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Original article

Accelerated infliximab induction therapy in inflammatory bowel disease

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Abstract

Introduction: Inflammatory bowel disease (IBD), which includes ulcerative colitis (UC) and Crohn's disease (CD), represents a therapeutic challenge, especially in moderate-to-severe cases and in patients with previous anti-TNF failure. Accelerated infliximab induction, using higher doses or shorter intervals than the standard regimen, has been proposed to achieve faster inflammatory control in patients with a high inflammatory burden or refractory disease. However, evidence in Latin American settings remains limited. The objective of this study was to describe the clinical outcomes of patients with IBD treated with accelerated infliximab induction at a Colombian referral center. **Methodology:** Observational, descriptive case series that included adult and pediatric patients with UC or CD treated between January 2022 and December 2023 with infliximab 10 mg/kg at weeks 0, 1, and 2. The study evaluated clinical remission, the need for therapeutic escalation, and the requirement for surgery through a review of medical records. **Results:** The study included five patients (four men; three adults and two pediatric patients), all with previous anti-TNF exposure. Three had UC and two had CD. Only one pediatric patient with CD achieved clinical remission. Four did not respond, three required a change in medication to tofacitinib or vedolizumab, and two underwent surgery. No serious adverse events were documented. **Conclusion:** In this highly refractory cohort, accelerated induction showed a low remission rate and a frequent need for therapeutic escalation.

Keywords

Inflammatory bowel diseases, infliximab, ulcerative colitis, Crohn's disease, biological therapy.

INTRODUCTION

Inflammatory bowel disease (IBD), encompassing Crohn's disease (CD) and ulcerative colitis (UC), remains a persistent therapeutic challenge owing to its chronic, relapsing, and potentially progressive course, as well as its substantial systemic inflammatory burden[1,2]. The clinical course is characterized by alternating periods of remission and exacerbation that frequently require therapeutic escalation and dynamic long-term treatment adjustments. Advances in the understanding of IBD pathophysiology have demonstrated a multifactorial etiology involving genetic susceptibility, environmental factors, alterations in the gut microbiome, and both innate and adaptive immune

dysfunction. However, this biological complexity has limited the development of universally effective therapeutic strategies. Treatment goals have evolved from symptom control toward mucosal healing and deep remission, outcomes associated with lower rates of hospitalization, surgical intervention, and cumulative disability[3]. Nevertheless, sustained achievement of these targets remains limited by marked phenotypic heterogeneity, variability in underlying inflammatory pathways, and differential treatment responses[4]. In addition, secondary loss of response, immunogenicity, and the need for combination regimens further complicate clinical management[5]. Although combining biologic agents with immunomodulators may enhance efficacy, it also increases the risk of adverse events and lacks clear standardization regarding optimal sequencing, dosing, and treatment duration[4,6,7].

Infliximab, a chimeric monoclonal antibody targeting tumor necrosis factor alpha (TNF-α), is a cornerstone therapy for moderate-to-severe IBD, particularly when rapid induction of remission is required[8,9]. The conventional induction regimen (5 mg/kg administered intravenously at weeks 0, 2, and 6, followed by maintenance therapy every 8 weeks) has demonstrated efficacy in inducing clinical response and improving quality of life in both UC and CD[9,10]. However, primary therapeutic failure remains common. Contributing mechanisms include the development of anti-drug antibodies (ATIs), pharmacokinetic alterations resulting in increased drug clearance and subtherapeutic exposure, and the presence of TNF-independent inflammatory pathways leading to mechanistic nonresponse[11,12]. These limitations have prompted investigation of dosing-optimization strategies aimed at overcoming pharmacokinetic failure and achieving more rapid inflammatory control in settings of high disease activity.

Accelerated infliximab induction refers to intensified regimens involving higher doses or shorter dosing intervals than the standard regimen, most commonly 10 mg/kg administered at compressed intervals during the induction phase. This strategy is intended to counteract increased drug clearance associated with a high inflammatory burden, hypoalbuminemia, and fecal drug loss, all of which have been linked to subtherapeutic serum concentrations. Evidence supporting accelerated induction in UC derives primarily from the PREDICT study, which did not demonstrate superiority over the standard regimen with respect to early clinical response, remission, or short-term colectomy rates[13]. In CD, the evidence is more limited and is largely extrapolated from studies of acute severe ulcerative colitis (ASUC), in which infliximab pharmacokinetics have been more extensively characterized[14,15]. Although some observational studies have suggested short-term benefits, such as a reduction in early colectomy rates, these findings have not consistently translated into long-term advantages[8,10]. Likewise, the ACT 1 and ACT 2 trials demonstrated comparable efficacy of 5- and 10-mg/kg doses for induction and maintenance in UC, without conclusive evidence of sustained superiority with dose intensification[16].

