LETTER







Peri-operative fluid strategy and post-operative acute kidney injury in cardiac surgery patients: any role for pre-operative statin therapy?

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Acute kidney injury (AKI) is a common complication associated with increased morbidity and mortality in cardiac surgery patients. In this context, the recent paper by Kim et al., showing that a peri-operative liberal 'saline- and starch-based' fluid management significantly enhanced the incidence of severe post-operative AKI in off-pump cardiac surgery patients, is to be acclaimed [1].

With regard to the potential culprit(s) triggering this AKI, the authors pointed at 'usual suspects' such as excess chloride and intrinsic starch-induced renal tubular lesions. However, it is noteworthy that patients in the atrisk group were less likely (p = 0.001!) to be receiving statins before surgery [1]. Statins may prevent kidney injury through inhibition of post-operative inflammatory processes. Compared with statin-naive subjects, cardiac surgery patients taking statins indeed had reduced levels

of circulating C-reactive protein, tumour necrosis factor alpha, myeloperoxidase, and pro-inflammatory interleukin (IL)-1, IL-6, and IL-8 and higher concentrations of the anti-inflammatory IL-10 [2]. The largest metaanalysis to date, including nearly 60,000 cardiac surgery patients, revealed a 13 % reduction of post-operative AKI in patients pre-operatively treated with statins [3].

Whether statins embrace the kidney as friend or foe is debatable but probably depends upon the type of study cohort (e.g. septic vs. non-septic), the intervention (e.g. surgery), and pre-existing chronic kidney disease. For example, agent-dependent 'high potency' statin doses increase the risk of AKI in a general patient population [4] whereas the use of corresponding high doses in cardiac surgery patients appears to exhibit a reno-protective effect [5].

Authors' response

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We thank Professor Honore and colleagues for their interest in our article [1] and their comment. Statin has been widely used in patients with cardiovascular disorders. We agree that the patients in the control group were less likely to be receiving statin before the surgery, which could have affected our results. However, although some clinical studies have suggested a renoprotective effect of statin in patients undergoing cardiac surgery [6, 7], there is a lack of evidence to support the beneficial effect of pre-operative statin on renal function after cardiac surgery. Previous studies have reported that pre-operative statin therapy is not associated with a reduced incidence of post-operative AKI [8], including after cardiac surgery [9]. Nevertheless, we tested the impact of pre-operative statin treatment on our results. Although pre-operative statin treatment was not associated with post-operative AKI (odds ratio 0.95 (95 % confidence interval 0.65–1.39), p = 0.78) in a univariate analysis, we

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forced the variable into the final model and there was still a significant association between the renal protective fluid management strategy and post-operative AKI (odds ratio 0.23 (95 % confidence interval 0.14–0.36), p <0.001). We can therefore definitely conclude that statins had no significant role to play in our observations.

Abbreviations

AKI: acute kidney injury; IL: interleukin.

Competing interests

The authors declare that they have no competing interests.

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