Methods This was a retrospective cohort study of all patients undergoing antireflux surgery from 2001 to 2009 in a tertiary centre under a single surgeon. Patients undergoing fundoplication and/or repair of paraoesophageal hernias were included. Patients underwent preoperative assessment by means of endoscopy, oesophageal manometry and 24 h oesophageal pH monitoring. Patients were grouped on the basis of their 24 h pH profile into upright refluxers (daytime increased oesophageal acid exposure) and mixed refluxers (night-time increased oesophageal acid exposure +/- daytime reflux). Primary outcomes included if the patient had stopped PPIs following surgery and the incidence of postoperative dysphagia, vomiting and reflux symptoms. Secondary outcomes included if the patients deemed the operation a success.

Results 120 patients were included, with a median age of 49 years (range 24–81) at time of surgery. 55% (n=63) were male and 45% were laparoscopic procedures. 100 patients (83.3%) had Nissen fundoplication and 16% had a combination of fundoplication and paraoesophageal hernia repair. A DeMeester score >15 was present in 97.8% (n=88) patients, with 21% (n=19) patients having upright reflux and 65.6% (n=63) having mixed reflux symptoms on pH studies. Mixed refluxers were nearly twice as likely to have significant eosinophilic oesophagitis (grade B+) and or Barrett’s oesophagus on preoperative endoscopy (mixed reflux vs upright reflux: p=0.051). Those patients with mixed reflux symptoms were significantly more likely to stop PPIs postoperatively (mixed reflux vs upright reflux: p=0.011). Further, the incidence of significant post operative dysphagia was doubled in the upright reflux group (upright reflux 53.3% vs mixed reflux 26.7%; p=0.053). Overall, 83.3% (n=76) deemed the operation to have been a success, 7.7% (n=7) a partial success and 8.3% (n=8) considered that surgery had failed to improve their symptoms.

Conclusion In addition to the DeMeester score for predicting outcomes in antireflux surgery, the presence of reflux only in the upright position may indicate a poorer outcome.

Competing interests None declared.

REFERENCES

Abstract PTU-191 Figure 1 Kaplan–Meier overall survival according to CRM.

Conclusion R1a and R0 CRM are associated with equivalent recurrence (local and distant) and survival rates to R0 in patients with oesophageal and GOJ adenocarcinoma following NAC and oesophagectomy. R1a CRM involvement does not adversely survival in patients with oesophageal and GOJ adenocarcinoma following NAC.

Competing interests None declared.
quadrantic biopsies. DNA content abnormalities (aneuploidy/tetra- ploidy); loss of heterozygosity (LOH) at 9p and 17p loci; RUNX3, HPPI and p16 methylation; immunohistochemistry (IHC) for p53 and Cyclin A were tested on targeted biopsies. Each biomarker was correlated with the dysplasia and AFI status.

**Results** 111 patients with 210 biopsy areas were included in the analysis (AFI+, n=120; AFI-, n=90). Univariate per-biopsy analysis showed that all biomarkers correlated with dysplasia (p<0.05), with exception of 9p LOH. Multivariate analysis showed that aneuploidy, p53 IHC and Cyclin A (3 biomarker panel) were independently associated with dysplasia with an AUC=0.93 (95% CI 0.88 to 0.98) for any dysplasia and AUC=0.95 (95% CI 0.89 to 1) for HGD/early cancer (EC). AFI positivity significantly correlated with aneuploidy, p16 methylation, cyclin A and p53 staining (p<0.05). After excluding dysplastic areas, aneuploidy (p=0.05) and p53 (p=0.04) staining retained statistical correlation with AFI positivity. Analysis of the 3 biomarker panel in patients with dysplasia showed significant biomarker enrichment in AFI+ compared to AFI- areas (p=0.001). Finally, 3 biomarker panel was used to predict prevalent dysplasia. Using a cut-off of ≥2 biomarkers, the panel when applied to AFI+ areas alone, showed sensitivity and specificity of 88% and 90% respectively for diagnosis of HGD/EC, and 64% and 96% respectively for diagnosis of any dysplasia, compared to overall histology.

**Conclusion** AFI increases detection rate for molecular biomarkers. A panel of 3 molecular biomarkers on a small number of AFI targeted biopsies can efficiently predict the dysplasia status and potentially inform therapeutic management of patients with BE.

