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

doi:10.1136/gutjnl-2013-304907.262

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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

doi:10.1136/gutjnl-2013-304907.263

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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

doi:10.1136/gutjnl-2013-304907.264

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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.