Methods Patients admitted to SWBH NHS Trust with AUGIB were recruited. Dyspeptic patients attending for diagnostic OGD were used as controls. To assess platelet activation, citrated whole blood was incubated at room temperature with monoclonal mouse antibodies against constitutively expressed platelet marker CD42a-PerCP, and markers of platelet activation PAC1-FITC, and CD62P-APC. Incubation was terminated after 15 minutes. Samples were analysed using a FACSCalibur flow cytometer. Platelets were identified on the basis of their forward and side scatter properties and the presence of the CD42a platelet-specific marker. CD62P and PAC1 expression were measured by the percentage of platelets expressing these markers.

Data are expressed as means±SD for normally distributed parameters and median (interquartile range) for non-normally distributed parameters. Statistical analysis was performed using SPSS 18.0 software.

Results A total of 24 patients with AUGIB and 18 controls were recruited. Patients were age and gender matched. The mean age of the AUGIB group is 66.4 ± 18.2 years, and the control group is 62.8 ± 6.1 years. Significant differences were seen in all markers of platelet activation (table 1).

Introduction Recently we showed that the length of cardiac mucosa in asymptomatic volunteers correlated with age and obesity defined by waist circumference (WC) and intra-abdominal fat on MRI (ref). To further investigate the aetiology of expanded cardi a, we have performed detailed histological and immunohistochemical studies comparing cardi a with other upper GI epithelia including long segment Barrett’s with or without intestinal metaplasia.

Methods Double oriented biopsies from SCJ of the 52 H. pylori negative healthy volunteers in the original obesity study were examined. To assess inflammation, the densities of polymorphonuclear (PMN), mononuclear (MN) cell infiltrations and reactive atypia were scored at squamous, cardi a and oxyntocardiac mucosae of SCJ, separately. Slides were also stained for CDX-2, Villin, TFF-3 and CD15. The immunoreactivity in each of the three types of mucosa were compared to additional biopsies from the antrum and gastric body in same subjects and biopsies from ten patients with long-segment Barrett’s demonstrating foci with and without intestinal metaplasia (IM).

Results The median scores of PMN and MN cell infiltrations were maximum in the cardi a mucosa compared to either proximal or distal adjacent tissues (all p values <0.001). The score of reactive atypia showed that the cardi a mucosa had similarities to the antrum and Barrett’s with IM; however, it was identical in all immunohistochemical aspects to non-IM Barrett’s mucosa (Table). Table The extent (%) of immunostaining with different antibodies in squamocolumnar junction, gastric body, antrum and Barrett’s

Conclusion Patients presenting with AUGIB have prolonged levels of platelet activation for at least 12 weeks following the index event. This phenomenon may be further prolonged and further studies are required. This may explain the excess of CVS events in AUGIB patients. In patients with high cardiovascular risk early re-introduction of aspirin should be considered.

Disclosure of Interest None Declared

REFERENCES

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REFERENCE

Oesophageal free papers

EXPANDED CARDIA MUCOSA ASSOCIATED WITH CENTRAL OBESITY IMMUNOHISTOCHEMICALLY RESEMBLES NON-IM BARRETT’S MUCOSA

doi:10.1136/gutjnl-2013-304907.025
1Institute of Cardiovascular & Medical Sciences, 2Institute of Cancer Sciences, University of Glasgow, Glasgow, UK

Abstract OC-025 Table 1 Platelet activation at 12 weeks

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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). 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 18–83) had mid-oesophageal biopsies taken. 25/388 (5.9%) were diagnosed with EO male; mean age 40; range 26–56). Endoscopy showed mucosal pathology in 12/23 (52%) patients with confirmed EO; oesophagitis (n = 5), 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 a greater number of biopsies taken (mean 6.14; range 2–12) compared to those without EO (mean 5.02; range 2–8; p = 0.022). 28% and 51% had ≤3 and ≤4 biopsies collected respectively. The mean (±SD) number of eosinophils/hpf in the EO group was 64.3 (51.5).

**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

**REFERENCE**


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**OC-027**

**DEFINING CANCER RISK IN BARRETT’S EOSPHAGUS USING A 90-GENE SIGNATURE**

**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 155 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 96K 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 80% 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

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**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**

**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 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.