oesophageal changes. A diagnosis can only be made when a dense eosinophilia is confirmed on histology in the context of typical symptoms (e.g. solid food dysphagia)¹. We prospectively assessed the prevalence of EO in patients presenting to endoscopy at a tertiary referral centre with solid food dysphagia over 2 years.

Methods Between Jan 2010 and Dec 2011, 746 patients with dysphagia (including food bolus obstruction) had high definition white light endoscopy performed. Patient demographics, symptomatology, endoscopic and histological findings were recorded. EO was defined as the presence of > 15 eosinophils per high power field

Results Patients with oesophageal malignancy (n = 65), Barrett's oesophagus (n = 48) and post-oesophageal surgery complications (n = 16) were excluded. Of the 628 remaining patients, 388 (62%) (254 male; mean age 59; range 18–88) had mid-oesophageal biopsies taken. 23/388 (5.9%) were diagnosed with EO (19 male; mean age 40; range 26–56). Endoscopy showed mucosal pathology in 12/23 (52%) patients with confirmed EO; oesophagitis (n = 3), red furrows (n = 3), distal narrowing (n = 2), corrugated rings (n = 2), mucosal tear (n = 1) and white exudates (n = 1). 250 of the remaining patients had grade A or B oesophagitis. Overall 17 patients had food bolus obstruction. 11/17 patients had biopsies taken and 5/11 (46%) showed histological evidence of EO. 4/5 patients with bolus obstruction had distal oesophagitis on endoscopy but EO was confirmed following ≥4 mid-oesophageal biopsies. There was a trend towards those with EO having had a greater number of biopsies taken (mean 6.14; range 2–12) compared to those without EO (mean 5.02; range 2–8; p = 0.082). 28% and 51% had ≤3 and ≤4 biopsies collected respectively. The mean (±SD) number of eosinophils/hpf in the EO group was 64.3 (51.3).

Conclusion Mid-oesophageal biopsies can diagnose EO in at least 1 in 16 cases of patients with unexplained solid food dysphagia. However, 1/3 of patients in whom EO should have been considered (including 6 with food bolus obstruction) did not have biopsies collected. Furthermore, 1/4 had less than the recommended minimum 4 mid-oesophageal biopsies. In summary, our experience has shown that EO detection is likely to improve further if all patients with symptoms conducive with EO (e.g. solid food dysphagia) routinely trigger an EO biopsy protocol of ≥4 from the mid-oesophagus regardless of endoscopic findings.

Disclosure of Interest None Declared

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OC-027 DEFINING CANCER RISK IN BARRETT'S OESOPHAGUS USING A 90-GENE SIGNATURE

doi:10.1136/gutjnl-2013-304907.027

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Introduction Barrett's oesophagus (BE) has a highly variable outcome with 0.12–0.5% of patients per year progressing to oesophageal adenocarcinoma (EA). The histopathological grading of dysplasia is used to detect cancer risk in BE; however, considerable variability exists in the reporting of dysplasia. Molecular biomarkers that can detect BE patients with dysplasia would improve risk stratification in BE, enabling clinicians to focus on high risk patients requiring treatment and reduce endoscopic surveillance in the low risk group. The aim of this study was to identify and validate a gene expression signature as a biomarker that can objectively determine dysplastic status and thereby determine the risk of cancer progression.

Methods Microarray gene expression profiling was done using 59 oesophageal samples with strict consensus diagnosis by expert pathologists (21 BE with no dysplasia, 10 BE with low grade dysplasia, 13 BE with high grade dysplasia and 8 EA). This data was used to identify a gene signature that separated non-dysplastic BE from high grade dysplasia. Gene expression data from publically available datasets were used to validate the signature. An independent set of 135 fresh frozen samples covering a spectrum of dysplastic Barrett's stages and control tissue (40 BE with no dysplasia, 21 BE with low grade, 33 BE with high grade dysplasia, 32 EA and 9 duodenum) were used for validation using the high throughput 96:96 microfluidic Fluidigm® chip on the BioMark™ PCR system.

Results A set of 90 genes was identified that separated BE with no dysplasia from BE with high grade dysplasia. This 90-gene signature was able to separate the remaining untrained samples on the microarray dataset (7 non-dysplastic, 10 low grade dysplasia and 8 EA). The signature also separated non dysplastic BE samples from EA samples on 2 external published datasets (p ≤ 0.0012). With the fresh frozen samples, the signature separated BE with no dysplasia from BE with dysplasia and EA with an area under the curve of 0.87 (95% CI, 0.80–0.93). Pathway analysis revealed that the RAN (RAS-related Nuclear protein) regulation pathway (p < 0.0001) was the most significant pathway in this gene set. Furthermore, MYC was found to be the most significant transcription factor regulating at least 30% of these genes (p < 0.0001).

Conclusion The 90 gene-expression profile can reliably identify BE samples with dysplasia and cancer. This approach has the potential to provide robust risk stratification in BE samples as it overcomes the problems with variability in the reporting of dysplasia.

Disclosure of Interest None Declared

OC-028 STATIN USE IS ASSOCIATED WITH IMPROVED SURVIVAL IN PATIENTS WITH OESOPHAGEAL CANCER: A SURVIVAL ANALYSIS USING THE UK GENERAL PRACTICE RESEARCH DATABASE AND NATIONAL CANCER REGISTRY

doi:10.1136/gutjnl-2013-304907.028

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Introduction Patients with oesophageal cancer (OC) commonly present with an advanced stage of disease, and are often only amenable to palliative therapies. Of the minority suitable for potentially curative surgery, up to 50% develop recurrence at one year. Statins demonstrate several anticarcinogenic properties in oesophageal adenocarcinoma (OAC) cell lines including reducing cell proliferation, stimulating apoptosis and potentially limiting metastatic potential. We investigated for the first time the hypothesis that statin use after diagnosis or post-oesophagectomy was associated with improved survival in patients with OC.

Methods Cases of OC diagnosed between 1st January 2000 and 31st December 2009 were identified from the UK General Practice Research Database (GPRD). The GPRD data was linked to the UK National Cancer Registry (NCR) to determine histological subtype. Cox proportional hazard regression analysis with time-dependent exposures, estimated the associations between statin use (versus non-users) from diagnosis and post-surgery on overall survival and disease-specific survival. Multivariate analyses were adjusted for age, gender, body mass index, diabetes mellitus, cardiovascular disease, oesophagectomy, chemotherapy, radiotherapy and ACE inhibitor use.

Results In total 4445 cases of OC were identified, of which 606 were OAC and 344 were OSCC (histology data was available for 21.4% of patients). Overall 585 (13.2%) patients underwent oesophagectomy. In total 609 (13.7%) of patients were statin users