**PWE-113 COELIAC SEROLOGY AT A LARGE DISTRICT GENERAL HOSPITAL, RESULTS IN 6394 PATIENTS**

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**Introduction** Coeliac disease has an estimated UK prevalence of 1% and is an important, common cause of many gastro-intestinal and non-gastro-intestinal symptoms. Coeliac serological blood tests are commonly performed in both primary and secondary care. Positive coeliac serology occurs in patients with: (i) Coeliac disease, (ii) Latent Coeliac disease, (iii) Dermatitis herpetiformis. The UK national institute for health and clinical excellence (NICE), 1 British society of gastroenterology guidelines (BSG) and American Gastroenterology Association (AGA)2 guidelines recommend that all patients with positive coeliac serology undergo duodenal biopsy since diagnosis of coeliac disease requires both positive serology and typical histological findings.

**Methods** We reviewed the results of all coeliac serology tests performed at our hospital laboratory in the 24 months. The case notes for all patients with positive results were reviewed.

**Results** 6394 endomysial antibody results were performed on adult patients between 1 October 2010 and 30 September 2011. 100 (1.6%) were positive. Of these 67 (67.0%) underwent biopsy. 50 (74.6%) had histological evidence of coeliac disease; 5 (7.5%) were inconclusive and 11 (16.4%) had no evidence of coeliac disease. Of those who did not undergo biopsy 11 (33.0%) were known to have CD or refused D2 biopsy. In 22 (21.6%) patients gastroenterological follow-up had not been arranged, of these 16 (72.7%) tests had been arranged in primary care.

**Conclusion** In this study 1.6% of those tested had serology suggestive of coeliac disease, this is marginally larger than expected by chance and suggests that testing was not appropriately targeted. Surprisingly 21.6% of positive tests did not have appropriate follow-up arranged. We suspect these findings are not confined to our institution. Our findings suggest that engagement and education of non-gastroenterology colleagues, particularly those in primary care is important in order that patients receive appropriate treatment and conform to AGA, BSG and NICE guidelines. We plan in future that all positive coeliac serology test reports be issued with the advice that referral to a gastroenterologist is recommended.

**Competing interests** None declared.

**REFERENCES**


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**PWE-114 DOES GASTROINTESTINAL INFECTION TRIGGER COELIAC DISEASE?**

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**Introduction** It is thought that a “second hit” is required to trigger coeliac disease in genetically susceptible individuals. Various infective agents have been postulated as the second hit but there is little evidence to support this. We aimed to establish the recall rate of antecedent gastrointestinal infection in patients with coeliac disease, and the prevalence of undetected coeliac disease in those with stool culture proven gastrointestinal.

**Methods** Group A comprised histologically proven patients with coeliac disease (n=233, 61 male, median 60 years) who were asked to complete a validated questionnaire and then compared to healthy controls (n=219, 79 male, median 46 years), and controls with inflammatory bowel disease (IBD) (n=196, 124 males, median 56 years). Group B were patients with stool culture proven gastroenteritis (n=101, 48 males, median 57 years) who underwent serologic testing for coeliac disease (endomyosal antibody [EMA], tissue transglutaminase [tTG], immunoglobulin A [IgA]). They were compared with healthy controls (n=1200, 447 male, median 46 years). Those with positive serology underwent endoscopy and duodenal biopsy.

**Results** In Group A 69/233 (29.6%) with coeliac disease, and 53/196 (27.1%) with IBD reported having a gastrointestinal infection within the 12 months prior to diagnosis. In both diseases this was significantly greater than in healthy controls 15/219 (6.8%) (p<0.0001). In Group B 94/101 (95%) were antibody negative. The demographics, serology and biopsy results of the seven stool-culture positive subjects with positive coeliac serology are shown in Abstract PWE-114 table 1. The prevalence of coeliac disease in patients with stool culture positive gastroenteritis was 2.97%. This was higher than in healthy controls (12/1200, 1%) (p=0.10). In Group B the gastroenteritis pathogen was identified as Campylobacter species in 96/101 (95.0%), Salmonella species in 4/101 (4.0%), and Shigella in 1/101 (1.0%). One participant had IgA deficiency. This individual had normal IgG titres, IgG EMA and IgG tTG.

