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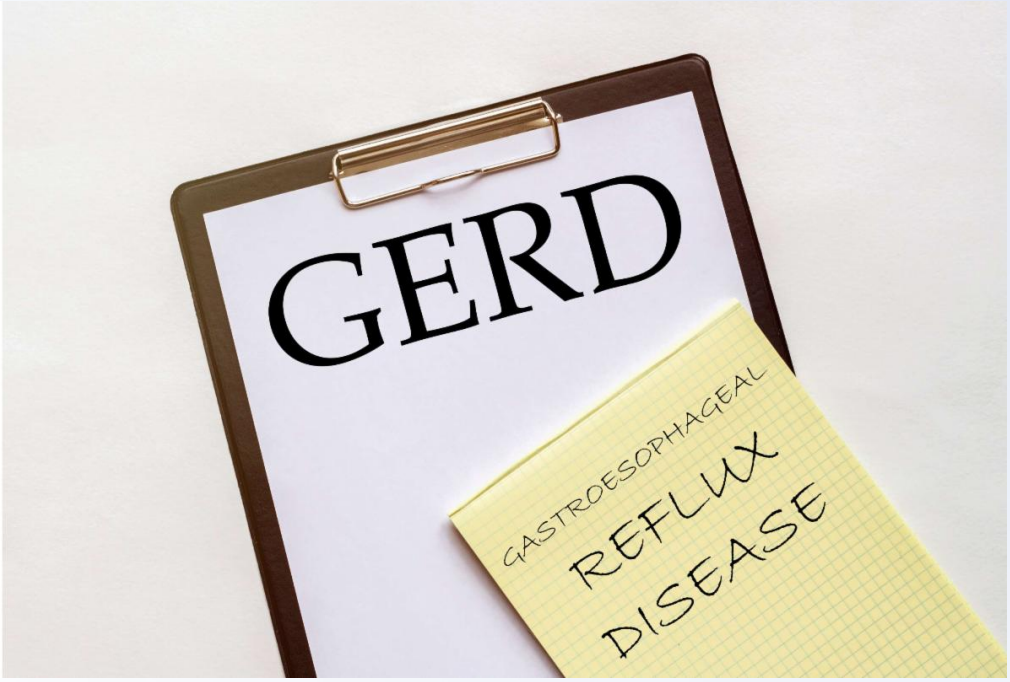


GERD Is a Complex Issue and Typically Difficult to Resolve

by Wayne Persky

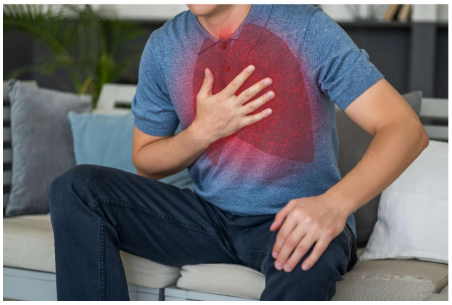
Gastric reflux, often leading to the development of gastroesophageal reflux disease (GERD) is a common problem among microscopic colitis (MC) patients. The medical approach to resolving this issue often leaves a lot to be desired, and progress toward the development of new medical treatments seems to be excruciatingly slow. The online website for medical professionals, Medscape, recently published an article describing new insights into the diagnosis and treatment of GERD (Yasgur, 2025, March 18).[1]

The article emphasizes that GERD is not a singular condition but a multifaceted clinical issue with overlapping symptoms, and a growing list of comorbidities. New research reveals not only its rising prevalence but also the need for refined diagnostic strategies and multidisciplinary care approaches.

> **GERD is multifaceted.**

> **It requires a multidisciplinary approach.**

GERD is defined as



the reflux of stomach contents into the esophagus causing symptoms and/or complications. It affects between 18% and 28% of North Americans and accounts for over 4.7 million medical visits annually in the U.S. Typical symptoms include:

- Heartburn
- Regurgitation

And for some patients, symptoms can include:

- Cough
- Sore throat
- Laryngitis
- Asthma
- Dental erosion
- Chest pain not associated with cardiac issues

But GERD typically involves much more than just acid reflux.

The disease involves a weakened lower esophageal sphincter (LES), impaired esophageal capacity, mucosal cellular changes, and in some cases, delayed gastric emptying (gastroparesis). Inflammation triggered by cytokine and chemokine release is typically involved, with microaspiration (the subclinical or unnoticeable entry of small amounts of fluid or food into the airways and lungs), and nerve reflexes mediated by the vagus nerve (the gut brain connection) that contribute to various abnormal esophageal issues.

Not only can GERD exacerbate asthma, but asthma can exacerbate GERD by weakening the LES. And it's known that certain asthma drugs such as beta agonists and steroids can decrease the strength of the LES. There can be overlapping symptoms with conditions like postnasal drip, vocal cord dysfunction, and laryngopharyngeal reflux (silent reflux, or asymptomatic GERD)

Research shows a bidirectional relationship between GERD and asthma.

