Results 51% of hospitalised IVH treated IBD patients met the WHO criteria of DM (CBG>11 mmol/L), while 20% and 6% had a CBG >14 mmol/l and >20 mmol/l, respectively. 8 patients had pre-existing DM, which was confirmed by admission HbA1c. RFR indicated disease severity score, duration of IVH, HbA1c and electrolyte imbalances (64%) were best predictors of hyperglycaemia. 49% were started on or switched biological therapy during admissions. 55% were discharged on prednisolone, 14% on budesonide and 34% on no GC. 48 patients had HbA1c checked at 3 month follow-up of which 4 were in the diabetic range. 1 was known DM with elevated CBG during admission whose insulin had been titrated, 2 had elevated CBG as inpatients with no prior DM discharged on gliclazide and insulin respectively and 1 was on long-term steroids for asthma who did not have CBG >11.0 mmol/L as inpatient. 4 other patients discharged on gliclazide for steroid induced DM had documented repeat HbA1c recorded, which were all in the normal range.

Conclusions Our data demonstrates that hyperglycaemia is common in IVH treated inpatients, therefore CBG monitoring should be routine practice. Predictive modelling (RFR) identifies more severe disease activity, duration of IVH treatment and HbA1c as risk factors for hyperglycaemia. The importance of IVH duration suggests hyperglycaemia risk may be physician-modifiable. Alternative treatment strategies such as earlier introduction of biologics, rapid steroid taper and nutritional support could be used to minimise medication associated metabolic instability in high risk patients.

Abstract PTH 105 Figure 1 Relative importance of input features of RFR model for prediction of highest CBG

PTH-106 PREDICTING RESPONSE TO VEDOLIZUMAB IN CROHN'S DISEASE – A SINGLE CENTRE EXPERIENCE

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Introduction There is a clinical need to develop personalised clinical biomarker scoring systems to direct biologic treatment for Crohn's disease (CD). Vedolizumab is a humanised anti- α 4 β 7 integrin biologic, inhibiting migration of lymphocytes to

the gastrointestinal mucosa. Dulai and colleagues [Gastroenterology, 2018; 155(3)] developed a new clinical prediction tool to determine probability of response to Vedolizumab. Our aim was to retrospectively determine whether this published clinical prediction tool could identify responders to Vedolizumab in our single tertiary IBD centre.

Methods Patients receiving Vedolizumab for CD between 2015–2018 were identified from the IBD database. The Dulai clinical score (>19 predicting response and $\leq$ 13 predicting non-response) was retrospectively calculated from baseline pre-treatment characteristics including absence of previous anti-TNF therapy (+3), no previous gastrointestinal surgery (+2), no fistulising disease (+2) along with pre-treatment CRP (mg/L) (variable) and albumin levels (g/L)(variable) as previously published. Clinical response at 26 weeks was determined based on documented improvement in clinical symptoms and no requirement for steroids. Radiological or endoscopic improvement in disease activity and reduction in faecal calprotectin were considered if data was available. Analysis was performed with SPSS V25 with Mann-Whitney U test, Fisher's exact test and ROC analysis, with significance $p < 0.05$.

Results Forty-nine patients with CD received Vedolizumab. 10 were excluded from analysis as they did not reach the 26-week endpoint. Following 26 weeks of Vedolizumab treatment, 13/39 (33.3%) CD patients had evidence of response. Responders versus non-responders had a median pre-treatment predictive score of 16.3 (8.2–20.8) and 14.1 (6.6–13.5), respectively ($p=0.222$). ROC analysis reported area under the curve of 0.621 (95% CI 0.44–0.81) ($p=0.222$). A score >19 had poor sensitivity [23.08% (95% CI 50.38, 53.81)] but good specificity [84.62% (95% CI 65.13, 95.64)] for predicting response to therapy. A score $\leq$ 13 had poor sensitivity [42.31% (95% CI 22.35, 63.08)] but good specificity [84.62% (95% CI 54.55, 98.08)] for predicting non-response. There was no association between predictive score >19 or $\leq$ 13 and patients who did ($p=0.666$) or did not ($p=0.151$) respond to Vedolizumab therapy, respectively.

Conclusions In this retrospective single centre tertiary IBD centre analysis, the previously published Dulai clinical score did not predict response to Vedolizumab in CD. Future prospective analysis and consideration of a modified score including additional parameters is warranted.

PTH-107 A UK NATIONAL SURVEY OF THERAPEUTIC DRUG MONITORING WITH ANTI-TNF MEDICATIONS IN IBD

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Introduction A growing body of evidence supports use of therapeutic drug monitoring (TDM) in improving efficacy and cost-effectiveness of anti-TNF therapy in patients with inflammatory bowel disease (IBD), supported by AGA guidelines.¹ Existing and evolving knowledge of TDM in clinical practice is less well understood. Our objective was to assess attitudes and barriers to TDM use with anti-TNF's in the UK.

Methods A 17-question survey was distributed to members of the British Society of Gastroenterology. Information on clinician characteristics, demographics, use and barriers towards