

diagnosis and AFIP scores to be independently associated with worse prognosis.

Conclusion A combination of surgery and imatinib may be necessary to provide a curative treatment for GISTs and prevent recurrence. Although our study is limited by small numbers, current risk-categorisation models appear to over-estimate recurrence risk with discrepancies in predicting behaviour for certain low-risk tumours. A weighted scoring system combining independent factors associated with poor prognosis may serve as a more accurate clinical prediction tool.

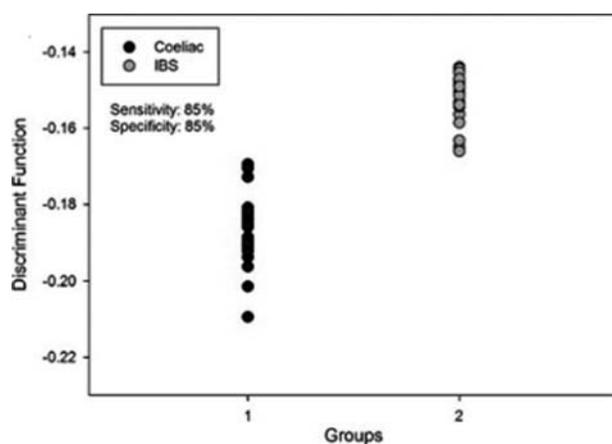
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PTU-163 URINARY VOLATILE ORGANIC COMPOUND ANALYSIS TO DISTINGUISH COELIAC DISEASE FROM IRRITABLE BOWEL SYNDROME: A PILOT STUDY

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Introduction Coeliac disease (CD), a T-cell-mediated gluten sensitive enteropathy, affects ~1% of the UK population, and in adults presents with a wide range of clinical features; often mistaken for irritable bowel syndrome (IBS). Heightened clinical awareness and serological screening identifies those likely to have CD; the diagnosis confirmed by histological features in small bowel/duodenal biopsies. Limitations to diagnosis are false negative serology (e.g., in IgA deficient patients, the young and the elderly) and reluctance to undergo biopsy. Examining the pattern of urinary volatiles offers a novel non-invasive approach. The gut microbiome is perturbed in several gastrointestinal disorders, resulting in altered gut fermentation patterns, and recognisable by analysis of volatile organic compounds (VOC) in urine, breath and faeces. The altered structure of the small intestinal mucosa, increased gut permeability and altered gluten peptide metabolism, we hypothesised, would change the



Abstract PTU-163 Figure 1

microbiome creating a unique “fermentome” pattern, distinguishable from IBS. We investigated this by examining the urinary VOC pattern using Field Asymmetric Ion Mobility Spectrometry (FAIMS).

Methods 47 patients were recruited, 27 with CD and 20 with diarrhoea-predominant IBS (D-IBS). Urine was collected and 10ml aliquots were stored frozen in universal containers. For assay, the containers were heated to $40 \pm 0.1^\circ\text{C}$. The headspace above the sample was then analysed by FAIMS. Linear discriminant analysis (LDA) was used for statistical evaluation.

Results LDA showed that FAIMS distinguishes the VOC pattern in CD vs D-IBS with a sensitivity and specificity of 85% respectively.

Conclusion This pilot study suggests that FAIMS offers a novel non-invasive approach to identify those likely to have CD, and distinguishes from D-IBS. It may have the potential to non-invasively track the progress of CD when on a gluten-free diet, to monitor adherence and observe changes.

Disclosure of Interest None Declared.

PTU-164 EVIDENCE OF TWO AETIOLOGIES OF GASTROESOPHAGEAL JUNCTIONAL CANCERS BASED ON GASTRIC PARIETAL CELL DENSITY

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Introduction Serum pepsinogen I:II ratio, a surrogate marker of atrophic gastritis, suggests that some adenocarcinomas at the gastroesophageal junction (GOJ) develop on a background of atrophic gastritis, similar to non-cardia gastric cancer, while others arise on a backgrounds of healthy, non-atrophic gastric mucosa similar to oesophageal adenocarcinoma. In this current study, we have directly the background gastric body mucosa in patients with junctional adenocarcinomas compared to oesophageal adenocarcinomas and non-cardia gastric cancers.

Methods 127 gastrectomy and oesophagectomy specimens for adenocarcinoma were identified for which clear topographic description allowed assignment to oesophageal, junctional (including cardia) and gastric non-cardia locations. In these gastric body mucosa specimens, well clear of the tumour margin, parietal cells were immunostained using anti- H^+/K^+ ATPase. Parietal cell density was counted in 3 to 5 well-oriented fields (1 mm^2 each) and expressed as mean parietal cell number per 1 mm^2 area. Total mucosal thickness, glandular thickness, intestinal metaplasia, inflammation indicated by polymorphonuclear (PMN) and mononuclear (MN) cells and reactive atypia (RA) were also scored. Non-parametric statistics were used to compare distributions.

Results Ten (8%) cases lacked well-orientated blocks of body mucosa. The remaining 117 patients included 34 oesophageal, 52 GOJ and 31 non-cardia gastric adenocarcinomas. Median (IQR) parietal cell densities were 836 (173), 602 (389) and 411 (334) per mm^2 in gastric mucosa of oesophageal, GOJ and gastric cancers, respectively (all differences $P < 0.001$). Using a parietal cell density of $630/\text{mm}^2$, 85% of oesophageal adenocarcinomas had a higher and 84% of non-cardia gastric cancers had a lower values. With the same cut-off, 50% of GOJ adenocarcinomas were gastric and remaining was oesophageal in origin.

