Introduction

HILI is common with a prevalence of 10% in US. Transient shift of intra hepatic hemodynamic compromise leads to tissue hypoxia and induces hypoxia induced protein (HIF), heat shock protein 70 (HSP24,70), Endothelial reticular stress (ER) leading to reperfusion injury (RI). Dramatic rise of transaminases, drastic reversal with restoration of perfusion in weeks follows. In cirrhotics HILI requires liver transplantation. This study evaluated spontaneous recovery and salvage in HILI utilising NAC.

Methods

Sixty patients (n = 60) with mean arterial pressure (MAP) <55% and normal LFTs at base line. Group A (n = 28) chronic liver disease (CLD) [alcohol-11/28 (39%), NASH-9/28 (32%), Hepatitis C-4/28 (14%), hepatitis B-2/28 (7%), PBC-1/28 (3%), AIH-1/28 (3%), Group B (n = 32) [respiratory failure-12/32 (37%), CHF-8/32 (25%), CVA-2/32 (6%), sepsis-6/32 (19%), post-op-4/32 (12%)]. Randomized into Placebo group- A1 (14) & B1 (16) and IV NAC for 48 hours - A2 (14) & B2 (16). Serum Transaminases, Bilirubin, INR, Liver function tests (LFTs) on 3rd (57%)6th day-(33%) 9th day-(19%) and 12th day-(14%). 1/14(7%) died of sepsis [NAC Groups A1-normalised LFTs 3rd day-(63%), 6th day-(25%) 9th day1/16(6%), one died]. B1(CLD) [LFTs 3rd day-(19%) 6th (44%) 9th 1/16(6%)] died. B2(CLD)[Normalized LFTs 3rd day-(63%), 6th day-(25%) 9th 1/16(6%), one died]

Conclusion

This Study postulates that IV NAC (A2, B2) has efficient spontaneous recovery and salvage in non-CLD subgroup B2 (65%) > A2(57%) in day 3, in CLD NAC (A2) > placebo (A1) clinical recovery over placebo at 3rd day, (44%) over (36%) - 6th day. Larger trial need to establish the routine usage of IV NAC in HILI.

Disclosure of Interest

None Declared.
cryoglobulin positive if a precipitate formed which disappeared on re-warming. Clinical features were correlated with presence of cryoglobulin.

**Results** Adequate samples were received in 65/75 HCV infected patients (31 G1, 34 G3). Of these, 35.4% (23/65) had detectable cryoglobulin. No cryoglobulin was detected in the healthy control samples. Clinical associations are listed below (p values from Fisher’s Exact Test unless otherwise stated).

### Abstract PWE-132 Table

<table>
<thead>
<tr>
<th>Clinical Parameter</th>
<th>Cryoglobulin Positive (n = 23)</th>
<th>Cryoglobulin Negative (n = 42)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (%)</td>
<td>13 (57)</td>
<td>34 (81)</td>
<td>0.04</td>
</tr>
<tr>
<td>Age (mean yrs)</td>
<td>44.6</td>
<td>45.8</td>
<td>0.73*</td>
</tr>
<tr>
<td>Genotype 3 (%)</td>
<td>15 (65)</td>
<td>19 (45)</td>
<td>0.10</td>
</tr>
<tr>
<td>Cirrhosis (%)</td>
<td>13 (57)</td>
<td>10 (24)</td>
<td>0.09</td>
</tr>
<tr>
<td>Renal Function (mean Cr)</td>
<td>67</td>
<td>72</td>
<td>0.17*</td>
</tr>
<tr>
<td>Viral load (mean IU/ml)</td>
<td>$5.2 \times 10^5$</td>
<td>$4.6 \times 10^5$</td>
<td>0.23*</td>
</tr>
<tr>
<td>Any symptoms (%)</td>
<td>12 (52)</td>
<td>16 (38)</td>
<td>0.30</td>
</tr>
<tr>
<td>Fatigue (median score/10)</td>
<td>5</td>
<td>5</td>
<td>0.75</td>
</tr>
</tbody>
</table>

* t test  
* Wilcoxon rank sum

There was no difference in prevalence of IVDU or Diabetes in those with cryoglobulins. No individual symptom was associated with cryoglobulin detection.

**Conclusion** Cryoglobulinaemia has a surprisingly high prevalence of 35% within our UK based cohort of HCV patients, being less common in males. Symptoms are non-specific and occur in the absence of detectable cryoglobulin with no association between symptoms and cryoglobulin positivity. There was a non-significant trend to association with cirrhosis and genotype 3 as shown in previous studies.

