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Management of Steroid-Refractory Immune Checkpoint Inhibitor Colitis: A Practical Guide to Advanced Therapies

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Key Takeaways

When to escalate: Patients with steroid-refractory or steroid-dependent immune checkpoint inhibitor colitis should receive advanced immunosuppressive therapy. Earlier escalation should be considered when endoscopy demonstrates high-risk features, particularly deep ulcerations, which predict steroid failure.

Which agent to choose: Infliximab and vedolizumab are the preferred advanced therapies and have comparable efficacy. Agent selection should be individualized based on the clinical context: infliximab is generally preferred for acute, severe disease requiring rapid control, whereas the gut-selective mechanism of vedolizumab may be favoured when immune checkpoint inhibitor rechallenge is anticipated.

How long to treat: At least three induction doses are recommended; however, treatment duration should be guided by objective markers of disease activity, such as fecal calprotectin levels and endoscopic mucosal healing, rather than by the number of doses alone. The intent to resume checkpoint-inhibitor therapy does not dictate the number of induction infusions but may support adding maintenance biologic therapy at the time of rechallenge to reduce recurrence risk.

Introduction

The advent of immune checkpoint inhibitors (ICI) has revolutionized cancer therapy by leveraging the immune system's ability to recognize and eliminate malignant cells.¹ These agents are engineered monoclonal antibodies that disrupt inhibitory immune checkpoints that normally restrain T-cell activity, thereby enhancing antitumour immune responses.² Despite their transformative impact, ICI can precipitate off-tumour immune-related adverse events (irAEs), which may involve any organ system. Among these, colitis, with or without enteritis, is one of the most common and clinically significant gastrointestinal (GI) side effects caused by ICI therapy, particularly in patients receiving cytotoxic T-lymphocyte-associated protein 4 (CTLA-4) agents.³

In patients who present with colitis or acute diarrhea, the initial step is to exclude alternative causes, particularly infectious causes

(e.g., *Clostridioides difficile*, invasive bacterial enterocolitis, and, in certain circumstances, cytomegalovirus colitis). Once the clinical picture and investigations support ICI colitis as the most probable diagnosis, severity should be graded according to the Common Terminology Criteria for Adverse Events (CTCAE) v5.0 (see **Table 1**). This system grades severity using stool frequency and features such as hematochezia and mucus, together with clinical parameters including the need for hospitalization or intensive care and hemodynamic instability.⁴ These criteria can both underestimate and overestimate the true severity of ICI colitis, as discussed below.⁵

Management of irAEs typically involves corticosteroids due to their broad immunosuppressive effects.⁶ However, corticosteroids come with their own set of well-recognized side effects including immunosuppression, hyperglycemia, and bone loss. Initial corticosteroid therapy varies by colitis severity: grade 1 disease is managed

Grade	Diarrhea	Colitis/Enterocolitis
1	Increase of <4 stools/day over baseline; mild increase in ostomy output	Asymptomatic; clinical or diagnostic observations only; intervention not indicated
2	Increase of 4–6 stools/day over baseline; moderate increase in ostomy output; limiting instrumental ADL	Abdominal pain; mucus or blood in stool
3	Increase of ≥7 stools/day over baseline; hospitalization indicated; severe increase in ostomy output; limiting self-care ADL	Severe or persistent abdominal pain; fever; ileus; peritoneal signs
4	Life-threatening consequences; urgent intervention indicated	Life-threatening consequences; urgent intervention indicated
5	Death	Death

Table 1. CTCAE v5.0 severity grading for diarrhea, colitis, and enterocolitis; *adapted from National Cancer Institute, Common Terminology Criteria for Adverse Events (CTCAE) v5.0, 2017 (public domain).*

Definitions: **Diarrhea:** increased frequency and/or loose or watery stools; colitis, inflammation of the colon; **Enterocolitis:** inflammation of the small and large intestine. A semicolon denotes “or” within a grade. **ADL:** activities of daily living. Grades are based predominantly on clinical symptoms and do not incorporate endoscopic findings.

