

1.90 and 1.95 umol/l respectively,  $p > 0.05$ ). There was no significant correlation of vitamin A with FGF19 or SeHCAT retention as a whole or within the subgroups. There were no significant differences in serum 25OH-vitamin D between the groups (38, 47, 46 nmol/l,  $p > 0.05$ ) There was no correlation of 25OH-vitamin D with FGF19 or SeHCAT retention as a whole, or within the subgroups.

**Conclusion** As vitamins A and D are known to induce FGF15 expression in mice, it was important to establish whether these vitamins could be associated with the abnormal FGF19 levels seen in human BAD. These results do not provide support to the possibility that deficiency of these vitamins is directly involved in the pathogenesis of BAD.

**Competing interests** None declared.

**PTU-158 COELIAC DISEASE INVESTIGATION AND FOLLOW-UP**

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**Introduction** Coeliac disease (CD) is an abnormal immune response to gluten affecting 1% of the UK population, resulting in small bowel villous atrophy, malabsorption and GI symptoms. A large number of patients are tested for CD and often repeat requests are made. This retrospective analysis looked at the prevalence of patients with positive CD serology in relation to subsequent gastroenterology referrals and/or small bowel biopsy.

**Methods** The data were collected from patients who had undergone serological CD testing. All patients with a positive anti-tissue transglutaminase, or a positive endomysial antibody test were recorded and notes analysed for referral or endoscopy. We excluded patients under 18 years of age and those with known CD. The data were obtained from the laboratory database at Brighton and Sussex University Hospitals over a period of 1 year.

**Results** We identified 7369 CD serology requests. The referral source for CD serology was a general practitioner, hospital specialist, gastrointestinal surgeon or gastroenterologist. Overall, 169 (2.3%) individuals had a positive result. From the total number of requests, 6.75% (498) were repeat requests. Of these, 8.23% (41) had a second positive test. 13.86% (69) of the patients who had a repeat serology done by a general practitioner or other hospital clinician were referred to gastroenterology. The overall prevalence of CD was 2.27% (167), of which 58% (97) were referred for a small bowel biopsy. Small bowel histology confirmed CD in 52% of the biopsy samples, that is, almost one third of the total number of cases with positive serology.

**Conclusion** Positive CD serology was recorded in 2.3% of the study group. Although small bowel histology is the gold standard in the diagnosis of CD,<sup>1</sup> this was performed on 58% of the patients with positive serology. It is apparent that not all cases with positive serology are being referred to a specialist. We recommend that

Abstract PTU-158 Table 1 Source of coeliac serology referral

	Total serology requests (%)	Positive serology result (%)	Negative serology result (%)
GP	4502 (61.1%)	82/4502 (1.8%)	4420/4502 (98.2%)
Other hospital specialist	1716 (23.3%)	41/1716 (2.4%)	1675/1716 (97.6%)
GI surgeon	102 (1.4%)	3/102 (2.9%)	99/102 (97.1%)
Gastroenterology	1049 (14.2%)	41/1049 (3.9%)	1008/1049 (96.1%)

patients with suspected or incidental diagnosis of CD should be referred to a gastroenterologist for assessment, confirmation of the diagnosis with small bowel biopsy and advice on gluten-free diet.

**Competing interests** None declared.

**REFERENCES**

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**PTU-159 SELF REPORTING OF GLUTEN SENSITIVE GI SYMPTOMS IN PRIMARY CARE: SHOULD WE ACCEPT THE DIAGNOSIS OF COELIAC DISEASE WITHOUT FURTHER INVESTIGATIONS?**

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**Introduction** Many unselected patients presenting to gastrointestinal (GI) clinics self-report that they have symptoms related to the ingestion of gluten. For this reason we undertook a prospective and systematic evaluation of this group of patients to ascertain the subsequent diagnostic yield.

**Methods** All patients were referred to a dedicated coeliac or gluten sensitivity clinic by GPs through a choose and book system. The referral criteria were "GI symptoms attributed to gluten ingestion." Investigations included baseline haematology, biochemistry, haematinics, C reactive protein and HLA status for the DQ2/DQ8 haplotypes. In addition, coeliac serology was performed: endomysial antibody (EMA), tissue transglutaminase antibody (tTG), immunoglobulins, as well as duodenal biopsies on a gluten containing diet. A diagnosis of coeliac disease was based on either the presence of villous atrophy or in cases with lesser degrees of the modified Marsh grading, an associated positive coeliac serology and/or HLA typing.

**Results** 140 patients were investigated over a 5-year period. 80% were women and the median age of presentation was 37 yrs (range 16–88). In patients self-reporting symptoms related to the ingestion of gluten, the diagnosis of coeliac disease was reached in 10% (n 14). 85% (n 119) did not have coeliac disease but fulfilled the ROME criteria for irritable bowel syndrome (IBS). These patients were given a clinical diagnosis of gluten sensitive IBS (GS-IBS). Importantly, organic pathology was found in 5% (n 7) all of whom had additional alarm symptoms—mesenteric ischaemia, bacterial overgrowth, lactose intolerance, bile salt malabsorption, lymphocytic colitis, ulcerative colitis and pyloric stricture. A positive coeliac serology ( $p < 0.0001$ , exact fisher test) was significantly associated with coeliac disease. All patients with coeliac disease were HLA positive compared to 44% of GS-IBS cases. There was statistically no significant difference in gender, clinical symptoms or baseline bloods (haemoglobin, vitamin B<sub>12</sub>, folate, ferritin, calcium or albumin) between the groups, ( $p > 0.05$ ).

**Conclusion** Self-reporting of gluten related GI symptoms only results in a diagnosis of coeliac disease in 10% of cases. The majority of patients do not have overt coeliac disease but may belong to the spectrum of GS-IBS, a relatively new and under researched concept. Moreover, 5% had significant underlying disease. An empirical trial of gluten-free diet prior to referral may be unhelpful and delay the diagnosis in this group of patients. This data suggests that patients who self-report gluten induced GI symptoms should be considered for further investigations.

**Competing interests** None declared.