refractory ulcerative colitis (UC). Registrational trials suggested lower efficacy among patients exposed to prior anti-tumour necrosis factor (TNF) agents. We aimed to examine the real-world treatment persistence, biomarker trends, corticosteroid use, and colectomy-free survival in anti-TNF refractory patients treated with tofacitinib.

Methods 20 patients with anti-TNF refractory UC commencing tofacitinib between January 2019 and May 2020 were retrospectively identified across 3 NHS Trusts. Baseline demographic and clinical parameters including treatment persistence, reasons for discontinuation, inflammatory biomarkers (faecal calprotectin [FC] and C-reactive protein [CRP]), corticosteroid use, and colectomy were recorded. Data were collected from tofacitinib initiation until discontinuation or the study end point, May 2021. Fisher’s Exact test was used to appraise the relationship between corticosteroid requirement and treatment persistence. An independent t-test was used to compare biomarker trends.

Results Of 20 patients with a median age of 37 years (range 19-74), 55% were male. Median baseline simple clinical colitis activity index was 7. Mean (SD) CRP was 17.4mg/L and FC was 1034 mg/g (SD). All patients were exposed to 1 anti-TNF agent, 6 patients (30%) to 2 anti-TNF’s, and 7 patients (35%) to vedolizumab. Treatment persistence rate was 45% (9/20) at 12-months (Figure 1). 9 of the 20 patients (45%) required corticosteroids after commencing tofacitinib; of these, 67% did not achieve treatment persistence at 12 months. The need for additional corticosteroids was not associated with treatment persistence (p=0.406). There was no significant difference in 2-month FC (p=0.401) or CRP (p=0.498) in those who persisted with therapy at 12 months compared with those who did not. There were no emergency colectomies. 4 patients underwent elective sub-total colectomy, all of whom discontinued tofacitinib therapy. Those who persisted with therapy at 12-months remained colectomy-free. Adverse events occurred in 10%: 1 patient discontinued treatment due to herpes zoster and another suffered a pulmonary embolus (both aged 49).

Conclusion Tofacitinib can be an effective therapy in those with anti-TNF refractory UC with 45% of patients in our cohort demonstrating treatment persistence at 12-months. Neither early normalisation of inflammatory biomarkers nor need for further steroid therapy were associated with treatment persistence but it is likely our study was under-powered. Colectomy-free survival was observed in all patients persisting with therapy at 12 months.
known small bowel (SB) Crohn’s disease; first assessment for presence of SB disease in IBD; & investigation for SB disease in patients without a known diagnosis of IBD. Obesity, complicated surgical history (>1 resection or strictureplasty involving different segments, or stoma), & known proximal SB disease were deemed unsuitable.

Results 105 MREs were performed in January 2018. 59 (56%) were deemed suitable for IUS instead of MRE. Most common reasons for unsuitability included complex surgical history (n=17, 37%), obesity (n=14, 30%), non-appropriate indication (n=12, 26%) & known upper gastrointestinal disease (n=10, 22%).

Of suitable cases, 32/59 (54%) had active inflammation detected including 17 (53%) isolated ileal, 8 (25%) ileocolonic, & 6 (19%) isolated colonic. In one case performed as first assessment for SB disease, both ileal & jejunal disease were found, the latter likely to be missed with IUS. No cases of isolated upper gastrointestinal inflammation were found. Regarding non-gastrointestinal findings in potential IUS patients, there were two cases of pancreatic cysts necessitating further investigation with serial MRIs & endoscopic ultrasound, yielding a side branch intraductal papillary mucinous neoplasm & a benign serous cyst adenoma. One case of multiple high T2 skeletal lesions was deemed clinically insignificant following further investigations. No other significant extra-intestinal findings not expected to be seen on IUS were identified.

Conclusion >50% of MREs could have been performed as IUS instead, with a potential annual cost saving of >£110,000. No instances of inflammation would have been missed based on distribution, although in one case the full extent of disease may not have been identified on IUS. Incidental non-gastrointestinal findings resulted in multiple investigations but were of limited clinical significance.

Methods 632 patients were identified from the Trust IBD database, who were initiated on biologics from January 2015 to December 2019. 465 matched our inclusion and exclusion criteria. Baseline characteristics, such as gender, biologic, type of IBD and number of biologics used previously, were matched to produce two cohorts, each of 98 participants comprising >60s & <60s.

Results Adverse effects, hospitalisation and need for emergency surgery was seen in 5.1% of under 60s (n=5) and 13.3% of over 60s (n=13). There was no significant difference in the proportion of patients who failed to complete 12 months of biologic treatment from the >60s (n=20, 20.4%) versus <60s (n=12, 12.2%) (OR 1.838, p=0.126). A larger proportion of biologic failure was seen in those on anti-TNFα vs. non-anti-TNFα biologics, but this was not significant (OR 2.35, p=0.051). IBD type (Crohn’s vs. Colitis) was not a predictor of biological failure (OR 1.856, p=0.176), nor was previous biologic use (OR 0.644, p=0.118); concomitant thiopurines/methotrexate (OR 0.46, p=0.134); co-morbidities (OR 0.834, p<0.752); smoking status (OR 0.956, p=0.888); severity score (OR 0.939, p=0.420); baseline CRP (OR 1.024, p=0.250); faecal calprotectin (OR 1.00, p=0.746). Risk of biological failure at 12 months was greater in women than in men (OR 2.5, p=0.023).

Conclusions Age is not an independent predictor of pooled biological failure at 12 months post-initiation. This finding may be factored in when personalised treatment approaches are sought for >60s who are not responsive to conventional therapy. However, more research is required in a higher-powered study to investigate whether treatment failure is influenced by various demographic factors and by biologic type.