supportively without steroids, whereas grade 2 receives prednisone or IV methylprednisolone 1–2 mg/kg/day and grade 3–4 receives IV methylprednisolone 1–2 mg/kg/day. Furthermore, up to 20% of patients requiring steroids for an irAE are steroid refractory, necessitating escalation to alternative immunosuppressive agents.⁷ Current management guidelines for such refractory cases rely largely on expert consensus and draw parallels from treatment paradigms in primary autoimmune diseases, notably inflammatory bowel disease for the management of ICI colitis.⁸ When dealing with ICI colitis, three key questions must be answered. What is the right time to start advanced therapy? Which agent is best to use? How long should advanced immunosuppression be maintained?

When to Start Advanced Therapy

Two clinical scenarios should prompt escalation beyond corticosteroids: steroid-refractory disease and steroid-dependent disease. Steroid-refractory ICI colitis is defined as the absence of meaningful clinical improvement after approximately 72 hours of adequate-dose intravenous corticosteroids (methylprednisolone 1–2 mg/kg/day).^{5,9} Steroid-dependent disease refers to the recurrence of colitis symptoms during or shortly after completing the glucocorticoid taper, which leads to re-escalation and consideration of steroid-sparing therapy.⁵ Both

the American Gastroenterological Association (AGA) Clinical Practice Update and the American Society of Clinical Oncology Guideline Update converge on these definitions, while the National Comprehensive Cancer Network (NCCN) guidelines similarly recommend additional immunosuppression if there is no response within 1–2 days for grade 3–4 colitis or within 3 days for grade 2 colitis.^{9,10} Importantly, the approximately 72-hour threshold is based on expert consensus, retrospective observation and extrapolated from the management of severe ulcerative colitis presentations rather than prospective trials designed to validate a specific time point. The consensus of the guidelines on this threshold reflects clinical pragmatism between allowing sufficient time to observe a steroid response while being short enough to avoid complications of uncontrolled inflammation.

The decision to escalate therapy should not be based solely on time. Baseline clinical and endoscopic features can identify patients at high risk of steroid failure before the 72-hour threshold arrives. Among these, colonic ulceration on initial endoscopy is the strongest and most consistently validated predictor of steroid-refractory disease. The AGA update identifies endoscopic ulceration as the only established factor associated with the need for secondary immunosuppression, and notably, CTCAE symptom grading is not predictive.⁵ The NCCN guidelines reinforce this finding, noting that endoscopic ulceration is associated with a greater likelihood of

Feature	Infliximab	Vedolizumab
Class/mechanism	Chimeric monoclonal anti-TNF- α antibody; broad suppression of TNF-mediated inflammation across tissues	Humanized anti- $\alpha 4\beta 7$ integrin antibody; blocks lymphocyte trafficking to the gastrointestinal mucosa
Typical dose	5 mg/kg IV	300 mg IV
Induction schedule	Weeks 0, 2, and 6 (IBD-adapted)	Weeks 0, 2, and 6 (IBD-adapted)
Onset of action	Rapid, often within days	Slightly slower, but comparable in prospective data (median time to improvement approximately 3 days)
Clinical remission	High (approximately 90% in the randomized comparison)	High (approximately 89% in the randomized comparison)
Key cautions/relative contraindications	Hematologic malignancy (rare lymphoma risk), severe congestive heart failure, concurrent ICI hepatitis (drug-induced liver injury) Screen for tuberculosis and hepatitis B; exclude bowel perforation	Theoretical interference with mucosal antitumor responses in primary GI malignancies or GI metastases; fewer systemic infectious concerns
Effect on antitumor immunity	Systemic mechanism; preclinical models suggest TNF blockade may not impair ICI efficacy	Gut-selective mechanism theoretically spares systemic antitumor immunity, though it may affect mucosal responses in GI tumours
Best-suited scenario	Acute, severe, or fulminant colitis requiring rapid control; concurrent extra-intestinal irAEs needing systemic immunosuppression	When gut-selective control is preferred: planned ICI rechallenge, higher infection risk, or concern for recurrence

