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Very early creatinine changes and 30-day mortality after cardiac surgery

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Editor,

With interest we have read 'Very early changes in serum creatinine are associated with 30-day mortality after cardiac surgery' by Bernardi *et al.*[1] In their observational cohort study among 7651 patients undergoing elective cardiac surgery [40% coronary artery bypass grafting (CABG), 6% off-pump CABG, 34% valve surgery and 20% combined procedures] they demonstrated an association between a rise in serum creatinine and postoperative 30-day mortality, which persisted after adjusting for fluid balance. The incidence of acute kidney injury (AKI) was 10, 2 and 6% (AKI stage 1, 2 and 3, respectively), and AKI necessitating renal replacement therapy (RRT) occurred in 5% of the patients. The authors demonstrated that very early and minimal changes in serum creatinine (0 to $<26.5\,\mu mol\,l^{-1}$, measured within 120 min after cardiac surgery), were relevant to outcome. The authors suggested that: 'Clinicians paying attention to such early increases in serum creatinine (SCrea) at least $26.5\,\mu mol\,l^{-1}$ may avoid the evolution of complications and further renal damage'. We agree with the authors that small changes in serum creatinine are relevant to outcome, which is in line with the results from our earlier studies based on first week serum creatinine changes after cardiac operations.[2] In addition, we demonstrated that the currently used AKI classification underestimates long-term mortality risk after cardiac valve operations. We revealed that a peri-operative rise in serum creatinine of more than $26.5\,\mu mol\,l^{-1}$ or 50% as compared with baseline (KDIGO AKI criteria: AKI 1) was associated with long-term (up to 17 years follow-up) all-cause mortality (hazard ratio 2.27, P less than 0.05 for valve; hazard ratio 1.65, $P < 0.05$ for valve + CABG; hazard ratio 1.56, $P < 0.05$ for CABG). Moreover, after valve operations, even a small rise in serum creatinine of at least 10–25% (i.e. below the threshold for AKI) was also strongly associated with long-term mortality (hazard ratio 1.39, $P < 0.05$), which was not the case after CABG operations. Although Bernardi *et al.* included the type of surgery as covariate in their regression analysis model, it is unclear whether the association between peri-operative changes in serum creatinine and mortality were similar or diverging for different types of cardiac operations, for example, CABG, valve or combined operations. In our work, we proposed to use a cut-off of serum creatinine increases of 10% to identify patients at risk of long-term mortality after valve surgery. How should the increased mortality among patients with a rise in serum creatinine of less than $26.5\,\mu mol\,l^{-1}$ be used in clinical practice? Finally, the authors discussed several factors (i.e. fluid changes, creatinine production and clearance) affecting serum creatinine level and concluded that patients in the group with the largest rise ($>26.5\,\mu mol\,l^{-1}$) upon ICU admission had AKI per definition. If serum creatinine production is not significantly affected by surgery, would adjusting the perioperative rise in serum creatinine for the time between both measurements allow even more precise identification of patients at risk of mortality?

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References

1 Bernardi MH, Ristl R, Neugebauer T, *et al.* Very early changes in serum creatinine are associated with 30-day mortality after cardiac surgery: a cohort study. *Eur J Anaesthesiol* 2020; **37**:898–907.
2 Bouma HR, Mungroop HE, de Geus AF, *et al.* Acute kidney injury classification underestimates long-term mortality after cardiac valve operations. *Ann Thorac Surg* 2018; **106**:92–98.

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Reply to: very early creatinine changes and 30-day mortality after cardiac surgery

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Editor,

We thank Bouma *et al.* for their comments[1] about our recently published article on very early creatinine changes and impact on mortality after cardiac surgery.[2] We have read with interest the comments and remarks they made.