**Abstract PWE-114 Table 1**

<table>
<thead>
<tr>
<th>Case number</th>
<th>Age</th>
<th>Sex</th>
<th>Pathogen</th>
<th>EMA</th>
<th>tTG</th>
<th>Duodenal biopsy</th>
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<td>Marsh 3c</td>
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<tr>
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<td>19</td>
<td>Marsh 0</td>
</tr>
<tr>
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<td>94</td>
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<tr>
<td>4</td>
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</tr>
<tr>
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</tr>
<tr>
<td>7</td>
<td>32</td>
<td>F</td>
<td>Campylobacter</td>
<td>0</td>
<td>169</td>
<td>Marsh 1</td>
</tr>
</tbody>
</table>

**Conclusion** Patients with coeliac disease have a recall rate of previous gastrointestinal infection similar to those with inflammatory bowel disease, and significantly greater than healthy controls. In coeliac disease gastrointestinal infection may well be the “second hit” required to trigger disease but further work is required.

**Competing interests** None declared.

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**PWE-115 A 13-YEAR, SINGLE-CENTRE EXPERIENCE OF GLUCOSE HYDROGEN BREATH TESTING**

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**Introduction** Historically, the gold standard for diagnosing small intestinal bacterial overgrowth (SIBO) has been qualitative culture of jejunal aspirate. However this test is costly and invasive. More commonly in clinical practice the glucose hydrogen breath test (GHBT) is used. We aimed to determine which clinical features and baseline laboratory investigations indicate a high likelihood of SIBO as defined by positive GHBT.

**Methods** We undertook a retrospective analysis of records for all patients referred for GHBT at a single teaching hospital over a 13-year period 1998–2010. Data collected included age, sex, baseline and peak hydrogen levels, previous surgical procedures,
comorbidities, haemoglobin levels, vitamin B₁₂, folate, ferritin and albumin levels. A positive result was a rise in hydrogen of at least 20 ppm, or methane of 12 ppm, over the baseline for each gas.

**Results** 447 patients were identified (120 male, median age 56 years, range 17–90). Overall 84/447 (18.8%) of tests were positive. The patient characteristics associated with a positive result were concurrent use of proton pump inhibitor (PPI) (p = 0.0005), previous partial gastrectomy (p < 0.0001), previous right hemicolectomy (p = 0.0004), and age over 75 years p = 0.0001. The laboratory investigations predictive of a positive result were low vitamin B₁₂ (p = 0.02) and low albumin <30 g/dl (p = 0.05).

**Conclusion** This is the largest single centre study of factors predictive of SIBO as defined by positive GBHT. Use of proton pump inhibitor, partial gastrectomy, right hemicolectomy, age over 75 years, low vitamin B₁₂ and low albumin were predictive of SIBO.

**Competing interests** None declared.

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**PWE-116 CAN THE NEW ESPGHAN DIAGNOSTIC GUIDELINES FOR COELLIAC DISEASE BE APPLIED TO ADULTS?**

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**Introduction** In adults, duodenal biopsy is mandatory for diagnosis of coeliac disease. This is usually preceded by serological tests for coeliac specific antibodies—anti-tissue transglutaminase (tTG) and endomysial antibodies (EMA). However, the recent guidelines produced by the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN)¹ in 2010 recommend that histological assessment (duodenal biopsy) might be omitted in cases of anti-tTG more than 10 times the upper limit of normal which have been verified by endomysial positivity, a typical history for coeliac disease and HLA DQ2 or DQ8 positivity. Our aim was to analyse our adult population of patients with possible diagnosis of coeliac disease to ascertain the proportion of patients with anti-tTG levels more than 10 times the upper limit of normal.

