levels of malondialdehyde (MDA)-TBA adduct, a naturally occurring product of lipid peroxidation.

Results 22 subjects were recruited: 10 AH (6 males; median MELD 12; DF 45.6); 5 ALD (2 males; median MELD 18) and 7 HVs (3 males). MDA was significantly higher in AH vs HVs (median 36.1μM vs 14.8μM; p<0.01) and in ALD vs HVs (median 28.6μM vs 14.8μM; p = 0.03) but similar between AH and ALD patients. In AH patients, there was no strong correlation between MDA levels with MELD or DF (r=0.14 and 0.57, respectively; both p>0.05) (figure 1).

Conclusion Oxidative stress as measured by lipid peroxidation is increased in patients with ALD and AH when compared to HVs.

**Discussion** A PPV of 15% suggests the automated count has little value in clinical practice. Its benefit lies in its strong NPV to rule out SBP, but reliance on this method results in inflated SBP rates and overtreatment with potentially harmful antibiotics.

**REFERENCE**
6. GIRFT review team (2018). Getting it right first time: Gastroenterology, NHS

**P79** AUTOMATED CELL COUNT FOR THE DIAGNOSIS OF SPONTANEOUS BACTERIAL PERITONITIS: IS IT USEFUL IN CLINICAL PRACTICE?

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Introduction Spontaneous bacterial peritonitis (SBP) is both a common and severe complication of ascites. It carries a mortality rate of 11–19.1%,1–3 thus early diagnosis and treatment is imperative in this vulnerable group.

The incidence of SBP ranges from 10–30% in hospital inpatients with cirrhotic liver disease.4 However, asymptomatic outpatients carry a much lower rate.5 A recent UK-wide report observing both inpatients and outpatients found a total SBP rate of 3.13%,6 though underreporting may have affected this.

The gold standard for diagnosing SBP is an ascitic fluid manual cell count (>250 mm3 polymorphonuclear leukocytes).7 Our trust does not have access to same day manual samples, there were 37 positive results. With a positive predictive value (PPV) 15%. It must be noted that the negative predictive value (NPV) was 100% (n=78). Of 103 automated only samples, there were 37 positive results. With a PPV of 15% we would expect a further 3.5 cases. Therefore, potentially 31.5 cases of SBP were over diagnosed due to our reliance on the automated result.

**Discussion** A PPV of 15% suggests the automated count has little value in clinical practice. Its benefit lies in its strong NPV to rule out SBP, but reliance on this method results in inflated SBP rates and overtreatment with potentially harmful antibiotics.

**REFERENCE**
6. GIRFT review team (2018). Getting it right first time: Gastroenterology, NHS

**P80** DIVERGENT GUIDELINES REGARDING METHOTREXATE PRESCRIBING IN THE UK: TIME FOR HEPATOLOGISTS TO PROVIDE DIRECTION?

1,2Lucy Turner*, 1Martin Veysey, 1John Hutchinson, 1Charles Millson. 1Hull York Medical School, UK

Introduction Low dose methotrexate (MTX), an effective treatment for immune-mediated diseases, has been used by multiple specialities since the 1960s. Historically, MTX-induced hepatotoxicity dictated its potential use; only being advocated in patients with ‘life-ruining’ disease and regular liver biopsies were mandatory.1 Guidelines have been divergent across various specialities since 1987, and this persists today.2–4

Aim To compare current guidelines regarding MTX prescribing, monitoring and action in the face of presumed hepatotoxicity.

Methods The archives of professional bodies and associations in rheumatology, dermatology and gastroenterology were searched for guidance pertaining to the use of methotrexate, dating back to 1950, within the UK, Europe and America.

Results A total of 17 guidelines related to MTX monitoring were published between 1972 and 2019 by dermatologists, rheumatologists and gastroenterologists. Guidelines differed across specialties to this day in regard to baseline investigations, monitoring and action required on liver blood test abnormality. The most recent of these are demonstrated in table 1.

