October 2019 for ALD. Data was available for 409 patients. Demographic and socioeconomic data was collected, alongside pre-transplant psychiatric assessment. Evidence of recidivism in those surviving longer than 3 months post-transplant and subsequent outcomes were recorded.

1,761 patients underwent liver transplantation in Scotland over this 27 year period, 26.8% for ALD. Median follow up was 70 months (IQR 38-113) during which 319 patients remained abstinent, and 90 relapsed. Of these, 33 drank harmfully, and 3 developed ALD related graft loss.

Rates of recidivism were highest those who at listing were; younger (55.6 vs 58.2 years p=0.0006), had a shorter pre-transplant abstinence (10 vs 15 months p<0.0001), no co-factor for liver disease (p<0.0001) and identified as high or moderate risk by psychiatry (59% vs 35% p<0.0001). Median time to recidivism was 21.4 months and this was not influenced by severity of drinking. Logistical regression identified several potential risk factors including; age (OR 0.959, [CI 0.922, 0.975]); presence of co-factor (OR 0.312, [CI 0.244, 0.604]); shorter pre-transplant abstinence (OR 0.953, [CI 0.944, 0.982]).

Here we present 27 years of follow-up data for all patients who underwent liver transplant for ALD related cirrhosis in Scotland, demonstrating a post-operative recidivism rate of 22%. Key factors that appear to increase risk of recidivism include younger age, absence of co-factor at listing, and shorter length of pre-transplant abstinence.

Oesophagus

**Abstract HFR-1**

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>TFF3: -</th>
<th>TFF3: +</th>
<th>TFF3: +/-</th>
<th>TFF3: +/4</th>
<th>TFF3: +</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>14</td>
<td>26</td>
<td>12</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

**Table 1**

<table>
<thead>
<tr>
<th>Endoscopy and histology findings</th>
<th>Endoscopy with in 12 months</th>
<th>Endoscopy within 3 months</th>
<th>Endoscopy within 3 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endoscopy</td>
<td>NDBE: 5</td>
<td>NDBE: 1</td>
<td></td>
</tr>
<tr>
<td>histology</td>
<td>ATYPIA: 1</td>
<td>LGD: 2</td>
<td></td>
</tr>
<tr>
<td>findings</td>
<td>INDEF: 2</td>
<td>IMC: 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LDG: 2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

40 patients (71%) had a either a low-risk result (TFF3 positive only – 26) or required a repeat Cytosponge® (TFF3/aty-pia/p53 negative or equivocal – 14). 16 patients (29%) needed an endoscopy with 3 months (cellular atypia – 12, p53 & cellular atypia – 4). 14 of these patients have since had an endoscopy of which 7 had a new diagnosis of dysplasia (indefinite – 2, low grade dysplasia, LGD – 4, intramucosal adenocarcinoma, 1) (Table 1).

**Conclusions** The Cytosponge® has proved to be an acceptable non-endoscopic tool for patients with BE under surveillance where endoscopy is not possible. Preliminary data are promising to detect dysplasia and triage patients to endoscopy early. Further large scale, longitudinal follow-up is needed.

Introduction

Endoscopic surveillance for Barrett’s Oesophagus (BE) has been indefinitely postponed due to the COVID-19 pandemic (Rees Clin Med 2020). As well as the potential for missed progression to dysplasia, the negative impact on patients’ quality of life is immeasurable.

The Cytosponge® is a minimally invasive cell sampling device which to date has been researched in screening for BE (Ross-Innes Lan Gastr Hep 2017). We describe the first worldwide use of the Cytosponge® outside a clinical trial to triage BE surveillance patients unable to undergo endoscopy due to COVID-19.

**Methods** Consecutive patients with non-dysplastic BE (NDBE) who were overdue endoscopy were invited to have the Cyto-sponge®. The sample was analysed for TFF3 (a marker of intestinal metaplasia), cellular atypia and p53. It was carried using minimal personal protective equipment (PPE) as it was not deemed to be an aerosol generating procedure (AGP).

**Results** 60 patients have undergone the Cytosponge® procedure to date. Most patients (58, 97%) swallowed the device and in 4 was a result not possible (2 - unable to swallow the Cytosponge®, 2 - sample not analysable/sponge did not enter stomach). No complications were encountered.

