Albuminemia <30 g/l (p = 0.031) resulted to be a risk factor for postoperative complications. HEQ had a better result for RALS (p = 0.019), while no differences resulted for SF36, BIQ and GIQLI.
Conclusions: Minimally invasive technique for CD is feasible, even for complicated and recurrent disease. Our study demonstrated low rates of short- and long-term postoperative complications. No difference between techniques were demonstrated. Further studies, with a larger and sample size and randomised controlled design, should be performed to assess the best surgical technique.

P600

Clinical utility of anti-IFI16 seroreactivity in the response to anti-TNF therapy in IBD

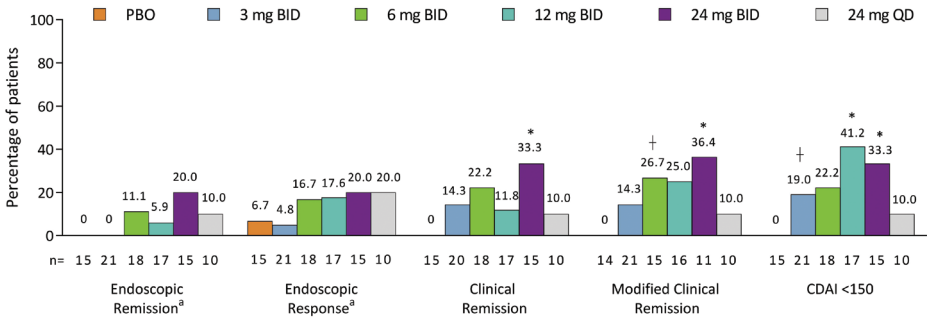
V. Caneparo[1,2], B. Verstockt[3,4], S. Vermeire[3,4], S. Landolfo[1], M. Gariglio[2], M. De Andrea[1,2]*
[1]*University of Turin, Medical School, Department of Public Health and Pediatric Sciences, Viral Pathogenesis Unit, Turin, Italy,* [2]*University of Piemonte Orientale, Department of Translational Medicine, Virology Unit, Novara, Italy,* [3]*University Hospitals Leuven, Department of Gastroenterology and Hepatology, Leuven, Belgium,* [4]*Translational Research in Gastrointestinal Disorders, Department of Chronic Diseases, Metabolism and Ageing, Leuven, Belgium*

Background: Although the cause of IBD remains largely unknown, available evidence suggests that the innate response is greatly involved in inducing gut inflammation. Aberrant expression of pattern recognition receptors (PRRs), including the IFI16 protein, in colonic biopsies from active ulcerative colitis (UC) and Crohn's disease (CD) patients has been independently described (Vanhove *et al.*, *Inflamm Bowel Dis* 2015; Caneparo *et al.*, *Inflamm Bowel Dis* 2016). Consistent with the fact that the IFI16 protein is a target for autoantibodies, we have also demonstrated that its de novo overexpression in the gut epithelial cells leads to the development of specific autoantibodies in IBD (Caneparo *et al.*, *Inflamm Bowel Dis* 2016). To substantiate the clinical utility of these autoantibodies, the anti-IFI16 seroresponse was evaluated in a new and larger cohort of IBD patients undergoing anti-TNF therapy.
Methods: Sera from 145 IBD patients (31 CD and 114 UC), prospectively harvested before and after infliximab (IFX) therapy, were assessed by ELISA for the presence of anti-IFI16 autoantibodies, alongside with 182 sera from healthy controls (HC). The patient's antibody statuses were qualitatively and quantitatively associated with disease phenotype and response to IFX therapy, endoscopically assessed after a median of 12 weeks after first IFX administration. Response was defined as the absence of ulcerations or clear endoscopic improvement compared with baseline endoscopy in CD, and a Mayo endoscopic subscore of 0 or 1 in UC.
Results: Higher prevalence of anti-IFI16 IgG, that tended to increase after therapy, was observed in both CD and UC patients when compared with HC (p < 0.001). With cut-off levels corresponding to the 95th percentile of the distribution in the control population (113 U/ml), 61% of CD patients before and 74% after therapy, and 55% of UC patients before and 61% after therapy tested positive for anti-IFI16 IgG antibodies (p = 0.21 for CD, p = 0.92 for UC, McNemar test) in comparison with 6% of HC. Univariate analysis showed that low levels of autoantibodies at baseline significantly correlated with response to IFX therapy in CD (p = 0.004, Mann–Whitney U test).
Conclusions: In this validation cohort of IBD patients undergoing anti-TNF therapy, we replicated that anti-IFI16 IgG prevalence is significantly higher in both CD and UC patients, compared with HC. Additionally, we established that low levels of anti-IFI16 IgG at baseline significantly correlate with endoscopic response to IFX. Even if larger prospective data are needed to justify the use of anti-IFI16 antibodies as predictive biomarker for IFX therapy, altogether these results substantiate the need to focus on the biological and clinical significance of the IFI16 protein and specific antibodies in IBD.

P601

Upadacitinib improves steroid-free clinical and endoscopic endpoints in patients with Crohn's disease: Data from the CELEST study

R. Panaccione[1]*, R. Atreya[2], M. Ferrante[3], M.C. Dubinsky[4], B.E. Sands[4], M.T. Abreu[5], F. Cataldi[6], J.V. Enejosa[6], Q. Zhou[6], B. Huang[6], A.P. Lacerda[6], A.L. Pangan[6]
[1]*University of Calgary, Calgary, Canada,* [2]*Friedrich-Alexander-University Erlangen-Nürnberg, Medical Clinic 1, Erlangen, Germany,* [3]*University Hospitals Leuven, Leuven, Belgium,* [4]*Icahn*



Endoscopic remission: SES-CD≤4 and ≥2-point reduction from BL, with no subscore >1.
Endoscopic response: SES-CD reduction >50% from BL or endoscopic remission.
Clinical remission: SF≤1.5 and AP≤1.0, and both not worse than BL.
Modified clinical remission: SF≤2.8 and AP≤1.0, both not worse than BL.
Modified clinical remission was analysed in patients with SF≥4.0, AP≥2.0 at BL; other endpoints were analysed in all randomised patients.
*, † significant at ≤0.05 and ≤0.1 level. [a]The follow-up ileocolonoscopy was performed at either week 12 or 16, per randomization schedule

Abstract P601 – Figure. Proportion of subjects who discontinued corticosteroid and achieved clinical and endoscopic endpoints at 16 weeks.