millions (ppm) using a QuinTron BreathTracker DP at baseline, then—after 75 g oral glucose—at 20 min time points up to 3 h or until positive. Positive test: fasting H2 ≥20 or CH4 ≥10 ppm or a rise in H2 ≥12 or CH4 ≥6 ppm. Some patients also had duodenal aspirates collected endoscopically. Positive result: >10^4 colony forming units/ml. Patients with positive tests were treated with antibiotics. Results were assessed retrospectively.

**Results**

126 patients, 66 males and 60 females, median group age 61 years (range 35–86), treated for gynaecological (30%), upper GI (25%), lower GI (9%), urological (23%), other cancers (10%)—myeloma, ependymoma, bronchial, breast and lymphoma—were re-biopsied within 12 months, 311 (85%) within 24 months. The Gut clinic are entered onto a database. Patients undergo repeat duodenal aspiration and biopsies when needed. Emerging evidence reports worse outcomes with lack of mucosal recovery, suggesting histological normalisation as the aim of treatment. However, few centres routinely re-biopsy and many clinicians depend on serology or symptoms for follow-up. This retrospective study evaluates the role of the follow-up biopsy in assessing response to treatment, and that the proportion of patients with positive serology at first biopsy, only 43% with VA on follow-up biopsy were positive for anti-TTG antibodies. Conversely, 13% without VA on follow-up had positive serology. The sensitivity, positive predictive and negative predictive values for serology compared to biopsy at follow-up are 43.4%, 68.3% and 70.5% respectively.

**Conclusion**

Response to diet in CD might be monitored by symptoms, serology or histology. Many patients diagnosed in adulthood report minimal or no symptoms at diagnosis and this is therefore not a credible marker of response. Here we show that:

- Anti-TTG antibody serum has a sensitivity of 87% at diagnosis.
- Anti-TTG serum has a poor sensitivity (43%) on follow-up and is therefore unreliable.
- Histological response rates are not increased after 1 year on diet.
- Approximately 28% of patients achieve normal duodenal mucosa, 58% show no or minimal signs of coeliac disease on follow-up biopsy.

We suggest that follow-up biopsy on diet is the only reliable form of assessing response to treatment, and that the proportion of patients achieving histological response could be used as a means of auditing quality of care in coeliac clinics.

**Competing interests**

None declared.

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**PTU-156**

**DIETARY RESPONSE IN COELIAC DISEASE SHOULD BE ASSESSED BY REPEAT DUODENAL BIOPSY**

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**Introduction**

Therapeutic goals in coeliac disease (CD) are poorly defined. Emerging evidence reports worse outcomes with lack of mucosal recovery, suggesting histological normalisation as the aim of treatment. However, few centres routinely re-biopsy and many clinicians depend on serology or symptoms for follow-up. This retrospective study evaluates the role of the follow-up biopsy compared to serology in treated coeliacs and is the largest study of paired biopsies reported.

**Methods**

Details of patients attending the Addenbrookes Adult CD Clinic are entered onto a database. Patients undergo repeat duodenal biopsy 9–12 months after commencing dietary therapy. Serum Anti-TTG antibodies and total IgA are measured at clinic visits. From the database of around 600 adult patients (30% male, 70% female, average age at diagnosis 40 and 44 yrs respectively) follow-up biopsy results were available for 433 patients and compared using a paired t-test.

**Results**

Paired initial and follow-up biopsies were available for 368/433 patients (no information about index biopsy in 65). 239 (65%) were re-biopsied within 12 months, 311 (85%) within 24 months. The proportion achieving normalisation of duodenal mucosa (Marsh 0) at 12 months, 12–24 months or after 24 months is 27%, 25% (p=0.61) and 25% (p=0.1) respectively. Those without villous atrophy (VA) (Marsh 0, 1 or 2) is 58%, 58% (p=0.98) and 52% (p=0.24) respectively. Excluding the 2.5% of patients with IgA deficiency, 13% of patients had negative anti-TTG antibody titres at the time of diagnostic biopsy. In the group of patients with positive serology at first biopsy, only 45% with VA on follow-up biopsy were positive for anti-TTG antibodies. Conversely, 13% without VA on follow-up had positive serology. The sensitivity, positive predictive and negative predictive values for serology compared to biopsy at follow-up are 43.4%, 68.3% and 70.5% respectively.

**Conclusion**

None declared.

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**PTU-157**

**A PROSPECTIVE STUDY OF SERUM VITAMIN A AND D LEVELS IN PRIMARY BILE ACID DIARRHOEA**

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**Introduction**

There is evidence that primary bile acid diarrhoea (BAD) is caused by disruption of the homeostatic mechanism controlling bile acid synthesis, with an overproduction of bile acids leading to chronic watery diarrhoea. Fibroblast Growth Factor 19 (FGF19) has been shown to be central to this homeostatic mechanism, providing negative feedback on CYP7A1, the rate limiting enzyme in bile acid synthesis. Median serum FGF19 levels have been shown to be lower in BAD patients than in diarrhoea controls. In mouse models, it has also been shown that expression of FGF15, the orthologue of FGF19, is induced by the active forms of vitamins A and D. The aim of this study was to investigate a possible causal relationship between low serum levels of vitamins A and D and BAD.

**Methods**

Patients with chronic diarrhoea were recruited prospectively. All patients underwent routine testing to exclude other causes of diarrhoea and had SeHCAT tests. Patients were classified as having primary BAD, secondary BAD, or unexplained chronic diarrhoea (CD). Serum 25OH-vitamin D was measured by LC-MS using standard procedures. Serum Vitamin A was calculated by HPLC. Results are expressed as medians. Mann–Whitney and Spearman rank correlation tests were used in analyses.

**Results**

Serum vitamin A levels were available in 145 subjects (primary BAD 46; secondary 29; CD 70). Serum vitamin D levels were available in 150 subjects (primary BAD 50; secondary 29; CD 71). There were no significant differences in the level of vitamin A between the groups with primary or secondary CD (2.10,