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INVITED COMMENTARY

Keep the pressure! Correlation of hemodynamic instability after reperfusion and severity of acute kidney injury following liver transplantation

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Acute renal dysfunction and kidney failure represent frequent complications in patients after liver transplantation. Indeed, postoperative acute kidney injury (AKI) may occur in up to 60% and is associated with significantly increased postoperative mortality [1]. However, the pathogenesis of AKI is yet not fully understood. To date, it is not possible to identify patients with an increased risk for AKI prior to transplantation; thus, the treatment remains clinically challenging.

In their article entitled "The Postreperfusion Syndrome Is Associated With Acute Kidney Injury Following Donation After Brain Death Liver Transplantation," Kalisvaart *et al.* [2] identify the postreperfusion syndrome (PRS) after orthotopic liver transplantation as a predictor for the development of an AKI. In fact, this finding is not entirely new, because several studies showed a correlation between PRS and AKI before [3,4]. With respect to the clinical relevance of this finding, however, the data of Kalisvaart well confirm that even patients with mild AKI present with a substantially higher risk for in-hospital morbidity and mortality after liver transplantation.

Addressing the pathogenesis of the hemodynamic instability during PRS, the authors were able to show that the extent in hemodynamic instability following reperfusion correlated with the severity of hepatic ischemia–reperfusion injury. Interestingly, and in line with this finding, a recent prospective cohort study confirmed hepatic ischemia–reperfusion injury after liver transplantation as a determinant for AKI [5].

Of main interest, Kalingsvaart *et al.* were able to reveal a direct correlation between the mean decrease in arterial pressure during PRS and the severity of postoperative AKI. It was shown before that patients with a need for more vasoactive drugs are at a higher risk for requiring renal replacement therapy after liver transplantation [6]. On top, we now know that the instability of the arterial pressure during reperfusion phase may be used to directly identify patients with increased risk for AKI after transplantation.

After being able to identify patients who are at risk for AKI after transplantation by hemodynamic instability, what are the clinical implications for our transplant setting? Taking into consideration that the hemodynamic instability represents a clinical manifestation of a severe hepatic ischemia–reperfusion injury, the therapeutic options will be limited by the time we already see the hemodynamic instability. Therefore, the clear target should be the attenuation of the liver graft preservation

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injury. To date, several factors influencing the severity of the hepatic ischemia–reperfusion injury have been identified and it is also well known that donor organ quality (heart-beating versus deceased donors) as well as cold and warm ischemia times have significant impact on hepatic ischemia–reperfusion injury and PRS [2,7]. However, confronted with donor livers with extended donor criteria and sometimes increased cold ischemia times due to long transportation ways, our possibilities to optimize these factors are clearly limited. Herein, it is noteworthy that the intraoperative stabilization of the arterial pressure during the phase of reperfusion was shown capable to decrease the incidence of PRS [8]. Maintaining stable intraoperative hemodynamic conditions should not be solely seen as duty of the anesthesiologist giving vasoactive medication. Much more, it requires an intense communication between surgeon and anesthesiologist and it is the surgeons' task to keep warm ischemia time short and minimize the intraoperative blood loss.

After all, the pathogenesis of AKI following orthotopic liver transplantation remains not fully understood and must be considered multifactorial. The reperfusion phase has substantial impact on postoperative morbidity and mortality of our patients, and the development of an AKI after orthotopic liver transplantation is strongly related to hemodynamic instability during this phase. However, this hemodynamic instability should be seen as clinical manifestation of hepatic ischemia–reperfusion injury.

In consequence, *keep the pressure*! In fact, we should keep up the arterial blood pressure during reperfusion phase to avoid AKI, but it is even more important that we put ourselves under pressure to achieve short cold ischemia time as well as short warm ischemia time with the best organ we can get.

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Conflict of interest

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