which results in poorer long-term graft survival rates compared with other liver diseases. Sustained virological response (SVR) after antiviral treatment has recently been shown to significantly improve liver histology and long-term survival[1].

Aim To describe our experience of the treatment of recurrent HCV post liver transplantation.

Method Retrospective case-note review of all patients transplanted for HCV.

Results 41 patients were transplanted in Newcastle for chronic HCV (10 had HCC) between 1993 and 2008. Up to 2002 our 5 year survival for patients with HCV was 45%. In order to try and improve this antiviral therapy was offered to patients from 2002. 15 patients (median age 50, range 38–68; 11 (73%) male) received individualised treatment with pegylated interferon (PEG-IFN) +/- ribavirin (RBV). All had liver biopsy showing recurrent HCV, 3 had cirrhosis and 1 had cholestatic hepatitis. 10 (66%) patients were infected with HCV genotype 1 (G1) and 5 with genotype 3 (G3). 4 patients (27%) achieved SVR following treatment (4 G3 and 1 G1) and 1 patient is currently on treatment and was HCV RNA negative at 12 weeks (G3). All patients who achieved SVR had =48 weeks treatment with PEG-IFN+RBV and had mild hepatic fibrosis. Of the four patients who had an SVR, 2 were taking ciclosporin and 2 tacrolimus. Adverse events were common and led to cessation of therapy in 6 patients (3 pancytopaenia, 1 refractory anaemia, 1 myocardial infarction, 1 hepatic decompensation). All cirrhotic patients stopped treatment due to adverse events. Severe anaemia was very common and 5 patients were treated with erythropoietin (2 had SVR).

Conclusion Treatment of recurrent HCV post-liver transplantation can be successful in selected patients, particularly patients with HCV G3 with mild hepatic fibrosis. This experience has led us to discuss anti-viral therapy at 6 months post liver transplant.

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P72 LOW RATES OF TESTING FOR DELTA HEPATITIS AMONGST A LARGE HEPATITIS B COHORT

doi:10.1136/gut.2010.223362.98

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Introduction Delta hepatitis is frequently neglected amongst hepatitis guidelines and publications. There are limited data on the prevalence of delta in the UK and this is likely to be a rapidly evolving area.

Aim The aim was to examine the rate of testing for delta hepatitis amongst all HBsAg (+) patients seen over a 3 year period in the large liver unit at St Mary's Hospital (SMH), London. Clinical characteristics of delta (+) patients were reviewed.

Method All HBsAg (+) results generated from the virology department from Jan 1 2007 to Dec 31 2009 were recorded. Duplicate, indeterminate and untraceable requests were excluded and the number of HBsAg (+) patients that reached hepatology specialist care established. All delta serology requests (ELISA) over the 3 year period were also reviewed. Further trawl of histology and clinical databases was made to identify any delta (+) patients diagnosed prior to 2007. The medical notes of delta (+) patients were reviewed.

Results 858 HbsAg (+) patients were identified that were seen in specialist hepatology clinics in the trust over the 3 years. Of these, only 56 had been tested for delta (6.5%). Delta Ab was (+) in four patients (7.1%). Delta testing was predominantly done in patients with abnormal liver function and low HBV DNA levels. A total of 13 delta (+) patients were found after the subsequent review of virology and histology records from 2000 onwards. Patients were young (mean age 36) with advanced disease (cirrhosis in 55%). The 13 patients were of varied ethnicity, having been born in 10 different countries.

Conclusion The rate of testing for delta was extremely low. The positivity rate was also low but equivalent to the rate reported amongst another London cohort where universal delta testing is undertaken amongst HBsAg (+) patients (Cross *et al J Med Virol* 2008). If the prevalence of delta is similar between the 2 London centres then 8.5% of the 858 patients at SMH would be expected to be delta (+) equivalent to 73 patients, rather than the four found during the 3 years of study. Prospective universal testing for delta is recommended to determine whether there is significant under-diagnosis occurring.

P73 REFERRAL OF HEPATITIS B SURFACE AG POSITIVE PATIENTS TO SPECIALIST HEPATOLOGY CARE: NEED FOR IMPROVEMENT

doi:10.1136/gut.2010.223362.99

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Introduction There are a number of obstacles to ensuring appropriate diagnosis and treatment of both hepatitis B and hepatitis C patients. A major potential problem is the failure of onward referral to specialist care following the finding that a patient is HBsAg (+). This is of particular concern due to existing misconceptions relating to supposed "healthy carrier states" and lack of knowledge regarding efficacy of current hepatitis B treatments.

