hyposplenism and the high prevalence of hyposplenism in CD, it is worth considering pneumococcal vaccination for all CD patients at diagnosis.

Disclosure of Interest J. Khan: None Declared, A. Jennings: None Declared, S. Subramanian Speaker bureau with: Shire, Abbott and Dr Falk pharma, Conflict with: Advisory board member for Vifor Pharma and Abbott

REFERENCE
1. Ludvigsson JF, Olén O, Bell M, Ekbom A, Montgomery SM. Coeliac disease and risk of sepsis. Gut 2008; 57(8):1074–80.

ENDOSCOPY PITFALLS IN CELIAC DISEASE DIAGNOSIS; A MULTICENTRE STUDY

Introduction The traditional diagnosis of celiac disease (CD) requires a small bowel biopsy to identify at histology the characteristic mucosal changes. The current biopsy practice amongst endoscopists for celiac disease is in most part unknown. The aims of this study were to compare the different diagnostic criteria in various centres in Italy, Iran, Lithuania, Romania and the UK, the methodological approach to the biopsy and to investigate the pitfalls of CD diagnosis.

To measure the number of specimens submitted during duodenal biopsy among patients in Italy, Iran, Lithuania, Romania and the UK, and to determine the incremental diagnostic yield of adherence to the recommended number of specimens.

Methods A total of 931 patients who underwent duodenal biopsy for CD were recruited prospectively at nine centres in European and Middle East countries. Small-bowel biopsies were obtained from the duodenal bulb and the second part of the duodenum (and from the duodenal bulb when it had a micronodular appearance). The histopathological appearances were described according to the modified Marsh classification.

Results The most frequent degree of villous atrophy amongst Iranian subjects was 3A and that of the rest of the study population was 3C. The most common number of biopsy specimens for Romanian subjects was 3C. The most common number of biopsy specimens for Romanians was 1 (52%) followed by 2 for Italian (56%), 3 for Lithuanian (66.7%) and British patients (65%) and 4 for Italian patients (48.3%). The main presenting symptom was anaemia (18.7%) followed by malabsorption (10.5%), diarrhoea (9.3%) and dyspepsia (8.2%).

Conclusion Taking less biopsy samples than recommended will have a negative impact in detecting massive number of undiagnosed cases. As CD is more common with atypical presentation, taking 4 duodenal biopsies is mandatory for an accurate diagnosis or its exclusion.

Disclosure of Interest None Declared

Abstract OC-023 Table

|                        | GI (n = 53) | Non-GI (n = 24) | p   |
|------------------------|------------|----------------|-----|
| Age (years)            | 56.4 ± 22.87 | 53.9 ± 21.9    | 0.64|
| Male:Female            | 43:10 (4.3:1) | 17:7 (2.4:1)   | 0.37|
| Time to endoscopy (days)| 1.23 ± 1.57 | 1.79 ± 2.93    | 0.38|
| Laparotomy             | 0          | 2 (8.3%)*      | 0.09|
| Mortality ascribed to UGIB | 3 (5.7%) | 2 (8.3%)*      | 0.64|

*different patients

Conclusion The length of stay of patients with UGIB is dramatically shorter when receiving specialist care. This was statistically significant even after adjusting for social issues. Further data regarding the specific management of each case will be forthcoming. In line with previous reports [3], we found that the incidence of UGIB was higher in males. There was a trend toward better risk assessment, shorter time to endoscopy, reduced need for surgery and mortality in the GI group. Mortality rates in both groups compared favourably to the national average.

Disclosure of Interest None Declared

REFERENCES
1. CG141
2. Scope for improvement: A toolkit for a safer Upper Gastrointestinal Bleeding (UGIB) service. www.bsg.org.uk
3. Lasan A, García-Rodríguez LA, Polo-Tomás M etal; Am J Gastroenterol 2009; 104:1633–41.
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-PecCP, 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 mean±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 62.8±6.1 years. Significant differences were seen in all markers of platelet activation (table 1).

