with increased complications and healthcare costs. Optimising preventative care reduces readmissions but many patients are readmitted before their outpatient review by hepatologists. Consequently, we established a novel care pathway with a nurse-led, structured early post-discharge clinic. Patients are seen within 2 weeks of discharge, with specific interventions for ascites, encephalopathy, varices, alcohol misuse and nutrition. The aim of our study was to assess impact on readmission and mortality.

**Methods** Following an index admission with decompensated cirrhosis, patients were seen in the nurse-led, early post-discharge clinic (intervention cohort) or received standard hepatology physician-led outpatient follow up (controls). Clinical data including demographics, liver disease aetiology, reason for admission and stage of liver disease were analysed. Outcome measures included time to first readmission, rate of readmissions and mortality up to 12 months.

**Results** There were 91 control patients and 78 in the intervention cohort. There were no significant differences in age, gender or aetiology of liver disease at index admission and mean MELD-Na on discharge was 18.1 and 19.8 (p=NS) in control and intervention groups respectively. Ascites was the most common decompensating event in both groups. Median time to readmission was 51 days in the control group and 98 days in the intervention group (p<0.01). The intervention cohort had significantly fewer early readmissions at 30 days (12% versus 30%, p<0.01) and 90 days (27% versus 49%, p<0.01) but not significantly at 12 months (58% versus 68%, p=0.16) with an overall reduction in bed day usage of 33%. Mortality for the control group was 4% at 30 days with no deaths in the intervention group. There were fewer deaths in the intervention group at 90 days (5% versus 15%, p<0.05) and 12 months (22% versus 41%, p<0.01).

**Conclusions** Following an index hospitalisation with decompensated cirrhosis, goal-directed early post-discharge care can be effectively delivered by specialist nurses, prior to outpatient review by hepatologists. This model was associated with significantly fewer unplanned readmissions, lower bed day usage and a reduced mortality. Our data suggest such nurse-led models of care deserve wider implementation and further evaluation.
OPPORTUNITIES TO INTERVENE AFTER EARLY AMINOBISPHONATES ENHANCE LIVER-RESIDENT GUT was lost. With decompensation within 3 months of first abnormal LBT those who did not (log rank p<0.001). Excluding patients – to decompensation in patients who engaged with alcohol serv-

The metabolomic signature determined by serum NMR in patients with IgG4-RD and more specifically IgG4-SC, is distinct from PSC and HC in our cohort. Metabolomic profiling has the potential to be incorporated as an additional criterion to improve the diagnosis of IgG4-RD and help distinguish IgG4-SC from PSC.

OPPORTUNITIES TO INTERVENE AFTER EARLY DETECTION OF ALCOHOL RELATED LIVER DISEASE Tseonge Jason Nyirenda*, Lucy Turner, Rebecca Bishop, Ian Rowe, Richard Parker. Leeds Teaching Hospitals NHS Trust, Leeds, UK 10.1136/gutjnl-2021-BASL.5

Alcohol related liver disease (ALD) is common worldwide and a frequent cause of ill health and death, particularly amongst younger people. We have previously shown that histological progression is slow. We examined the clinical progression of ALD and how interventions following detection of liver disease could alter the natural history.

The electronic health records of patients with ALD were examined retrospectively to identify: time of first liver blood tests (LBT), first abnormal LBT, time of diagnosis of liver disease and first episode of decompensation of liver disease. Decompensated liver disease was defined as: ascses, variceal bleeding, hepatic encephalopathy, alcoholic hepatitis or hepatorenal syndrome. Attendance at alcohol services and/or hepatology services was noted. Baseline characteristics including laboratory and anthropometric data were recorded. Patient’s postcode was used as a measure of deprivation. Data were analysed by multiparameter flow cytometry. γδ T-cell counts were determined by immunostaining. Long-lived persistence of intrahepatic γδ T-cells was examined using donor and recipient HLA-mismatched liver allografts (7–11 years post liver transplantation). Aminobisphosphonate (Zoledronic acid, ZOL) and IL-2 expanded blood Vγ2 T-cells, intrahepatic lymphocytes, and tumour-infiltrating lymphocytes, were co-cultured with human hepatoma cell-lines (HepG2, HuH7) pre-treated with ZOL to promote tumour-cell phosphoantigen accumulation for Vγ2 T-cell receptor activation.

Results Higher intratumoural γδ T-cell counts were associated with smaller HCC tumour size and greater 3-year patient survival (p<0.01). γδ T-cells exhibited a tissue-resident memory (TRM) phenotype (CD69+CD49a+) in human liver and HCC, with superior anti-tumour cytokine production and long-lived persistence in the liver (>10 years), an attractive profile to recapitulate with immunotherapy. A subset of γδ T-cells, Vδ2 T-cells, were selectively depleted within HCC but displayed the highest γδ TRM phenotype. In vitro expansion of blood Vδ2 T-cells using clinically approved ZOL and IL-2 induced a de novo TRM phenotype with improved cytotoxicity. Furthermore, direct sensitisation of hepatoma cell-lines with ZOL enhanced the anti-tumour function (IFNγ, TNFα) of co-cultured expanded Vδ2 T-cells and Vδ2 TRM cells isolated from HCC livers and tumours, with a significant increase in tumour-cell lysis.

Conclusion Liver-resident γδ T-cells possess beneficial and long-lived immunotherapeutic properties. Our findings indicate a novel immunotherapeutic strategy for HCC, combining the use of aminobisphosphonates to induce γδ TRM for potential adoptive cell transfer, with intra-tumoural delivery to sensitise HCC for more efficient γδ T-cell based targeting.