

# Abstract PTU-140 Table The effects of PPI on oesophageal acid reflux and symptoms (Off PPI/On PPI)

Fraction time pH < 4(%):	<b>Total</b> 10.45(7.2–15.5) /1.9 (0.6–4.9) ***	<b>Upright</b> 9.4(6.1–15.6) /2.5(0.9–5.3)***	<b>Supine</b> 9.7(5.0–16.1) /0.1(0–2.2)***
No. of reflux events associated with symptoms:	<b>HB</b> 8(2–15)/1(0–3)***	<b>RG</b> 2(1–8)/1(0–3) (P = 0.0975)	<b>CP</b> 3(2–6)/1(0–3)*

**Conclusion** Sequential 48hrs recording off/on PPI therapy using Bravo pH test is feasible in routine clinical practise. This technique documents the physiological and clinical response to PPI therapy on acid reflux and acid reflux associated symptoms (i.e. heartburn, chest pain). These preliminary findings suggest that this methodology could be of value in distinguishing symptoms related to acid reflux that respond to acid suppression and guiding medical therapy in reflux disease.

**Disclosure of Interest** None Declared

## PTU-141 CAMPYLOBACTER CONCISUS COLONISATION AND ONCOGENIC MARKER EXPRESSION IN BARRETT'S OESOPHAGUS

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**Introduction** *Campylobacter concisus* has recently been implicated in the pathogenesis of a number of gastrointestinal diseases. *In vitro* studies have shown that these bacteria can induce apoptosis in epithelial cells and their presence may induce altered expression of genes. Our group has reported that *C. concisus* was isolated from the Barrett's mucosa in 57% of affected patients but was not identified in normal oesophagogastric mucosa. From this series of cases of known *C. concisus* status we have examined the expression of a range of molecular markers associated with cancer development to correlate their pattern of expression with the presence of *C. concisus*. The aim of this study was to investigate whether *C. concisus* may contribute to malignant progression of BO, the major risk factor for the development of oesophageal adenocarcinoma (OA).

**Methods** Tissue microarrays (TMA) were prepared from samples of BO (n = 92), severe dysplasia (n = 39) and OA (n = 64). Paired biopsies were collected from a separate patient cohort with BO and formalin-fixed or used for Taqman PCR to determine *C. concisus* colonisation status (14 colonised and 13 uncolonised). The TMAs and BO slides were immunohistochemically stained for Ki-67, phospho-p53 Ser 15 and COX-2. Slides were semi-quantitatively scored 0–3 according to staining intensity of COX-2 and Ki-67. For phospho-p53, intensity of staining and percentage of glands stained were scored to derive a multiplicative phospho-p53 score. All slides were scored by a single blinded observer. Median scores were used in statistical testing, with p < 0.05 considered as significant. Results are presented in mean ± SEM unless stated otherwise.

**Results** Patient characteristics were similar between TMA samples [median age 70(24–91), 69.7% males] and the *C. concisus* BO cohort [median age 62(41–84), 66.7% males]. TMA staining patterns of Ki-67, phospho-p53 and COX-2 changed significantly from BO to OA, which is consistent with widely published results in the literature. This validates our method of scoring and staining. *C. concisus* colonised and uncolonised BO did not significantly differ in expression of Ki-67 (1.79 ± 0.21 vs. 1.92 ± 0.24) or phospho-p53 (0.15 ± 0.10 vs. 0.50 ± 0.25). However, *C. concisus* colonised BO (n = 8) was associated with lower expression of COX-2 compared to

uncolonised BO (n = 13), after the exclusion of samples from patients taking NSAIDs from the analysis (1.13 ± 0.13 vs. 1.69 ± 0.17, p = 0.0371).

**Conclusion** *C. concisus* status in BO was not associated with altered expression of phospho-p53 or Ki-67. However, the presence of *C. concisus* was associated with reduced COX-2 expression raising the possibility that the bacteria may induce a phenotype protective against the development of OA.

**Disclosure of Interest** None Declared

## PTU-142 HOW COMMON ARE DELAYS IN REFERRAL OF PATIENTS WITH OESOPHAGEAL CANCER FROM PRIMARY CARE?

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**Introduction** The UK has the highest age-standardised incidence of oesophageal cancer (OC) in Europe with a 5 year survival rate of only 11.6%. We have investigated a large primary care cohort to determine how common delays in referral are and associated factors.

**Methods** All subjects with OC from the Health Improvement Network (THIN) primary care database were studied. THIN includes over 6 million patients and is regionally and demographically representative of the UK.

A nested case-control study was performed with cases of 'delayed referral' defined as subjects who met NICE guidance for urgent referral (August 2004) with alarm upper gastrointestinal (UGI) symptoms (dysphagia, weight loss, abdominal mass, recurrent vomiting, UGI bleed, iron deficiency anaemia) or dyspepsia over 55 years but were not referred within 14 days. Control subjects were referred within 14 days. Logistic regression analysis assessed associations with delayed referral.

**Results** 4210 subjects had OC diagnosed after August 2004. 567 (377 (66.5%) male) had a referral date recorded and were analysed. Mean age 70.7 ± 11.2 years with 1.4 ± 0.8 mean consultations prior to referral. Presenting symptoms of OC: dysphagia 326 (57.5%), dyspepsia 174 (30.7%), anaemia 31 (5.5%), weight loss 23 (4.1%), recurrent vomiting 6 (1.1%), UGI bleed 6 (1.1%) and abdominal mass 1 (0.2%).

355 (62.6%) referred within 14 days with mean lag time of 1.9 ± 3.0 days. 212 (37.4%) had a potential referral delay: 60 (10.6%) 14–29 days; 103 (18.2%) 30 days–1 year; 49(8.6%) > 1 year; with mean lag time of 273.0 ± 433.1 days. Time from presentation to OC diagnosis in the controls was 63.8 ± 155.1 days compared with 3413 ± 469.7 days in the delayed referral group, with 0.8 ± 0.9 extra consultations prior to referral.

There was no difference in age at presentation (70.2 ± 11.0 years (delayed referral), 70.2 ± 11.3 years (controls), P = 0.5) and no association with male gender (1.15 (95%CI 0.8–1.6), p = 0.46) or Townsend index (group 1–2 versus group 4–5, 0.95 (95%CI 0.6–1.4), p = 0.8) for referral delay. Subjects with dysphagia were less likely to experience referral delay (0.15 (95%CI 0.1–0.2), p = < 0.0001) compared with other alarm symptoms. However, delayed referral did not significantly affect oesophagectomy rate (14.6% vs 16.9%, 0.72 (95%CI 0.4–1.2), p = 0.18) or 1 year survival (43.8% vs 44.1%, 0.98 (95%CI 0.7–1.5), p = 0.95).

**Conclusion** A third of patients were not promptly referred to secondary care despite meeting NICE criteria leading to diagnosis delays. 80% of subjects with dysphagia were referred promptly, but only 39% of subjects with other alarm symptoms or new dyspepsia. Surprisingly, delayed referral did not affect surgical resection or 1 year survival rate.

**Disclosure of Interest** None Declared