at a cost of £169. Mean (SE) costs from hospital discharge to 28 days were £293 (£22) per patient. The main cost driver post discharge was readmission to hospital; 12% of patients were readmitted within 28 days for a mean of 4.8 days. The mean cost associated with readmission across all patients was £127. HRQoL was on average (SE) 0.68 (0.01) at 28 days.

Conclusion The mean cost up to 28 days for patients presenting with AUGIB is £2,207. At 28 days, the mean HRQoL in patients who have experienced an AUGIB is well below the average population level of 0.86. This is the first study to provide detailed estimates of the costs and HRQoL associated with AUGIB in the UK. These data can be used by healthcare providers and researchers to inform the design of subsequent cost-effectiveness analyses of interventions for AUGIB.

Disclosure of Interest None Declared.

PTU-149 MALNUTRITION AND GASTROINTESTINAL (GI) SYMPTOMS IN PATIENTS WITH UPPER-GI CANCER

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Introduction Persistent GI symptoms and malnutrition have been associated with poorer quality of life in upper-GI cancer patients. This study aims to assess GI symptoms and nutritional status in patients undergoing modern treatment.

Methods Patients with newly diagnosed upper-GI cancer were prospectively reviewed at the time of diagnosis and at 3- and 12-months following radical treatment. Nutritional assessment was performed using the patient-generated subjective global assessment (PG-SGA), which is considered the 'gold-standard' for nutritional assessment and has been validated in the oncology setting (score ≥ 4 intervention needed; score ≥ 9 critical intervention needed). The gastrointestinal symptom rating scale (GSRS) was used to evaluate the presence/absence and severity of 22 GI symptoms using a 4-point response scale. Total scores range from 0–66, where 0 = all symptoms absent and 66 = all symptoms severe.

Results 61 males and 19 females, median age 66 (range 46–89) years were recruited (61% oesophageal, 33% gastric, 6% gastro-oesophageal junction tumours). Of these, 68 were reviewed at 3-months and 25 at 12-months. Mean (SD) body weight and body mass index (BMI) were 76.7 kg (17.4) and 26.7 kg/m^2 (4.7) at baseline, 74.4 kg (14.8) and 25.9 kg/m^2 (4.4) at 3-months and 72.1 kg (16.3) and 24.7 kg/m^2 (4.4) at 12-months. There was a significant mean difference in weight (-2.0 kg, p = 0.002) and BMI (-0.56 kg/m^2, p = 0.006) at 3-months compared to baseline. These reduced further by 12-months. Mean (SD) PG-SGA score at baseline 9.0 (6.3), 3-months 7.8 (5.6), and 12-months 7.4 (5.0) indicated that intervention was required. At baseline, 3- and 12-months 61%, 52% and 68% of patients respectively were considered moderately or severely malnourished. Mean (SD) total GSRS scores were 14.2 (10.8), 12.0 (9.4) and 15.5 (11.5) at baseline, 3- and 12-months respectively. The symptoms with the greatest increase in prevalence (% more patients) from baseline to 3-months (n = 68) were nausea (+24%), loose stool (+16%), urgency (+6%), flatulence (+6%) and early satiety (+6%). Those with the greatest decrease in prevalence (% less patients) during this time were difficulty swallowing (-24%), painful swallowing (-24%), regurgitation (-21%), belching (-15%) and acid reflux (-12%). Of the n = 25 followed up at 12-months, the most common symptoms reported were flatulence (76%), belching (72%), abdominal pain (68%), abdominal grumbling (56%) and early satiety (52%).

Conclusion After treatment commences there is progressive weight loss over time. Troublesome GI symptoms persist at 12-months and may be contributing to this weight loss. Optimising nutritional status and controlling GI symptoms is required throughout the treatment pathway.

Disclosure of Interest None Declared.

PTU-150 EO: ARE WE GETTING THE MESSAGE YET?

