(F2F) with daily consultant supervision, supported by a registered nurse and two medical student volunteers acting as health care assistants. F2F and virtual clinic reviews were offered. Patients were referred into the HAU from local GPs, consultant referrals, following ward discharge and via a direct patient hot line triaged by two clinical nurse specialists.

Results Data were collected from 23rd March to 23rd June 2020, comprising 136 patient encounters. 86 patient encounters were completed in the F2F, the remainder in the virtual clinic. 67% of patients were females and 56% had decompensated cirrhosis in the F2F clinic, with alcohol the most common aetiology (41%). The rest of the patients has a mixture of non-cirrhotic aetiology. 14 patients needed paracentesis and 4 patients had infusions (blood or iron). Of the patients with cirrhosis, 83% had Child – Pugh Score B (7–9) and 14% had Child Pugh C (10–15), 56% had a UKELD between 49–60. Majority of the patients were followed up in the consultant led virtual clinic (65%) and HAU virtual clinic (25%). One patient underwent a liver transplant and 2 patients were referred to other specialist clinics. 3 patients were discharged to the GP. There were 2 patients admitted directly to the hospital with variceal bleed and sepsis. None of the patients within the HAU clinic were infected with Covid-19, and there were no deaths.

Conclusion Our study shows that patients with advanced liver disease can be safely managed as outpatients in a well-supported closely-monitored unit. Given reports of significantly increased Covid-19 related morbidity and mortality in patients with cirrhosis, we have demonstrated an alternative and effective ambulatory model of care, which can be retained to deliver safe care to this vulnerable patient group in the future.

Conflicts of Interest The authors have no conflicts of interest or competing interests to disclose.

REFERENCE
1. https://www.journal-of-hepatology.eu/article/S0168-8278(20)30305-6/fulltext

P71 ABCB4 MUTATIONS CAN CAUSE A SPECTRUM OF CHOLESTATIC PHENOTYPES PRESENTING IN ADULTHOOD

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Background and Aims The ABCB4 gene encodes the floppase, multidrug-resistance p-glycoprotein 3 (MDR3), which transports phosphatidylcholine (PC) to the outer leaflet of the cell membranes lining the bile canaliculi. PC combines with bile acids in the canicular lumen to form micelles, thus preventing the emulsification action of bile acids damaging the canicular epithelium. Mutations in the ABCB4 gene are associated with failure of this process leading to cholestatic liver disease. Presentations range from progressive familial intrahepatic cholestasis type 3 (PFIC3), most commonly presenting in childhood, to less severe forms typically presenting in adulthood. Adult phenotypes are poorly characterised hence we sought to examine in detail a series of patients with ABCB4 variants presenting to our institution.

Methods Six unrelated adults with ABCB4 variants (four female, mean age 39 years) presenting with a cholestatic liver disorder were identified. In addition, three sisters with adult-onset cholestasis (labelled as PFIC3), one of whom was compound heterozygous for ABCB4, were studied. As well as case note review, detailed sequencing and histopathological analysis were performed.

Results Cases were sub-phenotyped as follows: drug-induced cholestasis, idiopathic adulthood ductopenia, refractory primary biliary cholangitis (PBC) and adult PFIC3. 6/9 had presented with gallstone complications and 5/7 females had a history of intrahepatic cholestasis of pregnancy (ICP). Liver transplantation was required for two out of these nine patients, with another currently wait-listed. Histologically, all cases demonstrated a degree of ductopenia, affecting the smallest interlobular ducts only, copper-associated protein and fibrosis. Portal inflammation was consistently present but of note non-ductocentric. At least one previously unreported pathogenic ABCB4 variant was observed (c.620T>G, p.Ile207Arg)) and ‘adult PFIC3’ was associated with compound, rather than simple, heterozygosity.

Conclusion We describe a range of adult phenotypes associated with pathogenic variants, including novel, in the ABCB4 gene. A distinct histological pattern was observed which differs from classical PBC and primary sclerosing cholangitis (PSC), in some cases overlapping with vanishing bile duct syndromes. Cholestatic liver disease in adults merits genetic analysis, particularly where there is a history of early gallstone disease or ICP, a relevant family history or where the histological profile described is present. Family members should be screened and liver transplantation may be required in more severe cases.

P72 ‘FIRST REPORT OF LIVER TRANSPLANTATION IN BLAU SYNDROME’

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Introduction Blau syndrome is a rare autosomal dominant inflammatory granulomatous disease caused by mutations in the NOD2 gene, classically presenting in childhood. Hepatic manifestations are recognized including cholestasis and granulomatous liver disease. We describe a novel NOD2 gene mutation c.1471A>C, p.(Met491Leu) in an adult presenting with decompensated granulomatous liver disease, requiring an orthotopic liver transplant, the first reported in this syndrome. Disease recurrence has since occurred and he is awaiting re-transplantation related to septic complications from ischemic cholangiopathy. Furthermore, we appraise the effectiveness of antibody therapies in halting disease progression.

