Increasing trans-hepatic caval pressure gradient is associated with acute kidney injury after liver transplantation, irrespective of surgical caval technique

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Background
During the anhepatic phase of liver transplantation (LT), inferior vena cava (IVC) clamping leads to decreased venous return and distal venous engorgement. This can impair renal perfusion pressure and result in acute kidney injury (AKI). Piggyback technique (PB) is proposed to offer some protection over caval replacement (CR) due to incomplete clamping, but clinically the degree of IVC occlusion varies during PB. We therefore propose that it is IVC pressure differential that determines likelihood of AKI and not solely surgical technique.

Aims
1. To describe IVC pressure gradients during the anhepatic phase in PB and CR techniques
2. To quantify the association of maximum transhepatic caval pressure differential (dPmax) with AKI

Methods
This was a retrospective, single-centre study of consecutive adult patients undergoing LT between January 2013 and June 2014. Exclusions were super-urgent transplants, preoperative creatinine ≥100μmol/L, or absent IVC pressure data. The outcome was development of AKI at 72hrs (AKIN ≥1). We fitted multivariable models to quantify the association of dPmax with AKI.

Results
After exclusions, 75 patients were included. PB was used in 48% of cases (22% of which used portocaval shunt) and CR in 52%. No patient underwent veno-venous bypass. In both CR and PB techniques, at least 25% of patients had a maximum anhepatic PD of >26mmHg (IQR 20-27mmHg and 9-26mmHg respectively).

Incidence of AKI was 39%. Controlling for donor and recipient factors, every 1mmHg increase in dPmax was associated with 18% increased likelihood of AKI (OR 1.18 (1.04-1.34), p= 0.01) (Figure 1).

Conclusion
Increasing pressure differential across the IVC during the anhepatic phase of LT is associated with increased risk of AKI at 72hours postoperatively. Substantial caval pressure gradients are encountered even when the piggyback surgical technique is employed.

Figure 1. Maximum anhepatic caval pressure differential and the probability of postoperative AKI