Conclusions High rates of sustained clinical response were observed to occur following a BI switch supported by the use of pre and post switch TDM. TDM dose escalation resulted in a statistically significant increase in TLs, this may account for the rates of continued clinical response

REFERENCE

1. Papamichael K. *Frontline Gastroenterol.* 2016 Oct;7(4):289–300.

PTH-134 EXPERIENCES OF USING VEDOLIZUMAB IN THE TREATMENT OF INFLAMMATORY BOWEL DISEASE IN THE EAST MIDLANDS

^{1,2,3}JR White*, ³S Din, ⁴R Ingram, ⁴S Foley, ⁵M Alam, ⁵R Robinson, ¹R Francis, ¹E Tucker, ⁶M Jalal, ⁶D Elphick, ⁷E Atallah, ⁷A Norman, ⁸M Amin, ⁸A Sajjad, ⁹N Heggs, ⁹S Meadowcroft, ^{1,2}GW Moran. ¹Nottingham Digestive Diseases Centre, University of Nottingham, UK; ²NIHR BRC at Nottingham University Hospitals and University of Nottingham, UK; ³Royal Derby Hospital, UK; ⁴Kings Mill Hospital, Mansfield, UK; ⁵Leicester General Hospital, UK; ⁶Chesterfield Royal Hospital, UK; ⁷Lincoln County Hospital, UK; ⁸Kettering General Hospital, UK; ⁹Takeda UK Ltd, High Wycombe, UK

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Introduction Randomised controlled trials have demonstrated efficacy of vedolizumab in Ulcerative Colitis (UC) and Crohn's Disease (CD). Further data in the real-world setting is needed to inform future practice.

Methods A multicentre retrospective observational chart review study was conducted with all pts initiated on vedolizumab across 7 UK hospitals between 1/11/14–30/11/16. The Health Research Authority approved the protocol (19/HRA/0008). Clinical disease activity was assessed at baseline, week 14, 30 & 52 using the Harvey Bradshaw Index (HBI) and partial Mayo Score (pMS). Clinical remission was defined as HBI≤4 or pMS <2 with a combined stool frequency and rectal bleeding subscore of ≤1. Clinical response was defined as ≥2-point decrease from baseline in pMS and ≥3-point decrease from baseline in HBI. The primary objective was to describe corticosteroid-free and clinical remission. Secondary objectives included effect on disease activity scores, biochemical markers, concomitant drug use, mucosal healing, hospital admissions and adverse effects.

Results 192 patients were included: 100 CD, 87 UC and 5 IBD unclassified (grouped with CD in this analysis). 46% of UC and 10% of CD patients were anti-TNF naïve. Median age was 44 (range 18–79) years; 49% male and median BMI was 25.7 (IQR 22.5–31.4). Exposure time was 38.0 (23.7–56.7) for UC and 30.9 (21.3–49.6) weeks for CD. Corticosteroid-free remission rates for UC and CD were 46% and 45%, while clinical remission rates were 52% and 44% respectively. Clinical response rates for UC was 49% and CD was 53%. The median time to corticosteroid free remission for UC and CD was 17.6 (8.7–29.6) and 14.1 (6.0–21.7) weeks and clinical remission was 15.1 (7.4–24.9) and 10.1 (3.1–21.0) weeks respectively. Time to clinical response for UC was 9.4 (5.7–15.4) and CD was 9.5 (6.1–18.2) weeks. Disease activity decreased from baseline at 14 weeks: pMS 5 (4–6) vs 3 (1–5) p=0.025 and 30 weeks pMS 5 (4–6) vs 2(1–5.5) p=0.032. Concomitant corticosteroid and immunomodulator use decreased in UC (48% vs 15% and 41% vs 18%) and CD (27% vs 10% and 26% vs 7%), respectively. The overall rate of IBD-related hospital admissions per patient per year was 1.3 (0–18.1). Adverse events were reported in 5.2% of patients.

Conclusions Results in our predominately anti-TNF experienced vedolizumab cohort mirror other published real-world data and demonstrate good clinical effectiveness and safety profile.

