When reviewing all 2WW referrals for gastroscopy the cancers pick up was 10% with the majority of examinations being normal or identifying insignificant findings.

Conclusion The two week wait referral system is often considered to be a poor method for detecting oesophagogastric cancer. In our data 10% patients referred in this manner had oesophagogastric cancer which is consistant with existing data. However when looking at all cases of of cancer diagnosed in this time period the 2WW represents the pathway for diagnosis for over half our malignancies (56%). Our cohort of patients showed similar TNM staging at the time of diagnosis irrespective whether they were refered routinely or on an urgent basis.

This suggests that the 2ww is an important pathway for referral of upper gastrointestinal malignancies but unfortunately does not identify patients at earlier stage. This is probably due to the lack of symptoms in early oesophogogastric cancer and strengthens the argument for identifying patients at an earlier stage perhaps by screening or surveillance of high risk groups.

Disclosure of Interest None Declared

PTU-170 OESOPHAGEAL PERFORATION RESULTING FROM BAND ACHALASIA – A DELAYED COMPLICATION OF LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING

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Introduction Laparoscopic adjustable gastric banding (LAGB) is a common bariatric procedure in the UK due to its relative technical ease and reversibility. The technique has been around since the 1990s and although its immediate complications have been evident and known, the longer term complications are still emerging and not yet completely understood. Oesophageal dysmotility post-LAGB is now increasingly being recognised as a long-term complication associated with LAGB. This paper presents a potentially life-threatening complication associated with oesophageal dysmotility more than a decade after LAGB placement.

Methods A 58yr old lady presented with chronic cough and mediastinal widening on chest X-ray. A computed tomogram (CT) revealed a mega-oesophagus with a collection in the mediastinum in keeping with a contained oesophageal perforation and a LAGB in situ. On further questioning, she mentioned that she had had a LAGB placed 12yrs previously.. She had been experiencing recurrent coughs, chest infections, weight loss and dysphagia for 2 yrs but had not sought medical help.

Results The LAGB was completely emptied (9mls of fluid). She was managed conservatively with nil orally, nasogastric drainage, antibiotics, parenteral nutrition over a period of 4 weeks and serial imaging was performed to monitor progress. She responded well to it, the perforation had completely healed, she resumed oral intake and was discharged.

Conclusion While oesophageal dysmotility is emerging as a long-term complication occurring around 5–7 yrs post-LAGB, its association with oesophageal perforation has not been described in the literature prior to this incident. It is likely that oesophageal dysmotility resulted in mega-oesophagus and the associated reflux caused frequent coughing in our patient. The valsalva manoeuvre during coughing which closes the cricipharyngeus proximally and the presence of LAGB distally may have generated a high pressure zone within the oesophagus leading to perforation. This was a potentially life-threatening complication. This re-inforces the importance of life-long commitment to follow-up in patients who undergo bariatric surgery. We suggest at-risk patients developing mega-oesophagus should be identified and timely band –emptying performed to avoid this serious complication. Further long-term cohort studies need to be performed to determine the exact prevalence of oesophageal dysmotility and such complications.

Disclosure of Interest None Declared.

PTU-171 BURIED BARRETT'S DYSPLASIA ('BBAD') STUDY: RESULTS FROM A LONG TERM FOLLOW UP STUDY OF BARRETT'S NEOPLASIA COHORT

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Introduction Buried Barrett's' or Subsquamous intestinal metaplasia (SSIM) refers to glands which are 'buried' underneath the squamous epithelium. High dose acid suppressive therapy and lack of acid exposure can result in squamous re-epithelialisation over the Barrett's mucosa. Buried Barrett's can pose significant diagnostic and surveillence challenges. Data on the prevalance of Buried Barrett's in endoscopic therapy naïve patients is limited. Like wise there is limted data on the prevalance of Buried Barrett's in patients following EMR. We aim to study and compare the prevalence of Buried Barrett's in these two groups of patients.

Methods

Inclusion Criteria:

- Patients with Barrett's referred for endoscopic treatment between June 06 and June 12
- Patients with Barrett's dysplasia following EMR procedure.