Abstract OC-024 Table 1 Platelet activation at 12 weeks

|                       | AUGIB                        | Controls                     | P-value |
|-----------------------|------------------------------|------------------------------|---------|
| CD62P %               | 16.77 (15.26–18.28)          | 12.95 ± 2.77                 | < 0.001 |
| PAC1%                 | 7.04 ± 3.67                  | 3.98 ± 1.78                  | 0.001   |
| CD62P+PAC1+ %         | 1.33 (0.70–1.97)             | 0.73 (0.60–0.87)             | 0.003   |

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

1. Sung JJ, Lau JY, Ching JY, Wu JC, Lee YT, Chiu PW, Leung VK, Wong VW, Chan FK. Continuation of low-dose aspirin therapy in peptic ulcer bleeding: a randomised trial. Ann Intern Med. 2010 Jan 5; 152(1):1–9.
2. Disney BR, Watson R, Blann A, Lip G, Tselepis C, Anderson M. Platelet activation in acute upper gastrointestinal bleeding. Gut 2012; 61 (Suppl 2): A361.

OC-025 EXPANDED CARDIA MUCOSA ASSOCIATED WITH CENTRAL OBESITY IMMUNOHISTOCHEMICALLY RESEMBLES NON-IM BARRETT’S MUCOSA

doi:10.1136/gutjnl-2013-304907.025

1. M H Derakhshan, E V Robertson, Y Y Lee, A A Wirz, J J Going, K E McColl. 1Institute of Cardiovascular & Medical Sciences, 2Institute of Cancer Sciences, University of Glasgow, Glasgow, UK

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 cardia, we have performed detailed histological and immunohistological studies comparing cardia 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, cardia and oxyntocardiac mucosas of SCJ, separately. Slides were also stained for CDX-2, Villin, TFF-3 and LI-Cadherin. 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 cardia mucosa compared to either proximal or distal adjacent tissues (all p values < 0.001). The score of reactive atypia was maximum at the most distal squamous mucosa. Immunohistochemistry showed that the cardia 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). The extent (%) of immunostaining with different antibodies in squamocolumnar junction, gastric body, antrum and Barrett’s was further investigated.

Table

| Antibody | Squamocolumnar Junction | Cardium | Cardia | Oxyntocardiac | Body | Antrum | Barrett’s(nonIM) | Barrett’s(IM) |
|----------|--------------------------|---------|--------|---------------|------|--------|------------------|---------------|
|          | Median                   | Squamous| Cardiac | Oxyntocardiac | Body | Antrum | Barrett’s(nonIM) | Barrett’s(IM) |
| CDX.2    | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| Villin   | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| TFF.3    | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| LI.Cadherin | 0.00                | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |

Disclosure of Interest None Declared

REFERENCE

Robertson et al. Gut 2012; 61(supp 2): A256–7.

Oesophageal free papers

OC-026 EOSINOPHILIC OESOPHAGITIS IN PATIENTS PRESENTING WITH DYSPHAGIA- A PROSPECTIVE ANALYSIS

doi:10.1136/gutjnl-2013-304907.026

1. M Kumar, F Khan, R Sweis, T Wong. ‘Gastroenterology, St.Thomas’ Hospital NHS Trust, London, UK

Abstract OC-025 Table

| Antibody | Squamocolumnar Junction | Cardium | Cardia | Oxyntocardiac | Body | Antrum | Barrett’s(nonIM) | Barrett’s(IM) |
|----------|--------------------------|---------|--------|---------------|------|--------|------------------|---------------|
|          | Median                   | Squamous| Cardiac | Oxyntocardiac | Body | Antrum | Barrett’s(nonIM) | Barrett’s(IM) |
| CDX.2    | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| Villin   | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| TFF.3    | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| LI.Cadherin | 0.00                | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |
| IQR      | 0.00                    | 0.00    | 0.00   | 0.00          | 0.00 | 0.00   | 0.00             | 0.00          |

Introduction Eosinophilic oesophagitis (EO) is a chronic relapsing-immune/antigen mediated disease of the oesophagus with rapidly increasing incidence and prevalence; however EO often remains under-diagnosed. Early detection and appropriate therapy improves quality of life and may prevent development of chronic