compliance with Spigelman scoring system. 4/14 had recurrent Duodenal adenomas after EMR. 3 patients were found to have Colon cancer. So it is essential that all patients with Duodenal adenomas have a colonoscopy.

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Disclosure of Interest None Declared.

PTU-178 CLINICAL UTILITY OF ENDOFASTER® IN PATIENTS ON CHRONIC PPI THERAPY UNDERGOING UPPER GI ENDOSCOPY

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Introduction Patients undergoing upper GI endoscopy (OGD) are often on chronic PPI therapy for dyspeptic symptoms or gastro-oesophageal reflux disease (GORD). Continued PPI therapy at the time of the endoscopy can influence the outcome of H. pylori testing via rapid urease test (CLO) or even histology. In addition, complete response to PPI in patient with GORD and/or Barrett's oesophagus (BO) is often not fully predicted by the clinical history. We hypothesised that rapid testing of H. pylori status and gastric pH by Endofaster® could provide the physician with real-time information useful to influence patient management.

Methods This pilot feasibility study included 135 consecutive patients at a single centre who underwent OGD for BO surveillance/endotherapy (n = 95), screening for hereditary diffuse gastric cancer (n = 11) and evaluation of GORD/dyspepsia (n = 29). The pH and the H.pylori status were measured on 4 mL of gastric aspirate using the Endofaster®, which connects to the suction port of the gastroscope. If clinically indicated, CLO test or gastric biopsies were performed. Endofaster® results were matched with the history of PPI intake (PPI type and time of last dose) and results of CLO and gastric histology.

Results Overall, 109 patients were on chronic PPI treatment and of these 74% presented with gastric hypochloridia (pH >4) at Endofaster® analysis. Forty-nine patients reported PPI intake on the same day of the OGD and 15% of these (n = 7) had acid gastric pH (<4). Fifty-nine patients had CLO test and 57 had gastric histology results available, while 26 patients had both. Only 1 patient was positive for H. pylori on histology, which was also positive at Endofaster®. Two patients had a positive CLO test, of which one was Endofaster® positive for H. pylori. Eleven patients were positive at the Endofaster® but not at CLO, of which 91% were on chronic PPI (n = 10).

Conclusion This feasibility study shows that a significant proportion of patients on chronic PPI therapy still have acidic gastric pH suggesting sub-optimal response to PPI. Endofaster® may detect H.pylori infection in patients on chronic PPI therapy, which are often false negatives when tested by CLO and histology. A prospective study matching Endofaster® data with gold standard tests for H. pylori status and gastro-esophageal reflux are needed to conclude on the clinical implications of these findings.

Disclosure of Interest None Declared.

PTU-179 SOCIAL DEPRIVATION IN BARRETT'S OESOPHAGUS?

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Introduction Squamous oesophageal carcinoma is more prevalent in those with lower socio-economic status. Little is known about this for oesophageal adenocarcinoma (OAC). Anecdotally, OAC has been observed in higher socio-economic groups. Barrett's oesophagus (BO) is the only known precursor of OAC. This study investigates the association between BO and social deprivation using the 2010 Index of Multiple Deprivation (IMD).

Methods *Patients*: 1076 BO diagnosed in Rotherham, between April 1978 and August 2012. *IMD:* The Office for National Statistics (ONS) divides England into geographical areas each with a similar population size termed Lower Layer Super Output Areas (LSOA). The 2010 IMD comprising a combination of 38 separate indicators is assigned to each LSOA. Every residential postcode is assigned to a LSOA and thus an IMD. This was applied to the postcodes of Rotherham patients at time of BO diagnosis. IMD Quintiles were derived by dividing the distribution of all IMD scores in England into 5 equal categories. The 6257 residential postcodes for Rotherham and the 1076 BO patients were placed in the quintiles relevant to their IMD score. *Analysis:* Chi square goodness of fit tests were used to compare the observed

Abstract PTU-179 Table 1

		BO diagnosed = 2000 n = 490			BO diagnosed = 2001 n = 586		
IMD quintile descending order of deprivation	Rotherham postcodes* n = 6257	Observed (O)	Expected (E)	Ratio (O/E)	Observed (O)	Expected (E)	Ratio (O/E)
Most deprived	2282	188	178.7	1.05	199	213.7	0.93
2nd most deprived	1659	142	129.9	1.09	128	155.4	0.82
3rd most deprived	988	64	77.4	0.83	89	92.5	0.96
2nd least deprived	1049	71	82.2	0.86	133	98.2	1.35
Least deprived	279	25	21.8	1.14	37	26.1	1.41
p value				*0.21*			*0.0001*

* Each postcode includes ~20 households.

(O) distribution of BO to the expected (E). Analysis was stratified into those diagnosed before 2001 and 2001 onwards, this being the median year of diagnosis.
Results (See Table) 2/3rds of all Rotherham postcodes fell into the 2 most deprived quintiles. The O/E IMD distribution of the BO cohort diagnosed before 2001 was similar to that of the Rotherham population but highly significant differences emerged later (p = 0.0001): the two least deprived quintiles had 170 BO patients against 124 expected (37% increase).
Conclusion No single factor is likely to explain the change observed in the last decade, however, the highly significant difference observed points to a strong association between *lower* deprivation and *increasing* risk of BO. To the best of our knowledge this is the first report showing a quantitative link between BO and socio-economic status, which may form a basis for the apparent socio-economic shift between squamous oesophageal cancer and OAC.
Disclosure of Interest None Declared.

