Abstract OC-063 Table 1

| | MMSE | Barthel | Anxiety | | | Pain | | |
|--------------------|-------------|------------|-----------|-----------|-----------|-----------|-----------|-----------|
| Assessment | | | Pre | 1h post | 24h post | Pre | 1h post | 24h post |
| Patient n = 49 | 28.1 (6.4) | 17.8 (5.0) | 3.5 (3.2) | 1.7 (2.3) | 2.8 (2.9) | 0.1 (0.3) | 2.0 (2.3) | 3.4 (2.9) |
| Clinician n = 21 | 6.2 (9.6) | 3.2 (4.3) | 0.3 (0.7) | 0.1 (0.2) | 0.2 (0.7) | 0 (0) | 0 (0) | 0.1 (0.7) |
| $Combined \; n=70$ | 21.6 (12.6) | 13.4 (8.3) | 2.5 (3.0) | 1.2 (2.0) | 2 (2.8) | 0 (0.3) | 1.4 (2.1) | 2.5 (2.9) |

Conclusion Pain at 1h post PEG placement was common in selfreporting patients and usually mild. By 24h, 41% reported moderate to severe pain often taking analgesia. Preprocedural anxiety did not predict post procedural pain. Clinician examination of all patients at 1h did correlate with self-reported discomfort or predict self reported pain at 24h. Clinician assessment at 1h and 24h where patients could not self assess failed to identify pain. After PEG placement patients should be offered advice on pain and given access to analgesia. It is likely that pain is not identified in debilitated patients and clinicians need to be more alert to its possible presence.

Disclosure of Interest None Declared

REFERENCE

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Radiology free papers



doi:10.1136/gutjnl-2013-304907.063

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Introduction Only some gastric cancers (GC) are detectable on PET imaging (PET avid). Few studies have assessed the molecular and biological differences between PET avid and non-avid GCs. Thus there may be prognostic differences between the two groups that have not been established.

The primary aim was to identify and measure clinical differences of PET avid and non-avid primary GCs. The secondary aim was to determine whether characteristic molecular differences exist between PET avid and non-avid primary GCs.

Methods Participants selected for this study were individuals (male and female, any age) with a diagnosis of GC that attended the Peter MacCallum Cancer Centre, Victoria, Australia, during the period of 1992–2002 who had agreed to partake in research.

A total of 52 primary GC cases who received an initial PET scan were identified and included in this study.

Results From the 52 cases identified 29 tumours were PET avid and 23 were non-avid.

PET avid tumours were mostly intestinal type, 86%; T stage 3, 46%, with no cases of T4. Avid tended to be N1, 47%. Suggesting they may spread via lymph rather than locally. An AJCC stage of Ib was the most frequent, 35%, but overall more were stage IIIa+b, 49%. Avid tumours were located along the greater and lesser curve of the body in equal proportion: 25% each.

Non-avid tumours were mostly diffuse type, 68%; T stage 3, 41%, with 9% of T4. Non-avid tumours tend to be N0, 46%. Overall, AJCC stage IIIa was the most frequent, 33%, with 9% at stage IV. Non-avid tumours were found more in the antrum, 40%. Signet ring carcinomas were found to be significantly more likely to be PET non-avid, p = 0.017. Non-avid tumours were significantly, p = 0.004, less differentiated than avid.

Progression free survival was significantly less in the avid group, survival of 808 versus 1208 days, p = 0.04. However, the overall

survival showed little difference. From these results it is difficult to determine true survival difference. Larger studies are needed to investigate this further.

The overall genetic profiles of the avid compared to non-avid were not significantly different.

Conclusion Our results suggest that PET avid tumours are diagnosed earlier or that they are less locally invasive, and that non-avid tumours are locally invasive.

PET does appear to provide valuable information regaurding the histological sub-type of the tumour, its likely differentiation, lymph node involvement and metastasis (stage). Information available from PET on the genomics of the tumour is still unclear but there does seem to be some difference in expression levels of genes in avid and non-avid tumours. Further studies with larger numbers are needed. The use of PET for diagnosis, preoperative staging and management planning is still uncertain.

