responses, and research is still far from developing cost-effective and feasible approaches to predict these responses.

Methods Using peripheral blood samples and colonic biopsies, we aim to stratify CD patients into different immunopathotypes. We quantified frequencies of leukocyte populations and cytokines in peripheral blood of patients and healthy controls, using flow cytometry analysis and ELISAs. Cytokine and leukocyte population levels were correlated using Sparse PLS Discriminant Analysis. Additionally, gene expression data was generated by RNAseq from colonic biopsies to identify genes that were differentially expressed between active and remission CD samples, and healthy controls.

Results Our initial analyses have identified 3 distinct clusters of CD patients based on expression patterns of peripheral blood mononuclear cells (PBMCs) and cytokines. Patient clusters were found to either upregulate pro-inflammatory cytokine only (Cluster A), upregulate pro-inflammatory cytokines with altered frequencies of leukocyte populations (Cluster C) or solely display altered leukocyte frequencies (Cluster C). In order to further understand potential distinct disease mechanisms, significantly differentially expressed genes from the RNAseq data were analysed identifying genes that varied in expression in the CD cohort, resulting in 13 targets. These gene targets are currently validated in additional CD colonic biopsies using qPCR. Together with clinical information about treatment response and disease severity, these data will be used to investigate the capacity of the blood phenotyping and biopsy gene expression signatures to act as biomarkers to predict treatment responses in CD.

Conclusions Based on our results it can be suggested that CD patients have different immunopathotypes, which might be associated with distinct treatment responses. Our ongoing work will assess these potential associations. Should strong associations be identified, their prognostic value will be assessed. Our eventual aim is to help ensure that CD patients receives the most appropriate treatment soon after diagnosis. This is likely to be crucial in limiting damage caused by ongoing inflammation.

PTH-076 SWITCHING FROM ORIGINATOR INFlixIMAB TO CT-P13: A UK SINGLE CENTRE EXPERIENCE

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10.1136/gutjnl-2019-BSGAbstracts.135

Introduction and Aims The infliximab biosimilar (CT-P13) received market authorization for inflammatory bowel disease in late 2016 with the aim of reducing cost and increasing access to therapy. The prospect of 'switching' patients from originator to CT-P13 has concerned clinicians. We present an experience of 'switching' from originator infliximab (IFX-O) to CT-P13 and present efficacy, safety and immunogenicity data from our cohort.

Methods We performed a retrospective review of patients switched from IFX-O to CT-P13 at our center. Disease demographics, clinical course and outcomes were analysed from electronic case records at 8 months and at last follow-up at 13 months.

Results Ninety-six patients (35 female) were 'switched' from IFX-O to CT-P13. Of these 44 had Ulcerative colitis (UC)

and 52 had Crohn's disease (CD) with a mean age at diagnosis of

34.7 years (median = 33, IQR = 24.5). Montreal phenotype for UC was Proctitis (E1) = 1, Left sided (E2) = 16, Pancolitis (E3) = 27 and for CD (L1 = 10, L2 = 12, L3 = 29, L4 = 1) and (B1 = 27, B2 = 14, B3 = 11), 9 patients had perianal disease.

Mean duration of IFX-O treatment before switching was 49.8 months (median = 44, IQR = 52) and on CT-P13 11.5 months (median 13). At switch, 76 patients had a normal CRP (UC = 33, CD = 43), and in 15 patients it was elevated (UC = 10, CD = 5).

At 8 months, 72 patients (UC = 34, CD = 38) were in clinical remission (pMayo < 2 and HBI < 5) and 80 patients remained in biochemical remission (UC = 35, CD = 45). In 14 patients (UC = 8, CD = 6) CRP increased. Of 51 patients (UC = 21, CD = 30) undergoing endoscopic assessment, 31 achieved mucosal healing (UC = 13, CD = 18).

At 13 months 69 patients remained on CT-P13. 28% discontinued the drug due to immunogenicity (n=10), loss of response (n=5), surgery (n=5), remission (n=5), side effects (n=2) and 1 patient died of hospital acquired pneumonia.

39 out of 96 patients had therapeutic drug levels checked within a median of 13 months from switch. 27 had sub-therapeutic levels (below 4ug/ml), 11 of which were switched to another biologic, 5 referred for surgery, 4 had dose escalated to 10 mg/kg, 5 continued CT-P13 (4 with no antibodies seen and 1 with antibodies of 127), one had immunomodulator added and another stopped CT-P13 being in remission. Antibodies to Infliximab were seen in 15 of 39 patients (38.5%), of whom 8 were switched to an alternative biologic, 2 had dose escalation (10 mg/kg IFX), 4 patients stopped IFX with no other intervention and 1 person continued treatment with low antibody titre of 6.

Discussion Biosimilar IFX (CT-P13) was well tolerated. Clinical efficacy and loss of response rates with CT-P13 appears to be similar to IFX-O. This holds promise for a wider adoption of 'switching' to fulfil the purported aims of wider access to treatment at a lower cost.

PTH-077 DE-ESCALATING THERAPY IN PATIENTS WITH CROHN'S DISEASE RECEIVING ADALIMUMAB: SUBGROUP ANALYSIS OF THE CALM STUDY

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10.1136/gutjnl-2019-BSGAbstracts.136

Introduction This analysis evaluated the impact of de-escalating therapy on mucosal healing 48 weeks (wks) after randomization in patients (pts) with Crohn's disease (CD) in the CALM study.