In Latin America, and particularly in Colombia, the management of severe IBD reflects both global therapeutic advances and regional structural limitations. Infliximab remains widely used and has demonstrated effectiveness in Colombian cohorts with moderate-to-severe UC[17]. However, increasing disease prevalence, barriers to access to biologic therapies, diagnostic limitations, and inequities within the healthcare system pose substantial challenges to therapeutic optimization[18,19]. Furthermore, real-world data on accelerated induction strategies in Latin American populations remain scarce.

Against this backdrop of clinical uncertainty and structural limitations, the present study aimed to describe real-world clinical outcomes among patients with IBD who underwent accelerated infliximab induction at a Colombian referral center. This study was not intended to establish causal relationships or determine comparative efficacy, but rather to provide rigorous observational evidence from a highly refractory cohort managed under routine clinical care conditions.

METHODS

Study design and participant selection

An observational, descriptive, retrospective case series based on convenience sampling was conducted, including adult and pediatric patients with a diagnosis of IBD who received care between January 2022 and December 2023 at a referral center for IBD in Colombia.

Eligibility criteria included an established diagnosis of severe ulcerative colitis (UC) or Crohn's disease (CD), confirmed according to clinical and endoscopic criteria. The indication for accelerated infliximab induction was determined on a case-by-case basis through specialized clinical judgment and shared decision-making between the treating physician and the patient. Patients with active *Clostridioides difficile* infection, documented bacterial enteric infections, or cytomegalovirus infection were excluded. No formal treat-to-target strategy was implemented. Therapeutic decisions during and after induction were made under routine clinical practice conditions and were based on individualized clinical assessment.

Treatment regimen

All patients received a fixed accelerated infliximab induction regimen consisting of 10 mg/kg administered intravenously

at weeks 0, 1, and 2. Selection of this regimen was based on clinical disease severity and specialized medical assessment. No alternative regimens were used within the cohort.

Data collection

The primary source of information was the institutional medical record. Sociodemographic variables and baseline clinical characteristics prior to infliximab initiation were recorded, including age, sex, IBD type (UC or CD), classification according to the Paris/Montreal criteria, prior exposure to anti-TNF therapy, concomitant corticosteroid use, and fecal calprotectin levels when available.

Given the retrospective, real-world nature of the study, potentially relevant variables such as body mass index (BMI), systematic baseline laboratory parameters, therapeutic drug monitoring (TDM), and standardized clinical disease activity indices were not collected according to a predefined protocol.

Follow-up and outcomes

Patients were followed from initiation of accelerated induction until the occurrence of a clinically relevant outcome, including clinical remission, need for treatment intensification or a change in medical therapy, surgical intervention, or the last clinical assessment available in the medical record. Follow-up duration varied among patients and was individualized, reflecting real-world clinical practice.

The primary outcome was clinical remission following accelerated induction. Clinical remission was defined as documented resolution of gastrointestinal symptoms according to the treating physician's medical record, including improvement in stool frequency, rectal bleeding, abdominal pain, and overall clinical status.

Secondary outcomes included the need for additional medical therapy and the need for surgery. Endoscopic assessment was not systematically performed as part of a protocolized post-induction strategy. Colonoscopy was requested according to individual clinical judgment and was not performed as part of a standardized algorithm.

Statistical analysis

The database was constructed using Microsoft Excel® version 2019. Information confidentiality was ensured through anonymization of the records, with no identifying information included. Data were independently verified by three investigators. Statistical analyses were performed using SPSS® version 25.0.

Given the small sample size and descriptive nature of the study, only descriptive analyses were performed. Results are presented as absolute numbers and, where applicable, percentages, solely for descriptive purposes and without inferential intent.

RESULTS

Five patients with IBD who received accelerated infliximab induction during the study period were included. Three were adults (aged 19, 37, and 18 years), and two were pediatric patients (aged 15 and 11 years). The cohort comprised four men and one woman. All patients had been previously exposed to anti-TNF therapy before undergoing accelerated induction. The mean age was 13 years in the pediatric subgroup and 24.6 years in the adult subgroup. Three patients had a diagnosis of UC and two had CD.

Outcome assessment was performed throughout the available follow-up period for each patient. Most clinically relevant events occurred during the early post-induction phase, allowing assessment of the initial clinical response and the need for therapeutic escalation.

