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 ≥ 2 cm. 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 ≥ 2 cm, respectively. The AUCs of the 8-factor panel for CLE and IM ≥ 2 cm 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 PEI¹. 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.

Abstract PTU-174 Table 1

	Severe, No. (%)	Moderate, No. (%)	Mild, No. (%)
High clinical likelihood supported by imaging	12 (52.2)	3 (16.7)	3 (15.8)
Clinically pancreatic cause no imaging/no evidence on imaging	7 (30.4)	4 (22.2)	2 (10.5)
Other diagnosis	4 (17.4)	6 (61.1)	14 (73.7)

In 2 patients the FE-1 was repeated; in 1 when treatment failed and FE-1 was still <100 ug/l (due to bile salt malabsorption), the other following treatment for coeliac disease and microscopic colitis (FE-1 147ug/l then normal). There was no difference in symptoms (steatorrhoea, diarrhoea, weight loss, abdominal pain) between the groups.

Conclusion This study shows that clinicians need to be aware that even in patients with FE-1 less than 100ug/l, the cause may be non-pancreatic in origin. FE-1 becomes a less reliable diagnostic tool in moderate to mild PEI parameters. FE-1 should be repeated if symptoms do not improve with pancreatic enzyme replacement. Symptoms may not be helpful in distinguishing pancreatic from non-pancreatic causes of low FE-1.

Disclosure of Interest None Declared

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PTU-175 QUADRUPLE, CLINICAL, RADIOLOGICAL, CYTOLOGICAL AND BIOCHEMICAL ANALYSIS OF PANCREATIC CYSTIC LESIONS ARE NECESSARY PRIOR TO THERAPEUTIC PLANNING

doi:10.1136/gutjnl-2013-304907.265

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Introduction Back ground Distinguishing benign from malignant or pre-malignant pancreatic cystic lesions is essential when formulating the surgical therapeutic strategy. Lack of a well-defined pre-operative predictability criteria makes therapeutic planning challenging.

Aims To study the correlation between pre-operative morphological and biochemical features of resected pancreatic cystic lesions and predictive power of these features in relation to biological behaviour and final histology.

Methods We systematically reviewed the literature to identify the relevant variables that are used to predict the nature of pancreatic cystic lesions and aid therapeutic planning; this was followed by designing a template encompassing all these variables to collate data of resected pancreatic cystic lesions from two centres. We collated clinico-pathological and biochemical data, pre-operative CT, MRI, EUS, PET CT, FNA analysis and final post-operative pathology reports.

Results 63 patients with pancreatic cystic lesions were identified; 3 were drained endoscopically out of which two were pseudocyst and 1 was abscess, 12 underwent resection, 3 were serous, 1 mucinous, 1 IPMN, 4 ductal adenocarcinomas, 1 endocrine neoplasm, 1 pseudocyst with abscess, one patient's final histology results was missing. Three patients had neoplastic cells on FNAC, 2 patients had FNAC results suspicious for neoplasm, 26 were reported to have benign

findings at EUS and FNAC and managed conservatively. 10 had elevated intra-cystic CEA levels, 3 patients had elevated CA 19-9 levels at FNAC. 1 patient was diagnosed having VHL, 1 patient had lymphatic cyst, 1 patient was diagnosed having Giardiasis, 1 patient was stented for palliation, 7 patients were undergoing further definitive treatment, and 1 patient with IPMN had therapeutic ERCP.

Conclusion Our interim results suggest that quadruple assessment including clinical, radiological

(CT/PET/MRI/EUS), FNAC and biochemical analysis is necessary prior to therapeutic planning.

Disclosure of Interest None Declared.

PTU-176 RETROSPECTIVE AUDIT OF ANTIBIOTIC USE IN PATIENTS ADMITTED TO INTENSIVE CARE UNIT (ICU) WITH SEVERE ACUTE PANCREATITIS

doi:10.1136/gutjnl-2013-304907.266

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Introduction The Atlanta classification divides patients with acute pancreatitis (AP) into two groups, mild and severe. The severe form (severe acute pancreatitis, SAP) is best managed in HDU or ICU setting and is associated with high morbidity and mortality despite best efforts at early diagnosis and timely intervention. The use of antibiotics in SAP is controversial. The aim of the audit is to compare antibiotic use and mortality of patients admitted to the ICU against national standards.

Methods Retrospective audit of management and outcome of consecutive patients admitted to ICU with SAP during the period of 2007–2010. The timing of antibiotic use, the agent(s) used and the site of infection were compared.

Results Of 51 patients, 42 received antibiotics. 63% (n = 32) of patients were started antibiotics within 48 hrs of hospital admission. The first choice antibiotics were cefuroxime and metronidazole in 45% of cases, co-amoxiclav and metronidazole in 12% of cases and tazocin ± metronidazole in 14% of cases. In 26% of cases a combination of imipenem ± fluconazole ± gentamicin ± vancomycin was used.

Three patients were confirmed to have pancreatic infections based on positive culture and 10 patients had extra-pancreatic infections. Four patients had bacteraemia, 4 had chest infections, 2 had UTI and one had *Clostridium difficile*. All extra-pancreatic infections had Gram negative bacilli as a causative organism.

Antibiotic use did not improve survival, nor was there any observed survival benefit when the different antibiotic agents were compared (p = 0.7 and 0.4 respectively). The timing of antibiotic use also does not appear to confer a survival benefit (p = 0.5). All patients with proven pancreatic infection died, there was not a significant difference in survival in those with extra-pancreatic infections (p = 0.2).

Conclusion Current guidelines recommend the use of antibiotics only in the presence of proven infection. Given that a majority of patients were commenced on antibiotics within 48hrs of admission and that only a minority were shown to have proven infection it would seem that adherence to this guideline has been poor. It has been suggested that early antibiotic use without proven infection may lead to increased antimicrobial resistance. This audit has demonstrated that early antibiotic use does not benefit survival in severe acute pancreatitis. The choice of agent is important as no single combination has been shown to be advantageous. The agent selected should be based upon clinical findings and laboratory evidence as to the organism(s) present and their susceptibilities. Further work could investigate whether antibiotic use might reduce morbidity in SAP. Other therapeutic strategies should be investigated in order to try to improve survival in this patient group.

Disclosure of Interest None Declared