Glandular mucosa was thicker in patients with GOJ cancer compared to gastric (0.735 vs. 0.600, $p = 0.005$) and thinner

than oesophageal cancer (0.735 vs. 0.900, $p < 0.001$). Inflammatory scores in GOJ cancers were lower than in gastric cancer and higher than in oesophageal cancer.

Conclusion This study provides direct evidence for marked differences in the gastric mucosal phenotype in the patients with oesophageal versus gastric non-cardia cancer, with the former being healthy and uninflamed, but the latter atrophic and inflamed. The background gastric mucosa of GOJ cancer supported them being two distinct aetiologies, one group resembling oesophageal adenocarcinoma and other gastric non-cardia cancer.

Disclosure of Interest None Declared.

PTU-165 WORLDWIDE EPIDEMIOLOGICAL EVIDENCE SUPPORTS A COMMON FACTOR PREDISPOSING TO NON-CARDIA GASTRIC CANCER AND PROTECTING FROM OESOPHAGEAL ADENOCARCINOMA

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Introduction During last three decades, global incidence of oesophageal adenocarcinoma has increased more rapidly than any other cancer. A concurrent reduction in the incidence of gastric cancer has been reported from some populations. We aimed to examine the geographical pattern of oesophageal adenocarcinoma versus gastric non-cardia cancer across the world where reliable cancer registry data were available.

Methods Data were abstracted from "Cancer Incidence in Five Continents" Volume 10. Oesophageal and gastric cancers were selected based on ICD-10 codes C15 and C16, respectively. Oesophageal adenocarcinomas were identified by ICD-O morphology codes. Datasets reporting >500 cases for total gastric cancer and >100 for total oesophageal cancer were selected. We examined correlation between age-standardised Incidence rates (ASR) of oesophageal adenocarcinoma and non-cardia gastric cancer using Spearman's non-parametric correlation coefficient (CC). We also allocated cardia cancers into oesophageal and non-cardia gastric adenocarcinoma categories based on gender ratio for oesophageal adenocarcinoma and non-cardia gastric cancer in each dataset.

Results Out of 424 datasets from 290 cancer registries, 206 datasets covering 40 countries met the selection criteria. There was a strong inverse correlation between oesophageal adenocarcinoma and gastric non-cardia cancer in males (CC = -0.768, $p < 0.001$) and females (CC = -0.705, $p < 0.001$). After dividing cardia cancer into two subtypes with potentially oesophageal or gastric origin and adding them to original oesophageal adenocarcinoma or gastric non-cardia groups, the inverse correlation remained strong in males (CC = -0.660, $p < 0.001$) and females (CC = -0.536, $p < 0.001$). Oesophageal adenocarcinoma only showed a rise when incidence of non-cardia gastric cancer fell below 9/100,000 person-years for males and 4.5/100,000 person-years for females.

Conclusion This cross-sectional study is consistent with a common underlying factor predisposing to non-cardia gastric cancer and protecting from oesophageal adenocarcinoma, such as *H. pylori* atrophic gastritis. If this is the case, then the incidence of non-cardia gastric cancer would need to fall to substantially lower levels than currently seen in Far Eastern populations before any rise in oesophageal adenocarcinoma would be apparent.

Disclosure of Interest None Declared.

PTU-166 DETECTION RATES OF GASTRIC CANCER AT THE QUEEN ELIZABETH HOSPITAL BIRMINGHAM 2009–2013

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Introduction Despite open-access endoscopy, previous series have suggested that between 8–20% of early gastric cancers (GC) are potentially missed at prior endoscopy.^{1,2} Although upper gastrointestinal alarm symptoms are more frequently associated with malignancy, this may represent advanced cancer with poorer survival rates, as patients with early GCs may be asymptomatic. The false-negative rate for the diagnosis of GC may also be a measure of quality for endoscopy services. This is based on a reported median duration of 37 months between endoscopic diagnosis of early GC and progression to advanced GC,^{2,3} so we assessed all oesophagogastroduodenoscopy (OGD) findings to assess detection of GC in a large tertiary hospital in the West Midlands.

Methods Patients with histologically confirmed GC were identified from histopathology and endoscopy records. Patients who had undergone at least one OGD before the diagnosis were studied. Detection of GC within 3 years of a negative OGD was interpreted as a false negative.

Results Between September 2009 and September 2013, 16823 OGDs were performed. GC was diagnosed in 75 (0.45%) patients (male/female ratio 1.78; median age 74; 85% Caucasian). Sixty-seven (89%) of the 75 patients with GC presented with alarm symptoms. 33% (25) were done as inpatients, with 43% (at least 32 of 50 outpatients) being referred as urgent outpatients. Five of the 75 (7%) patients had previous OGDs within three years preceding diagnosis. Only one of these was planned because of a suspicious gastric ulcerative lesion at the same site, with other causes being gastric polyps (2); normal (1) and gastritis (1). There were 53 (71%) deaths in total, 47 (89%) of these patients had alarm symptoms at diagnosis of GC.

Conclusion The absolute rates of GC are low (0.1%/OGD/year) and false-negative rates of 5% (within 3 years) for diagnosis of GC are reassuring with only a minority of preceding OGDs in this series demonstrating suspicious lesions. Whilst GC presents with alarm symptoms in the vast majority, the prognosis remains very poor, so continued quality measures in endoscopy will be required to ensure that early gastric cancers are not missed.

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Disclosure of Interest None Declared.

PTU-167 BARRETT'S OESOPHAGUS SURVEILLANCE STUDY (BOSS) UPDATE: SUCCESSFUL RECRUITMENT TO A LONG FOLLOW-UP RCT

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