PTH-135 AN INFLAMMATORY BOWEL DISEASE-SPECIFIC NUTRITION SCREENING TOOL (IBD-NST) FOR BETTER OUTPATIENT CARE

¹Catherine Wall, ^{1,2}Bridgette Wilson*, ³Jeremy Sanderson, ^{1,2}Miranda Lomer. ¹King's College London, Department of Nutritional Sciences, London, UK; ²Guy's and St Thomas' NHS Foundation Trust, London, UK; ³Guy's and St Thomas' NHS Foundation Trust, Department of Gastroenterology, London, UK

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Background Patients with inflammatory bowel disease (IBD) can have hidden nutrition risks that generic malnutrition screening tools, which place too much emphasis on body mass index (BMI), are not able to identify, therefore some IBD patients that would benefit from dietary interventions are missed. By identifying nutrition risk using an IBD-specific screening tool, early detection of nutrition risk may be able to prevent development of malnutrition and improve clinical outcomes.

Aim This study aimed to develop and test an IBD-specific nutrition self-screening tool (IBD-NST).

Methods IBD patients were recruited prospectively to independently complete IBD-NST and Malnutrition Universal Screening Tool (MUST). Subjective global assessment (SGA) and hand grip strength (HGS) were completed by a dietitian. The IBD-NST scored nutrition risk as low (0), moderate (1) or high (≥2), including questions on BMI, unintentional weight loss, and a combination of active disease and nutrition concerns. Scores were compared to sub-optimal (15% below the mean) HGS, mid-upper arm muscle circumference (MAMC), BMI, weight loss >5% and SGA. Chi² compared dichotomous outcomes and Receiver Operator Characteristic curves were used for prediction assessment with a cut off of AUC<0.7 for poor prediction.

Abstract PTH-135 Table 1 Patients identified at nutrition risk by IBD-NST and MUST

		MUST			
		Low	Moderate	High	Total
IBD-NST	Low	60	4	4	68 (67%)
	Moderate	0	9	0	9 (9%)
	High	12	1	11	24 (24%)
	Total	72 (71%)	14 (14%)	15 (15%)	101

Results 101/116 patients (87%) were recruited, 54 (53%) were female, 61 (60%) had CD, 33 (33%) had UC and 7 (7%) had IBD-U. Mean (SD) age was 40 (14) years and BMI was 24.6 kg/m² (4.3). SGA identified 11/91 (12%) with malnutrition and IBD-NST and MUST identified a similar number of patients at nutrition risk (table 1). Twelve patients were low risk for MUST but high risk for IBD-NST due to having a flare and concerns about their nutrition. Unlike SGA and MUST, IBD-NST nutrition risk was not predicted by BMI (AUC=0.286 (SE 0.06) (95% CI 0.17, 0.40)). No significant difference in suboptimal HGS was seen across BMI categories

of underweight (7/9 (78%)), healthy (28/46 (61%)), overweight (14/23 (61%)) and obese (6/12 (50%)) $p=0.642$. Patients with $>5\%$ weight loss (13/101 (13%)) or suboptimal HGS (56/101 (55%)) were significantly more likely to be at nutrition risk using IBD-NST, MUST and SGA. Patients with suboptimal MAMC (10/66 (15%)) were not significantly more likely to be at nutrition risk using IBD-NST or MUST.

Conclusion The IBD-NST identifies more patients with high nutrition risk and places less importance on BMI than SGA or MUST. Finally, we confirm that BMI is a poor indicator of HGS in IBD supporting a reduced importance in identifying nutrition risk. Further work is underway to test the repeatability of the IBD-NST.

PTH-136 INFLAMMATORY BOWEL DISEASE PATIENTS EXPRESS HIGHER LEVELS OF CIRCULATING GALECTINS-1 AND -3 THAN HEALTHY PEOPLE

¹Sreedhar Subramanian, ²Tony Yu*, ³Susanna Dodd, ⁴Lugang Yu. ¹Royal Liverpool University Hospital, Liverpool, UK; ²School of Medicine, University of Edinburgh, Edinburgh, UK; ³Department of Biostatistics, Institute of Translational Medicine, University of Liverpool, Liverpool, L69 3GS, UK; ⁴Institute of Translational Medicine, University of Liverpool, Crown Street, Liverpool L69 3BX, UK

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Introduction Non-invasive biomarkers currently available in clinic in (i) accurately diagnosing IBD and (ii) in determining disease activity in established IBD all show poor sensitivity and substantial limitation. In this study, we investigated whether levels of serum galectins, a family of 15 galactoside-binding proteins, could be used as potential non-invasive biomarkers for IBD diagnosis and for determining the disease activity.

