

Method 211 OLT recipients (median age 53 (20–70) years: 140 male; 71 female) for chronic liver disease were studied to assess longitudinal long term changes in renal function. The MDRD (version 4) equation was used to estimate glomerular filtration rate (eGFR) pre-OLT and at 1 week, 1 month, 6 months 1 year, 2 years and 5 years post-OLT. Acute Kidney Injury was defined according to Acute Kidney Injury Network criteria and CKD staging according to Kidney Dialysis Outcomes Quality Initiative criteria.

Results Median follow-up was 2023 (3–3188) days and median survival was not reached in this study (76% 5 year survival). Median eGFR at time of transplant was 67 (29–149) ml/min/1.73 m² falling to 59 (24–165) ml/min/1.73 m² at 6 months post-OLT ($p<0.001$, Wilcoxon signed test) and 57 (11–203) ml/min/1.73 m² at 5 years post-OLT ($p<0.001$; Friedman test for eGFR over study period $p<0.001$). A reduction in eGFR was noted at 6 months post-OLT in patients with post-OLT AKI ($p=0.010$, Kruskal–Wallis test) but this difference was not detectable at 5 years ($p=0.557$). 40% of patients had CKD 3 or more pre-OLT with 60% with CKD 3 at 5 years ($p<0.001$, χ^2 test). AKI stage >1 during the first week post OLT did not influence long term survival (HR 1.43 (95% CI 0.65 to 3.1, $p=0.315$, Kaplan–Meier method) neither did the use of renal replacement therapy post OLT (HR 1.56 (95% CI 0.60 to 4.1, $p=0.268$, KM method).

Conclusion In this cohort a decline in eGFR of 10 ml/min/1.73 m² was noted over the 5 year follow-up; twice the rate of decline expected in the general population. The higher proportion of patients at CKD stage 3 or more at 5 years compared to pre-OLT suggest greater awareness of this potentially important risk factor for cardiovascular disease is required. AKI post-OLT was not associated with a higher risk of long term reduction in eGFR beyond 6 months post-OLT; nor did the use of RRT post-OLT impact on 5-year mortality.

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POST-LIVER REPERFUSION HEPATIC VEIN PORTAL GRADIENT PRESSURE MEASUREMENT AND THE RISK OF GRAFT DYSFUNCTION IN WHOLE LIVER TRANSPLANTATION

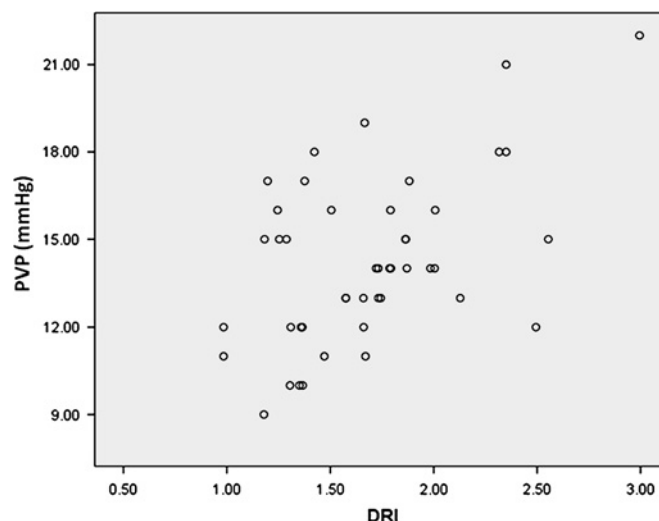
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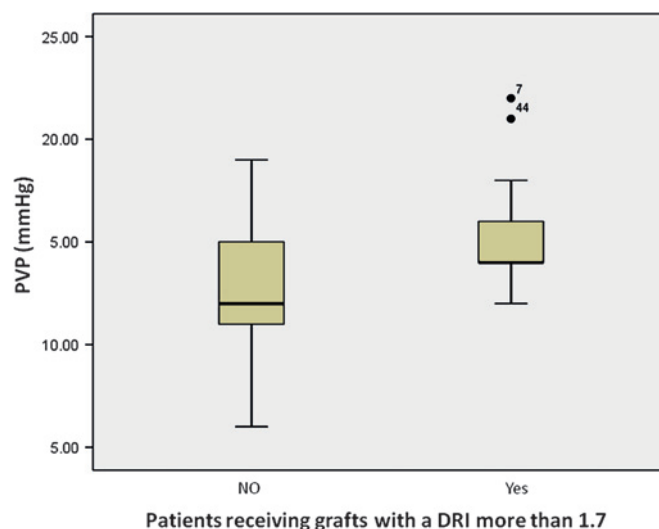
Introduction “Small for size liver syndrome” is relatively uncommon after whole liver transplantation. Portal hyperperfusion is thought to damage the hepatic microvasculature, and drive Small for size liver syndrome. Post-perfusion portal venous pressure (PVP) may be an indirect reflection of sinusoidal injury. Donor Risk Index (DRI) can be utilised to assess the quality of the graft, with those having a DRI >1.7 considered as marginal¹. This study looked at the association of portal pressure and graft quality in a whole liver transplant setting.

Method 50 adult patients who underwent liver transplantation underwent portal venous pressure measurements by direct pressure transduction during surgery. Central venous pressure was recorded at the same time points to be used to derive the Hepatic Venous Portal Gradient (HVPG) (HVPG=PVP-CVP). DRI was calculated for all the grafts used. The degree of fatty infiltration (%) was assessed on post-perfusion biopsies. Graft function was monitored post-operatively by recording INR (peak and days 1, 2, 3, 5 and 7), lactate (peak level and day 1 and 2) serum AST (peak and days 1, 2, 3 and 7) and serum bilirubin (days 1, 5 and 7). The presence of ascites, jaundice and coagulopathy were documented. Ascitic drain loss (ml/day) was recorded up to the time of drain removal.

Results A statistically significant correlation was found between PVP and DRI ($p<0.05$) (Abstract P97 Figure 1).



Abstract P97 Figure 1 Scatter plot graph showing a statistically significant correlation PVP with DRI. ($p=0.022$)



Abstract P97 Figure 2 Box plot graph showing the difference of PVP in liver grafts with a DRI >1.7 and those with DRI <1.7 . ($p=0.069$)

This was supported by higher mean and median values of PVP in the group receiving grafts with a DRI >1.7 $n=22$) (Abstract P97 Figure 2).

Patients in this group had a mean PVP of 15 (± 2.976) mm Hg and a median of 14 mm Hg (range: 8–22), in contrast to those receiving grafts of DRI <1.7 ($n=28$) who had a PVP mean of 13.89 (± 5.513) mm Hg and a median of 12.5 mm Hg (range: 6–34). No statistically significant correlation could be found between PVP and the degree of fatty infiltration. Post-perfusion HVPG had a significant correlation ($p<0.1$) with increased ascitic drainage on day 7.

Conclusion The HVPG should be evaluated as a tool for assessment of portal pressure post-reperfusion, particularly in patients transplanted with a high DRI, to predict morbidity post-transplant.

REFERENCE

1. Maluf DG, Edwards EB, Kauffman HM. Utilization of extended donor criteria liver allograft: is the elevated risk of failure independent of the model for end-stage liver disease score of the recipient? *Transplantation* 2006;**82**:1653–7.