only. There were no deaths or need for liver transplantation in either group.

Conclusion Acute hepatitis B usually runs a benign course but a proportion of patients can develop severe disease. Patients with acute severe hepatitis B may be safely treated with anti-viral drugs. Randomised controlled studies with newer antiviral agents are required to establish guidelines in treating patients with acute severe hepatitis B.

Competing interests None declared.

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PMO-180 | **RAISED SERUM IMMUNOGLOBULINS IN CHRONIC HEPATITIS C: INCIDENCE AND ASSOCIATION WITH GENOTYPE, LIVER FIBROSIS AND SUSTAINED VIRAL RESPONSE**

doi:10.1136/gutjnl-2012-302514b.180

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Introduction Serum Immunoglobulins (Igs) are commonly raised in Chronic Hepatitis C (HCV) but their clinical significance is not fully known. There is also little information on the normalisation of Igs post HCV treatment. We aimed to assess (1) the incidence of raised Igs in HCV patients, (2) the association between the most commonly raised Ig [Immunoglobulin (IgG)] and genotype [G] and liver fibrosis and (3) the association between normalisation of IgG in those achieving sustained viral response (SVR).

Methods Demographics, genotype, pre-and post-treatment Igs, Ishak liver fibrosis scores (F) and SVR of all patients undergoing treatment for HCV since 2006 was collected. Data from G2, G4, G6 and unknown genotype patients were not included in the analyses.

Results 295 (n) patients were treated in the study period {Genotype 1 [G1] 71, Genotype 3 [G3] 205, males 181 (mean age 45.4); females 114 (mean age 41.3)}.

1. 217/295 (73%) patients had raised pre-treatment Igs-either alone or in combination. Raised pre-treatment IgG, IgM and IgA were seen in 32%, 22% and 11% of G1 and in 56%, 16% and 8% of G3 patients respectively. A significant association between viral genotype and raised pre-treatment level was seen only with IgG ($p=0.0009$) and not with IgA ($p=0.46$) or IgM ($p=0.20$).
2. In G1, 43% of patients with advanced fibrosis (F>4) had raised pre-treatment IgG compared to 29% of patients with F≤4 [non-significant (NS) association, $p=0.66$]. However in G3 advanced fibrosis (F>4) was significantly associated with raised pre-treatment IgG [33/41 (80%) with F>4 vs 70/130 (54%) with F≤4, $p=0.0031$] suggesting that pre-treatment IgG can be a good predictor of advanced fibrosis in G3.
3. Overall SVR was achieved in 34% in G1 and 65% in G3. In those who achieved SVR, normalisation of raised IgG was seen more in G3 than in G1 [52% vs 44%, NS association, $p=0.72$].

Conclusion Our study confirms: (1) Presence of raised serum immunoglobulins, particularly that of IgG is common in both G1 and G3 patients. (2) Significant association between raised pre-treatment IgG and advanced fibrosis is seen in G3 but not in G1. In G3, pre-treatment IgG level can be good predictor of advanced

fibrosis. (3) Post-SVR normalisation of IgG is seen more in G3 than in G1.

Competing interests None declared.

PMO-181 | **LONG TERM FOLLOW-UP OF CHRONIC HEPATITIS B (HBV) PATIENTS TREATED WITH PEGYLATED INTERFERON: SINGLE CENTRE EXPERIENCE**

doi:10.1136/gutjnl-2012-302514b.181

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Introduction Pegylated Interferon (PEG IFN) is NICE approved for the treatment of chronic hepatitis B (HBV) but it is now known that the response is genotype dependent. To analyse the long term outcome of chronic hepatitis B patients treated with Pegylated Interferon.

Methods Retrospective analysis of our hepatitis B database to identify HBV patients treated with PEG IFN. The following data were obtained from the patient's records and our electronic reporting systems: demographics, length of treatment, ALT and viral load (HBV PCR) at various points during and after treatment.

Results 13 patients (9 males, average age 36.8 yrs; 4 females, average age 34.7 yrs) were treated from April 2007 with a mean follow-up of 33 months (133 weeks). There were 8 eAg +ve and 5 eAg −ve patients. None were co-infected with the Delta virus. In the eAg +ve group, there were 3 genotype D (South Asians), 3 genotype C (2 Chinese and 1 South Asian), 1 genotype B (South Asian) and 1 unknown genotype (white Caucasian). All had a raised ALT and mild changes (Ishak fibrosis score 0—2) on liver biopsy. 4/8 (50%)—3 Genotype C, 1 Genotype D—achieved eAg seroconversion to eAb +ve and 1/8 (13%)—unknown genotype—achieved sAg clearance at the end of treatment. ALT normalised only in those who sero-converted. HBV PCR was <200 IU/ml in 5/8 patients at week 24 and 4/8 patients had undetectable PCR at the end of treatment at week 48 (lack of data for two patients, one failed treatment and one HBV PCR 42 IU/ml). There were three cases of lamivudine resistance. Four patients relapsed within 1-year post PEG IFN (all genotype D) and required treatment with tenofovir (+/− lamivudine). In the eAg −ve group, there were four genotype D patients (all South Asians, two co-infected with HCV genotype 3a) and 1 unknown genotype (Chinese). The three non co-infected patients showed good response at the end of treatment but all relapsed within 1 year and all needed further treatment with nucleoside analogues with good viral response. The co-infected patients achieved sustained viral response for HCV and maintained a low HBV viral load.

Conclusion This study confirms good outcomes for non-genotype D patients treated with PEG IFN. However, eAg −ve patients with genotype D treated with PEG IFN tend to relapse after treatment. The use of HBsAg quantification will help to tailor treatment in the future.

Competing interests None declared.

PMO-182 | **HCV RESEARCH UK: A UK NATIONAL RESOURCE TO SUPPORT RESEARCH INTO HCV INFECTION**

doi:10.1136/gutjnl-2012-302514b.182

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Introduction Background: Hepatitis C virus (HCV) infection has been identified by the MRC and Department of Health MRC as a