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Introduction Eosinophilic oesophagitis (EO) is the underlying diagnosis in at least 10% of those with dysphagia. To make the diagnosis, oesophageal biopsies showing an eosinophil count >15 per hpf are required. It is most frequent in males under aged 50 years.

Methods All patients having a gastroscopy for dysphagia were identified retrospectively for 6 consecutive years from our endoscopy reporting system. Patient demographics, endoscopic findings and whether biopsies were taken were recorded together with histology results.

Results 3068 patients had a gastroscopy with an indication of dysphagia (1489 female, age 15–100 years, average 67.7 y). The number of patients varied little between years (486–550 patients/year). Common endoscopic diagnoses were normal (20.4%), benign stricture (12.6%), oesophagitis (18.1%), Barrett's (4.8%), dysmotility (3.7%) and hiatus hernia (10%). 1620 (52.8%) had oesophageal biopsies.

44 patients (1.5% of all patients) were diagnosed with EO, 32 of who were males. This equates to 2.8% of those who were biopsied and 4.7% of those biopsied without cancer, stricture or Barrett's. Although only 13.3% of those with dysphagia were aged 50 years or under, they equated to 45.4% of those diagnosed with EO. Of those with EO, 6 had food bolus, 6 "typical" EO changes e.g., feline oesophagus, ridges etc, 4 an irritable oesophagus and 3 Schatzki rings.

Conclusion EO is a relatively common cause of dysphagia but is almost certainly under-recognised due to lack of oesophageal biopsies at endoscopy. Reliance on endoscopic changes of EO at endoscopy will miss the majority of cases. Although biopsying only those under 50 years would be more cost effective than biopsying all, it would also miss the majority of cases. It may be appropriate for the BSG to use frequency of oesophageal biopsies in dysphagic patients as a quality assurance measure for upper GI endoscopy.

Disclosure of Interest None Declared.

PTU-151 PREDICTORS FOR COELIAC DISEASE IN CASES OF LYMPHOCYTIC DUODENOSIS

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Introduction Lymphocytic duodenosis (LD) is an early marker for coeliac disease (CD). However, the majority of cases are due to non-CD related conditions.

Aims To identify the predictors of CD when presented with LD.
Methods 215 LD patients had undergone prospective and systematic evaluation for CD and other recognised associations.

The gold-standard diagnosis of CD was based upon the presence of HLA-DQ2 and/or DQ8, persistence or progression of LD following a gluten challenge, symptomatic improvement on a gluten-free diet, and no alternate cause found.

Binary logistic regression models, adjusting for age and gender, were subsequently performed to compare presenting variables between CD and non-CD cases, and to determine their sensitivity, specificity, positive and negative predictive values (PPV and NPV).
Results CD was diagnosed in 47 cases (22%) and non-CD in 168 cases (78%). There was no statistical difference in demographics, clinical symptoms (i.e. diarrhoea, weight loss, abdominal pain), anaemia or haematinics between the CD and non-CD group.

Patients with CD, in comparison to non-CD, were significantly more likely to have a positive family history of CD (21.3% vs. 3.6%, OR 6.81; PPV 62.5%, NPV 81.4%, specificity 96.4%), positive HLA-DQ status (100% vs. 49.4%; PPV 36.2%, NPV 100%, specificity 50.6%), and presence of endomysial antibody [EMA] (49% vs. 0.6%, OR 159; PPV 96%, NPV 87%, specificity 99.4%); all $p \leq 0.001$.

A normal tissue transglutaminase antibody (TTG) level was seen in 29.8% CD and 82.7% non-CD cases (OR 0.086, $p < 0.001$; PPV 9.1%). There was no difference in the prevalence of TTG levels 1–2 x upper limit of normal (ULN) between the groups (29.8% CD vs. 14.3% non-CD; PPV 38%). However, TTG levels between 3–20 x ULN were significantly more prevalent in the CD group (31.9% vs. 3%; PPV 66.6% >87.5%), whilst a TTG > 20 x ULN was exclusive to CD (8.5%, $p < 0.001$, PPV 100%).
Conclusion At the outset, only the presence of positive EMA or TTG > 20 x ULN are highly predictive and specific for CD. However, as they have limited sensitivities, most patients with LD require further work-up prior to diagnostic confirmation.
Disclosure of Interest None Declared.