Methods A single centre prospective cross-sectional study was conducted in 168 IBD patients (97 CD and 71 UC) and 40 healthy controls. Disease activity was assessed using Harvey-Bradshaw Index (HBI) for CD and simple clinical colitis activity index (SCCAI) for UC. Active disease was defined as HBI >5 and SCCAI >2 . Serum levels of galectins-1, -2, -3, -4, -7 and -8 was analysed by ELISA and compared with that in active and non-active IBD patients and healthy controls.

Results Serum levels of galectins-1 and -3, but not galectins-2, -4, -7 and -8, were significantly higher in patients with CD ($p=0.0001$ and $p=0.0003$ for galectin-1 and -3, respectively) and UC ($p=0.0002$ and $p=0.0001$ for galectin-1 and -3, respectively) as compared to healthy controls. Twenty seven (27.8%) CD patients and 41 UC patients (57.8%) had clinically active disease. None of the serum galectins including galectins -1 and -3 showed significantly different in patients with active UC or CD compared to patients in clinical remission. Serum CRP was significantly higher in patients with active UC (mean $\pm$ SD, 7.3 ± 12.5 mg/L) compared to those in remission (4.8 ± 14.5 mg/L, $P=0.047$). Similarly, serum CRP was higher in patients with active CD (4.6 ± 8.2 mg/L) compared to those in remission (10.2 ± 12.1 mg/L, $P=0.047$).

Conclusions Serum levels of galectins-1 and -3 were significantly higher in IBD patients compared to healthy people and this difference however could not separate active from inactive IBD.

PTH-137 PHOSPHATE STUDIES: IMPORTANT CHANGES IN PHOSPHATE HOMEOSTASIS AND BONE METABOLISM AFTER IV IRON

¹Tariq Iqbal*, Myles Wolf, Philip Schaffalitzky de Muckadell, Heinz Zoller. ¹Queen Elizabeth Hospital Birmingham, Birmingham, UK; ²Duke University School of Medicine, North Carolina, USA; ³Pharmacosmos A/S, Holbaek, Denmark; ⁴Medical University of Innsbruck, Innsbruck, Austria

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Introduction Iron deficiency anaemia (IDA) is the most common extraintestinal complication of inflammatory bowel disease (IBD), and IV iron is commonly used to treat IDA in IBD. Of the two commonly used IV iron preparations, ferric carboxymaltose (FCM) and iron isomaltoside IIM, there is evidence that FCM can cause hypophosphatemia due to FGF23 mediated renal phosphate wasting, which has been associated with osteomalacia; the comparative effects of IIM are unknown.

Methods In two identical, open-label trials, 245 adults with IDA randomly received (1:1) IIM (1 $\times$ 1000 mg) or FCM (2 $\times$ 750 mg; 1 week apart). We compared the incidence, severity and duration of hypophosphatemia, and effects on fractional phosphate urinary excretion (FEPO₄), FGF23, parathyroid hormone (PTH), vitamin D (VitD), and bone turnover biomarkers in blood and urine.

Results In a pooled analysis, the incidence of hypophosphatemia <2 mg/dL was markedly higher in the FCM vs. IIM group (74.4% vs. 8.0%, $p<0.0001$). Hypophosphatemia persisted at day 35 in 43.0% of FCM-treated patients compared to 0.9% of IIM-treated patients ($p<0.0001$). Severe hypophosphatemia ≤ 1 mg/dL occurred in 11.3% of FCM patients compared to 0.0% of IIM-treated patients ($p<0.0001$). FCM infusion significantly increased intact FGF23 compared to IIM ($p<0.0001$): FGF23 after FCM rose from 49.9 pg/mL at baseline to 149.5 pg/mL one day post-infusion; and to 327.9 pg/mL by day 8. The corresponding figures for IIM were 59.9 pg/mL at baseline, 58.3 pg/mL by day 1 and 66.9 pg/mL by day 8. Compared to treatment with IIM, FCM significantly: increased FEPO₄; decreased serum 1,25-(OH)₂ vitD; decreased ionized calcium; and increased PTH, which persisted through day 35. Post-FCM changes were accompanied by significant increases in total and bone specific alkaline phosphatase that persisted through day 35. Correction of IDA was comparable between the two treatments. Significantly fewer adverse drug reactions occurred with IIM than with FCM. Serious or severe hypersensitivity reactions occurred in 0.8% in the IIM group and 1.7% in the FCM group.

Conclusion Compared to IIM, FCM-induced high rates of FGF23-mediated hypophosphatemia, which was frequently