Disclosure of Interest None Declared

OC-065 AUDIT OF RADIATION EXPOSURE IN CROHN'S PAST AND PRESENT

doi:10.1136/gutjnl-2013-304907.064

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Introduction The aim of this audit was to establish how much radiation patients with newly diagnosed Crohn's disease are exposed to and if this has changed over time within our practise. We hoped that with the increased availability of new imaging modalities such as ultrasound and MRI scanning the exposure of these patients to radiation may have fallen.

Methods Using the IBD database all Crohn's patients diagnosed from 1995 to 2011 were identified. We then collected data on 59 patients diagnosed from 1995 to 2001 and on 61 patients diagnosed from 2006 to 2011. These dates were chosen as ultrasound of the small bowel was introduced in UHL in 2002, and took a few years to become established as an imaging technique in Crohn's.

Data for each patient was collected only on studies performed in the first year of diagnosis, to prevent bias occurring due to the length of time that patients had the disease. We also felt the first year was likely to encompass studies performed to establish the diagnosis and extent of disease.

Results For patients diagnosed between 1995 and 2001 with Crohn's disease the average exposure to radiation in the first year was 1.83 mSv (range 0-12.3 mSv). For patients diagnosed between 2006 and 2011 we found an average exposure of 2.67 mSv (range 0-24 mSv), an increase of 46%.

Abstract OC-065 Table 1

| Dates | No of patients | MRI | US | СТ | AXR | Ba studies | Average dose (mSv) |
|-----------|----------------|-----|----|----|-----|------------|-----------------------|
| 1995–2001 | 59 | 0 | 24 | 4 | 24 | 35 | 1.83 |
| 2006–2011 | 61 | 25 | 65 | 19 | 45 | 5 | 2.67 |

Conclusion Radiation exposure in Crohn's disease appears to be increasing despite new modalities such as ultrasound and MRI. The increase is attributed to the increased use of CT scanning, as availability and accuracy of imaging via CT in Crohn's disease have improved in recent years. With the gradual introduction of low-dose CT scanning, we would hope these levels will fall again in the near future. Furthermore we observed an increase in the use of plain abdominal films of 87.5%. We feel this may be attributable to the shift in attitude towards treating unwell patients with the increasingly effective and available pharmacological therapies, rather than surgical options, although further audit should be carried out to establish if this is indeed the case.

Disclosure of Interest None Declared

Neoplasia and cancer pathogenesis free papers

OC-066 THE CANCER RESEARCH UK (CRUK) FUNDED ICGC OESOPHAGEAL ADENOCARCINOMA PROJECT: MRC RESEARCH CENTRE AND CRUK CAMBRIDGE RESEARCH INSTITUTE

doi:10.1136/gutjnl-2013-304907.065

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Introduction Esophageal adenocarcinoma (EAC) has one of the fastest rising incidences of any cancer in the western world. With a 5-year survival below 10% it is one of the most common causes of cancer death in US and UK. Currently little is understood about the genetic alterations that drive the development of OAC. Better understanding of these alterations may allow the development of novel therepeutic approaches

Methods We have performed whole genome sequencing on 22 cases. Targeted amplicon resequencing of 27 recurrently mutated genes was performed on a validation cohort of 100 further oesophageal adenocarcinomas.

Results In the discover set of 22 OACs we identified recurrent mutations (>3 tumours) in 31 genes including several implicated in tumorigenesis; TP53, CDKN2A, ARID1A. Strikingly in the validation cohort we observed that > 30% of EAC samples harbour mutation of one or both of the SWI/SNF complex members ARID1A and SMARCA4. In addition we identified highly recurrent mutations in several additional genes including TRIM58, SSTR4 and MYO18B.

Conclusion Whole genome sequencing provides an unbiased screen of mutational architecture of OAC. This has allowed the identification of several recurrently mutated genes not previously implicated in this disease providing a unique insight to it's pathogenesis **Disclosure of Interest** None Declared

OC-067 AN EXPRESSION SIGNATURE OF THE ANGIOGENIC RESPONSE IN GASTROINTESTINAL NEUROENDOCRINE TUMOURS: CORRELATION WITH TUMOUR PHENOTYPE AND SURVIVAL OUTCOMES

doi:10.1136/gutjnl-2013-304907.066

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Introduction Gastroenteropancreatic neuroendocrine tumours (GEPNETs) are heterogeneous with respect to biologic behaviour

and prognosis. Since angiogenesis is a renowned pathogenic hallmark as well as a therapeutic target, we aimed to investigate the prognostic and clinico-pathological role of tissue markers of hypoxia and angiogenesis in GEPNETs.