PTU-152 PEPSIN IN SALIVA FOR THE DIAGNOSIS OF GASTRO-ESOPHAGEAL REFLUX DISEASE

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Introduction Current diagnostic tests for GORD have moderate sensitivity/specificity and can be invasive and expensive. Pepsin detection in saliva has been proposed as an "office-based" method for GORD diagnosis. The aims of this study were to establish normal values of salivary pepsin in a large cohort of healthy asymptomatic subjects and to determine its value to discriminate patients with reflux-related symptoms (GORD, hypersensitive oesophagus) from functional heartburn.
Methods 100 asymptomatic controls and 111 patients with heartburn underwent MII-pH monitoring and simultaneous salivary pepsin determination on waking, after lunch and dinner. Cut off value for pepsin positivity was 16 ng/ml. Patients were divided into GORD (increased acid exposure time (AET) n = 58); Hypersensitive Oesophagus (HO) (normal AET and + SAP), n = 26) and Functional Heartburn (FH) (normal AET and – SAP, n = 27). Multiple group comparisons were performed using one-way ANOVA followed by with Tukey's Test for Gaussian distributed data and the Kruskall-Wallis Test with Dunns comparison for non-Gaussian data. Receiver Operator Characteristic curves were constructed to determine and compare the sensitivity and specificity of different pepsin cut-off concentrations.
Results 1/3 of asymptomatic subjects had pepsin in saliva at low concentration (0(0–59) ng/ml). Patients with reflux-related symptoms (GORD and HO) had higher prevalence (77–89%) and pepsin concentration than controls (HO, 237(52–311) ng/ml and GORD, 121(29–252) ng/ml) ($p < 0.05$). Patients with FH had low prevalence (33%) and concentration of pepsin in saliva (0 (0–40) ng/ml). The area under the receiver operating characteristic curve had a value of 0.8034 +/-0.04 (95% confidence interval 0.719 to 0.8873, $p < 0.0001$). A positive test had 77.6% sensitivity and 63.2% specificity for diagnosis of GORD/HO. When all saliva samples were negative, there was 80% probability that symptoms were not due to reflux (FH). One positive sample with >210 ng/ml pepsin suggested the presence of GORD/HO with 95% probability.
Conclusion In patients with symptoms suggestive of GORD, salivary pepsin can be used to confirm or reject the diagnosis before empirical PPI treatment. This may lessen the use of unnecessary anti-reflux therapy and the need for further invasive and expensive diagnostic methods.
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PTU-153 THE TIMING OF ONCOGENIC EVENTS IN THE EVOLUTION OF THE OESOPHAGEAL ADENOCARCINOMA GENOME AND IMPLICATIONS FOR CLINICAL DIAGNOSTICS

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Introduction A series of clonal expansions are thought to underlie the progression of Barrett's oesophagus (BE) to oesophageal adenocarcinoma (OAC). Each expansion carries with it somatic driver mutation (s) fixing it within a larger population and therefore increasing the likelihood of acquiring a second mutation. However, the precise order in which somatic variants occur remains unknown.
Methods We performed whole genome sequencing in 25 cases of OAC and 3 matched cases of BE. Findings were validated in a larger cohort of OACs (n = 90), metaplastic never-dysplastic BE (NDBE, n = 66 with a median follow-up of 58 months) and high-grade dysplasia (n = 43) using amplicon resequencing. Mutational signatures and gene-centric somatic mutations were determined using an in-house pipeline incorporating standard statistical methods and the publically available EMu pipeline.
Results There were 7 distinct mutational signatures present in both early (BE) and late disease (OAC). Fifteen genes were determined to be potential novel drivers of OAC development.