test only if presence of sepsis and 4% only if patients are encephalopathic. Only 28% have access to lab polymorphonuclear leucocytes in ascites to confirm SBP; 60% use white cell count (WCC) > 250 and 16% use WCC > 500 in ascitic fluid as criteria. Cefotaxime (52%) and Tazocin (32%) were the preferred antibiotic choices with treatment duration ranging between < 5 days (4%), 5–7 days (40%) and > 7 days (56%). Surprisingly, 8 out of 22 respondents (36.3%) do not administer albumin routinely for confirmed SBP but 100% prefer 20% human albumin compared to 4.5% human albumin. There was huge discrepancy in the albumin administration regimen between the members. One Consultant gives Terlipressin to all SBP patients but 24% will consider terlipressin if patients are at high risk of hepatorenal syndrome. 4% routinely repeat diagnostic ascitic tap after 48 hours on SBP treatment while 20% repeat only if infection not settling; 64% do occasionally and 12% ‘never’ repeat. A good 16.7% members do not start prophylactic antibiotics after an episode of SBP but with others, Ciprofloxacin (50%) and Norfloxacin (50%) are favourites. 96% are not in favour of primary antibiotic prophylaxis for ascites with low protein counts. Encouragingly, 45.5% would consider referral for liver transplantation in appropriate patients after an episode of SBP.

Conclusion Even among specialists dealing with chronic liver disease patients day to day, there is wide variation in management of SBP. It is alarming to note that doctors do not believe in secondary prophylaxis and do not administer albumin as part of treatment. There is serious need to standardise treatment and prevent improper management that can cause deterioration in liver function rendering them poor candidates for transplantation.

Disclosure of Interest None Declared

**REFERENCES**


**PTU-090** AUDIT ON FACTORS THAT PREDICT BONE DISEASE IN CIRRHOSIS

doi:10.1136/gutjnl-2013-304907.180

1 A Bond, K Koss, V Maddirala. ‘Gastroenterology, East Cheshire NHS Trust, Macclesfield, UK

**Introduction** Osteodystrophy is a recognised complication with cirrhosis. Female sex, cholestasis, low BMI are proven to increase risk of bone disease in cirrhotics17. Our aim is to audit our current practise of diagnosis and management of osteoporosis in liver patients and to identify other associated risk factors.

**Methods** Retrospective audit was done on 73 cirrhotic patients enrolled in the Hepatocellular Carcinoma (HCC) surveillance programme in our hospital. Our practise was compared with British Society of Gastroenterology (BSG) recommendations15. Data on demographics, aetiology of cirrhosis, alcohol or steroid intake, post menopausal state, presence of varices, investigations for osteoporosis done and treatment given were collected and analysed.

**Results** Only 54 patients had DEXA scan performed at any point, with average duration between cirrhosis diagnosis and first scan being 2.41 years. 14 had osteoporosis (T score < −2.5) at hip or lumbar spine and 11 were osteopenic (T of −1.5 to −2.5), showing a prevalence of bone disease at 73.5%. On analysis, except for female sex, no other variable increased risk of bone disease. Few patients were tested for Vitamin D (6.8%) or hormonal studies (8.1%) but 78% had thyroid tests and 100% had bone profile. Treatment for osteoporosis was given, as recommended in all subjects; however 2-yearly follow up scans happened only in 19.2%. Though 6/11 patients had varices, association was not statistically significant by Pearson chi-square test (p value of 0.058). Similarly, longer duration of cirrhosis did not increase risk as analysed by t-test.

**Conclusion** Our audit showed high prevalence of bone disease (osteoporosis and osteopenia) in chronic liver disease patients, however there is bias as DEXA scans were requested only in symptomatic patients. There was poor compliance with BSG guideline especially with surveillance intervals. Female sex increases risk but the association of bone disease with duration of cirrhosis and presence of varices are not statistically significant.

**Disclosure of Interest** None Declared

**PTU-091** TRANSIENT ELASTOGRAPHY AS A PREDICTOR OF OESOPHAGEAL AND GASTRIC VARICES

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1 A Bond, K Koss, V Maddirala. ‘Gastroenterology, East Cheshire NHS Trust, Macclesfield, UK

**Introduction** Screening for varices with two yearly gastroscopy is recommended practise for all patients with liver cirrhosis. Prevalence of oesophageal or gastric varices in patients with liver cirrhosis varies in the literature from 30–70%.

The Aim of this study was to analyse if the result of transient elastography (TE) can be used to predict the risk of developing gastric and/or oesophageal varices in cirrhotic patients. We compared this data with endoscopic and radiological evidence of portal hypertension.

**Methods** We analysed data of 807 patients who underwent TE examination at East Cheshire NHS Trust and identified 103 patients with suspected liver cirrhosis (liver stiffness score of >12.5 kPa). The cut off F4 liver fibrosis/cirrhosis varies between 14.5 kPa to 18.2 kPa for different aetiologies of liver cirrhosis. We evaluated results of TE against upper GI endoscopy and abdominal ultrasound/CT.

**Results** 26% of patients with F4 liver fibrosis/cirrhosis were found to have oesophageal/gastric varices at endoscopy. A total of 37% patients had evidence of portal hypertension on USS or CT. Only 41% of patients with splenomegaly were found to have oesophageal/gastric varices. 78% of those found to have varices had a liver stiffness score of >25 kPa.

**Abstract PTU-091 Table**

<table>
<thead>
<tr>
<th>Liver Stiffness (kPa)</th>
<th>Number of patients</th>
<th>Percentage of patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>70.1–75</td>
<td>12</td>
<td>44.44</td>
</tr>
<tr>
<td>40.1–70</td>
<td>4</td>
<td>14.81</td>
</tr>
<tr>
<td>25–40</td>
<td>6</td>
<td>22.22</td>
</tr>
<tr>
<td>20–24.9</td>
<td>2</td>
<td>7.4</td>
</tr>
<tr>
<td>12.5–19.9</td>
<td>3</td>
<td>11.11</td>
</tr>
</tbody>
</table>

**Conclusion** We postulate that TE helps us to stratify a patient’s risk of developing oesophageal/gastric varices, according to their liver stiffness score. The likelihood of developing oesophageal/gastric varices increases with increased liver stiffness. Patients with liver stiffness scores lower than 25kPa may avoid unnecessary upper GI endoscopy.

**Disclosure of Interest** None Declared