

**Results Patients:** To date 29 patients have been switched. The median time since switch is 9 months. 25 patients are >6 months post switch and included in the analysis. The median age was 61 (range: 28–81) years, 84% male, 60% Caucasian, 28% Black African and 12% Asian. At LT 6 were acute HBV with Liver failure, 7 had HCC and 4 had delta co-infection. At LT 22 had detectable HBV DNA, 11 were on lamivudine (LAM) and 1 was on LAM and adefovir (ADV). 52% patients had archived samples suitable for drug resistance testing. None had evidence of any drug resistant mutations.

**Results:** Since LT all had received HBIG IM with HBsAb levels of 170 (range 101–454) mIU/ml. 80% were receiving concurrent LAM, 4% LAM and ADV and 16% no oral anti-HBV agent. The median time from LT to switch was 10 (range 2.6–20.3) years. At switch HBsAg and HBV DNA was undetectable in all subjects. 92% were on calcineurin inhibitor based immunosuppressive regimens. Serum creatinine was 104 (range 62–170)  $\mu\text{mol/l}$ , estimated glomerular filtration rate (eGFR) was 65 ml/min, 24 h urine creatinine clearance was 76 (range 41–150) ml/min and total protein excretion was 81 (range 31–441) mg/day. Serum ALT was 26 IU/l, phosphate was 0.98 mmol/l and vitamin D was 14  $\mu\text{g/l}$ . 12 had hypertension and/or diabetes. 16 patients were switched to TDF, 9 patients with eGFR <60 ml/min or renal risk factors were switched to ETV. Six months after switch all patients remained HBsAg and HBV DNA undetectable and there was no difference in serum creatinine [103 (TDF 93, ETV 115)  $\mu\text{mol/l}$ ], eGFR [65 (TDF 70, ETV 57) ml/min], ALT (23 IU/l), phosphate [1.03 (TDF 1.00, ETV 1.05) mmol/l] and vitamin D [17 (TDF 16.3, ETV 18.5)  $\mu\text{g/l}$ ]. No therapy withdrawal/change was required due to adverse effects. The approximate drug cost saving made per patient/year from switching from HBIG to TDF or ETV is £11 000 and £10 000 respectively.

**Conclusion** A stratified conversion protocol, based on the assessment of virological parameters and renal co-morbidities, ensures patients can safely and effectively be switched from HBIG to TDF or ETV to prevent HBV recurrence post LT. HBsAg and HBV DNA remains undetectable and no deterioration in renal function has been observed to date. Significant drug cost savings can be achieved utilising this protocol.

**P78** **LONG-TERM ANTIBIOTIC PRESCRIPTION IN PATIENTS RELISTED FOR LATE HEPATIC ARTERY THROMBOSIS IS ASSOCIATED WITH GREATER WAITING LIST MORTALITY INDEPENDENT OF MELD**

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**Introduction** Optimal prioritisation and medical management of patients with late hepatic artery thrombosis (HAT) awaiting liver transplantation remains unclear.

**Aim** To examine the association of complications of late HAT and their interventions with liver transplant waiting list mortality.

**Method** Single centre study of 49 patients listed for late HAT 01/1995–06/2010. Late HAT was defined as occurrence >4 weeks following liver transplantation. Cox regression was adjusted for listing MELD score at all times. Despite increasing waiting time statistical analyses did not demonstrate any influence of listing time period.

**Results** Mean listing MELD score was 16 (SD 7). 29% of patients demonstrated biliary stricture/s, 20% cholangitis and 63% biloma/abscess/s. The estimated 3- and 12-month transplant-free survival following listing was 85% and 53%, respectively. 36 patients were regrafted, with a median time from listing to transplantation of 45 (IQR 13–167) days.

No relationship was demonstrated between the presence of biliary stricture/s ( $p=0.984$ ), cholangitis ( $p=0.770$ ) or biloma/abscess/s ( $p=0.143$ ), and wait-list mortality. Instead, an increasing number of biloma/abscess drain insertions ( $p=0.038$ ) and long-term (LT) prescription of antibiotics ( $p=0.029$ ) were linked with an increased risk of death. Multi-drug resistant bacteria (MDRB) were cultured in bile/blood more frequently in those receiving LT antibiotics (44% vs 8%,  $p=0.004$ ), and MDRB positivity was also a risk factor for waiting list mortality ( $p=0.033$ ). On multivariate analysis the only predictor of death was LT antibiotics (MELD, HR 1.23; 95% CI 1.04 to 1.44,  $p=0.013$ ; antibiotics, HR 24.20; 95% CI 1.28 to 455.88,  $p=0.033$ ).

Following regraft, LT antibiotics ( $p=0.025$ ) and MDRB positivity while listed ( $p=0.002$ ) remained predictors of patient mortality independent of the preoperative MELD score. The estimated 3- and 12-month post transplant survival of patients with MDRB positivity was 63% and 25%, respectively, and for those without 89% and 86% (log-rank  $p=0.001$ ).

**Conclusion** Patients listed for late HAT receiving LT antibiotics are a high risk group who require greater priority for liver transplantation. Our results raise the possibility that by increasing bacterial resistance LT antibiotics may have a detrimental effect on patient survival.

**P79** **PLASMA SUCCINYLAETONE IS RAISED AFTER LIVER TRANSPLANTATION FOR TYROSINAEMIA TYPE 1 AND IS ASSOCIATED WITH REDUCED PORPHOBILINOGEN SYNTHASE ACTIVITY SUGGESTING IT IS FUNCTIONAL**

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**Introduction** Tyrosinaemia type 1 (TT1) is a rare disorder of tyrosine metabolism leading to accumulation of toxic metabolites such as succinylacetone (SA) and a high risk of hepatocellular carcinoma. Children with TT1 traditionally required liver transplantation (OLT) and while the need for this has reduced since the introduction of nitisinone some still go on to require OLT. Circulating SA inhibits the enzyme porphobilinogen (PBG) synthase and its activity can be used as a marker of functional circulating SA. Elevated urinary SA post OLT thought to be due to local production has been reported.

**Aim** This study describes a novel finding of elevated plasma SA following OLT for TT1.

**Method** A retrospective analysis was performed of patients treated for TT1 at our institution from 1989 to 2010.

**Results** In patients who received nitisinone prior to OLT mean urinary SA was elevated at presentation (159.6 mmol/mol creatinine, ref. range <1) as was plasma SA (17.58 mol/l, ref. range <0.01) but both became undetectable on nitisinone prior to OLT ( $p<0.05$ ). This was associated with increased mean PBG synthase activity from 0.032 to 0.99 nkat/g Hb (ref. range 0.58 1.25). In patients who did not receive nitisinone, mean urinary SA was 274.6 mmol/mol creatinine immediately prior to transplant. Plasma SA levels/PBG synthase activity prior to OLT were not available in this group. Following OLT in patients treated with nitisinone, mean urinary SA levels quickly rose and remained elevated for the duration of follow-up. Plasma SA levels also rose with a progressive decrease in mean PBG synthase activity to low-normal levels. In patients who had not received nitisinone, mean urinary SA fell quickly by 1 year post OLT but remained above normal at levels similar to those seen in the nitisinone treated group. No data were available for plasma SA/PBG synthase activity in this group until