Table 2. Comparison of infliximab and vedolizumab for advanced (steroid-sparing) therapy in immune checkpoint inhibitor colitis; *courtesy of Rami Habib, MD, Wilson H. Miller Jr., MD, PhD, and Khashayar Esfahani, MD.*

Abbreviations: GI: gastrointestinal; IBD: inflammatory bowel disease; ICI: immune checkpoint inhibitor; irAE: immune-related adverse event; IV: intravenous; TNF: tumour necrosis factor.

corticosteroid failure and that initiating biologic therapy early in these patients may reduce the risk of disease recurrence.¹⁰ In addition to endoscopic assessment, stool biomarkers can be used as a non-invasive stratification layer. The NCCN recommends measuring fecal calprotectin and lactoferrin at initial assessment across all colitis grades, with serial calprotectin monitoring every 2 months to guide treatment duration.¹⁰ Taken together, these data support a risk-stratified approach. In patients with deep or extensive ulceration, markedly elevated calprotectin, or other high-risk features, there is a rationale for pre-emptive escalation to selective immunosuppressive therapy (SIT) rather than waiting for formal steroid failure. This approach represents the strongest opportunity to improve outcomes, because the current “fail-then-escalate” approach exposes the highest-risk patients to the longest steroid courses and a greater risk of complications.

The strategy of upfront biologic therapy, defined as initiating infliximab or vedolizumab concurrently with or shortly after corticosteroid treatment rather than sequentially, has gained support in observational studies. A large propensity-matched real-world analysis comparing early biologic initiation (within 3 days of colitis diagnosis) to delayed initiation (days 3–14) found numerically favourable outcomes with early therapy across mortality, hospitalization, and long-term corticosteroid use, although these differences did not reach statistical significance.¹¹ A single-centre retrospective study of prophylactic vedolizumab in high-risk patients, including those with prior severe ICI colitis or pre-existing inflammatory bowel disease (IBD), demonstrated markedly reduced diarrhea severity and eliminated the need for corticosteroid treatment.¹² Prospective trials, including NCT04407247, are needed to confirm whether these findings represent a true therapeutic benefit or reflect unmeasured confounding.¹³ Until those

data mature, the following points remain relevant: guidelines endorse early escalation in patients with endoscopically severe disease, observational data suggest benefit from upfront biologic therapy, and the existing evidence base remains insufficient to justify a universal change in practice.

Choice of Advanced Therapy

Infliximab and vedolizumab represent mechanistically distinct approaches to steroid-refractory ICI colitis (see **Table 2** for a detailed comparison). Infliximab broadly suppresses tumour necrosis factor (TNF)-mediated inflammation, whereas vedolizumab selectively blocks lymphocyte trafficking to the GI tract.^{5,14} Both agents are typically administered on an IBD-adapted induction schedule at weeks 0, 2, and 6; however, responses in ICI colitis occur far more rapidly than in IBD, generally within one week.⁵ Preliminary results from the first randomized controlled trial comparing the two agents (NCT04407247) have been reported in abstract form. Among 19 of 22 evaluable patients, clinical remission rates at 2 weeks were similarly high (infliximab 90% vs. vedolizumab 88.9%), with a median time to symptom improvement of 3 days in both treatment arms. Steroid-free remission at one month was achieved in 80% and 77.8% of patients, respectively, and recurrence rates did not differ significantly (40% vs. 33.3%).¹³ These findings are consistent with a meta-analysis of eight retrospective studies, which demonstrated no significant difference in remission rates between the two monotherapies. Although vedolizumab was associated with a higher rate of colitis recurrence (odds ratio [OR] 0.32, 95% confidence interval [CI] 0.19–0.53) it was also associated with a significantly lower overall corticosteroid usage (mean difference –18.29 days).¹⁵ Similarly, the largest single-centre retrospective study (n = 1,151 patients with ICI colitis) reported comparable efficacy with infliximab producing a shorter time to clinical response.¹⁶ (See **Table 2**.)

