Abstract P223 Figure 1

During the last admission 29% patients had a discussion documented (1 declined, 5 HE or low Glasgow coma score). 67% of NOK had a discussion documented during the last admission (1 no NOK). 3 patients were referred to palliative care. 68% of the patients had indicators suggesting a mortality more than 81% within 1 year. 10 had refractory ascites (50% survival at 1 year). 12 had previous HE (mortality 42% at 1 year).

Conclusions In this single centre retrospective audit, a significant proportion of patients and/or their NOK are not being informed about the advanced nature of their condition and poor prognosis nor is there sufficient and timely involvement of palliative care or advanced care planning. Further prospective longitudinal studies need to be undertaken across multiple sites to ascertain the extent of this potential gap in care provision.

REFERENCES

Results 214 patients had a fibroscan reading in 2018 on the basis of abnormal LFTs or known liver disease. 121 (56%) were female. The modal aetiology was non-alcoholic fatty liver (42%), followed by alcoholic pathology (20%). Other indications included autoimmune, metabolic, viral or idiopathic liver disease.

Of this cohort, 111 had OGD results available. Within this group, 33 patients were identified as at-risk according to the Baveno criteria. In the at-risk group, 10 patients were found to have oesophageal varices. Additionally, two patients from the at-risk group were found to have gastric varices and two patients were found to have portal hypertensive gastropathy (PHG).

In the patients who satisfied the criteria and were deemed not to be in need of variceal screening, one patient had grade 1 oesophageal varices. One patient was found to have gastric varices and a two were found to have PHG.

Six patients were identified as being in need of variceal screening but had not undergone an OGD.

Only two patients in the cohort had undergone upper GI endoscopy for a suspected acute upper GI bleed - one was found to have PHG; another was found to have grade II varices with red signs which were banded.

Conclusions The Baveno VI criteria have a negative predictive value (NPV) of 98.7% (95% CI 92.3 to 99.8%) in the assessment of oesophageal varices. If extended to include gastric varices, the criteria have a NPV of 79.2% (95% CI 73.9 to 83.6%).

This retrospective analysis of a local cohort demonstrate that fibroscan and platelet count can be used as a non-invasive method to stratify patients with liver disease according to their risk of having oesophageal varices.

We would advocate for greater use of TE in patients with liver disease in the hope of minimising the costs and risks associated with unnecessary endoscopic screening.

REFERENCE

P224

AVOIDING INVASIVE VARICEAL SCREENING: DOES THE BAVENO VI CONSENSUS CRITERIA APPLY IN A LOCAL COHORT?

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Introduction Variceal bleeding is a common complication of portal hypertension and gastroenterologists often request variceal screening for patients with a new diagnosis of cirrhosis. However, OGD is unpleasant for patients and though generally considered safe is not without complications (e.g. aspiration, perforation, bleeding). The Baveno VI consensus states that in patients with a Fibroscan (transient elastography, TE) reading of <20 kPa and a platelet count of >150, screening endoscopy can be safely deferred. We aim to demonstrate that these criteria can be validated in our local cohort.

Methods We extracted data from the trust’s fibroscan clinic logbook and Unisoft database. Results were limited to OGDs undertaken in the trust for any indication. We compared TE results, platelet count and endoscopic findings against the Baveno VI criteria.

P225

PARTIAL SPLENIC ARTERY EMBOLISATION FOR PORTAL HYPERTENSION – A SINGLE CENTRE EXPERIENCE

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Introduction Variceal Haemorrhage (VH) which is refractory to medical and endoscopic secondary prophylaxis can be a challenge. When TIPS is not possible, as is sometimes the case in the setting of mesenteric venous thrombosis, partial splenic artery embolization (SAE) has been demonstrated as an effective rescue therapy. However, serious complications have been reported in up to one third of patients.

Methods A radiology database search revealed 143 splenic embolisation procedures performed between September 2008 and December 2019. Following exclusion for splenic haemorrhage in trauma or splenic artery aneurysms in patients with pancreatitis, 8 patients received partial splenic artery embolisation for portal hypertension related indications.

Results 8 patients received partial SAE (targeting 50% of splenic volume) to treat complications of portal hypertension between November 2015 and September 2019. The median
age was 46, aetiology of portal hypertension was, extrahepatic portal venous thrombosis (n=5), PSC (n=1), PBC (n=1) and obliterator portal venopathy (n=1). 2 patients previously had liver transplants. The indications for embolisation were spleno-megaly associated abdominal pain (n=1), ascites (n=1) and recurrent VH (n=6). One patient had ascites (grade 3) pre-procedure. Post-embolisation median platelet and total white cell counts increased from 67 to 105 × 10^9/L and 2.1 to 4.7 × 10^9/L respectively and median bilirubin reduced from 26 umol/L to 16 umol/L. After the procedure 0/6 patients embolised for VH had a recurrence. 7 out of 8 patients developed post-embolisation syndrome and 2 patients developed pleural effusions which did not require drainage. 1 patient had a puncture site haematoma treated conservatively. The patient embolised for ascites developed SBP and decompen-sated further, requiring transplantation 23 days after embo-lisation. 2 of 8 patients died following embolisation, one after 5 months from liver abscesses in a failing graft and the other 15 months later from an unrelated cause.