PTU-180 ROLE OF BODY COMPOSITION AND METABOLIC DYSFUNCTION IN BARRETT'S OESOPHAGUS AND PROGRESSION TO CANCER

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Introduction Oesophageal adenocarcinoma (OAC) arises within Barrett's oesophagus (BE). Obesity is associated with metabolic syndrome (MS) and cancer progression. Body composition has a direct impact in obesity-related diseases. Normal weight individuals with increased fat mass are considered metabolically obese.
Methods To evaluate the prevalence of obesity, altered body composition and metabolic indexes in patients (pts) with and without BE; and association with cancer progression in BE. In sequential pts undergoing gastroscopy, MS, waist/hip ratio (WHR) and body fat% (BF by bioimpedance analysis) were obtained. In BE pts, histological findings were correlated with metabolic data. Pts were classified according to Body Mass Index (BMI), abdominal obesity (AO by WHR) and in females, Normal Weight Obese (NWO). Identified risk factors significantly associated with BE at univariate analysis were subsequently entered into a multivariate logistic regression analysis.
Results 250 cases and 230 controls (F/M: 193/287) were enrolled. Age (cut off: 57 years) and male gender (M/F 193/57; OR 5.01, p < 0.0001) were identified risk factors for BE. AO (76 *vs* 51%; OR 3.13; p < 0.001), increased BF% (30.7 *vs* 17.6%; p = 0.001), higher BMI (overweight: 39.6 *vs* 30%; OR 2.09; p = 0.0008; obese: 32 *vs* 22%; OR 2.3; p = 0.004) and MS (33.2 *vs* 20%; OR 1.95; p = 0.0017) were significantly associated with BE. A positive trend, possibly related to the small number of female cases, was demonstrated for NWO (28.1 *vs* 19.1%; OR 1.06; p = 0.1). More cases were affected by hypertension (37.4 *vs* 21.3%; OR 2.4; p < 0.001) and hyperlipidaemia (72.8 *vs* 53.9%; OR 2.28; p < 0001) but not diabetes. When adjusted by gender, age and race into a multivariate analysis, independent risk factors for BE were BF% (OR 1.90; p = 0.01) and AO (OR 1.67; p = 0.03). Metaplasia and dysplasia were present in 57.2 and 42.8%. AO was the only metabolic parameter independently correlated with high grade dysplasia (38 *vs* 21%; OR 2.44; p = 0.001).
Conclusion Abdominal obesity, and body fat mass are strong risk factors for BE. A positive trend association was demonstrated in NWO. Furthermore, abdominal adiposity plays a role in progression to OAC. BE might therefore be considered in the metabolic syndrome spectrum and as such, in this group screening interventions may be considered.
Disclosure of Interest None Declared.

PTU-181 RELATIONSHIP BETWEEN BARRETT'S OESOPHAGUS AND ABDOMINAL ADIPOSITY OR BMI AND ACTIVATED PATHWAYS IN PROGRESSION TO CANCER

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Introduction Barrett's oesophagus (BE) remains the strongest risk factor for oesophageal adenocarcinoma (OAC). Several studies describe an association between BE and obesity through mechanical and metabolic consequences. Visceral fat is a recognised endocrine organ. Adipokines and insulin resistance impact upon obesity-related diseases and cancer pathways. Our aims were to evaluate the relationship between BE, abdominal adiposity/BMI and pathways in the progression to cancer.
Methods Height, weight, waist-hip ratio, blood pressure assessment and fasting blood samples were obtained from sequential patients (pts), undergoing gastroscopy. BMI, fasting glucose and insulin, lipids, leptin and adiponectin were measured. Pts were then classified as normal-weight, overweight or obese and the presence of abdominal obesity (AO) and/or metabolic syndrome (MS, defined by WHO criteria) documented., to evaluate the relationship between BMI and abdominal adiposity with metabolic indices and adipokines in BE compared to controls. Biopsies were obtained from BE and histological progression to cancer was correlated with metabolic indexes. Chi square, Fisher, t-Student test and logistic analysis were used for comparison.
Results 480 patients were enrolled (250 cases: F/M: 57/193; mean age: 63.7; 230 controls: F/M: 136/94; mean age: 51.9). Metabolic derangements were more common in BE compared to controls; Metabolic syndrome (33.2 *vs* 20%; OR 1.95; p = 0.0017), insulin levels (10.2 *vs* 7.2µIU/ml; p = 0.001), HbA1c (5.8 *vs* 5.1%; p < 0.01), insulin resistance (47 *vs* 27%; OR 1.54; p < 0.01), dyslipidaemia (72.8 *vs* 53.9; OR 2.3; p < 0.0001) and hypertension (37.4 *vs* 21.3%; OR 2.4; p < 0.001). MS was present in 39.7 *vs* 34.2% (OR 3.05; p < 0.001), 43.7 *vs* 21.9% (OR 5.2; p < 0.001), 92.1 *vs* 54.9% (OR 8.08; p < 0.0001), in overweight, obese, AO pts with BE and controls, respectively. Insulin resistance was present in 39.2 *vs* 33.8% (OR 1.3; p < 0.05), 38 *vs* 22.3% (OR 1.7; p < 0.01) and in 82.5 *vs* 54.5% (OR 1.5; p < 0.001) in overweight, obese and AO pts, respectively. A trend was observed for decreased adiponectin levels in BE *vs* controls while leptin levels showed no correlation. In BE pts, the presence of dysplasia was associated with MS (42 *vs* 25%; p = 0.005) and and insulin resistance (51.4 *vs* 34.0%; p = 0.005).
Conclusion BE association with insulin resistance and MS suggests activation of specific metabolic pathways in pts with abdominal obesity or BMI. Progression to cancer appears driven by metabolic dysfunction in MS and a carcinogenic insulin pathway.
Disclosure of Interest None Declared.