**Competing interests** None declared.

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**GASTRO-OESOPHAGEAL REFUX (GORD) AND COELIAC DISEASE: A BIDIRECTIONAL STUDY**

**Introduction** There are limited data assessing the relationship between coeliac disease and GORD. We aimed to establish the prevalence and nature of GORD symptoms in patients with coeliac disease, and the prevalence of undetected coeliac disease in those presenting to endoscopy with GORD symptoms.

**Methods** Group A were histologically proven patients with coeliac disease (n=225) who were asked to complete a validated reflux questionnaire and then compared to age/sex-matched controls (n=548). Group B were consecutive GORD patients undergoing endoscopy who had duodenal biopsies and coeliac serology taken. (n=851) Furthermore, patients with newly diagnosed coeliac disease underwent manometry and pH studies prior to commencing gluten free diet (n=35).

**Results** In Group A the prevalence of GORD was greater in coeliac disease (66%) than in healthy controls (50%) p=0.0001. Coeliac patients also report reflux of greater severity: coeliac disease OR=6.8, 95% CI=3.6 to 12.7, p≤0.001. In Group B at endoscopy the prevalence of undetected coeliac disease was 1.66% (14/851). In Group C 31/33 were able to tolerate manometry and complete testing (2 had partial investigation before the catheter was removed) At manometry 50/33 (91%) had a normal lower oesophageal sphincter pressure (LOS). 2/33 had a hypertensive LOS. 21/33 (64%) had normal oesophageal motility. However 10/33 had a hypocontractile oesophagus, 1 was hypertensive and 1 showed functional oesophageal junction obstruction. During manometry 6/31 (19%) demonstrated significant reflux, 6/31 (19%) had some reflux and the final 19/31 (61%) had no reflux episodes. In these coeliac patients neither the presence of symptoms nor abnormal oesophageal study findings was related to histological grade of coeliac disease, (villous atrophy) or serology findings.

**Conclusion** Up to two thirds of patients with coeliac disease report reflux symptoms and one third have demonstrable abnormalities of oesophageal motility and reflux. However, in an unselected population at endoscopy, reflux symptoms are not predictive of coeliac disease.

**Competing interests** None declared.

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**POSTERS**

**PTU-194**

**DOES METABOLIC SYNDROME IMPACT TUMOUR PATHOLOGY IN OESOPHAGEAL ADENOCARCINOMA?**

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**Introduction** Obesity is an established risk factor for both the increased incidence of oesophageal adenocarcinoma cancer (OAC), and adverse outcomes by increasing risk of recurrence and reducing survival in obese patients post oesophagectomy. The exact mechanism of this relationship is unclear but the pattern of fat distribution pattern is likely important. Abdominal obesity more closely reflects an increased visceral fat area and is associated with alterations in metabolic risk profile. The clustering of central obesity, hypertension, and raised plasma glucose, triglycerides and HDL cholesterol is also known as the metabolic syndrome (MetS). The processes underlying the metabolic syndrome especially insulin resistance and increased leptin, can provide a favourable growth environment for malignant cells and may have a role in cancer progression.

**Methods** The aim of this prospective observational study of OAC patients was to examine the incidence of MetS and its relationship to tumour pathology in an Irish population. Patients underwent a metabolic and nutritional assessment prior to initiation of treatment. Visceral fat area was measured using CT scans. MetS was defined according to the International Diabetes Federation definition.1

**Results** 83 OAC patients (71 male: 12 female) were recruited with a median age of 64.6 years ± 10 (range 48–86). All patients underwent an oesophagectomy, with 42% (n=35) receiving neo-adjuvant chemoradiotherapy. 58% of patients were either overweight or obese with a further 60% centrally obese. Males had significantly greater visceral fat area (p=0.031) despite no difference in total abdominal fat compared to females (p=0.757). The incidence of obesity may be underestimated as 41% of patients reported unintentional weight loss with 18% losing >5% of their usual body weight. MetS was diagnosed in 59% patients, which exceeds the population norms reported at 21%.2 The presence of MetS was not associated with tumour length, depth of invasion, lymph node positive disease, clinical or overall pathological stage in males or females. Individual features of MetS were also not significantly related to the pathological staging of oesophageal cancer.

**Conclusion** We report an increased prevalence of MetS and central obesity in a cohort of Irish patients with OAC. Dysphagia and weight loss are common in the presentation of oesophageal cancer and may mask the effect of obesity and metabolic syndrome on the clinical pathological features of OAC in this cohort. Further research is needed to fully understand the underlying biological mechanisms linking obesity to oesophageal cancer.