Abstract O34 Table 1 EUS results and follow up according to size of lesion

EUS Diagnosis	<10 mm	10–20 mm	>20 mm
Probable GIST	3	17	32
Benign lesion	5	15	1
Indeterminate	0	0	8
Pancreatic rest	6	11	0
Lipoma	1	7	1
Suspicion of malignancy	0	0	5
Cyst	0	4	5
Polyp	2	4	0
Diverticulum	0	0	1
Follow up	<10 mm	10–20 mm	>20 mm
Repeat EUS	2	23	8
Resection	0	2	14
OGD Surveillance	5	4	4
Imatinib	0	0	3
Repeat CT/MRI	0	2	13
Other chemo/surgery	0	0	2
Died other cause	0	3	1
No follow up	12	26	4
Diagnostic laparoscopy	0	0	1
No data/patient declined	0	1	1

18 (13.6%), 58 (43.9%), and 54 (40.9%) lesions were <10 mm, 10–20 mm and >20 mm respectively. Three patients had EUS reported as normal/submucosal thickening only.

78 of the 81 biopsy results of SELs at initial endoscopy provided no diagnostic value. 47 (35.6%) patients underwent FNA of lesion, (0%,12% and 72% of patients for size <10 mm 10–20 mm and >20 mm respectively). 5 (3.8%) SELs were not sampled due to patient factors. 27 (57%) of EUS-FNAs were diagnostic: 20 (42.5%) were proven GIST, 3 (6.3%) leiomyoma, 3 (6.3%) other malignancy and one lipoma. Only 2 (28%) FNAs of lesions <20 mm were diagnostic.

All patients with SELs <20 mm all survived, except four who died of unrelated causes. Two had a resection and 36 underwent surveillance. Of the 53 patients with SELs >20 mm, 17 had resection/Imatinib, 25 underwent surveillance and two died of other causes.

Conclusion EUS is a useful tool in the assessment, diagnosis and follow-up of small SELs. Management of lesions <20 mm remains controversial, however our patients with SEL <20 mm had no clinical sequelae and no EUS findings of concern during follow-up of at least one-year. Diagnostic yield of FNA for lesions <20 mm was low which suggests a more conservative, surveillance approach may be appropriate.

O35 RE-INITIATION OF ANTIPLATELET AGENTS AFTER UPPER GASTROINTESTINAL BLEEDING IN THE UK BETWEEN 2000 AND 2017

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Introduction Death after upper gastrointestinal (GI) bleeding is commonly due to cardiovascular disease and early re-initiation of anti-platelet therapy after haemostasis is associated with a reduction in all-cause and cardiovascular mortality. 2012 NICE upper GI bleeding guidelines advocate early re-initiation of antiplatelet agents for primary or secondary prevention of cardiovascular disease.

Methods The Health Improvement Network (THIN) is a UK primary care database containing data from over 15 million subjects. Subjects were identified with an existing diagnosis of ischaemic heart disease (IHD) or stroke, who were taking antiplatelet agents (aspirin, clopidogrel, ticagrelor, prasugrel or dipyridamole) and had an upper GI bleed between 2000 and 2017. Re-initiation of antiplatelet agents (defined as a record of at least one prescription after the upper GI bleeding event) within 28, 56 and 90 days was examined. Interrupted time series analysis (ITS) was performed to investigate changes in prescribing practices before and after the 2012 NICE guidelines, with prescription of statins over the same time period used as a negative control.

Results 6,372 subjects prescribed antiplatelet agents and 9,615 prescribed statins with a clinical code for upper GI bleeding and IHD or stroke were identified. Between 2000 and 2017, the proportion of subjects who had their antiplatelet agents re-initiated post-upper GI bleed increased from 33.9 to 55.6% ($p<0.001$), 41.2 to 71.3% ($p<0.001$) and 45.8 to 75.0% ($p<0.001$) at 28, 56 and 90 days respectively. There was no significant change in re-initiation of statins within 90 days (77.8 to 81.6%, $p=0.60$). The ITS revealed that the 2012 NICE guidelines had no significant effect on re-initiation of antiplatelet agents at 90 days (incidence rate ratio for post-trend change 1.001 (95% CI 0.999–1.003), $p=0.55$).

Conclusions There has been a progressive and statistically significant increase in early re-initiation of antiplatelet agents after upper GI bleeding in the UK between 2000 and 2017. However, the 2012 NICE upper GI bleeding guidelines appeared to have no significant effect on prescribing practices.

O36 INVESTIGATING CLONAL EXPANSIONS IN THE NORMAL STOMACH AND THE 3D ARCHITECTURE OF OXYNTIC GASTRIC GLANDS

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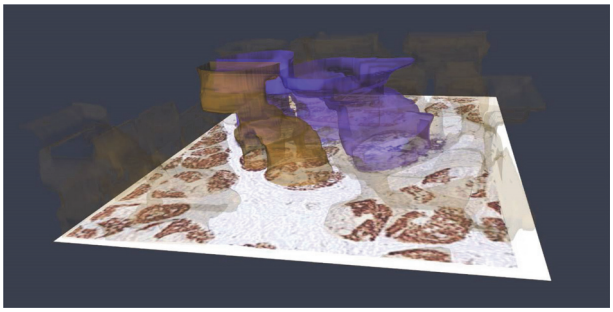
Introduction Gastric epithelium is exposed to constant environmental insults, with *H. Pylori* the primary carcinogen of non-cardia intestinal type gastric cancer. How mutations clonally expand in normal and inflamed epithelium prior to the development of cancer is relatively unknown. In this study we use neutral clonal markers to characterise clonal expansions in normal epithelium. We investigate the impact previous *H. Pylori* exposure has on clonal expansions in gastric epithelium. We then develop a new method of 3D reconstruction to visualise the structure of individual gastric glands.

