nurse specialists. Proformas identifying treatment centre, patient characteristics, viral response, treatment compliance and outcome were completed and entered into a database for further analysis. Viral data from genotype 1 HCV patients identified those who relapsed, had viral breakthrough or were non-responders. Non-responders were further categorised into partial and null responders.

Results 361 patients from five centres (Plymouth, Exeter, Truro, Torbay and Barnstaple) were identified. 164/361 patients (45.4%) had genotype 1, of which 11.6% (n=19) were cirrhotic. 40.8% (n=67) achieved SVR, 15.9% (n=26) relapsed and 4.3% (n=7) had viral breakthrough. Of 33 (20.1%) non-responders, 20 were null responders, 11 partial responders and 2 had insufficient viral load data. 9.1% (n=15) stopped treatment early, 7.9% (n=13) were lost to follow-up and 1.8% (n=3) had no post-treatment viral load data available. 17 genotype 1 patients were treated in prison, of which, 11.7% (n=2) stopped treatment early and 41.1% (n=7) were lost to follow-up. Of the 97 (59.2%) genotype 1 cases who did not achieve SVR, at least 44/164 (26.8%) would have a very clear benefit from re-treatment with PIs.

Conclusion A significant number (26.8%) of genotype 1 treatment-experienced patients treated in the South West would benefit from re-treatment, with addition of a PI to their HCV treatment. Adherence with treatment and reliable follow-up of patients are crucial for safe treatment with PIs. Despite the provision of a good HCV service, a significant number of cases did not attend for prearranged reviews. At present, 7.9% of cases treated were lost to follow-up, with prisoners disproportionately unlikely to attend planned clinics.

Competing interests M Saunders: None declared, C Sieberhagen: None declared, L Taylor: None declared, F Fry: None declared, M McKenna: None declared, S Needs: None declared, R Chimakurthi: None declared, M Cramp grant/research support from: Unrestricted educational grants from Roche, MSD and Janssen, Conflict with: Served on advisory boards for Janssen, Roche, MSD and Gilead.

REFERENCE

1. Ghany M, et al. An Update on Treatment of Genotype 1 Chronic Hepatitis C Virus Infection: 2011 Practice Guideline by the American Association for the Study of Liver Diseases. *Hepatology* 2011;54:1433–44.

PMO-166 EARLY EXPERIENCE WITH TELAPREVIR FOR PATIENTS WITH ADVANCED FIBROSIS OR CIRRHOSIS

doi:10.1136/gutjnl-2012-302514b.166

M E Cunningham,* J Schulz, L Payaniandy, Y Kallis, P Kennedy, P Kooner, R Marley, G R Foster. *Barts and The London NHS Trust, London, UK*

Introduction The direct-acting HCV protease inhibitor telaprevir has recently been licensed for treatment of chronic genotype 1 HCV infection, and promises significant improvements in sustained virological response for these patients. However the patients who may benefit most from novel HCV therapies, namely those with advanced fibrosis or cirrhosis who have previously failed to respond to pegylated interferon (pegIFN) and ribavirin treatment, are relatively poorly represented in the telaprevir clinical trials. Efficacy, safety and tolerability were assessed in patients with genotype 1 HCV and advanced fibrosis/cirrhosis who have received telaprevir-containing treatment at the Royal London Hospital.

Methods Laboratory results and case notes were reviewed for all patients treated with pegIFN, ribavirin and telaprevir at the Royal London Hospital between September 2011 and January 2012.

Results Eight patients with genotype 1 HCV had commenced telaprevir-containing treatment. All had advanced fibrosis/cirrhosis (median Ishak score 5, range 4–6). One was treatment-naïve, three had previously failed to respond to pegIFN/ribavirin and four had

relapsed after therapy. All patients had completed at least 4 weeks of telaprevir-containing therapy. With one exception, all patients achieved undetectable HCV RNA at week 4 of treatment; the patient who did not had a viral load of 168 IU/ml at week 4 and undetectable HCV RNA at week 8. One patient had completed 12 weeks of therapy, with undetectable HCV RNA. The most common side effects were fatigue (8/8), pruritis (4/8), rash (3/8), anal pain (3/8), depression (3/8), nausea (3/8), gastrointestinal disturbance (2/8) and oral candidiasis (2/8). Most side effects were successfully managed, although telaprevir was stopped in two patients at week 8 due to worsening rash and one patient withdrew from all therapy at week 4 due to tolerability. The most common laboratory abnormality was an early, transient rise in bilirubin (3/8). Significant anaemia (Hb).

Conclusion Telaprevir in combination with pegIFN and ribavirin appears efficacious in patients with advanced fibrosis or cirrhosis, who have previously failed treatment with pegIFN and ribavirin alone. However, the incidence of significant side effects in this subgroup of patients is high and necessitates frequent follow-up with medical support. Side effects, particularly rash, may limit duration of telaprevir treatment. Whether this impacts on sustained virological response remains to be seen.

Competing interests M Cunningham: None declared, J Schulz: None declared, L Payaniandy: None declared, Y Kallis: None declared, P Kennedy: None declared, P Kooner: None declared, R Marley: None declared, G Foster grant/research support from: Roche, Janssen, Tibotec, Novartis, Consultant for: Abbott, BI, BMS, Chughai, Janssen, Merck, Novartis, Roche, Tibotec.

PMO-167 PRESENCE OF VIABLE HCV RNA IN MONOCYTES AT THE END OF TREATMENT PREDICTS RELAPSE IN GENOTYPE 3 HCV INFECTION

doi:10.1136/gutjnl-2012-302514b.167

¹M E Cunningham,* ²A Javaid, ¹J A Waters, ¹G R Foster. ¹Blizard Institute of Cell and Molecular Science, Queen Mary, University of London, London, UK; ²Vale of Leven District General Hospital, Glasgow, UK

Introduction Although genotype (G)3 HCV is generally regarded as “easy to treat”, based on clinical trial data showing response rates of up to 80%, real world studies have shown substantially lower rates of treatment response (45%), particularly in patients with advanced fibrosis or cirrhosis. Most patients who fail treatment for G3 HCV initially respond to antiviral therapy, but relapse after the end of treatment. HCV RNA has been demonstrated in peripheral blood mononuclear cells from patients with chronic HCV, but whether viral replication occurs in these cells remains controversial. This study tests the hypothesis that viable HCV in monocytes at the end of treatment predicts relapse in patients with G3 HCV.

Methods CD14 (+) monocytes from patients at the end of treatment for G3 HCV were isolated and fused with HuH7 cells. The fused cells were maintained in tissue culture for up to 5 days, before extraction of HCV RNA and quantification by PCR. p Values were derived using the Mann–Whitney U test for comparison of non-parametric data. Results are expressed as mean ± SEM.

Results HCV RNA increased up to fivefold in fused compared to unfused monocytes. Viral protein production was demonstrated in fused cells by indirect immunofluorescence, confirming that viral replication occurs in the fused cells. Fused monocytes from patients who relapsed after treatment showed a significantly greater increase in HCV RNA than those from patients with a sustained virological response (246.8 ± 103.9%, compared to 5 ± 33.9%, p=0.02).

Conclusion These data demonstrate that the presence of replication-competent HCV in monocytes at the end of treatment predicts