In clinical practice, agent selection is driven by clinical context rather than by clear efficacy differences. Infliximab is used in severe and urgent cases given its faster onset of action and greater clinical experience in this setting.¹⁷ However, infliximab should be avoided in patients with hematologic malignancies (given the role of TNF- α in antitumor immune surveillance), in

those with severe congestive heart failure (given demonstrated cardiac harm even with short-course use), in patients with concurrent ICI hepatitis (given the risk of drug-induced liver injury) and weighed carefully in patients with significant comorbidities or elevated infection risk. While these complications vary in frequency, vedolizumab is recommended as a gut-selective alternative that avoids these risks.⁵ The gut-selectivity of vedolizumab is its main theoretical advantage. By confining immunosuppression to the GI tract, vedolizumab may preserve systemic antitumor immunity and is therefore particularly attractive for patients in whom ongoing or future ICI therapy is planned.¹⁴ However, because vedolizumab acts locally in the gut, the AGA update cautions that it could attenuate mucosal antitumor immunity in patients with tumours in the GI tract, namely primary GI cancers or GI metastases, and flags this as a relevant consideration in treatment selection.⁵ For patients with concurrent extra-intestinal irAEs requiring systemic immunosuppression, infliximab's broader mechanism may offer the advantage of treating multiple toxicities simultaneously. Infection risk should also be considered. Unlike inflammatory bowel disease, where steroids are used at lower doses but maintenance immunomodulator therapy and prolonged tapering are the norm, ICI colitis is treated with higher weight-based steroid doses as detailed above but a deliberately shorter taper, typically over 4–6 weeks after symptoms improve to grade ≤ 1 , and maintenance immunosuppression is far less often required.¹⁸ When a biologic (infliximab or vedolizumab) has been added, guidelines recommend compressing the taper further, to under 2–4 weeks, specifically to limit cumulative steroid exposure and the resulting risk of infection.¹⁰

For patients refractory to both infliximab and vedolizumab, the evidence base narrows considerably. The NCCN suggests tofacitinib, a Janus kinase inhibitor, and ustekinumab, an anti-interleukin-12/23 antibody, as potential treatments for dual-refractory colitis.¹⁰ In addition, fecal microbiota transplantation has demonstrated approximately 75% efficacy in a small series of 15 refractory cases.¹⁹ Other therapies, including calcineurin inhibitors (tacrolimus) and extracorporeal photopheresis have been described in case-based evidence.^{5,17} Given that many of these options carries a risk of interfering