Only one patient, belonging to the pediatric subgroup with CD, achieved clinical remission following accelerated induction. The remaining four patients did not achieve remission. Of these, three required treatment intensification or a change in medical therapy because of lack of response, including transition to tofacitinib or vedolizumab.

Two patients ultimately required surgical intervention because of persistent therapeutic failure. One underwent right hemicolectomy with terminal ileal resection, while the other underwent total colectomy with ileostomy.

No infusion reactions, infections, or other serious adverse events were documented during the induction process. Baseline clinical characteristics and individual patient outcomes are presented in detail in **Table 1**.

DISCUSSION

The present case series describes the clinical outcomes of accelerated infliximab induction in a cohort of patients with IBD who had been previously exposed to and had documented failure of anti-tumor necrosis factor (anti-TNF) therapy. Prior anti-TNF failure is a recognized predictor of reduced response to subsequent biologic therapies, particularly when switching within the same therapeutic class, and therefore represents a key consideration in the appropriate interpretation of our findings[1-3].

Anti-TNF failure is a heterogeneous phenomenon that may result from pharmacokinetic mechanisms, including increased drug clearance, fecal drug loss, immunogenicity, or subtherapeutic serum drug concentrations, or from true pharmacodynamic or mechanistic nonresponse, in which TNF is no longer the primary driver of inflammation[4-6].

Table 1. Baseline clinical characteristics and short-term outcomes of patients undergoing accelerated infliximab induction (n = 5)

Characteristic	Case 1	Case 2	Case 3	Case 4	Case 5
Sex	Female	Male	Female	Male	Male
Diagnosis	UC	CD	CD	UC	CD
Age (years)	15	18	11	19	37
Paris/Montreal classification	E4S1G1	A1L1B2B3	A1aL3pG1	E3S3	A2L2B2
Calprotectin (µg/g)	1000	1155	5534	Not available	Not available
Prior anti-TNF therapy	Yes	Yes	Yes	Yes	Yes
Infliximab dose (mg/kg)	10 (weeks 0–1–2)	10 (weeks 0–1–2)	10 (weeks 0–1–2)	10 (weeks 0–1–2)	10 (weeks 0–1–2)
Clinical remission	No	No	Yes	No	No
Concomitant corticosteroid use	Yes, three courses in the previous year	Yes, two courses in the previous year	Yes, one course in the previous year	Yes, two courses in the previous year	No
Required additional medical therapy	Yes, tofacitinib	Yes, vedolizumab	No	Yes, tofacitinib	No
Required surgery/type	No	Yes, right hemicolectomy and terminal ileal resection	No	No	Yes, total colectomy with ileostomy

Anti-TNF: tumor necrosis factor inhibitors; CD: Crohn's disease; UC: ulcerative colitis; n: number. Table prepared by the authors.

This distinction is not merely academic but has direct clinical implications: whereas underexposure may warrant dose optimization or a within-class switch, mechanistic failure is associated with a low likelihood of benefit from a second anti-TNF agent and may instead necessitate a switch to a different therapeutic target[7,8].

In our cohort, all patients had experienced prior anti-TNF failure and received accelerated infliximab induction in the setting of severe disease, which in many cases was refractory to corticosteroids. In this clinical setting, particularly in acute severe ulcerative colitis (ASUC), rapid control of inflammation is critical to prevent major complications or urgent colectomy[9,10]. Severe systemic inflammation, hypoalbuminemia, and increased fecal drug loss have been associated with subtherapeutic infliximab concentrations during standard induction, providing a biologically plausible rationale for the use of intensified regimens in selected patients[11,12,22].

Nevertheless, the absence of systematic therapeutic drug monitoring (TDM) and routine assessment of anti-drug antibodies limited our ability to distinguish pharmacokinetic failure from mechanistic resistance. This limitation is particularly relevant in patients with prior anti-TNF exposure, in whom early identification of underexposure versus mechanistic nonresponse is essential to guide timely therapeutic intensification or transition to alternative mechanisms of action[13,23].

The low remission rate observed should not be interpreted as conclusive evidence of the ineffectiveness of accelerated infliximab induction. Rather, it reflects real-world outcomes in a highly refractory population with advanced disease and limited opportunity for early objective reassessment. Previous studies have shown that patients with prior anti-TNF failure, particularly those with mechanistic nonresponse, have lower remission rates when treated with a second agent from the same class, even with intensified dosing regimens[14-16].