**Methods** Retrospective collection of data were obtained from the electronic clinical patient database from 2008 to 2011 at York Hospital. Inclusion criteria were all patients with positive anti-tTG and available duodenal histology results while on normal diet. York Hospital uses the Orgentec Anti-tTG IgA kit.

**Results** 113 (70%) of the 161 patients were female. The median age was 49 years (Range 16–89 years). 52 patients (32%) had anti-tTG levels greater than 10 times the upper limit of normal (ie, with a value ≥100 u/ml). All 52 had positive EMA. 51/52 had typical symptoms (chronic diarrhoea, weight loss, fatigue, anaemia) while one patient had leg weakness and a positive family history for coeliac disease. All 52 patients had biopsies consistent with coeliac disease. One patient had HLA DQ2/DQ8 status checked, which was positive. The test was done due to equivocal IgA levels, weakly positive anti-tTG and Marsh I findings.

**Conclusion** In our local adult population of patients with known positive anti-tTG and duodenal histology, about a third of the patients had anti-tTG levels greater than 10 times the upper limit of normal. This group had biopsies consistent with coeliac disease. Therefore, the ESPGHAN diagnostic guideline recommending the omission of duodenal biopsies in patients with anti-tTG levels greater than 10 times the upper limit of normal may be applicable for a significant proportion of the adult population (other similar studies² ³ have found rates of 45–58%).

**Competing interests** None declared.

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**REFERENCES**


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**PWE-117 RESPONSES TO DIETARY INTERVENTION GUIDED BY FOLLOW-UP DUODENAL BIOPSY IN COELLIAC DISEASE**

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**Introduction** British Society of Gastroenterology guidance on repeating the intestinal biopsy in coeliac patients on diet is ambiguous and suggests that dietary advice is the same irrespective of the follow-up biopsy results. Addenbrooke’s Coeliac Disease Clinic has a policy of routine re-biopsy at 9–12 months after commencing gluten withdrawal. The following graded dietary interventions are introduced if villous atrophy is present on repeat biopsy—interview and dietary history; food diary; withdrawal of oats; withdrawal of “Codex” products, barley malt extract, (“supersensitive diet”—SSD); liquid diet. Response is assessed by re-biopsy as we have shown serology to be unreliable in this setting.

**Methods** Information was retrieved from the Addenbrooke’s Adult Coeliac Disease database of over 600 patients with coeliac disease (30% male, 70% female, average age at diagnosis 48 yrs and 44 yrs respectively). 170 patients with persisting villous atrophy on follow-up biopsy were identified. Dietetic interventions and outcomes based on subsequent biopsy results were reported.

**Results** Of 170 patients with persisting villous atrophy, 84 did not undergo re-biopsy after dietitian intervention and were therefore excluded from further analysis. In 57 patients interview or food diary analysis revealed a likely source of gluten ingestion as the cause of persistent villous atrophy and advice was given to eliminate the likely source (Dietary Advice—DA). In 29 patients, no potential gluten source was identified and a “supersensitive diet” was recommended (SSD). Further biopsy revealed complete normalisation of the duodenal mucosa (Marsh 0) in 14 (24.5%) of the DA group and 8 (27.5%) of the SSD group. Normal or minor changes (Marsh 0, 1, or 2) were seen in 31 (54%) of the DA group and 18 (62%) of the SSD group. Intensive dietary intervention revealed two additional patients who concealed deliberate gluten ingestion. 11 patients who remained persistently seronegative, had no identifiable source of gluten and showed no response to SSD were deemed to be histologically refractory and assigned to careful clinical follow-up.

**Conclusion** Contrary to current BSG guidance, dietary advice is not irrespective of the outcome of the follow-up biopsy on gluten free diet. Dietitian intervention is effective in over half of the patients who showed persistent villous atrophy despite following a gluten free diet. This strategy also identifies patients who are histologically refractory and at high risk of subsequent complications. These results strongly support a policy of assessment by follow-up biopsy and appropriate specialist dietitian intervention in the management of coeliac disease.

**Competing interests** None declared.