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Contrast “induced” versus “associated” acute kidney injury: take care with the definition

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Dear Editor,

We have read with great interest the large retrospective study by Hoste et al. [1] on the epidemiology in ICU patients of acute kidney injury (AKI) after radiological procedure with parenteral administration of contrast medium (CM). In accordance with recent smaller published series using different case-mix populations [2, 3], they found a 16 to 22% incidence of AKI according to the definition used. They also identified risk factors and underlined the negative impact of such subsequent AKI on patient outcomes, including the need for renal replacement therapy, length of ICU and hospital stays, and mortality.

However, because of the well-known multifactorial origin of AKI in the ICU, the authors used the term contrast-associated AKI (CA-AKI) instead of the generally accepted term contrast-induced AKI (CI-AKI). In their study, no restricted inclusion criteria were used to assess kidney function stability before the radiological procedure. Thus the impact of CM injection on subsequent renal function impairment remains doubtful for the following reasons: first,

patients who developed CA-AKI were more critically ill at ICU admission as assessed by higher APACHE II score, and they are likely to have more organ dysfunction at baseline as assessed by the higher rate of patient under vasopressors and mechanical ventilation. The evolution of the Sequential Organ Failure Assessment (SOFA) score [4] between ICU admission and CM infusion is lacking and it would be very interesting to compare the delta SOFA score of patients with either CA-AKI or no CA-AKI. Second, serum creatinine levels increased between ICU admission and inclusion in the “CA-AKI group”, whereas it decreased in the “no CA-AKI group”. Thus, we are unclear about either the natural history of renal function in cases or the true effect of CM administration on kidney function in these more severely ill patients. Adding the changes in serum creatinine concentrations within 48–72 h before CM administration would be very informative to evaluate the clinical stability of the kidney function before the radiological procedure. And third, no control group of patients who were submitted to CT without CM was analyzed and compared with their population. This is a major flaw of the study because an increase in serum creatinine concentration has already been described in non-critically ill patients who had a CT scan without CM administration [5]. Performing such analyses would allow one to estimate the attributable impact of CM injection on renal function impairment. Finally, in unselected and non-stabilized critically ill patients, it might be thought that CA-AKI and its relatively poor outcomes more likely reflect the initial severity of the acute disease as well as the secondary clinical impairment rather than a direct relationship with CM administration.

Altogether, CI-AKI in the intensive care setting remains unclear but emerges as a real hazard event depending on various risk factors. We think that a consensual definition of CI-AKI in the ICU is necessary to homogenize the future literature on this topic, and that further prospective well-designed studies on CI-AKI incidence and prevention in critically ill patients are now warranted.

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