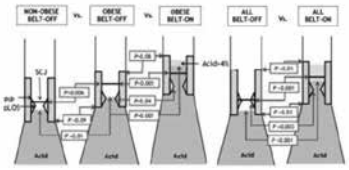

Abstract PTU-138 Figure

Conclusion Our findings indicate that in asymptomatic volunteers, central obesity and waist belt cause partial hiatus herniation and that waist belt also causes short segment reflux.
Disclosure of Interest None Declared

Oesophagus

PTU-139 AN UNUSUAL COMPLICATION OF MYELODYSPLASIA

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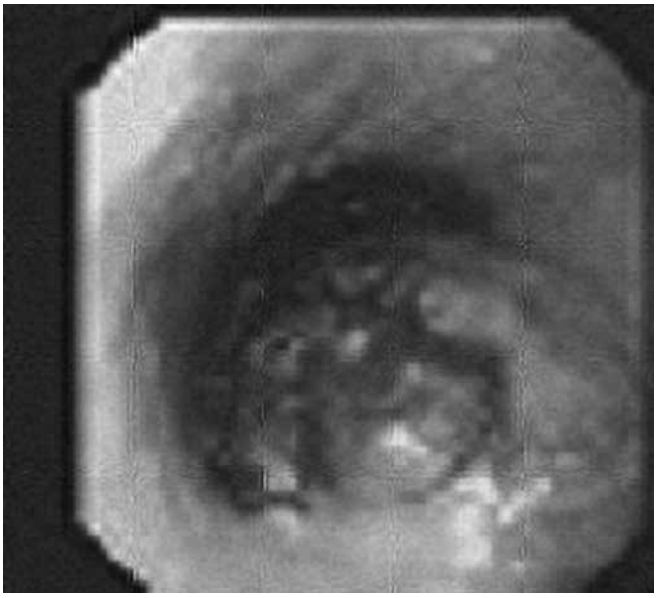
Introduction Oesophageal haematoma is a rare clinical entity which is part of the spectrum of oesophageal injuries that includes Mallory-Weiss tear and Boerhave's syndrome. We present a case of oesophageal haematoma as the only complication of myelodysplasia.
Methods Case presentation An 81 year old gentleman presented to the emergency department with sudden onset chest pain. He ate a scone and went to bed. Half an hour later, he developed a sharp, retrosternal pain,radiating to the neck. The pain was worse by breathing and swallowing. His past medical history included myelodysplasia and ischemic heart disease. Physical examination was unremarkable. Laboratory investigations showed platelets count of 59×10^3, D. Dimer of 2746 ng/ml. CT pulmonary angiography didn't show any pulmonary embolus but obstructed oesophagus,filled with food up to the middle part. Gastroscopy showed large bluish red mass extending between 23 cm and 40 cm in the Oesophageal lumen, originating from the submucosa. The mass didn't look malignant, therefore, biopsies were not taken. Clinical impression was submucosal oesophageal haematoma. Aspirin was stopped and he was treated conservatively, with liquid diet. Dysphagia improved and he was discharged 4 days later. Complete resolution was shown on follow up CT thorax and gastroscopy 6 weeks later.
Results Discussion The first published report of Oesophageal haemtoma,from Marks and Keat in 1968, described a small mucosal breach as a constant feature. It is a result of haemorrhage within the oesophageal wall, usually in the submucosa and more often in the distal oesophagus as it is least supported by adjacent structures. The pathogenesis is unclear, Criblez and colleagues reviewed 91 patients with submucosal haematoma in literature, Precipitating factors were vomiting in 22% of cases, instrumentation in 17%, haemostasis abnormalities in 21% and spontaneous occurrence in 37%.Acute chest pain is a common presentation that should be differentiated from other causes as myocardial infarction. The typical triad of chest pain, hematemesis and dysphagia occurred only in one third of patients.CT typically, shows symmetric or asymmetric oesophageal thickening with concentric or eccentric oesophageal mass with well-defined borders. Gastroscopy is the diagnostic test of choice. Most cases resolve spontaneously within 1–3 weeks with conservative approach. Surgery is only indicated in complicated cases. Angiography is usually considered when endoscopic therapy failed or when surgery is risky.
Conclusion Diagnosis of Oesophageal haematoma can be achieved by interpreting symptoms in conjunction with imaging and endoscopy findings at presentation and follow up. Porgnosis is favourable, as it usually resolves with conservative management.
Disclosure of Interest None Declared



Abstract PTU-139 Figure

PTU-140 THE EFFECT OF PROTON PUMP INHIBITORS (PPIS) ON OESOPHAGEAL ACID REFLUX USING A PROLONGED WIRE-LESS BRAVO PH MONITORING

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Introduction Recently prolonged catheter-free pH monitoring (Bravo®, Given Imaging, Yoqeam, Israel) up to 96 hours has become possible which enables more physiological evaluation of oesophageal acid exposure and its response to therapeutic interventions. This study applied this technology to measure acid reflux, and reflux related symptoms in patients with proven gastro-oesophageal reflux disease on and off high-dose PPIs. The potential utility of this methodology in guiding medical therapy was assessed.
Methods Patients with reflux symptoms were recruited prospectively from Mar.2012 to Oct.2012. PPI was stopped for 7 days prior to the Bravo capsule insertion. The 1st 48hr pH recording was performed off PPI and the 2nd 48hr was on twice daily PPI. The 48hr pH and symptom data for the two periods were compared, including percentages of acid exposure in total, upright and supine periods and symptom–reflux association for which heartburn (HB), chest pain (CP) and regurgitation (RG) were analysed. Data were expressed as median & interquartile. Wilcoxon signed-rank and Mann Whitney tests were used for statistical analysis (*P < 0.05; **P < 0.01; *** < 0.001).
Results Data from prolonged pH monitoring up to 96 hours were available from 89patients of whom 36 (22 males, mean age 50, range 26–76 years old) with complete studies and pathological acid exposure in the 1st 48 hours were studied in detail. Acid reflux measurements on PPI therapy were greatly reduced when compared to those recorded off therapy (Table). 27/36 (75%) patients had normal acid exposure on PPI therapy. The overall number of HB and CP reported in the 2nd 48hr period was reduced by almost two thirds (**13 (4–26) vs.5 (2–14)****); however the number of these symptoms that were actually associated with acid reflux events was almost completely abolished (**4 (2–14) vs.1 (0–3)*****). No effect on volume RG was present.

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

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