Conclusion Escalating PIFN2a monotherapy is associated with HCV clearance and a reduction in liver related mortality in this small RCT. The differences from HALT-C and EPIC, and similarity to COPILOT may relate to marked differences in methodology (specifically the omission of therapy in the control arm), cirrhosis stage or sample size. These findings warrant further investigation of PIFN2a for patients with advanced cirrhosis for whom there is no other treatment and where transplantation is associated with graft infection and rapid progression to cirrhosis.

Acknowledgements Roche provided free drug and support for pharmacovigilence and viral diagnostics. The study was supported by the British Liver Disease Clinical Interest Group, BASL, BSG and the Wellcome Trust through the Southampton Clinical Research Facility.

Abstract P68 Table 1 Results

	SVR n (%)	Liver-related morbidity	Liver-related mortality	All-cause mortality
Intervention n=17	3 (18)	2 (12)	0	0
Control n=22	0	6 (26)	5 (23)	6 (27)
p Value	>0.001	0.23	0.035	0.019

P69 HEPATITIS DELTA RNA LEVEL AND GENOTYPE, AND HEPATITIS B SURFACE ANTIGEN TITRE PREDICT RESPONSE TO PEG-INTERFERON IN THE TREATMENT OF CHRONIC HEPATITIS DELTA VIRUS

doi:10.1136/gut.2010.223362.95

S Hughes, I Carey, D Shang, M Horner, M Bruce, I Fletcher, T J S Cross, A Suddle, K Agarwal, P M Harrison. *Institute of Liver Studies, King's College Hospital, UK*

Introduction Hepatitis Delta Virus (HDV) infection is associated with more severe liver disease in individuals with hepatitis B virus (HBV). The only treatment for HDV shown to be of benefit is α-interferon, but predictors of response to therapy are not known.
Aim To evaluate the efficacy of PEG-interferon α 2a (PEG-IFN) therapy and factors predicting viral response in patients with chronic HBV/HDV co-infection.
Method Between 2005 and 2010, 14 patients (71% female, median age 32, 57% Black African, 36% Caucasian, 7% Oriental, 79% HBeAg −ve, 100% HDV RNA +ve, 64% HDV genotype 1, 29% HDV genotype 5, 7% HDV genotype 6, 43% cirrhotic) were treated with PEG-IFN 180 mcg/week for a median of 48 weeks. The median follow-up post treatment was 16.5 months. A retrospective analysis was undertaken to assess clinical and virological factors predictive of outcome. HDV RNA was measured by an in-house real-time quantitative PCR assay (range 6.4×102 to 6.4×107 copies/ml), and genotyping was performed by comparison of nucleotide sequences of HDV RNA with previously reported sequences of HDV genotypes 1–8. HBV DNA was measured using the Roche COBAS Ampliprep/TaqMan assay. Hepatitis B surface antigen (HBsAg) titres were measured using the Abbott Architect assay.
Results In response to PEG-IFN, 64% cleared HDV RNA by end of treatment (EOT), 1 patient with genotype 1 HDV relapsed and 54% remain HDV RNA −ve beyond 24 weeks post-treatment (sustained virological response; SVR); 2 patients subsequently cleared HBsAg. Baseline HDV RNA was significantly higher in non-responders compared to those with SVR (2.1×106 vs 1.3×104 copies/ml, p=0.003) and predicted treatment response (AUROC=1.0, p=0.003). A HDV RNA of >1.96×105 copies/ml predicted treatment failure (p=0.001, positive predictive value 100% and sensitivity 100%). There was a strong correlation between HDV RNA level and HBsAg titre (r=0.82, p<0.001). Baseline HBsAg titre was significantly higher in non-responders compared to those with SVR (10 067 vs 5820 IU/ml, p=0.007) and also predicted treatment response (AUROC 0.95, p=0.007). A cut-off HBsAg titre of >9000 IU/ml predicted treatment failure (p=0.021, positive predictive value 100% and sensitivity of 67%). All patients with HDV genotype non-1 achieved SVR, compared with only 25% of those with HDV genotype 1 (p=0.02). Responders were similar to non-responders with respect to gender, age, liver histology staging and pre-treatment ALT.
Conclusion PEG-IFN was an effective treatment for chronic HDV, with 54% achieving SVR. Levels of HDV RNA >1.96x105 copies/mL and HBsAg titres >9000 IU/ml predicted treatment failure, whereas HDV genotype non-1 predicted long-term viral clearance.

