



Corticosteroid Use in IBD: Focus on Budesonide and Localized Therapies

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Received: July 04, 2025

Published: July 28, 2025

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Abstract

Corticosteroids remain a cornerstone in the management of acute flares of inflammatory bowel disease (IBD), including ulcerative colitis (UC) and Crohn's disease (CD). Despite their effectiveness in inducing remission, systemic steroids are associated with substantial side effects, prompting the development of locally acting corticosteroids such as budesonide and rectal formulations. This article reviews the indications, benefits, and limitations of corticosteroid use in IBD, with a focus on appropriate selection and precautions in clinical practice.

Keywords: Corticosteroid; IBD; Budesonide; Localized Therapies

Introduction

Inflammatory Bowel Disease (IBD), encompassing Crohn's disease (CD) and ulcerative colitis (UC), is a chronic immune-mediated condition characterized by relapsing-remitting gastrointestinal inflammation. The mainstay of induction therapy in moderate to severe flares includes corticosteroids, which provide rapid symptom control. However, systemic corticosteroids (e.g., prednisolone) are associated with significant adverse effects and are unsuitable for maintenance therapy. In this context, budesonide, a second-generation corticosteroid with high topical anti-inflammatory activity and low systemic bioavailability, has emerged as an attractive option for induction therapy in specific phenotypes of IBD.

Systemic corticosteroids in IBD: indications and limitations

Systemic corticosteroids remain highly effective in inducing remission in IBD. A Cochrane meta-analysis by Ford, *et al.* (2011) [1] found that systemic corticosteroids induce clinical remission in 60–80% of patients with active Crohn's disease within 4–8 weeks. Similarly, they remain the standard of care in acute severe ulcerative colitis (ASUC), with intravenous hydrocortisone or methylprednisolone being first-line treatment as per ECCO and AGA guidelines.

However, systemic corticosteroids are burdened by a well-established profile of adverse effects including hyperglycemia, hypertension, osteoporosis, adrenal suppression, infections, and psychiatric disturbances. Prolonged use beyond 12 weeks is strongly discouraged, and their inability to induce mucosal healing or prevent relapse limits their role to short-term induction only.

Budesonide in crohn's disease

Budesonide is a potent corticosteroid with approximately 90% first-pass hepatic metabolism, significantly reducing systemic exposure. It is formulated as

- Budesonide EC (Entocort®): targeting terminal ileum and right colon.
- Budesonide MMX (Cortiment®): extended-release formulation for colonic delivery.

In mild to moderate ileocecal Crohn's disease, budesonide 9 mg daily is approved for induction therapy. In a pivotal trial [2], budesonide showed superior efficacy over placebo (remission rates 51% vs 20%) and comparable efficacy to prednisolone (60% vs 58%) with significantly fewer glucocorticoid-related adverse events (28% vs 60%).

A 2008 Cochrane review [3] concluded that budesonide is superior to placebo and 5-ASA, but less effective than conventional systemic corticosteroids in inducing remission in CD. However, due to its favorable safety profile, it is recommended as first-line therapy for localized, non-fistulizing ileocecal CD, especially in steroid-dependent patients or those at risk of steroid-related complications.

Budesonide in ulcerative colitis

Historically, corticosteroids were reserved for moderate to severe UC flares unresponsive to 5-ASA therapy. However, budesonide MMX, due to its colonic release profile, has been investigated as an oral option in UC.

The CORE I and CORE II trials [4] evaluated budesonide MMX 9 mg vs placebo and mesalamine in patients with mild-to-moderate UC. Budesonide MMX demonstrated statistically significant higher remission rates compared to placebo (17.9% vs 7.4%; $p = 0.0143$) and was non-inferior to mesalamine. Moreover, the incidence of glucocorticoid-related side effects remained low and comparable to placebo.

This led to the approval of budesonide MMX for induction of remission in mild-to-moderate UC, particularly in patients intolerant to or unresponsive to 5-ASA. It is not recommended for use in acute severe UC or in extensive colitis with deep ulcerations.

Topical corticosteroids: Rectal enemas, foams, and suppositories

Topical corticosteroids are crucial in managing distal UC (proctitis or left-sided colitis). Rectal hydrocortisone and budesonide enemas or foams offer direct anti-inflammatory action with minimal systemic absorption.

A study by Marteau, *et al.* (1998) [5] compared budesonide enema with 5-ASA enemas in active distal UC and found similar efficacy in achieving remission, with significantly fewer side effects in the budesonide group. Further trials have shown budesonide foam to be superior to placebo and non-inferior to mesalamine suppositories in proctitis, offering improved patient comfort and compliance.

Clinical recommendations support combining rectal corticosteroids with oral 5-ASA in moderate distal disease or using them as monotherapy in mild proctitis. The optimal frequency is once daily administration, preferably at bedtime to enhance retention.

Precautions and Monitoring

When prescribing corticosteroids in IBD:

- Screen for latent infections (TB, hepatitis B) in patients at high risk.
- Counsel patients on short-course duration (<8-12 weeks).
- Provide bone protection if systemic steroids are used >3 months.
- Taper gradually to avoid adrenal insufficiency.
- Monitor glucose, BP, psychiatric symptoms, and infection signs.

Patients on budesonide typically do not require bone protection due to low systemic exposure, though adrenal suppression can occur with prolonged or repeated use.

Conclusion

Corticosteroids, including systemic and locally acting agents, are foundational in the induction of remission in IBD flares. Budesonide offers a targeted, safer alternative to traditional corticosteroids, particularly in mild-to-moderate ileocecal CD and colonic UC. Local rectal corticosteroids remain effective for distal disease. Appropriate selection based on disease location, severity, and patient risk profile is essential to optimize outcomes while minimizing harm. Long-term strategies must transition to steroid-sparing agents such as immunomodulators or biologics.

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