



EDITORIAL COMMENT

Predicting contrast-induced nephropathy in patients with acute myocardial infarction: Can it be avoided?



Previsão de nefropatia induzida por contraste em pacientes com enfarte agudo do miocárdio: pode ser evitado?

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Copeptin, the C-terminal part of pro-arginine vasopressin, has been extensively analyzed in different settings as a marker of acute severe biological stress, and has been linked to worse heart- and kidney-related clinical outcomes.¹

In their article published in the current issue of the *Journal*, Yildirim and Cabbar² hypothesized that copeptin is a sensitive biomarker to predict acute kidney injury after primary percutaneous coronary intervention (PCI) and found an association between copeptin levels and contrast-induced nephropathy (CIN) in patients with ST-elevation myocardial infarction (STEMI). They conclude that copeptin level at hospital admission has added value as an independent predictor of CIN.

In an era when older and sicker patients, with more complex heart disease, are being treated percutaneously with more complex procedures, it is fair to assume that more contrast media will be used. Even if currently available contrast media are safer than previous types, in older patients with glomerular filtration rate below 40 ml/min, CIN should be always an important concern for every interventionalist. It can affect as many as 25% of STEMI patients treated with primary PCI,³ it is the third most common cause of acute renal failure in hospitalized patients,⁴ and has a major

impact on patient prognosis as well as on morbidity and quality of life.^{5,6} These reasons are sufficient to bring CIN to the forefront of our concerns every time we decide on a contrast-based procedure, whether diagnostic or therapeutic.

The clinical value of Yildirim and Cabbar's results in preventing CIN in the setting of STEMI is doubtful, since primary PCI is an emergent procedure in which the main concern is to obtain TIMI 3 flow in the culprit lesion as soon as possible, regardless of previous blood marker results, which are usually not available. But the availability of quick results from point of care testing⁷ may in the future be helpful in choosing safer treatment strategies for STEMI, as in the current controversy surrounding multivessel PCI in STEMI,⁸ in which it could lead to safer procedures, postponing non-culprit multivessel PCI, if possible.

The question is whether such results also apply to other clinical settings such as non-STEMI or even elective coronary procedures. It is reasonable to assume that they may, although further research is certainly needed. In such scenarios, upfront knowledge of an increased risk for CIN could be an important factor in the decision-making process, avoiding further acute kidney injury and CIN, as the real incidence of CIN in these patients may be underestimated due to the fact that they are discharged early, long before the 3-4 day peak incidence of CIN.⁹

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Yildirim and Cabbar have provided a tool to predict CIN after PPCI. Whether it has a better performance than the current score by Mehran et al.³ is still unknown, but is certainly simpler, a good predictor of adherence by interventionalists.

Conflicts of interest

The author has no conflicts of interest to declare.

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