P70 CAN ANTIVIRAL THERAPY FOR HEPATITIS C REDUCE THE PREVALENCE OF HCV AMONG INJECTING DRUG USER POPULATIONS? A MODELLING ANALYSIS OF ITS PREVENTION UTILITY

doi:10.1136/gut.2010.223362.96

M Hickman, N Martin, P Vickerman, S Hutchinson, D Goldberg, G Foster. *Bristol University, UK*

Introduction HCV antiviral treatment (peginterferon and ribavirin) is effective for individual patients, but few active injecting drug users (IDUs) are treated.
Aim We considered the utility of antiviral treatment for reducing HCV transmission amongst active IDUs.
Method An HCV transmission model amongst IDUs was developed, incorporating HCV antiviral treatment. We projected the chronic prevalence reductions resulting from different treatment rates over 5–40 years. Treatment efficacy was varied for three genotype scenarios (mixed, genotype 1, and genotype 2/3) and assumed to result in IDUs becoming susceptible (75%) or resistant/immune (25%). Two models were considered with treatment non-responders either allowed (unrestricted model) or not allowed (restricted model) to be retreated with the same success rates.
Results In the unrestricted model with mixed genotype, annually treating 10 infections per 1000 IDUs results in a relative decrease in HCV prevalence over 10 years of 31%, 14% or 7% for baseline (untreated endemic chronic infection) prevalences of 20%, 40% or 60%, respectively. Prevalence reductions are lower (by ~25% at this treatment level) for populations with all genotype 1 and similarly higher for genotype 2/3 populations. Reduction of prevalence to negligible levels within 20 years could be achieved by treating 21 infections per 1000 IDUs annually with 20% baseline prevalence, increasing to 53 or 99 in the 40% and 60% prevalence situation. Restricting retreatment does not alter the short-term (<5 year) projections with low treatment (<20 per 1000 IDUs annually), but reducing prevalence to negligible levels takes longer and becomes impossible at high prevalences (>55%). Lastly, the HCV free life years gained from treating active IDUs are projected to be higher than from treating non-IDUs for prevalences below 60%.
Conclusion Despite the possibility of re-infection, modest rates of HCV treatment amongst active IDUs could effectively reduce transmission. Evaluating and extending strategies to treat HCV among active injectors is warranted.

P71 ANTIVIRAL TREATMENT OF RECURRENT HEPATITIS C AFTER LIVER TRANSPLANTATION—THE NEWCASTLE EXPERIENCE

doi:10.1136/gut.2010.223362.97

S McPherson, M Hudson, D Sheridan, C Miller, K Baxter, M Bassendine. *Hepatology Department, Freeman Hospital, Newcastle-upon-Tyne, UK*

Introduction Recurrence of hepatitis C (HCV) post liver transplantation is universal and may follow a rapidly progressive course,

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.

REFERENCE

1. **Selzner N,** Renner EL, Selzner M, *et al*. Antiviral treatment of recurrent hepatitis C after liver transplantation: predictors of response and long-term outcome. *Transplantation* 2009;**88**:1214–21.

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

doi:10.1136/gut.2010.223362.98

B Smith, I Brown, D Muir, P Nutter. *Imperial College, Healthcare NHS Trust, London, UK*

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

B Smith, I Brown, D Muir, P Nutter. *Imperial College, Healthcare NHS Trust, London, UK*

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