Methods Pts with moderate-to-severe CD naïve to immunomodulators and biologics were randomized 1:1 to a tight control group (TCG) or clinical management group (CMG) after 8 wks of prednisone therapy. Treatment was escalated from no

treatment to 40 mg adalimumab (ADA) every other wk (EOW) to 40 mg ADA every wk (EW) to 40 mg ADA EW +2.5 mg/kg azathioprine (AZA)/day at 12, 24, and 36 wks after randomization based on failure criteria (CD Activity Index [CDAI]=200 or decrease <70 points [1 wk pre-randomisation] or <100 points versus baseline [at wk 11, 23, 35] and prednisone use for the CMG; CDAI=150, C-reactive protein=5 mg/L, faecal calprotectin=250µg/g, and prednisone use for the TCG). At 24 and 36 wks, if failure criteria were not met, patients de-escalated ADA EW dose to 40 mg ADA EOW. Pts re-escalated if failure criteria were met at the next visit. The primary endpoint (mucosal healing [CD Endoscopic Index of Severity <4] and no deep ulcers at 48 wks post-randomisation) was evaluated in pts who de-escalated treatment. Analyses were performed in pts who completed the study and did not move to rescue therapy. Non-responder imputation was used for missing data.

Results 15 pts in the CMG and 31 pts in the TCG de-escalated treatment during the study. Of those, 2 pts in the CMG and 8 pts in the TCG re-escalated to 40 mg ADA EW. Overall, 54% of pts in the CMG and 61% of pts in the TCG who de-escalated to 40 mg ADA EOW ± AZA achieved the primary endpoint (table 1). Of the pts who re-escalated to ADA EW, 0% in the CMG, and 75% in the TCG achieved the primary endpoint (table 1). The overall adverse event rates have been previously reported (Colombel, 2018)

Conclusions Our data suggest that repeated dose optimization based on tight control is a more refined approach resulting in mucosal healing compared with clinical management. Larger data sets are needed to confirm our observation on repeated dose optimization.

Abstract PTH-077 Table 1 Proportion of patients who reached the primary endpoint who de-escalated and/or re-escalated treatment

	CM n/N (%)	TC n/N (%)
De-escalated and remain de-escalated		
40 mg ADA EW to ADA EOW	7/13 (53.8)	7/13 (53.8)
40 mg ADA EW +2.5 mg/kg AZA/day to ADA EOW +2.5 mg/kg AZA/day	0	7/10 (70.0)
Re-escalated		
40 mg ADA EW to ADA EOW to ADA EW	0/2	6/8 (75.0)

REFERENCE

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PTH-078 VEDOLIZUMAB POST RESECTION FOR CROHN'S DISEASE: A 4 YEAR REAL WORLD EXPERIENCE

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10.1136/gutjnl-2019-BSGAbstracts.137

Introduction Vedolizumab is a gut specific anti integrin biologic therapy that NICE guidance indicates can only be used only in moderate to severe Crohns disease if an anti-TNF has failed, cannot be tolerated or is contraindicated and should be given as a planned course of treatment until it stops working or surgery is needed. There is no documentation regarding vedolizumab post resectional surgery. This audit utilises 4 years of data of using vedolizumab in a tertiary centre and assesses response rates, the use of vedolizumab post surgery

and observations regarding TNF naïve and patients with a history of prior cancer on vedolizumab.

Methods Utilising the vedolizumab database from the IBD department, the last 4 years of data was analysed, specifically identifying those who had resectional surgery with vedolizumab post surgery for prevention of recurrence or confirmed anastomotic recurrence. Data included indications for vedolizumab and Rutgeerts scoring at the anastomosis for those who had TI surgery.

Results 81 patients' data were on the vedolizumab database and their clinical notes were audited by the IBD team. Clinical remission was achieved in 49% of patients in our cohort, with the other 51% having a loss of response, side effects or primary non responders.

37% of those whose Crohns was in remission post vedolizumab were anti-TNF naïve compared to 12% of anti-TNF naïve patients in whom vedolizumab failed.

21 out of 81 patients had surgery for Crohns disease. Clinical remission was achieved in 66% (n=14) of patients started vedolizumab post resectional surgery due to being high risk of recurrence, or a Rutgeerts score of i2 or greater. This is compared to 33% of patients (n=7) who failed vedolizumab treatment post surgery. There was no statistical difference between Rutgeerts scoring (when appropriate) in those who went into clinical remission and those who did not.

On a further observational note; indications for vedolizumab differed between the patients in remission and those who had failed vedolizumab. 40% of the patients in remission had either cancer or a contraindication to an anti-TNF e.g. cardiac failure, or severe opportunistic infection on infliximab (PCP or mycobacterial infection). Only 9% of patients who failed vedolizumab treatment had cancer.

Conclusions Over 4 years, we achieved clinical remission in 49% of patients with Crohns disease using vedolizumab. 34% of these patients had had resectional surgery for their Crohns disease and were in remission. This real world data indicates that vedolizumab is a useful biological therapy in maintaining remission in patients post resectional Crohns surgery. The data also suggest, in concordance with the current literature, that being anti-TNF naïve affords a better prognosis with vedolizumab, than having failed treatment with an anti-TNF prior to vedolizumab.

PTH-079 IBD VS IBS REFERRAL PATHWAY : OUTCOMES FROM THE IBD NURSE LED RAPID ACCESS CLINIC

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10.1136/gutjnl-2019-BSGAbstracts.138

Introduction The role of the IBD nurse in reviewing suspected new patients is evolving with the onset of more rapid access clinics. The purpose of the study was to evaluate how reliable the use of a specific pathway from primary care was in identifying IBD patients vs IBS using the IBD nurse to reduce the referral to diagnosis time.

Methods A retrospective examination of data from a nurse-led rapid access clinic was used to determine if the use of an IBD vs IBS pathway was successful in reducing referral to treatment times but also to highlight if improvements to the service enabled a more satisfying patient experience?

Over an 18 month period, 400 patients (M=140, F=260) between the ages of 16–45 were reviewed in the rapid access