Aim The aim of this study was to establish what proportion of patients found to be HBsAg (+) by both primary care and hospital clinicians were referred to, and attended specialist hepatology clinics.

Method All HBsAg (+) results obtained by the virology department at St Mary's Hospital over a 3 year period from Jan 1 2007 to Dec 31 2009 were identified. Duplicate tests, equivocal serology and unidentifiable patients were removed. The source of the request was recorded: primary care, hospital out-patient, in-patient, Accident and Emergency or ante-natal clinic. The patient administration system at SMH was used to determine how many of these patients attended at least one hepatology clinic at SMH. For patients who failed to attend hepatology clinics the hospital notes were reviewed (when available) to try to establish reasons for lack of onward referral.

Results Initially 2698 HBsAg (+) results were found. This was reduced to 1094 patients by excluding duplicate requests (including from within hepatology), indeterminate (n=18) and untraceable confidential hospital numbers used by the sexual health clinic (n=459).

The Abstract P73 Table 1 indicates patients tested in primary care were far less likely to reach specialist care, Antenatal patients were the commonest group tested in the hospital setting who failed to reach hepatology clinics (22 patients in total) but they also made up the largest group of patients tested in secondary care. It was not possible to exclude the possibility that some patients may have been attending hepatology clinics outside SMH, but this was not documented in notes as an explanation for lack of referral.

Abstract P73 Table 1 Results

Request site	No.	Did not reach hepatology clinic
Hospital	912	81 (9%)
Primary care	182	151 (83%)

Conclusion This retrospective review is imperfect and may underestimate the number of hepatitis B carriers reaching specialist care. Nonetheless a major problem clearly is still occurring despite very

directive information appearing on virology result slips; the vast majority of patients found to be hepatitis B carriers in the community are not referred for appropriate follow-up. In-hospital referral rates are significantly better. This is consistent with a concerning survey of London GPs regarding knowledge of hepatitis and indications for referral (Taylor *et al Gut* 2009;**59**(Suppl1):PTU-072) and indicates the need for improving education in this area.

P74 CD161 EXPRESSING CD8+ T-CELLS; ELUSIVE PLAYERS IN VIRAL HEPATITIS

doi:10.1136/gut.2010.223362.100

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Hepatitis B (HBV) and C (HCV) -specific CD8+ T-cells are characterised by expression of the NK receptor CD161. CD8+ T cells with high levels of CD161 (CD161++) make up a mean of 12% of CD8+ T-cells in healthy controls and have distinct properties; they express the gut and liver homing chemokine receptors CCR6 and CXCR6, cytokines IL-17, IFN-γ and IL-22 and have narrow TCR Vb usage (predominantly Vb 7.2 and 13), linking them to the mucosal-associated invariant T-cells of the gut. In healthy controls ~40% of this CD161++ subset does not express the co-receptor CD8 alpha-beta (CD8ab), but the co-repressor CD8 alpha-alpha (CD8aa), yet share key functional and phenotypic features of the subset.

Aim We aimed to the study the distribution and phenotype of CD8ab and CD8aa subsets in chronic hepatitis C (cHCV) and hepatitis B (cHBV).

Method Flurochrome-labelled antibodies were used for multi-colour FACS analysis of lymphocytes in whole blood from 24 cHCV, 6 e-antigen (Ag) +ve HBV and 14 eAg−ve HBV patients and 19 healthy controls (HC). Liver infiltrating lymphocytes (LILs) (obtained from explant material; 4 HCV patients with paired PBMCs, eight alcoholic liver disease and 1 PBC) were included in the study. FACS data were analysed using FloJo software (Tree Star, Inc) and statistics were performed using PRISM (Graftpad software, Inc).

