71% had achieved SVR and 34% had advanced fibrosis/cirrhosis. 53% of patients had a diagnosis of depression. With the exception of the hepatitis specific limitations scale (HLIM), patients diagnosed with depression and those with a reduced level of physical activity scored significantly lower in all components of the HQLQv2 questionnaire (all p<0.05). The median physical functional component score was significantly lower in patients with advanced fibrosis/cirrhosis (38.6) compared to those without (47.4, p=0.012). Patients who achieved SVR scored significantly higher on the positive wellbeing score, HLIM, and the hepatitis specific health distress scale (HHD) compared to those with detectable HCV RNA (p=0.035, p=0.029 and p=0.004, respectively). Multivariable linear regression adjusted for age, gender, BMI and level of physical activity illustrated that both the presence of depression (aOR-19.85 95%CI -34.37—5.32, p=0.008) and achieving SVR (aOR 20.63 95%CI 4.02–37.24, p=0.016) were independently associated with the HHD score.

Conclusions Our data, from a real world setting, suggests that achieving SVR is associated with an improvement in QoL by reducing physical, mental and emotional limitations associated with HCV. Depression is also highly prevalent in this population and independently impacts upon patients QoL. This suggests the importance of utilising a holistic approach when assessing these patients.

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Background and Aims Features of primary biliary cholangitis (PBC) and autoimmune hepatitis (AIH) coexist in some patients; termed PBC-AIH overlap syndrome. The Paris Criteria are used for diagnosis. Treatment is determined by the predominant disease and those with active inflammation may respond to immunosuppression. This study aimed to review the characteristics and treatment response to immunosuppression in PBC-AIH overlap patients.

Method All prevalent patients with a clinical diagnosis of PBC-AIH overlap between 2010 and 2018 at a single tertiary centre were retrospectively reviewed. Patients who met the Paris criteria were termed 'True Overlap' and those who did not were classed 'Clinical Overlap'. The 2 groups were reviewed for clinical course and treatment outcome.

Results There were 39/66 (59%) patients with True Overlap. Approximately 97% of patients were female in both groups. Median age at diagnosis was 55 and 56 years in the True and Clinical Overlap groups, respectively, with median ALT (144 vs 114,p=0.07) and ALP (175 vs 203,p=0.37) at presentation. 36% (14/39) of True Overlap patients had advanced disease at diagnosis or progressed to cirrhosis, compared to 18% (5/27) in Clinical Overlap. More patients with True Overlap had moderate-severe interface hepatitis on biopsy than Clinical Overlap (38 vs 18, p= 0.001) but there was no difference in the presence of florid bile duct lesions. Immunosuppressants improved ALT (p<0.001) in both groups. 61%(19/31) of True

overlap patients on immunosuppression achieved biochemical remission(normal ALT and IgG) for the AIH component as compared to 52%(10/19) of Clinical Overlap. Severe interface hepatitis (p<0.05) at presentation and ductopenia (p<0.001) were associated with incomplete response to immunosuppression in both groups.

Conclusion In our unit, patients are treated clinically as having PBC-AIH overlap syndrome without having to meet the current Paris Criteria. Both groups had similar baseline biochemical characteristics with improvement in markers of inflammation with treatment but those with True Overlap had more severe inflammation and poorer clinical outcomes. Approximately half of the Clinical Overlap patients treated with immunosuppressant achieved biochemical remission despite not meeting Paris Criteria and 39% of True Overlap patients failed to achieve biochemical remission. This study suggests that the criteria for diagnosing overlap syndromes would benefit from refinement so that we can better delineate these disease phenotypes to ensure that patients who will benefit from immunosuppression get the appropriate therapy.

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Introduction Although only 20% of people with Alcohol Use Disorder (AUD) develop liver fibrosis/cirrhosis, those affected experience high morbidity and mortality. Better strategies are required to identify cases of advanced liver fibrosis amongst people with AUD. The Enhanced Liver Fibrosis (ELF) test has been used to good effect in NAFLD to identify people with liver disease. There has been concern that recent alcohol intake may elevate ELF scores, confounding diagnostic performance. We have investigated the relationship between ELF scores and alcohol consumption in people with AUD referred to a hospital-based alcohol specialist nurse (ASN).

Method Prospective service evaluation of liver fibrosis in consecutive patients referred to the ASN at the Royal Free Hospital from Nov' 2018-Dec' 2019. Patients were excluded if they were already known to have liver disease. Five ml of blood was collected and analysed for ELF score on an Advia Centuar. Data recorded included demographics, blood test and imaging results and self-reported alcohol history. Data were analysed using SPSS.

Results We included 100 patients (69% male, mean age 53.15 ± 14.3). Average BMI was 26.52 (± 5.94) and 85% were current or past smokers. Median alcohol intake was 140 units/week (IQR 79.1–280), with duration of excess alcohol of 15 years (IQR 10–29). The vast majority (97/100, 97%) were drinking alcohol within the last month prior to ELF test. Liver function tests were abnormal in 64/96 (66.7%) patients. ELF scores ranged from 6.87 to 13.78, median 9.66 (IQR 8.94–10.6). Of the total cohort, 29/100 (29%) had an ELF score of ≥10.5 indicating advanced fibrosis, and 14/100 (14%) had ELF scores ≥11.3 indicating cirrhosis. ELF score increased with age (p = 0.037). Alcohol intake was not
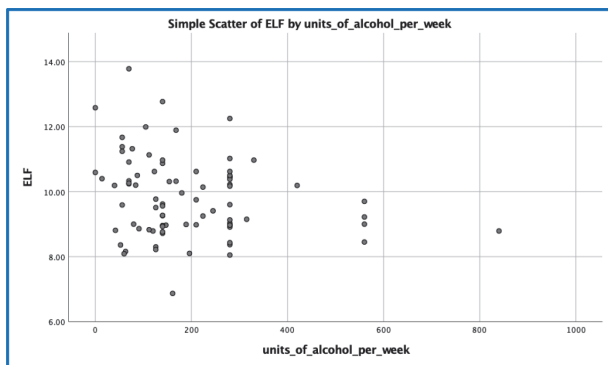
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associated with ELF score in multiple linear regression analysis, p = 0.168 (when adjusted for age, ALP, ALT, MCV, platelets and bilirubin) or when using binary ELF threshold of 10.5 (p = 0.366, OR 0.996, 95% CI 0.988–1.004). Neither BMI nor deprivation decile were associated with ELF score.

