ANTI-TNF DOSE AUGMENTATION FREQUENTLY OCCURS IN THE ABSENCE OF OBJECTIVE EVIDENCE OF DISEASE ACTIVITY

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Background: Anti tumor necrosis factor therapy (anti-TNF) is widely used in patients with moderate to severe CD and UC. When symptoms persist, anti-TNF dose is often increased or the dosing interval shortened. Evidence supporting the effectiveness of dose augmentation is of low quality and dose augmentation imparts a significant additional cost of care. Persons with persistent gastrointestinal symptoms ascribed to IBD may not have evidence of ongoing inflammation; hence, dose augmentation may not be expected to benefit those without inflammation.

Aims: We sought to determine the extent to which clinicians routinely assessed the presence of active inflammation prior to undertaking anti-TNF dose augmentation.

Methods: Medical records of all IBD patients prescribed anti-TNF therapy from 2007-2016 by 8 of 23 Manitoba gastroenterologists were reviewed and demographics, disease characteristics, and IBD treatments recorded. Patients who underwent anti-TNF dose augmentation were further reviewed for the presence of any objective assessment of inflammatory activity, including laboratory investigations (CRP, ESR, albumin, ferritin, hemoglobin, fecal calprotectin), cross-sectional abdominal imaging, and endoscopy.

Results: 529 patients receiving anti-TNF therapy were identified, of whom 151 had anti-TNF doses increased on 195 occasions (117 CD, 34 UC). 51 patients (43.6%) with CD had penetrating disease while 16 patients (47.1%) with UC had pancolitis. Mean age at diagnosis was 25.5 years and 50.3% of patients were female. There was no difference in demographics, disease phenotype, or baseline lab values between patients who received dose augmentation and those who did not.

Patients were assessed for biochemical evidence of disease activity in the 90 days preceding dose augmentation on 134 of 195 occasions (68.7%), however the results of these investigations were abnormal in only 23 cases (11.8%). Cross-sectional imaging was performed in 11 cases (5.6%) and revealed active disease in 8 (4.1%). Endoscopy was performed prior to dose augmentation on 28 occasions (14.4%) with 24 (12.3%) revealing active disease. Overall, objective evidence of inflammatory activity was present in only 48 of 195 dose augmentation events (24.6%), no objective evidence of inflammation was present in 95 (48.7%), and in 52 (26.7%), anti-TNF dose was increased without any investigation being performed.

Conclusions: Anti-TNF dose augmentation routinely occurs in the absence of objective evidence of active inflammatory disease. This represents a target for ongoing quality improvement to optimize care of persons with IBD requiring anti-TNF based therapies, given the significant economic burden of unjustified and potentially unnecessary dose augmentation.
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