Microscopic Colitis Foundation

Newsletter Archive

Wayne's Blog

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We feel that it will be helpful to provide an overall summary of the various treatments for microscopic colitis (MC), and their reported effectiveness. Gathering this important information together in a comprehensive series of articles will hopefully give patients a basic understanding of the various treatment options, and how they might work together to achieve improvement of symptoms and attain remission.

PART 2

A Closer Look at Medications Used To Treat MC

by Wayne Persky

As we discussed in Part 1 of this series, there have been precious few changes in the medical treatment of MC in the last couple of decades. Here's what the official medical guidelines for treating microscopic colitis (MC) recommend

treating microscopic colitis (MC) recommend:

First-line drug — oral budesonide.

Multiple societies (the American Gastroenterological Association in the US, and the European Microscopic Colitis Group) recommend budesonide as initial therapy for active MC and, when needed, for maintenance (American Gastroenterological Association, 2015, November 13)[1].

Why is budesonide effective for controlling MC?

Because it's a corticosteroid, budesonide suppresses inflammation, primarily by suppressing mast cell activity. Entocort EC is no longer available, but it was designed as controlled-release capsules to target the terminal ileum and the colon, so that systemic effects are minimized. The patent on Entocort EC expired years ago, so it has now been replaced by generic versions.

Uceris (a form of budesonide labeled for UC) is designed as a controlled-release capsule that targets the colon. For most patients, budesonide enhances bile acid absorption (which takes place in the terminal ileum), and reduces the malabsorption problem associated with MC. This combined effect not only suppresses the symptoms of MC, but allows many patients to actually gain weight.

> **Budesonide is the most common initial therapy.**

> **Controlled release capsules target this steroid to the intestine, minimizing side effects.**

Nutrient absorption improves because:

Even though most nutrients are absorbed in the small intestine (especially the duodenum and jejunum), active MC can interfere with digestion in several ways:

- While MC is “defined” as colonic inflammation, research (especially older work) shows that many patients also have low-grade inflammation in the ileum or even jejunum. This can impair absorption of fats, fat-soluble vitamins, bile acids, and minerals.
- Rapid transit due to active diarrhea shortens contact time between food and absorptive surfaces, so even if the mucosa itself is intact, less nutrient absorption occurs.
- Mucosal barrier disruption involves inflammatory mediators and increased permeability in the colon that can have systemic effects that disrupt small-intestinal permeability and production of digestive enzymes.

By reducing inflammation and normalizing motility, budesonide indirectly:

- Allows food to stay in the intestine longer.
- Restores a more normal mucosal environment, improving digestion and uptake in the small bowel.
- Improves bile acid handling, reducing fat malabsorption.

Note that:

Budesonide does not directly “increase absorption” of nutrients. Instead, it reduces the inflammatory process and rapid transit that interfere with nutrient absorption. When symptoms are reduced (less diarrhea, less permeability, less immune activation), the small intestine can go back to doing its job more effectively.

Most gastroenterologists usually prescribe:

9 mg of oral budesonide once daily, for 6 to 8 weeks, taken first thing in the morning, in order to get the maximum response. 70–80% of MC patients typically experience a cessation of watery diarrhea within the first few weeks. But after the initial treatment is ended, relapse is common, and retreatment is often attempted. If relapse occurs, patients may undergo repeat induction or transition to a lower-dose maintenance regimen (often 6 mg/day or 3 mg/day, sometimes alternating days).

Some gastroenterologists tailor this regimen based on patient age, comorbidities, and tolerance, but 9 mg of budesonide daily, for 6–8 weeks, remains the most widely used starting point for newly-diagnosed MC patients.

How effective is budesonide?

Randomized trials show about 80–90% of MC patients achieve clinical remission by 6 weeks in collagenous or lymphocytic colitis (Miehlke et al., 2009)[2]. Various published studies show that when a budesonide treatment is ended, relapse rates range from roughly 40–80%.

Why do so many MC patients relapse

when their initial budesonide treatment is ended? This hasn't been explored by researchers so there's no medical data to support the possibilities listed here, but it's very likely that relapse is caused mostly by one or more of 3 possibilities:

1. Tapering of the budesonide dose is done too rapidly (as the treatment is ended), causing a relapse of the disease due to mast cell rebound, resulting in a spontaneous elevated mast cell spike as mast cell populations attempt to rebuild.
2. Because inflammation is often the most concentrated in the terminal ileum of MC patients, their BAM may have been masked by the budesonide, and diarrhea returns after the BAM is no longer suppressed by the budesonide treatment.
3. Food sensitivities may show up, causing some patients to relapse because their immune system begins to detect gluten or other food sensitivities that were masked while they were taking budesonide.

Regarding dose tapering of budesonide:

When ending a budesonide treatment, although most clinicians recommend tapering the dose, most of them don't recommend taking the dose reduction tapering far enough to be optimally effective. The experiences shared by many budesonide users who are members of our discussion and support forum, show that longer, more drawn out dose tapering is far less likely to result in a relapse.

