The median age at baseline in the placebo group was 55 years; 93% were female, and 93% received daily UDCA (median dose, 15 mg/kg). After 1 year of standard-of-care (SOC), placebo patients demonstrated a slight increase in predicted risk of ESLD (table 1). After 1 year of OCA, predicted risk of ESLD at 5, 10, and 15 years was reduced to below baseline levels; these were sustained through the 60-month OLE.

In POISE, the UK-PBC risk score predicted an increased risk of ESLD in PBC patients treated with SOC and placebo for 12 months. OCA treatment led to sustained reductions in predicted risk of ESLD through 60 months.

The global pooled prevalence of NAFLD is 25% (Youssoni 2016). Genetic factors have been shown to play a significant role in determining the risk for the development and progression of NAFLD in Caucasian/East Asian populations where robustly replicated associations have been found, many at genome-wide significance, with variants in PNPLA3, MBOAT7, TM6SF2, GCKR and INSulin Receptor Substrate 1 (IRS1). Domain 1A (BAZ1A), Fibroblast Growth Factor 21 (FGF21), and Insulin Receptor Substrate 1 Substrate 1 (IR51). Variants in previously associated loci viz PNPLA3, MBOAT7, TM6SF2, GCKR and HSD17B13, showed much lower significance than expected (p value range 1.93 × 10⁻⁵ to 5.83 × 10⁻¹⁰) including SNPs in chromosome 20 open reading frame 78 (C20orf78), Transmembrane Protein 63C (TMEM63C), Bromodomain Adjacent To Zinc Finger Domain 1A (BAZ1A), Fibroblast Growth Factor 21 (FGF21), and Insulin Receptor Substrate 1 (IR51). Variants in previously associated loci viz PNPLA3, MBOAT7, TM6SF2, GCKR and HSD17B13, showed much lower significance than expected (p value range 1.20 × 10⁻³ to 0.730).

These findings suggest that there may be divergence in the genetic risk profile for developing NAFLD in the South Asian population compared with European/East Asian populations. This is concordant with the observed divergence in NAFLD phenotype, but needs to be validated in further, larger cohorts.

ND, JC, GM share first authorship. MM, KS, GA share senior authorship.

No associations were identified with NAFLD at genome-wide significance (p < 5 × 10⁻⁸). However, a number of genetic variants implicated in liver-related and metabolic pathology showed suggestive evidence of association (p value range 1.93 × 10⁻⁵ to 5.83 × 10⁻¹⁰) including SNPs in chromosome 20 open reading frame 78 (C20orf78), Transmembrane Protein 63C (TMEM63C), Bromodomain Adjacent To Zinc Finger Domain 1A (BAZ1A), Fibroblast Growth Factor 21 (FGF21), and Insulin Receptor Substrate 1 Substrate 1 (IR51). Variants in previously associated loci viz PNPLA3, MBOAT7, TM6SF2, GCKR and HSD17B13, showed much lower significance than expected (p value range 1.20 × 10⁻³ to 0.730).

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Abstracts

Abstract P25 Figure 1 Relation between MELD score and lifespan of drain complications were reported. Following LTAD, 15 patients (5/15 had pre-LTAD diagnosis) developed SBP at median 60 (20–423) days. Post-LTAD SBP was treated with antibiotics but 5 died. In 10 patients LTAD was removed after median 10 days of antibiotics and only 4 were replaced. For those who had replacement, 2 of 3 patients given prophylaxis suffered recurrent SBP. Other indications for removal were (leak 2; blockage 2). Patients needed hospitalization for median 19 (2–40) days in the 6 months prior to LTAD, and 12 (0–34) days in the following 6 months. In 11 of 20 patients with MELD score less than 21 (figure 1), the drain remained for 90 or more days while the median lifespan of LTAD in the whole cohort was 67 (6–463).

Conclusions In some patients, LTAD achieved long term palliation without hospital admission but many developed SBP post-insertion. Nevertheless, there was still a reduction in hospital stay. It was not possible to identify factors which might predict a successful outcome from this small cohort. Further research should focus on the impact of LTAD on quality of life measures, the role of antibiotic prophylaxis and better defining when LTAD is best employed in the natural history of patient’s with ascites.

P26 METHYL PREDNISOLONE IN TREATMENT OF SEVERE ALCOHOLIC HEPATITIS NOT RESPONDING TO PREDNISOLONE

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Introduction Prednisolone treatment of severe alcoholic hepatitis reduces mortality from 18% to 14% at d28 (STOPAH) but not at d90 (30%). Non-response may be due to steroid-resistance which might be overcome by using intravenous methylprednisolone (MePred).

Methods All patients with mDF>32 treated with MePred over a 4 year period were reviewed. Patients were treated with prednisolone 40mg daily for 7 days. Prednisolone non-response was defined as d7 Lille model >0.45 and severe alcoholic hepatitis confirmed by biopsy. Non-responders were given MePred 500mg daily for 3 days followed by prednisolone 40mg daily for 25 days and prophylactic antimicrobials.

Results Prednisolone was stopped in 7 non-responders, and MePred was given after biopsy. 5/7 had Lille score <0.45 at d7 post-MePred; one died after intracerebral haemorrhage and 4 survived beyond d90. 2/7 had Lille >0.45; one failed to respond and died at d9, the other died of liver failure at d28 despite a fall in bilirubin d7-431 to d28-297 (figure 1). No infective complications were reported. Mortality d28 29% and d90 42%.

Conclusions In patients with severe alcoholic hepatitis and prednisolone non-response, methylprednisolone leads to clinical and biochemical response and 58% had at least 90 days survival.

P27 MULTI-TEAM APPROACH TO APPLYING A PATIENT CARE BUNDLE IN DECOMPENATED CIRRHOSIS IMPROVES OUTCOMES

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At our DGH multiple audits have identified that there is poor compliance to an existing evidence based care bundle for patients with decompensated liver cirrhosis despite previous attempts to improve consistent use. Varying applicability of the bundle causes variation in the quality of care patients receive.

Presence of the existing bundle in the format of a sticker within the patient’s medical notes was audited along with application of the 6 main cirrhosis care bundle domains. Data was collected prior to and following intervention. A questionnaire was sent to junior medical staff to ascertain knowledge of the bundle and competency of performing paracentesis. Length of stay and 28 day mortality were used as patient outcome measures.

It was shown that adherence to the cirrhosis care bundle was poor. No patients had all of the recommended investigations carried out and none of the patients with ascites had an attempt to perform a diagnostic paracentesis. When asked 74% of junior doctors reported not feeling confident to perform paracentesis unsupervised. 45% of junior doctors were unaware of the existence of the cirrhosis care bundle. 48% of survey responders were foundation doctors.

The cirrhosis care bundle was redesigned into a printable format that can be accessed via the trust intranet because of concerns that the sticker might not always be available in clinical areas. The layout of the bundle was altered to improve usability and tick boxes were added to encourage the user to consider and complete each step in the bundle.

Doctors rotating between specialties and between trusts was also highlighted as an explanation of the high rates of