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 polyarthritides, 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 cirrhotic complications including recurrent oesopha- geal 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, Azathioprine 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-necrot- ing 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.

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**HEPATIC SARCOID: UK EXPERIENCE IN OUTCOMES FOR ORTHOTOPIC LIVER TRANSPLANTATION FOR ADULT GRANULOMATOUS LIVER DISEASE**

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**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 transplantation. NHSBT data between 2008 and 2019 shows that patient survival for liver transplant recipients with this condition 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 immu- nosuppression. Graft failure due to disease recurrence was observed in one case in the UK cohort.