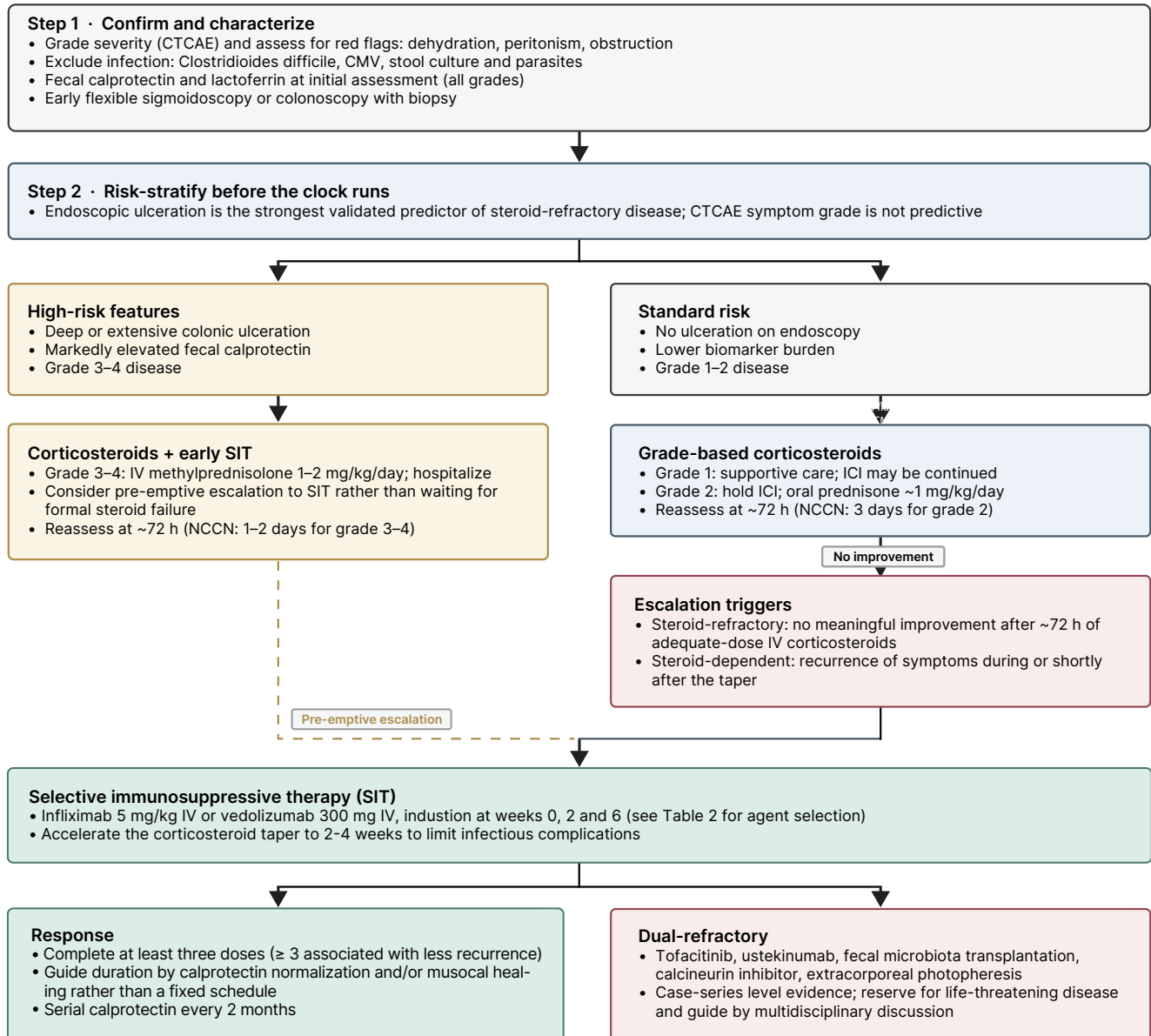


Figure 1. Risk-stratified escalation flow diagram in immune checkpoint inhibitor-induced colitis; *courtesy of Rami Habib, MD, Wilson H. Miller Jr., MD, PhD, and Khashayar Esfahani, MD.*

Abbreviations: ICI: immune checkpoint inhibitor; CTCAE: Common Terminology Criteria for Adverse Events; CMV: cytomegalovirus; IV: intravenous; SIT: selective immunosuppressive therapy; NCCN: National Comprehensive Cancer Network.

with antitumor immunity and that the evidence remains at the case-series level, their use should be reserved for life-threatening, multiply refractory disease and guided by multidisciplinary discussion.

Duration of Therapy

The optimal duration of biologic therapy for ICI colitis is not firmly established; however, retrospective data and guidelines support

a minimum of three infusions (at weeks 0, 2, and 6) as the standard induction course. The NCCN guidelines note that “receipt of three or more doses has been associated with less frequent colitis recurrence.”¹⁰ Corticosteroid tapering should proceed in parallel. When corticosteroids are used alone, guidelines recommend tapering over 4–6 weeks, whereas with coadministration of infliximab or vedolizumab, an accelerated taper over 2–4 weeks is recommended to reduce

