

Assessing risk factors of acute kidney injury after liver transplantation

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To the Editor:

We read with interest the recent article by Barreto, *et al.*¹ evaluating the risk factors for acute kidney injury (AKI) and 30-day mortality after liver transplantation (LT) in a single-center retrospective cohort study. They showed that viral hepatitis, longer warm ischemia time and high levels of serum lactate were risk factors for AKI. Given AKI is associated with significantly increased short- and long-term morbidity and mortality after LT, their findings have potentially clinical implications. In our view, however, a limitation of this study design is that some important perioperative factors affecting postoperative AKI are not included in multivariable regression analyses evaluating variables that are independently associated with development of postoperative AKI after LT.

First, preoperative albumin levels, body mass index and race were not provided. It has been shown that preoperative hypoalbuminemia is highly common among patients undergoing LT and is independently associated with postoperative AKI.² Furthermore, increased body mass index and non-Caucasian race have been shown as independent risk factors of AKI following LT.³

Second, this study only provided incidences of intraoperative bleeding and blood transfusion, but not volumes of intraoperative blood loss and blood transfusion. Actually, both vast blood loss (for ex-

ample, $> 60 \text{ mL/kg}$) and more than five blood transfusions in the intraoperative period has been shown as the independent risk factors of postoperative AKI in the patients undergoing LT.^{2,4} Furthermore, intraoperative hemodynamic instability, use of vasoressor and urine output were not included in the adjusted confounders for postoperative AKI. The available evidence suggests that intraoperative hypotension, use of noradrenaline and low urine output (for example, $< 60 \text{ mL/h}$) are independently associated with increased risk of AKI following LT.^{4,5} In addition, it was also unclear whether hydroxyethyl starch was used for intravascular volume resuscitation in the perioperative period. It has been shown that compared with patients receiving 5% albumin, patients receiving hydroxyethyl starch have an increased risk of AKI following liver transplantation.⁶

Third, we were not provided with postoperative complications. It has been shown that hypotension, hypoalbuminemia, postreperfusion syndrome, hepatic allograft dysfunction, reoperation, sepsis and surgical complications in the early postoperative period are significantly associated with occurrence of AKI after LT.^{2,4,7-9} Thus, we argue that not taking the above perioperative factors into account would have tampered with the inferences of the multivariable regression analyses for the risk factors of AKI following LT in this study.

Finally, in this study, AKI was defined according to the Acute Kidney Injury Network criteria as an increase more than two times in serum creatinine (stage 2 or 3 AKI) in the first 72 h after surgery. Furthermore, AKI was diagnosed only based on serum creatinine component of the Acute Kidney Injury Network criteria since data on urinary output was not available for all patients. However, the Acute Kidney Injury Network criteria require to use a 48 h time window, and a 24-h urine output collection is a better alternative to estimate glomerular

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filtration rate than serum creatinine.¹⁰ We are concerned that these factors would have confused incidence of postoperative AKI reported in this study. In addition, we would like to know why this study did not include stage 1 AKI, although it is more common than stage 2 or 3 AKI in the patients undergoing LT.⁸

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