When the prescribed treatment tapering ends (one 3 mg capsule per day), most users begin taking a 3 mg capsule every other day, for a week or so, followed by a capsule every 3rd day for a week or so, followed by a capsule every 4th day for a week or so. And many users go on to take a capsule every 5th day, or 6th day, or even longer. And any patients who don't feel secure when tapering at that rate, tend to stay at each dose level longer, for example, at least a couple of weeks before going on to the next level.

Maintenance strategies using budesonide:

Historically, budesonide was stopped after the initial 6–8 week course. But so many people relapse within weeks to months after the treatment is ended that many patients cycle through repeated treatments. Growing evidence supports the continuous use of low-dose budesonide in patients who relapse. Typical treatment regimens consists of:

- 6 mg per day
- 3 mg per day
- 3 mg every other day (for highly sensitive patients or those concerned about side effects)

Long-term studies show this approach can keep 70–80% of patients in remission. Some physicians reduce the dose gradually after induction, from 9 mg to 6 mg to 3 mg over several weeks., With the goal of finding the lowest effective dose that maintains remission. Note that not all patients need long-term therapy — some are able to remain in remission after tapering off budesonide.

A few gastroenterologists allow patient directed retreatment.

This involves restarting budesonide when the patient suffers a relapse and then ending the treatment. This is less common, but sometimes preferred by patients who are reluctant to take daily steroids

Regarding maintenance treatments:

If symptoms recur after tapering the dose and then stopping the treatment, the lowest effective maintenance dose (often 3–6 mg/day) is used. A 2024 meta-analysis of 35 studies based on 1657 MC patients treated initially with budesonide, and 146 patients subsequently treated for maintenance, found pooled maintenance remission rates of approximately 84%, and relapse rates of approximately 53% after stopping. In other words, about half will flare if budesonide is withdrawn (Tome, Tariq, Hassett, Khanna, and Pardi, 2024)[3].

Long-term budesonide use is relatively safe.

Budesonide has low systemic absorption, and side effects are much milder than with systemic steroids, but long-term use can still affect:

- Bone density
- Glucose metabolism
- Eye health (cataracts, or glaucoma are possible)

Therefore, regular monitoring is recommended for patients on prolonged low-dose therapy.

> **BAM (bile-acid malabsorption) is common.**

> **Bile-acid sequestrants are helpful.**

Bile acid sequestrants:

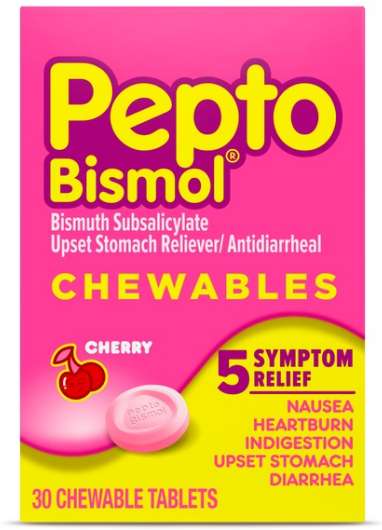
Bile acid sequestrants such as cholestyramine, colesevelam and colestipol are useful when bile-acid malabsorption (BAM) coexists with MC (which is common), or when patients are budesonide-dependent. Contemporary studies suggest about two-thirds respond, and bile acid sequestrants may reduce budesonide dependence in approximately 50% of patients (Pass, 2023, October 11)[4].

Detailed treatment information regarding the use of bile acid sequestrants was described in the September, 2025 newsletter, and because the discussion is somewhat long, rather than repeating all that information in this newsletter, here is a link where that newsletter can be read or downloaded:

https://www.microscopiccolitisfoundation.org/uploads/5/8/3/2/58327395/newsletter_september_2025.pdf

Antidiarrheals:

Loperamide (Imodium) can be used as a first-line treatment in mild disease, or as an add-on to help suppress urgency (when leaving the house to run errands), or for reducing the risk of nocturnal stools. Loperamide does not suppress the inflammation that causes MC, but it slows down motility to reduce the urgency associated with MC.



Bismuth:

Bismuth subsalicylate has mild anti-inflammatory properties. It was the first treatment shown by a published medical trial to be effective for controlling MC, almost 3 decades ago. In the trial, eight chewable 262-mg bismuth subsalicylate tablets per day for 8 weeks, brought resolution of diarrhea for 75% of those who remained in the trial (9 of the 12 from the original 13 who started the trial), and remission remained in effect after a 7–28 month follow-up, with no patients reporting adverse effects (Fine and Lee, 1998)[5]. Average time to respond to the treatment was 2 weeks.

Note however, that Dr. Fine (co-author of the study) no longer recommends the use of bismuth subsalicylate to treat MC due to the risk of neurological problems caused by a possible bismuth accumulation in the body. Bismuth subsalicylate (the active ingredient in Pepto-Bismol) can accumulate in the body, mainly when its normal clearance pathways are impaired, or when excessive doses are used for a prolonged period. There are at least 4 ways that can happen:

Impaired kidney (renal) function:

Both bismuth and salicylate components are excreted through the kidneys. In people who have kidney disease, or reduced glomerular filtration rate (GFR), elimination slows dramatically, leading to a buildup. Accumulated bismuth can deposit in the brain, causing neurotoxicity (encephalopathy, confusion, tremor, muscle twitches) and sometimes darkening of the gums, or skin.