Biopsy:

- Biopsies were first obtained from any suspicious looking area. Following this, biopsies were then obtained from the neosquamous area. Finally, random biopsies were obtained. These were sent in separate cassettes. Histopathology was reported by two independent GI pathologists and was prospectively recorded on a central pathology database.
- Buried Barrett's was defined as any metaplastic or glandular tissue beneath the squamous epithelium. Pathology specimens were reported by 2 independent, accredited GI pathologists.

Results

Abstract PTU-171 Table 1 Buried Barrett's with and without dysplasia

	Buried Barrett's in endoscopic therapy naïve patients	Buried Barrett's in patients post EMR procedure
Total	16/83 (19%)	22/83 (26.5%)
Buried Barrett's with no dysplasia	2/83 (2.4%)	9/83 (10.8%)
Buried Barrett's dysplasia	14/83 (16.8%)	13/83 (15.6%)
HGD	9/83 (10.8%)	4/83 (4.8%)
IMC	3/83 (3.6%)	4/83 (4.8%)
HGD + IMC	12/83 (14.5%)	8/83 (9.6%)
LGD	2/83 (1.2%)	5/83 (6%)

Conclusion

Our study shows that in the pre-EMR cohort, there was an overall prevalence of 15.7% of buried Barrett's and a 14.5% prevalence of buried Barrett's with high grade neoplasia (HGD or IMC).

Our results in the post EMR cohort shows an overall prevalence of 33.7% of buried Barrett's with 9.6% prevalence of buried high grade neoplasia (HGD or IMC) suggesting that a third of patients undergoing EMR for Barrett's dysplasia harbour buried Barrett's and a third of these patients harbour high grade neoplasia. This has significant implications for post EMR endoscopic assessment and surveillance.

The results from our study shows that there is a need to develop and maintain proficiency in sampling techniques in patients with Barrett's oesophagus. It also shows that the biopsies particularly from those with dysplasia should be carefully reviewed by

gastrointestinal pathologists who devote specific attention to identifying buried Barrett's and buried dysplasia.

Disclosure of Interest None Declared

PTU-172 C-MYC AS A BIOMARKER IN BARRETT'S OESOPHAGUS

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Introduction Identifying Barrett's oesophagus (BE) patients at risk of progressing to oesophageal adenocarcinoma (EA) remains a challenge. Diagnosis of low grade dysplasia is limited by considerable intra and inter-observer variability and inflammation in biopsy samples can lead to a diagnosis of indefinite for dysplasia. Immunostaining using molecular biomarkers would therefore be useful as a diagnostic adjunct in the assessment of dysplasia.

The Aim of this study was to identify and validate a molecular biomarker that can objectively determine dysplasia status and thereby determine cancer risk in BE.

Methods Biomarkers of interest were identified through mining of a microarray gene expression dataset from 59 oesophageal samples with strict consensus diagnosis by expert pathologists [21 BE with no dysplasia (NDBE), 10 BE with low grade dysplasia (LGD), 13 BE with high grade dysplasia (HGD) and 8 EA]. Subsequent validation was performed on a BE tissue microarray (TMA) (60 NDBE, 19 LGD and 29 HGD), and EA TMA (n = 278).

Results Seventy eight genes were differentially expressed between NDBE and HGD. c-MYC was selected as a top target as the expression levels progressively increased with dysplasia stage and cancer with a fold change of 2.46 between NDBE and HGD. mRNA expression levels were significantly higher in HGD and EA compared to NDBE ($p < 0.0001$). Immunostaining for c-MYC was positive in 25 out of 29 cases of HGD with a sensitivity 86% of and a specificity of 97% (Table 1). 10 out of 19 LGD stained positive for MYC. 254 out of 278 (91%) EA cases showed significant c-MYC protein expression. Among the 19 LGD samples, 9 had HGD in the BE segment and 7 of these were positive for c-MYC. Strong c-MYC staining was also observed in areas regarded as indefinite for dysplasia which were adjacent to areas of HGD.

