(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,[1] 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.

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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 canalicular lumen to form micelles, thus preventing the emulsification action of bile acids damaging the canalicular 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