and the volume of contrast medium administered during the procedure.[4]

Among patients with STEMI, the role of contrast agents has been questioned in acute kidney injury (AKI).[6] In this study, Caspi et al. concluded that the risk for AKI was similar among STEMI patients regardless of exposure to contrast material. Independent predictors of AKI in patients who underwent primary PCI included age ≥70 years, insulin-treated diabetes, diuretic therapy, previous infarction, baseline estimated glomerular filtration rate, and variables related to the presence of pump failure (higher Killip class, intra-aortic balloon pump use) and reduced left ventricular ejection fraction but not contrast agent dose.

In this issue Fatih Aksoy et al.[7] assessed the association between total oxidant status (TOS), total antioxidant capacity (TAC) and CIN in patients with STEMI. They assessed a cohort of 341 patients. In a multivariate regression analysis, they concluded that high-sensitivity C-reactive protein, uric acid and oxidative status index predicted the development of CIN. They also suggest that incorporating these variables into existing risk scores like the Mehran risk score[8] could improve its accuracy. Nevertheless, only 16.7% of these

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patients developed CIN, which the authors acknowledged could have influenced their results.

The results suggest, nonetheless, that TOC, TAC and oxidative stress index are associated with the development of CIN, reinforcing the possibility that oxidative stress may be partially responsible for the development of CIN, as already suggested by others.[9,10]

Preventive measures for CIN, such as fluid administration, pharmacological strategies, use of different contrast agents and renal replacement therapies, have been extensively assessed in many trials.

There seems to be some consensus on fluid administration as a means of protecting from CIN, but the results from trials are conflicting.[11,12] The addition of sodium bicarbonate may provide additional kidney protection by alkalinizing renal tubular fluid and thereby minimizing tubular damage.[13]

Fluid administration possibly associated with furosemide forced diuresis seems to be a good strategy, although there is the risk of hypovolemia. The fear of giving too much volume, mainly in patients with low ejection fraction can, on the other hand, lead to suboptimal protection. Renal Guard, a device enabling volume administration to be balanced with urine output, showed that a high urine output was achievable, while avoiding hypovolemia, thus protecting from CIN.[14]

Randomized trials with N-acetylcysteine had conflicting results but this drug can have a protective effect.[15]

The authors tried to identify further markers of increased risk of AKI. Still, measuring TOC and TAC in daily practice does not seem feasible. The result of this study may however contribute to further investigation on the mechanisms of CIN and possibly lead to novel therapeutic options to prevent CIN.

Currently, hydration and possibly sodium bicarbonate/N-acetylcysteine seem to be the best and only way to prevent CIN.

Conflicts of interest

The author has no conflicts of interest to declare.

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