Cryoglobulinaemia may have been underdiagnosed previously due to practical difficulties with testing and it should be considered in any patients with renal dysfunction and HCV.

**REFERENCES**


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**PWE-133 OSSOPHAGEAL VARICES SCREENING - ARE WE MEETING THE GUIDELINES?**

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**Introduction** Oesophageal varices develop and enlarge in cirrhotic patients at a rate of 6–8% per year and haemorrhage occurs at a rate of 5–15% per annum, causing significant morbidity and mortality. Of the 16,000 deaths/year attributed to cirrhosis, 53% are due to variceal bleeding. Despite improvements in therapy and prophylaxis the mortality from bleeding varices has remained static. Current BSG guidelines recommend screening with OGD at diagnosis and then 1–3 yearly depending on endoscopic findings.

**Methods** A retrospective review of clinic letters for all gastroenterologists at STDH from Aug-Oct 2012 was performed. Those eligible for variceal screening i.e. established cirrhosis, decompensated liver disease or evidence of portal hypertension on imaging were identified. Demographic details and liver disease aetiology were recorded. The endoscopy reporting system was reviewed to identify OGDs performed within the last 3 years and the indication for OGD. If an OGD report was absent, the appointment system and case notes were reviewed to establish if the patient refused or failed to attend (FTA) for OGD. Where no evidence of FTA or refusal was found, clinician failure to refer was documented.

**Results** 84 eligible patients were identified, 64 (76.2%) had an OGD recorded within the last 3 years. Table 1 shows results according to liver disease aetiology. In the group with a NAFLD/NASH or ‘other’ aetiology, all 16 cases who had not had OGD had not been referred. Of the alcohol aetiology, 7/16 had not been referred for OGD, and 9/16 FTA or were documented to have refused the test.

### Abstract PWE-133 Table 1

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Number</th>
<th>OGD within 3 years (%)</th>
<th>Screening/ surveillance</th>
<th>Bleeding</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>67</td>
<td>51 (76.1)</td>
<td>37 (55.2)</td>
<td>9 (13.4)</td>
<td>5 (7.5)</td>
</tr>
<tr>
<td>NAFLD/NASH</td>
<td>8</td>
<td>5 (62.5)</td>
<td>3 (37.5)</td>
<td>1 (12.5)</td>
<td>1 (12.5)</td>
</tr>
<tr>
<td>Other*</td>
<td>8</td>
<td>7 (87.5)</td>
<td>5 (62.5)</td>
<td>1 (12.5)</td>
<td>1 (12.5)</td>
</tr>
</tbody>
</table>

*autimmune hepatitis, primary biliary cirrhosis, hepatitis C, cryptogenic cirrhosis, and unknown or still under investigation

**Conclusion** Three quarters of patients eligible for varices screening have had an OGD within the maximum time frame suggested by the BSG guidelines. However, excluding OGDs performed for acute bleeding or other indications only 45/84 (56.6%) have been appropriately screened.

The majority of cases in this audit are secondary to alcoholic liver disease and their high FTA rate reiterates the known difficulties in engaging this group of patients, although numbers are small. This audit suggests a need to improve rates of screening for oesophageal varices; the main reasons suggested by this audit that could be targeted to improve screening rates are appropriate referral by clinicians and reluctance to attend for the test particularly in liver disease secondary to alcohol.

**Disclosure of Interest** None Declared.

**REFERENCE**


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**PWE-134 OUTCOMES FOR LIVER DISEASE PATIENTS ADMITTED TO A DISTRICT GENERAL HOSPITAL**

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**Introduction** In the UK, liver disease is the 5th commonest cause of death. A recent BSG commissioning report states that there were 45,694 hospital episodes due to liver disease with a mortality rate of 15.5% and median age of death of 59. The guidelines recommend that all liver disease should be managed by a hepatologist. Newham University Hospital (NUH) serves a socially deprived and ethnically diverse population of 290,000.

**Methods** All patients with a primary diagnosis of liver disease admitted to NUH from 1 April 2012 to 31 October 2012 were included in this study. Patients were identified from the on-call admission lists and electronic patient records and admission notes were checked for suitability. Patients admitted with alcohol withdrawal but without underlying liver disease were excluded. We examined the outcomes of all liver patients admitted during this time period.

**Results** 78 patients were admitted, of which 9 had ≥2 admissions. The demographic data and outcomes are listed in Table 1. The ethnic variation reflected that of the local community. The main causes of liver disease were alcoholic liver disease (56%), viral hepatitis...