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