Case report Having originally been treated for juvenile idiopathic arthritis and uveitis since the age of three, our
patient remained well until the age of 21 when he presented with cholestatic liver enzyme derangement, ascites and weight loss. Imaging suggested portal hypertension and a liver biopsy revealed epithelioid granulomas with no central necrosis and multinucleate giant cells with peri-venular and peri-portal fibrosis. Chronic liver screen and mycobacterial testing was negative. Around this time his daughter developed polyarthritis, uveitis and hepatosplenomegaly at the age of 4 years. She was diagnosed with Blau syndrome after genetic testing revealed the hitherto unreported patho-

logical variant, c.1471A>C, p.(Met491Leu), in the NOD2 gene. Genetic testing confirmed the presence of the same mutation in her father, consistent with a diagnosis of Blau syndrome.

At the age of 31, despite selective immunotherapy he developed ciedrhotic complications including recurrent oesopha-

gal bleeding and spontaneous bacterial peritonitis. He was accepted onto the liver transplant waiting list and subsequently received a Donation after Circulatory Death (DCD) graft in March 2019. Progress following transplantation was satisfac-
tory and immunosuppression consisted of Tacrolimus, Azathio-

prine and Prednisolone.

Three months later he was treated for septic complications from ischemic cholangiopathy. Imaging revealed a degree of hepatic artery stenosis and bile duct stricturing, thought to be ischemic in nature. He underwent liver biopsy which showed biliary features as well as focal portal and lobular non-necrotiz-
ging granulomatous inflammation identical to that seen in his native liver explant, thus in keeping with disease recurrence in his graft. Following his initial grafting he is awaiting re-

transplantation.

Review of Antibody Therapies in Blau Syndrome

Of 84 Blau patients treated with antibody therapy, 5 hep-

atic cases responded to anti-TNF therapy, with promising results if instigated before decompensation occurs.

Conclusion We report the first case of liver transplantation for Blau syndrome, in an adult case of Blau syndrome with a novel NOD2 mutation.

Methods and Materials Patients listed for liver transplantation with a primary diagnosis of hepatic sarcoidosis were identified from the UK Transplant Registry between 2008 and 2019 (NHS Blood and Transplant (NHSBT) Data). Data from this cohort was examined including demographics, graft and patient outcomes.

Results In the UK, 30 patients have been listed for liver transplantation due to hepatic sarcoidosis in the last decade. 18 patients received a liver transplant, 14 of whom are still alive today. Four patients died whilst on the waiting list. The mean age and mean United Kingdom Model for End Stage Liver Disease (UKELD) score at time of listing were 51.0 years (± 10.3 years) and 56.0 (± 4.2), respectively. The median patient survival was 1091 days, with both the 1- and 3-year patient survival being 89%. Graft failure occurred in 4 of the transplanted cases, and of these cases one was a result of recurrent disease, and another a result of biliary tract stenosis. In total 6 cases of sepsis were observed in the transplanted cohort. Details on the causes of death were unavailable.

Conclusion Hepatic sarcoid is a rare indication for liver transplant-

ation. NHSBT data between 2008 and 2019 shows that patient survival for liver transplant recipients with this condi-
tion in the UK was satisfactory in the short to medium term. US data between 1987 and 2007 suggested 1- and 5-
year patient survival of 78% and 61% respectively, which is worse than the UK outcomes, but might reflect recent advances in the field. Although not common, recurrent sarcoidosis in the donor liver does occur and may respond to increased immuno-
suppression. Graft failure due to disease recurrence was observed in one case in the UK cohort.

P74 HEPATITIS C VIRUS HIGH INTENSITY TEST AND TREAT HMP LEEDS

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Background and Aims Prisons are a high prevalence site with high throughput of people who in inject drugs. A High Inten-
sity Test and Treat (HITT) initiative is one potential approach to try and micro-eliminate HCV in prisons. Leeds is a medium secure local prison and was chosen as a site to trial this approach. The aim was to test and treat as many prisoner-

s as possible over a two weeks period.

Method Prior to testing commencing a publicity campaign was conducted by peers within the prison and prisoners were incentivized to be tested by providing chocolate bars and telephone access cards. Over the course of a weekend period in July 2019 an attempt was made to test all inmates in the prison for HCV, Hepatitis B (HBV) and Human Immunodeficiency Virus (HIV) with a point of care Matrix test. Positive antibody tests were further tested with capillary blood PCR tests performed in the local laboratory with test result turn-

around of 24 hours. Prisoners testing PCR positive were immediately informed of the diagnosis and a review of their current drug history made to check for drug drug interactions. If a genotype was already known they were treated with an appropriate genotype specific drug or if no genotype was available they were treated with the pan-genotypic drug Maviret®.