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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PM0-180 RAISED SERUM IMMUNOGLOBULINS IN CHRONIC HEPATITIS C: INCIDENCE AND ASSOCIATION WITH GENOTYPE, LIVER FIBROSIS AND SUSTAINED VIRAL RESPONSE

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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.

PM0-181 LONG TERM FOLLOW-UP OF CHRONIC HEPATITIS B (HBV) PATIENTS TREATED WITH PEGYLATED INTERFERON: SINGLE CENTRE EXPERIENCE

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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 seroconverted. 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.

PM0-182 HCV RESEARCH UK: A UK NATIONAL RESOURCE TO SUPPORT RESEARCH INTO HCV INFECTION

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

priority area for research and development to meet the clinical challenges posed by the scale of the infection in the UK. There has been a perceived lack of connection between clinicians and basic scientists working on HCV in the UK to address this problem. Aims: To create a multi-disciplinary consortium comprising clinicians and non-clinical scientists to encourage translational research. To establish a cohort of 10 000 patients with HCV infection across the UK, together with clinical database and biorepository—"HCV Research UK". To make this resource available to researchers, both academic/commercial, UK-based and abroad.

Methods Aims: Our objective has been to create a multi-disciplinary consortium comprising clinicians and non-clinical scientists to encourage translational research into the factors that determine outcome of infection, treatment response and disease progression. We aim to establish a cohort of 10 000 patients with HCV infection from across the UK that is supported by the necessary systems to make clinical data and specimens available to academic and commercial researchers, both in the UK and abroad.

Results Progress: HCV Research UK has been funded by the Medical Research Foundation (£1.92 million) to establish an infrastructure that connects 18 clinical centres who will recruit 10 000 HCV-infected patients. The key elements of the infrastructure are a bespoke clinical research database, which is linked to a biorepository of samples that will hold serum, PBMCs and DNA from patients. Access to data and samples is managed by a Tissue and Data Access Committee who have the authority to grant ethical approval for research using the resource. The study has been given CLRN portfolio status.

Conclusion Future plans: Recruitment of patients will begin in early 2012 and grant applications have been submitted to (1) Wellcome/Department of Health Innovation Challenge fund (2) MRC call for Stratified Medicine (3) BLT Research call (4) NIHR Programme Development award schemes for research using the resource. It is hoped that portfolio support will provide a mechanism for new centres to join the consortium.

Competing interests None declared.

PMO-183 ROLE OF IL28B POLYMORPHISM IN PREDICTION OF RESPONSE TO THERAPY IN PATIENTS WITH GENOTYPE 1 CHRONIC HEPATITIS C INFECTION

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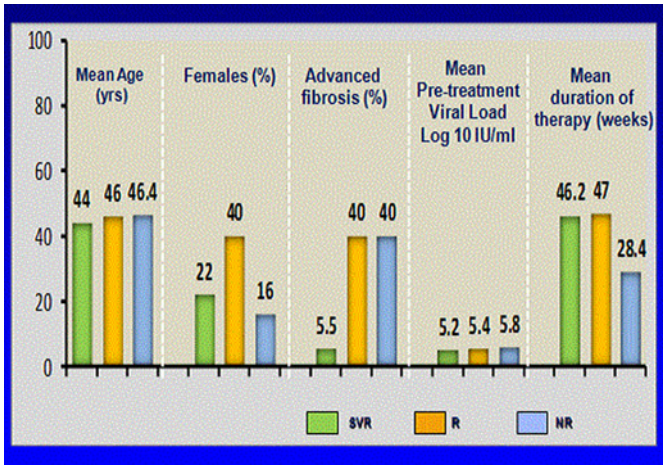
Introduction Patients with chronic hepatitis C virus (HCV) infection have a variable response to antiviral therapy with pegylated interferon and ribavirin. Influences include age, gender, viral genotype, viral load, severity of liver disease and coinfection. Around 45% of patients with viral genotype 1(G1) infection respond compared with 70%–80% with genotype 2/3. Recently a human IL28B polymorphism has been found to predict response in patients with G1 infection. There is little data on this from Europe and a study of IL28B polymorphisms in patients with G1 infection treated in Glasgow was conducted.

Methods Sequential Caucasian patients with G1 chronic HCV who had been treated with combination antiviral therapy were studied. Responses were classified as sustained viral response (SVR), relapse (R) or non-responder (NR). None had coinfection. Data on age, gender, viral load, duration of therapy and severity of liver disease (Ishak fibrosis stage <4 or ≥4) were collected. Individuals were genotyped for IL28B polymorphism rs12979860 using TaqMan®, Drug Metabolism Genotyping Assays and reported as CC, CT or TT.

Results 63 patients were classified (number, mean age, females, advanced fibrosis) by treatment response as SVR (18, 44, 4, 1), R (20, 46, 8, 8) and NR (25, 46.4, 4, 10). Mean pre-treatment viral load was similar in the three groups (5.2, 5.4, 5.8 $\log_{10}$ IU/ml) and mean duration of therapy shorter for NR (46.2, 47.2, 8.4 weeks) who often fulfilled an early stopping rule. The IL28B genotype was highly predictive of response (Abstract PMO-183 table 1). CC individuals have a much greater likelihood ($p<0.002$) of being in the SVR group than CT or TT individuals. Poorer response was also seen in patients with advanced fibrosis.

Abstract PMO-183 Table 1

	Total	CC	CT	TT
SVR	18	12	4	2
Relapse	20	9	10	1
Non-responder	25	2	16	7



Abstract PMO-183 Figure 1 Characteristics of study subjects

Conclusion The IL28B polymorphism is a useful and cheap assay allowing some prediction of response to antiviral therapy in patients with G1 chronic HCV infection.

Competing interests None declared.

Endoscopy I

PMO-184 COLONOSCOPIC TATTOOING OF COLORECTAL NEOPLASIA: A CHANGE IN PRACTICE

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Introduction Quality Assurance Guidelines for colonoscopy in the Bowel Cancer Screening Programme recommend tattooing of all lesions that may require later surgical or endoscopic localisation, using local protocols as guidance.[1] The St. Mark's Hospital colonoscopic tattooing protocol stated that all suspicious lesions should be tattooed, with the exception of those in the caecum and within 20 cm of the anal verge. Three tattoos should be placed (120° apart, close to the lesion) and distal to lesions proximal to the splenic flexure (SpFlx). Left sided lesions should have tattoos placed proximal to the lesion. Our aim was to audit compliance with the tattooing protocol in patients undergoing surgery for colorectal neoplasia.