Discussion Divergent guidelines regarding low dose MTX, particularly in relation to its apparent hepatotoxicity, have persisted for decades. Liver blood tests are a poor indicator of liver dysfunction and the advent of non-invasive measures of liver fibrosis provide a potential alternative. Hepatologists have stopped short of clear advice and guidance in this area.
Abstract P80 Table 1

<table>
<thead>
<tr>
<th>British Society of Rheumatologists 2017</th>
<th>British Association of Dermatologists 2016</th>
<th>Inflammatory Bowel Disease advisory group 2017</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline investigations</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height and weight</td>
<td>FBC</td>
<td>History</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>U&amp;E</td>
<td>Examination</td>
</tr>
<tr>
<td>GFR</td>
<td>LFTs</td>
<td>Daily alcohol intake</td>
</tr>
<tr>
<td>ALT and/or AST</td>
<td>Hepatitis B and C</td>
<td>FBC</td>
</tr>
<tr>
<td>albumin</td>
<td>U&amp;E</td>
<td>U&amp;E</td>
</tr>
<tr>
<td>Comorbidity</td>
<td>Varicella zoster status</td>
<td>LFTs</td>
</tr>
<tr>
<td>assessment</td>
<td>+/- COR &amp; exam</td>
<td>&quot;Non-invasive evaluation of liver fibrosis may be useful&quot;</td>
</tr>
</tbody>
</table>

| **Immediate monitoring**               |                                          |                                               |
|                                      |                                          |                                               |
|                                      |                                          |                                               |

| **3/12 after dose stabilised**         |                                          |                                               |
|                                      |                                          |                                               |
|                                      |                                          |                                               |

**Immediate investigations**

- **Baseline investigations**
  - FBC
  - U&E
  - LFTs

- **Immediate monitoring**
  - Every 2 weeks
    - FBC
    - U&E
    - LFTs
  - Every 1-2 weeks
    - FBC
    - U&E
    - LFTs
  - At 4 weeks:
    - FBC
    - U&E
    - LFTs

**3/12 after dose stabilised**

- **Every 3 months**
  - FBC
  - LFTs
  - U&E

**Alcohol**

- Not stated

**Action if abnormal LFTs**

- **Contact**
  - Withhold/decrease dose of MTX and urgently consider with a gastroenterologist
  - Treatment if...

- **INTERUPTION in treatment if...**
  - ALT/AST > 2 fold
  - ALT/AST > 2 fold +/− TB

**Unexplained albumin < 30g/L**

- LFTs in 2-4 weeks

**Platelets < 140 x 10^9/L**

We have developed an algorithm for patients commencing MTX and receiving this drug long-term, which we hope will provide some consistency.

**REFERENCES**


**P82 ESTIMATING THE PREVALENCE OF WILSON’S DISEASE USING ROUTINE LABORATORY AND CLINICAL DATA**

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**Introduction** The true prevalence of Wilson’s disease (WD), remains unknown. The estimated genetic prevalence in the UK (142/million) is higher than the clinical prevalence (15/million) reported in other European studies. We aimed to (1) estimate the prevalence of WD in Nottingham, (2) assess the utility of readily available laboratory and clinical data to identify patients with WD, and (3) propose a system to identify patients with WD nationally.

**Methods** Patients with WD attending Nottingham University Hospital (NUH) 2011–2018 were identified using multiple sources of case ascertainment: (1) serum ceruloplasmin level <0.2 g/l (2) 24-hour urinary copper measurement (3) ’Wilson’ in a liver biopsy report (4) hospital prescription for Penicillamine, Trientine or Zinc and (5) admission to NUH coded with ICD-10 Code E83.0 ‘Disorder of copper metabolism’.

We identified potential cases of WD using the Leipzig score, confirmed the diagnosis in hospital records and calculated the point prevalence with Poisson confidence intervals using the Office for National Statistics mid-2017 population estimates for the denominator population.

**Results** We identified 1,794 patients from ≥1 source, and 13 patients had WD. The overall prevalence of WD was 12.6/million (95%CI 6.7–21.6); males 15.6/million (95%CI 6.7–30.8) and females 9.6/million (95%CI 3.1–22.5). Patients with confirmed WD were followed up by: Hepatology 12 (92.3%), Neurology 6 (46.2%), Psychiatry 4 (30.8%), and Renal 1 (7.7%). 5 (38.5%) were being managed for cirrhosis secondary to WD. 1 (7.7%) had received a liver transplant.

Additionally, 23 (1.3%); males n=19) patients had a low (<0.2 g/l) serum ceruloplasmin level and an elevated 24 hour urinary copper, but had not been investigated further for WD. These patients, if confirmed to have WD, would increase the prevalence of WD to 34.9/million (95%CI 24.5–48.4).

**Discussion** This is the first UK population-based study of WD prevalence. The prevalence found in this study is lower than the previous UK population-based genetic study, but comparable to European population-based clinical studies. The significant difference in prevalence between genetic and clinical studies may be due to under-diagnosis of WD or variable genetic penetrance. The lower prevalence of WD among females indicates that more cases of WD are ’missed’ in females than males. The method of case ascertainment used in this study may be a cost-effective method of identifying patients with WD, and similar practices could be adopted nationally.