In the setting of ASUC, accelerated induction remains a widely used rescue strategy, supported by pharmacokinetic rationale and observational data suggesting potential short-term benefits in selected patients[9,11,17]. However, the optimal identification of candidates for this strategy remains uncertain. Contemporary evidence supports a personalized approach incorporating early clinical assessment, objective biomarkers, and, when available, TDM to avoid prolonged exposure to ineffective therapies[18,23].

From a regional perspective, real-world data on biologic optimization in Latin America remain limited. Most available evidence originates from North America and Europe, whereas Latin American studies have focused primarily

on standard treatment regimens and challenges related to access and healthcare coverage[17,19]. This underscores the importance of reporting local clinical experience, particularly in highly refractory populations managed within the structural constraints of regional healthcare systems.

The central message of this case series is not to discourage the use of accelerated infliximab induction, but rather to highlight the complexities inherent in its application to patients with prior anti-TNF failure. In this setting, accelerated induction should be considered a selective, individualized strategy, particularly when pharmacokinetic failure is suspected and immediate inflammatory control is required. Early objective reassessment and TDM-guided optimization may be critical for timely identification of patients with a low likelihood of sustained response and for facilitating transition to other biologic classes or surgical management[13,19].

Prospective studies incorporating standardized outcomes within treat-to-target strategies, systematic pharmacokinetic monitoring, and stratification according to the mechanism of prior anti-TNF failure are needed to more precisely define the role of accelerated induction in highly refractory IBD populations.

CONCLUSIONS

This descriptive case series provides a real-world assessment of clinical outcomes associated with accelerated infliximab induction in a small, highly selected cohort of patients with IBD and prior anti-TNF treatment failure. In this setting of severe and highly refractory disease, the intensified regimen was associated with a low rate of clinical remission and a frequent need for therapeutic escalation or surgical intervention.

These findings should be interpreted with caution given the retrospective study design, limited sample size, and absence of a standardized treat-to-target strategy or systematic therapeutic drug monitoring. Accordingly, the results are not generalizable and do not permit causal inferences regarding the efficacy of accelerated infliximab induction. Rather, they should be regarded as preliminary, exploratory, and hypothesis-generating data reflecting the challenges inherent in the management of highly refractory IBD populations under routine clinical practice conditions.

Accelerated infliximab induction may remain a reasonable and selective therapeutic option in specific clinical scenarios, particularly in severe disease requiring immediate inflammatory control. However, its use should be accompanied by close monitoring, early objective reassessment, and, when feasible, pharmacokinetic-guided optimization.

Prospective studies with larger sample sizes, clearly defined clinical outcomes, and systematic therapeutic drug monitoring are warranted to more precisely delineate the role of accelerated induction strategies in patients with prior anti-TNF failure.

Ethical considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki, 2013 version, Fortaleza, Brazil[20], and Resolution 8430 of 1993 of the Colombian Ministry of Health[21]. The study was classified as research involving no more than minimal risk, given its retrospective observational design and the absence of interventions beyond routine clinical care.

Confidentiality of the collected information was ensured. Given the retrospective nature of the study and the absence of direct intervention, formal informed consent was not required. Nevertheless, during administration of the data collection instrument, participants were informed of the objectives of the study.

Data availability

The data supporting the findings of this study are available from the corresponding authors, VPI and JSFO, upon reasonable request. The data are not publicly available because of ethical and privacy restrictions, as they contain information that could compromise the confidentiality of research participants.

Informed consent

This study was reviewed and approved by the ethics committee and institutional review board of each participating institution. The study design adhered to the principles established in the Declaration of Helsinki, 2013 version, Fortaleza, Brazil, and was classified as research involving no more than minimal risk. The confidentiality of patient information was ensured.

Conflicts of interest

The authors declare no conflicts of interest.

Use of artificial intelligence

The authors declare that no generative artificial intelligence tools were used at any stage of the preparation of this manuscript.

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Author contributions

- Study conception and design: Viviana Parra-Izquierdo, Sergio Romero-Arias, Carlos Augusto Cuadros-Mendoza, and Juan Sebastián Frías-Ordóñez.
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- Manuscript drafting: Viviana Parra-Izquierdo and Juan Sebastián Frías-Ordóñez.
- Critical revision of the manuscript: Viviana Parra-Izquierdo, Sergio Romero-Arias, Carlos Augusto Cuadros-Mendoza, Johon Francisco Garcés-Camacho, Silvia Hoyos, Gilnary Orduz, Luis Felipe Murcia-Cárdenas, and Juan Sebastián Frías-Ordóñez.

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