Abstract PTU-172 Table 1

Sample (n)	c-MYC Protein expression (%)
NDBE (62)	2 (3.2%)
LGD (19)	10 (53%)
HGD (29)	25(86%)

Table 1. Immunohistochemistry for c-MYC in oesophageal samples. NDBE: Non-dysplastic Barrett's oesophagus, LGD: Low-grade dysplasia, HGD: High grade dysplasia.

Conclusion Immunohistochemistry for c-MYC is a promising molecular biomarker in Barrett's oesophagus. It could also enable better risk stratification in patients with samples in the LGD and indefinite for dysplasia categories.

Disclosure of Interest None Declared.

PTU-173 GORD SYMPTOMS AND DEMOGRAPHIC FACTORS AS A PRE-SCREENING TOOL FOR BARRETT'S OESOPHAGUS

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Introduction Barrett's oesophagus (BO) occurs as consequence of reflux and is a risk factor for oesophageal adenocarcinoma (OAC). The current "gold-standard" for diagnosing BO is endoscopy which remains prohibitively expensive and impractical as a population screening tool. Therefore, we aimed to investigate the epidemiological factors and symptoms associated with BO in order to develop a pre-screening tool to aid decision making for diagnostic referrals.

Methods A prospective (training) cohort of 1603 patients attending for gastroscopy was used for identification of risk factors to develop a risk prediction model. Factors significantly associated with BO in the univariate analysis were selected to develop a prediction model that was validated in an independent, external cohort of 504 patients in primary care. We used two definitions of BO in the current study: 1) columnar lined epithelium of oesophagus (CLE) of any length reported in the endoscopy report with columnar epithelium on biopsy 2) an intestinal metaplasia confirmed on histopathological assessment with endoscopic length of BO ≥2 cm (IM ≥ 2 cm).

Results An eight-factor panel, including age, sex, smoking status, heartburn, acid reflux, chest pain, abdominal pain, anti-reflux medication, was identified from the training cohort with an area under the ROC curve (AUC) of 0.74 (95%CI: 0.70–0.77) for CLE, and 0.80 (95% CI: 0.75–0.84) for IM ≥ 2cm. This panel was significantly associated with BO in the external cohort, and the odds ratios for each factor increase were 1.43 (95%CI: 1.02–2.01) and 1.30 (95%CI: 1.04–1.62) for CLE and IM ≥ 2cm, respectively. The AUCs of the 8-factor panel for CLE and IM ≥ 2cm in the external cohorts were 0.85 (95%CI: 0.77–0.92) and 0.78 (95%CI: 0.71–0.84), respectively. After controlling for over-fitting, the AUCs dropped to 0.65 (95% CI: 0.54–0.77) and 0.67 (95%CI: 0.60–0.74), respectively.

Conclusion An eight-factor panel can help predict the presence of BO and has the potential to be applied in the general population with GORD symptoms as a pre-screening tool to help select patients for further investigation.

Disclosure of Interest None Declared.

Pancreas

PTU-174 LOW FAECAL ELASTASE IS NOT ALWAYS DUE TO PANCREATIC INSUFFICIENCY

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Introduction Faecal elastase-1(FE-1) is a widely available, simple, cheap indirect pancreatic function test used in patients in whom pancreatic exocrine insufficiency (PEI) is suspected. Sensitivity is 73–100% for moderate to severe, but 0–63% for mild PEI [1]. Specificity of the test (80–100%)[1] is compromised in patients who have other causes of diarrhoea or type 1 diabetes[2,3]. The aim of this observational study was to examine the causes of low faecal elastase in our hospital.

Methods A retrospective analysis of FE-1 tests performed between April 2010 and April 2012 was undertaken. The electronic medical notes of each patient with a low FE-1 (< 300 ug/l) were examined.

Results Of the 288 samples received by the laboratory, 23 patients had FE-1 of less than 100 ug/l (severe). 18 patients had FE-1 of 100–199 ug/l (moderate) and 19 patients had FE-1 of 200–299 ug/l (mild). The results were grouped as shown in Table 1. In 5 of the moderate PEI group there was insufficient information to determine the diagnosis. Non-pancreatic causes included microscopic colitis, coeliac disease, bile salt malabsorption, irritable bowel syndrome and infection.