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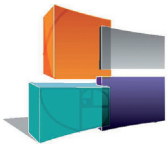

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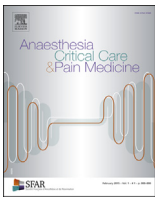

Letter to the Editor

[TIMP-2]*[IGFBP7] for Predicting Early AKI, response to Prof. Tyagi

To the editor,

We wish to thank Dr Luthra and Prof. Tyagi for their observations related to our investigation [1]. In their letter to the editor, they shared interesting comments concerning the definition of Acute Kidney Injury (AKI) and the role of [TIMP-2]*[IGFBP7] to predict AKI. Since these observations are essentials pillars of our article, we thank Dr Luthra and Prof. Tyagi for giving us the chance to clarify these important semantic points that are necessary to master when it comes to conduct and discuss an investigation on AKI.

First, we used the KDIGO criteria to define AKI. These criteria clearly mention that a patient is considered to have developed AKI when serum creatinine (sCr) increases by 0.3 $mg.dl^{-1}$ or more within the first 48 hours or rises to at least 1.5-fold from baseline within seven days after the renal aggression [2]. Thus, every study conducted following the KDIGO criteria should rigorously follow such criteria to determine whether AKI occur or not. To the best of our knowledge, KDIGO criteria were also adopted to validate the role of [TIMP-2]*[IGFBP7] to predict AKI in other trials conducted in the cardiac surgery setting with this same time frame [3]. In an effort to replicate the results reported from these previous investigations and to certify their validity providing more evidence to the literature, we have decided to assess AKI occurrence within the first seven days after the surgery. Also, one should bear in mind that the population studied in our trial is fragile and scheduled for a complex procedure requiring most of the time cardiopulmonary bypass [4]. Thus, the cardiac surgery population could not be compared to the critically ill ICU population, as Dr Luthra and Prof. Tyagi have done in their letter. Therefore, we strongly disagree with them on this point stating that evaluation for AKI developing within seven days following strictly the KADIGO criteria for clinical relevance is methodologically incorrect. In addition, Dr Luthra and Prof. Tyagi are wrong when they state that urinary [TIMP-2]*[IGFBP7] can predict only stage 2 and 3 AKI. Some trials conducted in the cardiac surgery setting have found that the occurrence of AKI stage 1 was associated with a significant early rise of [TIMP-2]*[IGFBP7] [3]. As indicated in our discussion [1], we did not grouped patients with no AKI with those having an AKI stage 1. Indeed, predicting the occurrence of AKI stage 1 is clinically fundamental, as every single kidney insult is potentially harmful and associated with negative clinical outcome [5]. Finally, the limitation of the rise in sCr associated, or not, with a low urinary output to define AKI are now well known [2,6,7]. Considering the significant progress that have been made in detection and validation of new biomarkers for AKI to replace or complement sCr measurement, the AKI definition is currently under revision [2]. Consequently, it could be affirmed that coupling functional and damage renal markers help physicians to diagnose, and not predict, AKI earlier [2].

We hope that this letter and our response will lead to more discussion in this area of investigation.

Disclosure of interest

The authors declare that they have no competing interest.

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