were significantly higher during therapy than when compared to both before therapy (p<0.00001) and after therapy (p=0.0002) but levels before therapy were not significantly different from post therapy. Elevated scores on therapy were attributable to an increase in mean HA (p<0.0002) and F3NP (p<0.01) levels on therapy, whereas a significant change in mean TIMP-1 during therapy was not seen. These elevations were seen in all patients regardless of changes in histological fibrosis after therapy (n=20 decrease, n=25 no change, n=35 increase in Ishak stage). However, individual changes in TIMP1 (t=0.259, p=0.04) and changes in ELF (t=0.515, p=0.004) from pre- to post-therapy levels were found to correlate with the change of Ishak fibrosis stage before and during therapy.

Conclusion During interferon-based therapy, levels of HA and F3NP and the ELF score rose globally and subsequently fell to values similar to those seen prior to therapy regardless of fibrosis evolution, while TIMP1 levels remained unaffected. However, ELF scores pre and post therapy did accurately reflect changes in histology. This suggests that ELF scores and non-invasive panels incorporating HA and F3NP should be interpreted with caution during interferon-based therapy.

Results In total 207 treatment naïve patients received antiviral therapy. Mean age at time of treatment was 45 years (20–66); 74% (154) were male and 67% (139) acquired CHC through injection drug use. G1 patients represented 49% (102) of the cohort; 5% (6) were Hepatitis B/HIV co-infected and 95% (196) were Caucasian. A clinical or histological diagnosis of cirrhosis was present in 8% (16). In total 12% (24) moved out of area or were lost to follow-up within 24 weeks of completing treatment. Based on intention to treat, Sustained Virological Response rates (undetectable HCV RNA in serum 24 weeks post treatment) were comparable to those derived from RCT data (Abstract PMO-173 table 1). Non-response was observed in 11% (11/102), 5% (5/98) and 14% (1/7) of G1, G2/3 and G4 patients respectively. Breakthrough or relapse was observed in 18% (18/102), 13% (13/98) and 14% (1/7) of G1, G2/3 and G4 patients respectively. Overall 1% (5) of patients discontinued treatment as a result of a laboratory abnormality and 12% (24) because of other medical complications or side effect intolerance. These proportions are comparable to those observed in RCTs (p=0.753, p=0.146).

Abstract PMO-172 Figure 1 Fibrosis evolution compared to changes in mean ELF score during interferon therapy.

Competing interests None declared.

PMO-173

DISTRIBUTION GASTRO ENTEROLOGY NETWORKS CAN PROVIDE SAFE AND EFFECTIVE HEPATITIS C TREATMENT: RESULTS OF A 4-YEAR AUDIT

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Introduction Chronic Hepatitis C (CHC) treatment is well described in the context of Randomised Controlled Trials (RCTs). Whether these findings can be extrapolated to treatment programmes delivered by nurse specialists working in District General Hospitals (DGHs) is unclear.

Methods The Dorset Viral Hepatitis Network has a catchment area of 750,000 people. Patients are assessed and treated in three DGHs by a team of nurse specialists working under the supervision of four lead clinicians. Between January 2007 and January 2011 standard of care for CHC treatment was Ribavirin and Pegylated Interferon 2a given for 24 weeks (G2/3 patients) to 48 weeks (G1/4). A retrospective analysis of the network’s reference database was undertaken focusing on treatment naïve patients.

REFERENCE


PMO-174

INTRAHEPATIC NATURAL KILLER CELL PHENOTYPING AND FUNCTIONAL ANALYSIS BY FINE NEEDLE ASPIRATION IN CHRONIC HCV INFECTION

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Introduction Hepatitis C virus (HCV) infection results in chronic infection in the majority of subjects, indicating viral immunoevasion strategies. Treatment with interferon alpha (IFNα) stimulates the immune system but the role of NK cells remains unclear.

Methods Intra-hepatic NK cells were obtained from 20 HCV infected donors prior to treatment and 16 non-viral chronic liver disease (CLD) patients along with paired peripheral blood samples. NK phenotype (CD16, NKp30, NKp46, NKG2D and NKG2A) and functional profile (Ki67, CD107a, IFN-γ and Granzyme B) was assessed by flow cytometry. In a separate cohort of 9 HCV patients, who had completed treatment, rate of viral clearance was calculated and pre-treatment peripheral blood NK phenotype and CD107a expression in response to increasing stimulation was measured. At low-level stimulation peripheral blood mononuclear cells (PBMCs) were incubated overnight with 50 u/ml IFNα and exposed to Huh7. Five target cells and at maximal stimulation PBMCs were incubated with 1000 u/ml IFNα and K562 target cells.

Abstract PMO-173 Table 1 Comparison of SVR rates between centres

<table>
<thead>
<tr>
<th>Genotype</th>
<th>SVR in DGH practice</th>
<th>SVR in RCT</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genotype 1</td>
<td>46% (47/102)</td>
<td>41% (241/583)</td>
<td>0.37</td>
</tr>
<tr>
<td>Genotype 2/3</td>
<td>60% (59/98)</td>
<td>68% (194/285)</td>
<td>0.16</td>
</tr>
<tr>
<td>Genotype 4</td>
<td>57% (4/7)</td>
<td>58% (14/24)</td>
<td>0.96</td>
</tr>
</tbody>
</table>

*51% Received Peg IFN α2a. †Derived from χ² test.