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Contrast induced nephropathy an elusive disease entity – More questions than answers

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Contrast-induced nephropathy (CIN) is defined as an acute deterioration in kidney function associated with the administration of iodinated contrast media, in the absence of another etiology [1,2]. This type of acute kidney injury is frequently encountered as a complication of percutaneous coronary intervention (PCI) with a reported incidence of as high as 14% and is associated with adverse short and long-term outcomes [1,3].

The possibility of CIN occurrence may impact clinical decision making at the catheterization laboratory as patients at high risk may undergo additional preventive measures, a delayed intervention strategy, staged or event-tailored procedures, and a prolonged follow-up [4,5]. As contrast volume used has been identified by several studies as a risk factor for CIN development [1] it is logical to hypothesize that patients undergoing more complex procedures associated with longer catheterization sessions and increased amount of contrast volume used, are prone to this type of acute kidney injury. Furthermore, this group of patients characterized by demanding lesion angiographic characteristics, unfavorable hemodynamic conditions and higher comorbidity burden in view of the associated high CIN risk, represent an ideal population among which a scrutinized decision-making with regards to procedural strategy or renal protective measures may be more beneficial. In sharp contrast, there is a scarcity of published data regarding CIN incidence in patients undergoing complex PCI procedures.

In this issue of the "International Journal of Cardiology" Azzalini et al. present data on the incidence of CIN in 2660 patients undergoing complex PCI procedures [6]. Complex PCI was defined according to an expanded version of a recently published definition, as a procedure with at least one of the following characteristics: 3 vessels treated, ≥3 stents implanted, bifurcation treated with two stents, total stent length > 60 mm, chronic total occlusion PCI, saphenous vein graft PCI, left main PCI, protected PCI or rotational or laser atherectomy [7]. CIN was defined as an increase in post-PCI creatinine of ≥0.3 mg/dl or ≥50% from baseline.

The authors reported similar CIN incidence between complex vs. non-complex PCI patients (12.1% vs. 11.5%, $p = 0.630$), as well as similar incidence rates of patients requiring in-hospital dialysis (0.5% vs. 0.2%, $p = 0.250$). Furthermore, complex PCI procedures were not associated with increased risk of CIN in multivariable analysis. However, increased rates of acute kidney injury were reported in subjects presenting with acute coronary syndromes, in cases presenting extreme procedural complexity (>4 complexity characteristics), and in those undergoing protected PCI (use of mechanical circulatory support device). As discussed by the authors this observed association could lie on the increased incidence of contrast volume used in these patient sub-groups as well as on the frequently encountered hypotensive episodes during the aforementioned clinical settings.

Certain limitations of the present study warrant mentioning before interpreting the reported results. Firstly, the study is inherent to various biases associated with the retrospective, observational, single-center design of the study. Secondly, the definition of complex PCI procedure was more or less arbitrary including characteristics that could be associated with a simple, straight-forward procedures such as saphenous vein graft revascularization or not including characteristics suggestive of complexity such as procedural time. Moreover, the time-frame of post-PCI creatinine assessment spanned only between 24 and 72 h. It is well known that CIN typically manifests within 13 days of contrast medium administration, peaks within 3–5 days and resolves within 10–21 days [8,9]. Therefore, there is a possibility of not capturing acute renal injury events happening at a later stage [10]. Another limitation is that for the most part of the analysis only a relative 'liberal' definition of acute kidney injury was used. There is a considerable number of alternative definitions and cut-off values for serum creatinine levels (absolute or relative changes) used to define CIN in current literature. It is now established that the observed incidence as well as its association with clinical outcomes depend on the definition used [1,9]. Finally, there is a possibility of selection bias as the under-investigation patient population represents roughly 45% of the initially included study population. Although the authors present data of the non-included patients (patients with no post-PCI creatinine available -n = 3163) there are differences in several clinical or angiographic characteristics suggesting of possible confounding.

In conclusion, we should congratulate Azzalini et al. for elucidating the incidence rate of CIN in a group of patients with complex PCI procedures who are often not included in previously published clinical trials.

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Furthermore, their results suggest that all complex PCI procedures are not similar relevant to CIN risk and that certain sub-groups (acute coronary syndrome patients, highly complex procedures, and a hypotensive clinical setting requiring a mechanical circulatory support device) could benefit more from staged- or carefully planned procedures.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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