responders, three of eight CDAI remission, and four of eight with SES-CD reduced >25%. Nine serious adverse events occurred in five of eight patients, with one early termination. In cohort II, there were three of eight CDAI-70 responders, one of eight CDAI remission, and two of seven with SES-CD reduced >25%. Five serious adverse events occurred in four patients, with two early terminations. In both cohorts, all SAEs were CD-related (i.e. worsening of the CD) except for one device-related postoperative infection and one device deficiency at PE.

Conclusions: VNS induced clinical and biomarker and endoscopic improvement in a significant proportion of drug refractory CD patients.

P575

Vedolizumab trough levels predict clinical outcomes in inflammatory bowel disease

L. Guidi¹,*, D. Pugliese¹, T. Panici Tonucci², B. Tolusso³, C. Felice¹, C. Di Mario³, A. Papa¹, E. Gremese², A. Gasbarrini¹, G.L. Rapaccini¹, A. Armuzzi¹

¹Fondazione Policlinico A. Gemelli, Catholic University, Internal Medicine and Gastroenterology Unit, Piazzale Clivio, Rome, Italy, ²Fondazione Policlinico A. Gemelli, Catholic University, Rheumatology Unit, Piazzale Clivio, Rome, Italy, ³Fondazione Policlinico A. Gemelli, Catholic University, Internal Medicine and Gastroenterology Unit, Rome, Italy

Background: Vedolizumab is an α4β7 integrin antagonist for the treatment of inflammatory bowel disease (IBD). The role of drug monitoring, based on the assessment of Vedolizumab trough levels (VTL) and anti-Vedolizumab antibodies (AVA) has not been clarified. In this study we investigated the correlation between VTL and AVA and clinical outcome.

Methods: We prospectively enrolled consecutive IBD patients starting VDL. Each patient underwent 300 mg infusion at Weeks 0, 2, 6, and 14; additional doses at Week 10 and then every 4 weeks were given to non-responders at Week 6. We assayed VTL and AVA by ELISA (Theradiag, Marne La Vallee, France) at Weeks 6 and 14. Limits of detection for VTL and AVA were 2 μg/ml and 35 ng/ml, respectively. Clinical response was defined as at least 30% reduction of Harvey Bradshaw Index (HBI) and partial Mayo score (pMayo) from baseline while remission was defined as HBI ≤5 or pMayo < 2. Statistics was performed by Mann–Whitney test, Spearman’s rho, receiver operating characteristics (ROC) curve analysis.

Results: We included 66 patients (mean age 46, 1 year; male 60%) with Crohn’s disease (CD, n = 34) and ulcerative colitis (UC, n = 32). Forty-seven (71%) IBD patients had previous anti-TNFα. Patients were followed up to a median of 36 week. Median VTL (IQR) at Weeks 6 and 14 were 32.4 (20.2–48.7) and 16.9 (10.6–22.7) μg/ml, respectively. We detected higher median VTL in responders/remitters at all concomitant and subsequent analysed time points (Table 1). Week 6 VTL were inversely correlated with CRP (rho = −0.32, p = 0.011). By ROC curve analysis we identified a value for VTL at Week 6 of 40.3 μg/ml indicative of response at Week 6 (AUC 0.714, p = 0.0009). Remission at Week 14 was predicted by a Week 6 VTL of 24.3 (AUC 0.682, p = 0.03), remission at Week 22 by a Week 6 VTL of 44.3 (AUC 0.713, p = 0.008) and remission at Week 36 by a Week 6 VTL of 52.9 μg/ml (AUC 0.666, p = 0.04), respectively. We also identified a cut-off of Week 14 VTL for remission at the different time points: 18 μg/ml (AUC 0.683, p = 0.01, AUC 0.729, p = 0.0049, AUC 0.714, p = 0.03, respectively, for remission at Week 14, Week 22, and Week 36). AVA were detected in 1.5% at Week 6 and in 3% at Week 14 and were not correlated with clinical outcome.

Conclusions: Our data suggest that Week 6 VTL is correlated with clinical response and could predict clinical remission at Week 14, Week 22, and Week 36. Week 14 VTL correlate with clinical response and could predict clinical remission at Weeks 22 and 36. Immunogenicity of VDL is low in our patients.

P576

Impact of improved access to biologic therapies and physician engagement on excess steroid exposure: Results from a UK audit of 3561 patients

C. Selinger¹, G.C. Parkes¹, M. Adamson¹, A. Bassi¹, F. Donovan¹, A. Fraser¹, B. George¹, L. Grey³, V. Hall¹, J. Lindi³, H. Ludlow¹, I. Parisi¹, P. Patel¹, R. Pollock¹, S. Salunke¹, J. Saunders¹, G. Scott¹, M. Smith¹, T. Raine²

¹Leeds Teaching Hospitals, Leeds, UK, ²Royal London Hospital, Barts Health, London, UK, ³Salisbury NHS Foundation Trust, Salisbury, UK, ⁴St Helens and Knowsley Foundation Trust, St Helens, UK, ⁵Kingston Hospital NHS Foundation Trust, Kingston upon Thames, UK, ⁶Bristol Royal Infirmary, Bristol, UK, ⁷Torbay and South Devon NHS Trust, Torbay, UK, ⁸Wirral University Teaching Hospital, Wirral, UK, ⁹North Manchester General Hospital, Manchester, UK, ¹⁰University Hospital Llandough, Cardiff, UK, ¹¹University College London Hospitals, London, UK, ¹²Epsom and St Helier University Hospitals NHS Trust, Epsom, UK, ¹³St George’s University Hospitals NHS Foundation Trust, London, UK, ¹⁴Forth Valley Royal Hospital, Larbert, UK, ¹⁵Royal United Hospitals Bath NHS Foundation Trust, Bath, UK, ¹⁶East Kent Hospitals University NHS Foundation Trust, Margate, UK, ¹⁷Brighton and Sussex University Hospitals