the risk of infectious complications.¹⁰ Rather than adhering to a fixed treatment duration, objective endpoints should guide the decision to discontinue therapy. The NCCN endorses serial fecal calprotectin monitoring (every 2 months) and repeat endoscopy to assess mucosal healing as tools to guide treatment duration, although neither are mandatory.¹⁰ Neither guidelines nor prospective data define a fixed interval for repeat endoscopy; it is reserved for confirming mucosal healing when it will change management, i.e. before ICI rechallenge and for re-evaluating refractory, persistent, or recurrent disease (with ileocolonoscopy when a prior limited exam was unrevealing).¹⁴ In clinical practice, treatment is continued until symptoms improve to grade ≤ 1 and, ideally, until calprotectin normalizes or endoscopic remission is confirmed. Patients presenting with deep ulceration or recurrent flares may require maintenance therapy beyond the standard three-dose induction.

Whether to continue maintenance biologic therapy when restarting the checkpoint inhibitor is among the most clinically consequential and least evidence-supported decisions in the management of ICI. The NCCN Principles of Immunotherapy Rechallenge recommend considering concurrent vedolizumab on immunotherapy resumption for patients with prior grade 2–3 colitis.¹⁰ Supporting this approach, a multicenter retrospective study of 138 patients has shown that concurrent SIT (infliximab or vedolizumab) during ICI rechallenge was independently associated with a reduced risk of severe colitis recurrence (OR 0.34, 95% CI 0.13–0.92) without a negative impact on survival outcomes.²⁰ Across ICI classes, the overall risk of recurrent ICI colitis upon rechallenge is approximately 30–37%, with a higher risk when switching from anti-programmed death ligand-1 (PD-1)/PD-L1 to anti-CTLA-4.¹⁷ Given the paucity of prospective data, clinical decisions must be individualized based on the severity of the initial colitis episode, the oncologic necessity of continued ICI therapy, and the availability of alternative cancer treatments.

An important concern in managing ICI colitis is whether immunosuppressive therapy compromises the antitumor efficacy of checkpoint inhibitor therapy. For corticosteroids, preclinical and clinical data suggest that prolonged exposure to high-dose courses may dampen antitumor responses, and one retrospective analysis reported an association between higher initial corticosteroid doses and reduced survival when

controlling for age and cancer type.²¹ In contrast, preclinical mouse models have paradoxically shown that TNF- α blockade can improve antitumor responses to ICI therapy, possibly by reducing TNF-induced apoptosis of tumour-specific T cells.^{22,23} For vedolizumab, the gut-selective mechanism is theoretically expected to spare systemic antitumor immunity, though it may interfere with mucosal antitumor responses in GI malignancies.⁵ At present, adequately controlled human studies are lacking, and the effects of either agent on antitumor responses remain uncertain.^{5,22}

Practical Synthesis

Three questions provide a practical framework for managing ICI colitis: when to escalate therapy, which agent to select, and how long treatment should be continued (**Figure 1** outlines the escalation pathway). Escalation to SIT should be triggered by steroid-refractory disease (no improvement after ~72 hours of intravenous corticosteroids) or steroid-dependent disease (recurrence on taper). However, earlier escalation should be considered in patients with endoscopic ulceration or markedly elevated stool biomarkers. The choice between infliximab and vedolizumab should be guided by clinical context: infliximab for acute, severe presentations requiring rapid control; vedolizumab when gut-selective immunosuppression is preferred, particularly if ICI rechallenge is planned or systemic infection risk is a concern. A minimum three-dose induction course is recommended, with treatment duration guided by calprotectin normalization and/or endoscopic remission rather than by a fixed schedule.

For patients resuming ICI therapy, concurrent maintenance vedolizumab should be considered to reduce recurrence risk. Throughout all stages of management, the overarching goal is to minimize cumulative steroid exposure.

The ongoing randomized trial comparing infliximab and vedolizumab (NCT04407247) is expected to provide the first prospective comparative efficacy data in ICI colitis. However, the currently available preliminary results are limited to 19 patients, and longer follow-up is needed.¹³ Whether upfront biologic therapy in high-risk patients improves outcomes over the current sequential approach requires further evaluation; the real-world data remains hypothesis-generating but not definitive.

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