Results CD8aa cells are exclusive to the CD161++ subset in HCs, cHCV and cHBV. In cHCV and eAg−ve cHBV there is a significant reduction in the proportion of cells in the CD161++CD8+ subset compared to HCs ($p \leq 0.05$). Within the CD161++CD8+ subset there is a further reduction in the fraction of CD8aa cells in cHCV patients (18.5% vs 34.13%, p=0.0086) compared to HCs. No difference is observed in cHBV. CD8ab and CD8aa CD161+ populations are found within human LILs in HCV, ALD and PBC. The CD161+CD8aa cell subset constitute a mean of 9.9% of the total CD8+ LILs. Relative enrichment of CD161+CD8aa cells is seen in the liver of patients with cHCV compared to peripheral blood (p=0.0079). In eAg−ve cHBV a distinct CD8a+blow population can be identified within the CD161+ and CD161− subsets. These populations are not seen in HCs, eAg+ve HBV and HCV ($p<0.05$).

Conclusion CD161++ CD8+ T-cells are lost from the peripheral blood in cHCV and eAg−ve HBV. Maintenance of this subset in eAg +ve HBV may reflect immuno-tolerance to virus at this stage of infection. In chronic HCV there is a relative enrichment of the CD161+CD8+ subset in LILs, indicating recruitment to and retention in the liver. The role of these cells in health, immunity and disease outcome in viral hepatitis requires further study. The emergence of a CD161+/CD161-CD8a+blow subset in eAg−ve HBV may reflect activation or exhaustion of these cells; their phenotype and function requires investigation.

P75 INFLUENCE OF VITAMIN D SUPPLEMENTATION ON OUTCOME IN THE TREATMENT OF CHRONIC HEPATITIS C

doi:10.1136/gut.2010.223362.101

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Introduction Vitamin D, acting as an immune modulator, has recently been shown to increase the sustained virological response (SVR) in genotype 1 patients.

Aim To retrospectively examine the outcome of patients treated in our institution using pegylated interferon and ribavirin, and compare the effects of treatment with a Vitamin D preparation.

Method All patients in our treatment database who have received treatment for Hepatitis C using Pegylated Interferon were identified. Only those patients who were greater than 6 months post treatment were included. Data collected included genotype, fibrosis score (Ishak) and if they were prescribed Vitamin D preparations, The primary outcome was to attain a SVR, defined as persistently negative HCV PCR status 6 months after cessation of anti viral therapy.

Results Data were available for 206 patients treated over a 3 year period. Total SVR by genotype was as follows, Genotype 1−39 % (n=44), Genotype 2−71 % (n=8), Genotype 3−72% (n=151) and Genotype 4−100% (n=3).

27.5 % (n=57) of our patients received Vitamin D supplementation with Calcichew D3 Forte (Shire Pharmaceuticals, Hampshire, UK) during the course of treatment, an observed SVR rate of 72 % was seen in those receiving supplementation compared to 64 % in those not supplemented (p=0.281).

When examining patients by genotype, no patients with genotype 1 received Vitamin D therapy. Of Genotype two patients 25 % (n=2) were treated with Vitamin D, achieving a 50 % SVR compared to 83.3% for those not treated with Vitamin D. 34 % of Genotype 3 patients received Vitamin D (n=52) achieving an SVR in 77% of cases, compared to those who did not receive supplementation (n=99) with an SVR of 71% (p=0.414).

From the subset of genotype three patients, the SVR for fibrosis scores <4 and 5/6 were 78% and 53 % respectively. When these groups were analysed considering Vitamin D supplementation those with fibrosis scores of <4 receiving supplementation achieved an SVR of 87% compared to 74% in those not (p=0.183). Patients with fibrosis scores of 5/6 achieved an SVR of 53 % in both supplemented and non-supplemented groups.

Outcomes were also analysed using fibrosis scores, as expected those with less significant fibrosis achieved SVR more frequently, no significant differences were detected when the data were analysed using treatment with Vitamin D as a variable.

Conclusion Our data show that Vitamin D supplementation could improve the SVR in Genotype three patients with mild/moderate fibrosis, this has not been reported so far. We suggest routine testing of vitamin D levels prior to combination therapy and replacement during treatment for chronic hepatitis C.

P76 LENTIVIRAL VECTORS CO-EXPRESSING HEPATITIS B CORE AND VFLIP INDUCE POTENT CD8 T-CELL AND ANTIBODY RESPONSES IN HLA-A2 TRANSGENIC MICE

doi:10.1136/gut.2010.223362.102

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Introduction The failure to clear persistent Hepatitis B viral (HBV) infection is characterised by an insufficient CD8 T-cell response to