Methods Tissue microarray (TMA) blocks were constructed with 86 tumours diagnosed from 1988 to 2010. TMA sections were immunostained for Hypoxia inducible factor 1α (Hif- 1α), Vascular Endothelial Growth Factor A (VEGF-A), Carbonic Anhydrase IX (CaIX) and Somatostatin receptors (SSTR) 1 to 3 and Ki-67. Biomarker expression was correlated with clinico-pathological variables and tested for survival prediction using Kaplan-Meier and Cox regression methods.

Results Eighty-six consecutive cases were included: 51% male, median age 51 (range 16-82), 68% presenting with a pancreatic primary, 95% well differentiated. Forty-four cases (51%) had distant metastases (liver 72%, lymph nodes 28%). Median tumour size was 3.0 cm (range 0.6–12.5). Vascular invasion and necrosis were present in 20 (23%) and 18 (21%) of the specimens. Median overall survival (OS) was 8.8 years (range 0.1–13.5). Overexpression of CaIX was observed in 10% of the specimens, VEGF-A in 78%, Hif-1 α in 59%, SSTR1 in 17%, SSTR2 in 31% and SSTR3 in 1%. Ki-67 index was obtained in all cases and scored as G1 in 84%, G2 in 13% and G3 in 4%. SSTR2 overexpression was predominant in pancreatic NETs (p < 0.01), whilst Hif-1 α was predominant in non pancreatic NETs (p = 0.05). Higher Ki-67 labelling was associated with larger tumour size (p < 0.001) and necrosis (p = 0.03). Overexpression of Hif-1 α and VEGF-A correlated with the presence of liver metastases (p < 0.001). Patients with Ki-67 count > 1% (p = 0.02), high Hif-1 α and low SSTR2 expression (p = 0.03) displayed significantly shorter OS times. **Conclusion** We have identified a coherent expression signature by immunohistochemistry that can be used for patient stratification and to optimise treatment decisions in GEPNETs. Tumours with low proliferation index, preserved SSTR2 and low Hif-1 α expression have an indolent phenotype and may be offered less aggressive management and less stringent follow up.

Disclosure of Interest None Declared

OC-068 ALGORITHMIC MANAGEMENT OF RADIATION-INDUCED GI SYMPTOMS IS HIGHLY EFFECTIVE: THE ORBIT RANDOMISED CONTROLLED TRIAL

doi:10.1136/gutjnl-2013-304907.067

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Introduction Chronic gastrointestinal (GI) symptoms after pelvic radiotherapy are common. Most affected patients never see a GI specialist. We developed a comprehensive algorithm to direct management of new GI symptoms after pelvic radiotherapy. A 3 arm randomised controlled trial was performed to test 2 hypotheses: (1) Algorithm directed intervention is beneficial compared to no intervention (2) outcomes are not worse when patients are managed by a nurse rather than a gastroenterologist.

Methods Patients treated with pelvic radiotherapy > 6 months earlier with persisting GI symptoms were randomised to management according to the algorithm by 1. a GI nurse or 2. gastroenterologist or 3. the self help Macmillan booklet "Pelvic Radiotherapy: Possible Late Effects". After 6 months, booklet arm patients with persisting symptoms could ask to see the gastroenterologist. Patients in the nurse arm were transferred to the gastroenterologist if they had problems beyond the algorithm's scope. The primary end point was change in the modified Inflammatory Bowel Disease Questionnaire – bowel sub score (IBDQ-B) at 6 months. Follow up continued until 12 months. The trial had 80% power to answer the 1st hypothesis after randomising 196 patients and the 2ndafter closing the booklet arm, and randomising 22 more patients to gastroenterologist or nurse.