

(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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10.1136/gutjnl-2014-307263.254

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; $p < 0.0001$) 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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10.1136/gutjnl-2014-307263.255

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 7.2 μ IU/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 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.