**Conclusions** In selected cases partial splenic embolisation can ameliorate portal hypertension (as evidenced by increasing white cell and platelet counts) and prevent recurrent VH. The majority of patients will develop post-embolisation syndrome and serious complications occurred in 3 of 8 patients. Further investigation into splenic embolisation as a treatment for portal hypertension in selected patients may be beneficial.

**Oesophagus**

**P226 UNDERDIAGNOSIS OF EOSINOPHILIC OESOPHAGITIS IN PATIENTS WITH DYSPHAGIA IN A DISTRICT GENERAL HOSPITAL**

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**Introduction** Eosinophilic Oesophagitis (EoE) has an annual incidence estimated at up to 20 new cases per 100,000 inhabitants. It must be considered in the differential diagnosis of patients with dysphagia or a food bolus. Our previous audits have suggested it is underdiagnosed in our institution. The aim of this study is to evaluate the adherence to European guidelines in the detection of EoE.

**Methods** We retrospectively reviewed the electronic patient records of all patients presenting for an upper gastrointestinal endoscopic procedure with an indication of dysphagia or a finding of food bolus obstruction over 1 year. The study was undertaken in a district general hospital in the south of England. Data was then collected for sex, age, macroscopic findings, quality and location of biopsies as well as histology. This was measured against European guidelines in the diagnosis of EoE, including at least six oesophageal biopsies from different locations and a histological diagnosis documenting 15 eosino-phils/hpf in the oesophageal mucosa. We applied percentages, means and standard deviations to analyse the data.

**Results** 1 year of endoscopies were reviewed (n=249). 46% of patients were male, the mean age was 68 (range 17–97). At the time of endoscopy only 42.2% of patients had oesoophageal biopsies. When we excluded patients with an endoscopic diagnosis of oesophageal malignancy this reduced further to 40.0%. In this group only 9.4% had the recommended six biopsies. The average number of biopsies for each patient was 3.7 (standard deviation 2.5). There were 7 patients with a histological diagnosis of EoE, of whom 6 had a documented eosinophil count of 15 eosinophils/hpf on histology.

**Conclusions** Considering the incidence of EoE, our data suggests that it is very likely underdiagnosed in patients with dysphagia or a finding of food bolus. This may be attributed to lack of awareness of the condition leading to insufficient biopsies and/or the lack of awareness for the number of biopsies required. Our data suggests that histological analysis is largely adhering to guidelines. These practices could be commonplace among trusts nationwide and further work must be done to improve awareness and diagnosis of this treatable condition. This is particularly relevant with the recent addition of an orodispersable budesonide specifically for its management.

**REFERENCE**


**P227 A EUROPEAN COMPARISON OF BARRETT’S VERSUS SQUAMOUS OESOPHAGEAL RESECTIONS: IS STRicture RISK RELATED TO PATHOLOGY?**

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**Aims** ESD is a minimally invasive therapeutic option for early oesophageal neoplasia, however is not without risk. In Europe, the complication profile is most established for Barrett’s neoplasia, being the predominant pathology, and stricture risk has been shown to be related to lesion circumference. Our aim was to compare the safety of ESD between Barrett’s and squa-mous neoplasia in a Western population.

**Methods** This was a retrospective analysis of all oesophageal ESDs performed within 3 tertiary referral centres in Europe. The primary outcome was post procedure stricture rate.

**Results** 226 oesophageal ESDs from 201 patients were included, consisting of 167 Barrett’s and 59 squamous neoplasia. Average age was 70.7 in Barrett’s and 68.5 in squamous neoplasia, with lesion size 34.6 mm and 34.2 mm and en bloc resection rate 96.6 and 94.6% respectively. The complication rate was 3/167 perforations or delayed bleeds and 7/167 strictures in Barrett’s, with 1/58 perforations or delayed bleeds and 15/58 strictures in squamous (1 patient lost to follow up). Circumferential lesion involvement did increase stricture

**Abstract P227 Table 1 Stricture Risk Stratified by Circumferential Lesion Involvement**

<table>
<thead>
<tr>
<th>Lesion circumference (%)</th>
<th>Strictures in Barrett’s ESD (n,%</th>
<th>Strictures in Squamous ESD (n,%</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤1/3</td>
<td>0/98 (0.0%)</td>
<td>2/3 (13.0%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>&gt;1/3–2/3</td>
<td>1/56 (1.8%)</td>
<td>6/26 (23.1%)</td>
<td>0.001</td>
</tr>
<tr>
<td>&gt;2/3</td>
<td>6/13 (46.2%)</td>
<td>6/9 (66.7%)</td>
<td>0.354</td>
</tr>
</tbody>
</table>