Conclusion In this cohort of patients with AUD, the amount of alcohol ingested was not associated with the ELF score suggesting that alcohol ingestion does not directly influence ELF results in AUD. ELF testing indicated that over a quarter of this cohort had advanced fibrosis, and 14% had cirrhosis (ELF≥ 10.5 and ≥11.3 respectively) in line with the literature. Further studies examining effects of alcohol unit thresholds on risk of liver fibrosis would be beneficial.

<table>
<tr><td>P211</td><td>ALCOHOL USE DISORDERS AND LIVER FIBROSIS – CASES ARE MISSED THROUGH FAILURE TO TEST</td></tr>
</table>

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Background and Aims Alcohol Use Disorders (AUD) account for 7.2% hospital admissions per year in the UK. While a proportion of these people are recognised to have liver disease and are managed by liver specialists, many are managed by a wide range of physicians and their liver disease may be missed even if their AUD is recognised. We aimed to use non-invasive tests for liver fibrosis to investigate the prevalence of occult liver disease in patients recognised to have AUD but not known to have liver disease.

Methods Prospective service evaluation of liver fibrosis in consecutive patients referred to the Alcohol Specialist Nurse (ASN) at the Royal Free Hospital from Nov' 2018-Dec' 2019. Patients were excluded if they were already known to have liver disease. Liver fibrosis was assessed using the Enhanced Liver Fibrosis (ELF) test performed on serum extracted from 5 ml of blood, analysed on an Advia Centuar. Patient demographic, blood test and imaging data were recorded along with alcohol histories. Patients with ELF scores ≥10.5 were invited for fibroscans and outpatient hepatology assessments.

Results We included 100 patients (69% male, mean age 53.15 ± 14.3). Median alcohol intake was 140 units/week (IQR 79.1–280), with duration of excess alcohol of 15 years (IQR 10–29). The commonest reason for presentation

to hospital was symptomatic alcohol withdrawal (n=36/100). Other reasons included falls/trauma (13%), pancreatitis (9%), mental health (12%), GI bleed (5%) and 'other' (25%). None had a prior history of liver disease. Four patients had documented signs of CLD. Liver function tests, checked in 96/100 patients were abnormal in 64/96 (64%). ELF scores ranged from 6.87–13.78, median 9.66 (IQR 8.94–10.6). Of the total cohort, 29/100 (29%) had an ELF score ≥10.5. Of these, 29.6% had normal LFTs. 76% had previously attended A&E in the last 5 years, (median number of presentations = 4, IQR 2–9) without assessment or diagnosis of liver disease.

Conclusion Over a quarter of patients in this cohort with AUD had evidence of advanced liver fibrosis that had been undetected prior to 'opportunistic' ELF testing. The vast majority had had recent hospital attendances representing additional missed opportunities for investigating liver disease. LFTs cannot be relied upon to for detection of liver disease in AUD. We propose that clinicians consider using non-invasive tests to assess liver fibrosis in all patients admitted to hospital with AUD.

<table>
<tr><td>P212</td><td>EXPLORING BIOCHEMICAL AND IMMUNOLOGICAL PREDICTORS BETWEEN ACUTE AUTOIMMUNE HEPATITIS AND DRUG INDUCED LIVER INJURY</td></tr>
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Introduction Differentiating between acute autoimmune hepatitis (AIH) and acute drug induced liver injury (DILI) remains a major diagnostic challenge, as there are no definite pathognomonic biochemical, immunological or histological features for either condition. We aimed to explore markers that may help to ascertain the correct diagnosis and thereby prevent unnecessary long term use of immunosuppression in DILI.

Methods A retrospective case-note review of patients presenting with acute hepatitis at University Hospital Birmingham from 2010–2018. Data are reported with p-values from Fisher's exact tests or as a median with Mann-Whitney tests as applicable. Significance was set as p<0.05. Histological analysis is ongoing.

Results A total of 28 patients with acute presentation of AIH and 42 patients with DILI were identified. The age at presentation was similar in the two groups (median: 56 vs. 55 years), with a preponderance of females in the AIH group (79% vs. 48% of DILI, p = 0.013). AIH patients were significantly more likely to be ANA (82% vs. 17%, p < 0.001) or SMA (68% vs. 17%, p < 0.001) positive and to have significantly higher IgG (median 19 vs. 10 g/dl, p < 0.001).

At presentation, AIH and DILI patients had similar levels of AST, ALT and GGT. DILI patients had significantly higher ALP (median 258 vs. 126 U/L, p = 0.006), bilirubin (median 213 vs. 32 mg/dl, p < 0.001) and MELD scores (median 19 vs. 10, p = 0.001) but significantly lower ALT/ALP ratios (0.6 vs. 2.1, p = 0.048). Resolution of liver enzymes took significantly longer in the AIH group (median 54 vs. 13 weeks, p = 0.024). Liver histology is under review in both cohorts. All patients with acute AIH were treated with steroids, compared to 24% of those with DILI.