Methods We collected tissue from patients undergoing sleeve gastrectomy for weight loss (n=15). Gastric corpus tissue was harvested and embedded *en face*, then labelled using enzyme- & immuno-histochemistry for neutral clonal markers (Cytochrome C Oxidase (CCO) & MTCO1) to identify clonal patches. Quantification was performed using digital



Abstract O36 Figure 1 3D reconstruction of gastric gland with CCO positive (brown) & negative regions (blue) visible

pathology software (Qupath), to analyse clonal patch sizes. Serial tissue sectioning was performed to trace CCO/MTCO1 mutated glands of interest for 3D reconstruction. Briefly, registration using a rigid and non-rigid B-spline transformation was applied, followed by a denoising step. Segmentation of glands was done by modelling using a Gaussian distribution, extraction of closing maps and applying an ellipsoidal fitting model. Cubic interpolation was then used for 3D modelling.

Results Patient ages were 31–65 years. Histologically, 8 were normal, 2 had active *H. Pylori* infection, 4 had evidence of previous infection with chronic inflammation, atrophy and intestinal metaplasia. CCO and MTCO1 clones were seen as wholly mutated glands and partially mutated glands. Overall clonal expansions were small, patch size analysis showed clones were most frequently singular glands, and rarely small patches (mean patch size = 1.65 glands). *H.Pylori* infection or chronic inflammation increased the frequency and size of patches compared to non-exposed tissue. 3D reconstruction (figure 1) allowed visualisation of the structure of the oxyntic gland, and tracing of CCO lineages allowed visualisation the functional architecture.

Conclusions This data describes the pattern of clonal expansions occurring in normal gastric epithelium. *H.Pylori* exposure and chronic inflammation lead to an increase of up to ten fold in frequency and size of clonal expansions. We observed a smaller increase in clonal expansions with advancing age. 3D reconstruction enabled tracing of mutant lineages in oxyntic glands, demonstrating for the first time the functional 3D architecture of the gastric stem cell unit. This work may help inform a model of pre-tumour progression in the chronically inflamed stomach.

Pancreas and neuroendocrine

O37 SCREENING FOR PANCREATIC CANCER IN HIGH RISK INDIVIDUALS: EXPERIENCE FROM A SPECIALIST CENTRE

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Introduction Two groups of high-risk individuals (HRI) for pancreatic ductal adenocarcinoma (PDAC) have been defined. 1) Individuals from familial pancreatic cancer (FPC) kindreds and 2) individuals with identified genetic syndromes (GS) due to a germline mutation. Screening of HRI has been proposed to identify premalignant lesions and early stage malignancy with the aim of improving outcomes. Screening criteria have been formulated by a number of organisations including the international Cancer of the Pancreas-Screening consortium (CAPS) and the Italian Society for the Study of the Pancreas (IASP). Recent CAPS and IASP publications have reported a significant yield. A prior meta-analysis concluded that 135 patients with HRI were needed to be screened to identify one high risk lesion. The aim of this study is to review compliance with guidelines and the yield of HRI screening in our screening programme.

Methods The study is a retrospective review of a prospectively maintained database of HRI. EUS, was the preferred annual screening method. MRI and CT were used in some patients due to intolerance of endoscopy or preference. Data was cross-checked with the endoscopy database and electronic patient record.

Results A total of 110 individuals (71F) median age 46 (IQR, 41–57.75) were enrolled and underwent at least one screening procedure between January 2006 and January 2019. 108 (98.2%) met either or both CAPS/IASP criteria: 58 were classified as FPC and 50 GS. The 2 who didn't meet criteria were a patient with idiopathic juvenile onset chronic pancreatitis (CP) and a patient with idiopathic CP and one first degree relative with PDAC. 487 screening procedures were performed. 407 (83.6%) EUS, 49 (10.1%) CT and 23 (4.75%) MRI with a median of 4 [IQR, 2–6] procedures per individual and median follow up 4.3 years [IQR, 2–7.75]. 9 (8.2%) had solid or cystic abnormalities identified on EUS and underwent tissue sampling. Two patients subsequently underwent distal pancreatectomy. The first (60 yr old female with hereditary pancreatitis) had a 20 mm cystic lesion in the tail of pancreas on her 2nd EUS. Resection histology was mucinous cystic neoplasm (follow up 11 years). The 2nd (48 yr old male, FPC) had a 14 mm nodule in body of pancreas. Histology was low grade pancreatic intraepithelial neoplasia (follow up 11.5 years). There were no adverse events consequent on screening.

Conclusions In a large cohort of HRI undergoing screening, compliance with international criteria was good with no screening related adverse event. However, the yield to date has been low with only one high grade precursor lesion resected from 487 screening procedures.

O38 RICOCHET: A TRAINEE-LED NATIONAL PROSPECTIVE STUDY OF THE DIAGNOSTIC PATHWAY FOR SUSPECTED PANCREATIC CANCER

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Introduction Pancreatic cancer is a deadly disease with a poor prognosis. Variations in the diagnostic pathway nationally may affect outcome, therefore a prospective study is necessary to map variation.

Methods Trainee-led prospective UK national study of the diagnostic pathway for suspected pancreatic cancer. Including all patients presenting within a 3-month study period, with 90-days follow-up. All investigation and MDTs were recorded in the REDCap database with a unique OpenPseudonymiser