Prolonged or high-dose use:

Even with normal kidneys, long-term daily dosing can cause gradual tissue accumulation, because a small fraction of bismuth remains in the body after each dose. Cases of bismuth encephalopathy have occurred after months to years of chronic use, particularly with older high-dose bismuth formulations. Not surprisingly, the FDA-approved over-the-counter duration limit (for example, Pepto-Bismol) is no more than 2 days for diarrhea without medical supervision.

Metabolic or drug-interaction issues:

Accompanying salicylate drugs (aspirin, diflunisal, for example) raise systemic salicylate levels and may enhance toxicity. Chelating agents (such as dimercaprol, penicillamine) can alter bismuth distribution and sometimes worsen central nervous system accumulation. Acidic GI conditions or low dietary sulfur reduce bismuth precipitation in the gut, increasing absorption fractionally.

GI permeability or inflammatory disease:

Conditions that increase intestinal permeability (IBD, including celiac disease and microscopic colitis) can enhance absorption of compounds that are normally minimally absorbed — including bismuth. Normally, less than 1% of ingested bismuth enters circulation but intestinal inflammation may increase that.

Laboratory or clinical signs of bismuth accumulation:

- Urine or blood bismuth levels rise long before symptoms.
- Toxicity usually appears when plasma levels exceed about 50 µg/L.
- Symptoms can include confusion, ataxia (balance or coordination issues), tremor, myoclonus (involuntary, sudden, and brief muscle contractions),dark lines on gums, and sometimes kidney damage.

Nevertheless, the American gastroenterological Association still recommends bismuth subsalicylate when budesonide isn't feasible (American Gastroenterological Association, 2015, November 13).

Other anti-inflammatories — mesalamine and systemic steroids:

The American Gastroenterological Association additionally recommends mesalamine (5-aminosalicylic acid [5-ASA]). Mesalamine is a relatively old drug that's significantly less effective than budesonide, but it's sometimes tried if budesonide cannot be used. Prednisone/prednisolone can help short-term when budesonide is not an option, but adverse-side effects mean that it cannot be used for anything other than short-term treatments.

Immunomodulators and biologics are sometimes used for refractory disease.

For patients who fail or cannot maintain remission on budesonide, the treatment guidelines support thiopurines (azathioprine/6-MP) or biologics (anti-TNF or vedolizumab) in selected cases. The use of Methotrexate is discouraged (Miehlke, et al., 2021, February 22)[6].

What about a surgical option?

The United European Gastroenterology and European Microscopic Colitis Group still recommends surgery in some extreme cases. They mention that diversion or subtotal colectomy, for example, is rare and reserved only for severe, treatment-refractory MC (Miehlke, et al., 2021, February 22). And indeed, old (published 3 decades ago) research data claims that fecal stream diversion can resolve collagenous colitis (Järnerot, Tysk, Bohr, and Eriksson, 1995)[7]. But isn't this a bit like burning down the barn to get rid of the rats?

In summary, the basic steps involved in the medical treatment of MC include:

1. Confirm diagnosis with segmental colon biopsies (rectal biopsies alone can miss MC) and rule out infections, celiac disease, and other causes of watery diarrhea.
2. Stop potential triggers (NSAIDs, PPIs, SSRIs/SNRIs, and other suspected medications) where clinically safe.
3. For mild disease, start loperamide, make dietary and lifestyle adjustments, assess for BAM.
4. For moderate to severe disease (3 or more stools/day or one or more watery stools/day), budesonide at 9 mg daily is recommended for 6–8 weeks. Tapering the dose should be used if the diarrhea is controlled.
5. If relapse occurs, consider a maintenance dose of 3–6 mg/day of budesonide. Discuss bone protection and monitoring with your health care provider.
6. If budesonide-dependence or intolerance is encountered, consider bismuth or a short course of prednisone, if needed. Check and treat coexisting conditions (BAM, SIBO, for example) (Nielsen, Fernandez-Banares, Sato, and Pardi, 2022)[8].
7. Refractory cases can be referred for thiopurines or biologics — very rarely surgery (Miehlke, et al., 2021, February 22).

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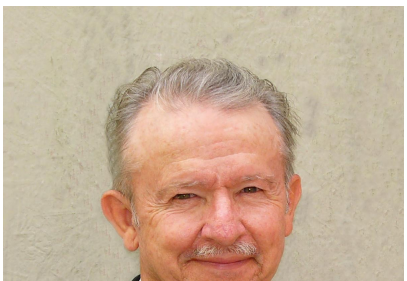

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ARE WE GETTING ENOUGH VITAMIN B12?

HORMONE REPLACEMENT THERAPY

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ALTERNATIVES TO ANTIBIOTICS

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*** The availability of the iPhone app in England and Europe was delayed because of extra time needed for approval. If you couldn't download earlier, try again.

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