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 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

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

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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 mucosae 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).

Table The extent (%) of immunostaining with different antibodies in squamocolumnar junction, gastric body, antrum and Barrett's

Conclusion Cardia mucosa which is extended proximally in H. pylori negative healthy volunteers with central obesity, is immunohistochemically identical to non-IM Barrett's mucosa. This is consistent with the expansion of cardia mucosa having similar aetiology to Barrett's mucosa and being due to metaplasia of the most distal oesophageal mucosa resulting from short segments reflux.

Disclosure of Interest None Declared

REFERENCE

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

Oesophageal free papers

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

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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

Abstract OC-025 Table

Antibody		Squamocolumnar Junction				Body	Antrum	Barrett's(nonIM)	Barrett's(IM)
		Squamous	Cardiac	Oxyntocardiac					
CDX.2	Median	0	1.0	0		0	1.0	5.0	90.0
	IQR	0.0	0.10	0.0		0.0	0.1	0.10	78,90
Villin	Median	0	30.0	5.0		1.0	90.0	35	90.0
	IQR	0.0	20,70	0.10		0.1	81,90	20,48	90,95
TFF.3	Median	1.0	80.0	30.0		10.0	70.0	70.0	90
	IQR	0.5	70,90	10,30		0.13	30,70	60,80	90,90
LI.Cadherin	Median	5.0	15.0	10.0		5.0	17.5	10.0	90.0
	IQR	1,5	5,25	5,15		0.9	11,24	5,30	89,91