

therapy had no change in their daily bowel frequency (7.0 vs 5.35, $p = 0.40$). The main reason for discontinuing medical therapy in this group was poor tolerability of the prescribed bile acid sequestrant (Colestyramine/ Colestipol). Crohn's disease was the only alternative diagnosis established in 10% (2/20), accounting for potential persisting symptoms.

Conclusion This is the first longitudinal study to assess patients with BAM and identifies factors predictive of this condition. Our findings suggest BAM is a chronic condition, which best improves with bile acid sequestrants. Given the problems with tolerability of older bile acid sequestrants, consideration should be given to Colesevalam, which may have a better tolerability profile.

REFERENCE

- 1 Kurien M, et al. Bile acid malabsorption: An under-investigated differential diagnosis in patients presenting with diarrhea predominant irritable bowel syndrome type symptoms. *Scand J Gastroenterol* 2011 Jul;46(7-8):818–22

Disclosure of Interest None Declared.

PTH-111 "NON CELIAC GLUTEN SENSITIVITY" (NCGS) IS UNCOMMON IN PATIENTS SPONTANEOUSLY ADHERING TO GLUTEN FREE DIET (GFD), AND IS OUTNUMBERED BY "FODMAPS SENSITIVITY"

B Zanini*, R Baschè, A Ferraresi, F Lanzarotto, M Marullo, C Ricci, A Lanzini. Dept Clinical and Experimental Sciences, University of Brescia, Brescia, Italy

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Introduction It is controversial whether symptoms in patients fulfilling the clinical criteria for NCGS¹ are specifically triggered by gluten or by cereal components other than gluten and specifically FODMAPs, or are attributable to a placebo effect². Our aim was to test assess gluten or FODMAPs dependence of symptoms in patients diagnosed as NCGS.

Methods NCGS patients referred to our Clinic were randomised to a double blind cross over study involving challenge with 10 g gluten Vs 10 g gluten free flour containing FODMAPs for 10 days each with 2 weeks wash-out in between (challenge stage). Patients were subsequently kept on a low FODMAPs diet for 8 weeks (low FODMAPs stage) Endpoints: patients were asked to indicate by symptom recurrence the gluten phase of challenge; correct identification was taken to indicate NCGS and incorrect identification accompanied by reduction of GSRS score during the low FODMAPs diet were taken to indicate FODMAPs sensitivity.

Results Twenty-five patients without celiac disease (age 42+9 years, M/F = 2/23, 10 HLA DQ2/8 positive, 13 negative, 2 unknown) on strict GFD entered the study. During the challenge stage, the gluten phase was correctly identified by 8 patients thus fulfilling criteria for NCGS (4 with HLA DQ2/8). Scores for the 3 dimensions of GSRS (pain $p = 0.03$; indigestion $p = 0.02$; and diarrhoea $p = 0.02$) were higher in NCGS patients during the gluten than gluten free flour challenge. Twelve patients thought they were challenged gluten while on gluten free flour indicating gluten independent symptom recurrence (gluten insensitive). GSRS scores in these patients were higher (pain $p = 0.004$; reflux $p = 0.013$; indigestion $p = 0.014$, constipation $p = 0.014$) during challenge with gluten free flour than with gluten. Five patients reported mild symptoms during both phases suggesting a placebo effect. During the low FODMAPs stage the score of indigestion dimension (comprising borborygmus, bloating, eructation, flatus) was significantly reduced ($p = 0.011$) in the gluten insensitive patients suggesting

FODMAPs sensitivity. There was no significant change in the 5 dimensions of the GSRS in NCGS patients.

Conclusion We conclude that the population of patients reporting intolerance to gluten containing diet is a mixed population of NCGS and of FODMAPs sensitive patients. NCGS is uncommon and is outnumbered by FODMAPs sensitivity in patients spontaneously adhering to GFD. Distinction between these 2 conditions is clinically relevant in relation to dietary counselling.

REFERENCES

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- 2 Gibson and Muir. *Gastroenterology* 2013;145: 693

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PTH-112 A SINGLE CENTRE EXPERIENCE OF TREATMENT OF REFRACTORY CELIAC DISEASE TYPE 2

^{1,1}I Nasr* ¹S Donnelly, ²C Ho-Yen, ³T Mitchell, ²F Chang, ¹P Ciclitira. ¹Gastroenterology, Guy's and St Thomas' NHS Foundation Trust, London, UK; ²Pathology, Guy's and St Thomas' NHS Foundation Trust, London, UK; ³Molecular Diagnostic Services, Guy's and St Thomas' NHS Foundation Trust, London, UK

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Introduction Refractory celiac disease (RCD) is a persistent malabsorption and villous atrophy despite adhering to a strict gluten-free diet (GFD) for at least 6–12 months in the absence of other cause¹. It is a rare complication of celiac disease (CD). RCD is classified based on the T-cells in the intraepithelial lymphocyte (IEL) morphology into type 1 with normal IEL and type 2 with aberrant IEL. RCD1 is managed with strict nutritional and pharmacological management. RCD2 can be complicated by ulcerative jejunitis or enteropathy associated lymphoma (EATL), the latter having a 5-year mortality of 8–20%. It is therefore necessary to investigate and manage RCD2 which has a less predicted response and has a poor prognosis due to the associated complications. Treatment options vary due to the low incidence of RCD2 and hence the small numbers of randomised control trials.

We present a single centre's experience in the treatment of RCD2.

Methods We performed a single centre retrospective study of all cases of RCD2 using the celiac database in a single centre between 2000 and 2013. Case notes, biological and histological data were reviewed for patients with a diagnosis of RCD2 diagnosed between 2000 and 2013. All patients were treated with prednisolone, 20 mg, and azathioprine, 2 mg/kg/day with repeat small bowel biopsy and T cell receptor analysis by PCR at 4 monthly intervals.

Results Fourteen out of twenty patients with RCD2 were successfully treated with prednisolone and azathioprine to become either type 1 refractory celiac disease, in 12 patients, or celiac disease, in 2 patients, with a better 5-year survival. None of the type 2 refractory patients developed lymphoma on this treatment.

Conclusion Prednisolone combined with azathioprine can be used successfully to treat RCD2. Our experience shows it is a safe and successful approach to improve prognosis. We successfully treated 7 out of 10 patients with RCD2 with this regimen.

REFERENCE

- 1 Alberto Rubio-Tapia, Joseph A Murray. Classification and Management of Refractory Celiac Disease. *Gut* 2010 April; 59(4):547–557

Disclosure of Interest None Declared.