**Abstracts**
in BE (high grade dysplasia (HGD)/intramucosal adenocarcinoma) and patients with non-dysplastic BE (NDBE). Endoscopic resection margins/targeted biopsy site histology served as the ground truth for dysplasia in videos. Videos were annotated for definite visual presence of dysplasia. We trained a convolutional neural network with a Resnet101 architecture to classify video frames into dysplastic or non-dysplastic using randomly selected frames from annotated videos.

**Results**

58 patients each with high quality video frames of magnification areas of BE (34 dysplasia, 24 NDBE) were included. Performance was evaluated using a 15-fold cross validation methodology. 76,496 (47,438 dysplasia, 29,058 NDBE) magnification video frames were analysed by the neural network. All dysplastic and non-dysplastic frames were included.

We used an exponentially weighted moving average of consecutive frames to make a diagnosis of dysplasia. The network achieved a per frame sensitivity of 82%, specificity of 82%, and Area under the ROC of 90%. The mean assessment speed per frame was 0.0135 seconds (standard deviation, + 0.006) (Figure 1).

**Conclusion**

The neural network can characterise BE dysplasia with high accuracy and speed on magnification endoscopic images. Whole video frames were used to train and test the data moving it towards real time automated diagnosis. This will potentially aid endoscopists to make key decisions regarding endoscopic sampling and resection in BE during the same endoscopic session.

**HFR-3**

**PATIENT REPORTED OUTCOMES ON SYMPTOM IMPROVEMENT- A GOOD PREDICTOR OF HISTOLOGICAL REMISSION IN EOSINOPHILIC ESOPHAGITIS?**

1Pavit Luthra*, 2Konstantinos Giaslakiotis, 3Sami Hoque. 1Department of Gastroenterology, Barts Health NHS Trust, London, UK; 2Department of Pathology, Barts Health NHS Trust, London, UK

**Introduction**

Eosinophilic esophagitis (EoE) is a chronic immune-mediated inflammatory condition of the oesophagus. Randomized control trials frequently use histological remission as a definition of treatment effect, although in clinical practice there is often de-escalation of therapy once there is patient reported improvement in symptoms. However, persistent mucosal inflammation still risks tissue re-modelling and may lead to fibrosis and stricture formation. We aim to assess if patient reported outcomes (PRO) of symptom improvement were a good predictor of histological remission.

**Methods**

We performed a retrospective observational analysis and identified 30 patients with EoE at Barts Health NHS trust between 2016-2020. EoE was defined as symptoms of dysphagia and/or food bolus obstruction with eosinophil count ≥15 per high power field in proximal and distal oesophageal biopsies. All patients had subsequent follow up with clinical, endoscopic and histologic re-assessment after a minimum eight week period of treatment. Dichotomous outcomes were compared between patient reported global improvement in symptoms (improvement or no improvement) and histological remission (eosinophil count <15 eos/hpf).

**Results**

19/30 (63.33%) patients were male. The median age was 35.9 years (age range 23-56). 18/30 (60%) patients had atopic disorders whilst 8/30 (26.67%) had IgE mediated food allergies. 22/30 (73.33%) received PPI as first line therapy, 6/30 (20%) received topical steroids and 2/30 (6.67%) had combined therapy with PPI and inhaled fluticasone. All patients had a minimum of 8 weeks treatment prior to reassessment. The mean duration of therapy was 38 weeks (+/- 24.28). There was no association between PRO of global symptom improvement and histological remission across all treatment groups (p=0.55). There was a mean increase of 10.33 eos/hpf on repeat biopsies. When specifically assessing outcomes following PPI therapy; 18/22 (81.8%) reported improvement in symptoms, however only 4/22 (18.2%) achieved histological remission (p=0.55).

**Conclusions**

Microscopic disease activity can persist despite patient reported symptom improvement. Hence histology is the ideal marker of inflammation for disease activity. Our findings support European guidelines reinforcing that targeting mucosal healing should be the goal of therapy. Treatment should not de-escalated if there is ongoing mucosal inflammation. Standardised definitions of histologic response and remission using validated scoring systems, as well as a biopsy procurement protocol are needed to further guide clinical practice and decision making.