In two related studies, it was shown that patients who had GERD had a 1.46 times higher hazard ratio (HR) for developing asthma, and patients who had asthma had a 1.36 times higher HR for developing GERD (Kim, Min, Oh, and Choi, 2020).[2]

GERD can cause hidden risks.

Adding to the growing body of evidence connecting GERD with pulmonary complications, a 2019 case-

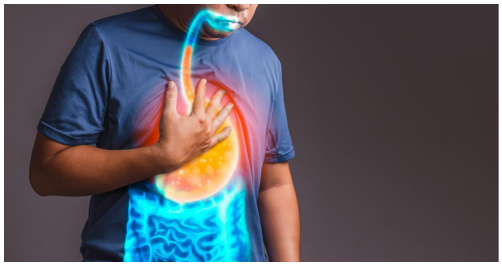
control study published in Gastroenterology and Hepatology From Bed to Bench revealed that even GERD patients without any respiratory symptoms may have subtle, yet significant, lung function impairments. This finding challenges the long-held assumption that respiratory involvement in GERD is limited to symptomatic individuals and underscores the disease's broader physiological reach (Nazemiyeh, Nouri-Vaskeh, Somi, Saeedi, and Sharifi, 2019).[3]

In this study, 78 GERD patients who had no history of pulmonary complaints were compared to 93 healthy controls using detailed pulmonary function tests. While overall lung volumes were within normal ranges in both groups, notable differences emerged in airway dynamics. Specifically, the GERD group showed a significantly lower FEV_1/FVC ratio (Forced Expiratory Volume in the first second/Forced Vital Capacity) and reduced maximal mid-expiratory flow (MMEF), which are markers that indicate small airway dysfunction. Moreover, airway resistance at 5 Hz (R5Hz) was significantly elevated in GERD patients, suggesting increased obstruction in the peripheral airways.

> **GERD can cause pulminary issues.**

> **Lung function screening is important.**

These findings imply that GERD may contribute to subclinical small airway disease, possibly via mechanisms such as microaspiration or inflammation mediated by vagal nerve activation, even in the absence of overt respiratory symptoms. This aligns with other evidence of GERD's role in worsening asthma and other airway conditions. Taken together, this study adds refinement to GERD diagnosis and management, emphasizing the need for clinicians to consider lung function screening in GERD patients, particularly those with comorbidities or at risk for respiratory disease.



The American College of Gastroenterology (ACG) recommends the following treatments:

- An eight week PPI trial for typical symptoms
- Endoscopy for patients with symptoms such as dysphagia (difficulty swallowing) or weight loss

- Ambulatory reflux monitoring (measuring the frequency, duration, and acidity of reflux events in the esophagus over a period of time, typically 24 hour) for refractory or atypical cases

Certain lifestyle modifications can be used to prevent reflux.

- Avoiding meals at least two hours before bedtime
- Elevating the head of the bed
- Avoiding trigger foods such as fried, spicy, caffeine, alcohol, for example
- Never sleep on your right side because this places the LES below the stomach
- Smoking cessation
- Weight loss may help in some cases, but that's usually not a problem for MC patients.

Medication options include:

- Proton pump inhibitors (PPIs)
- H2 receptor antagonists (H2 blockers) such as cimetidine, famotidine, nizatidine, and ranitidine.
- Potassium competitive acid blockers (P-CABs) such as vonoprazan and tegoprazan

Note that H2 blockers have to be taken more often than PPIs (generally, with each meal) but they reduce stomach acidity without totally neutralizing it, and they don't weakened the LES. Therefore, they can be used to wean off PPIs. P-CABs are relatively new, and show some promising advantages over PPIs, such as working faster and more effectively than PPIs, and lasting longer, while presumably not causing the major disadvantage of weakening the LES (but further studies are needed).

PPIs have been shown to:

- Suppress gastric acid production by irreversibly blocking the proton pump action of the stomach, which exchanges potassium from the contents of the digestive system with hydronium from cytoplasm in parietal cells in the stomach, and produces the enzyme primarily responsible for the acidification of the stomach contents and the activation of the digestive enzyme pepsin
- Reduce acidity of the stomach contents, which makes any refluxed material less irritating to the esophagus
- Help heal acid-related damage, such as esophagitis, and ulcers caused by exposure to highly acidic gastric contents.

Unfortunately, PPIs also:

- Do not stop reflux from occurring (they only mask the problem by reducing the acidity
- Tend to reduce the clamping strength of the LES, and in some cases, make reflux episodes more frequent, albeit less acidic.
- Negatively affect the digestion of food and the absorption of certain nutrients and proteins because of

- Negatively effect the digestion of food and the absorption of certain nutrients and proteins because of their suppression of gastric acid production
- Trigger reactions for many MC patients, and PPIs can increase the risk of developing MC

In other words, PPIs not only do not stop acid reflux, but they make the problem more likely to be chronic by weakening the muscle strength of the LES, and interfering with digestive capabilities. Consequently, the use of PPIs is an iatrogenic recipe for creating a state of hypochlorhydria (low stomach acid, and in some cases, increasing the risk of developing MC.

The FDA recommends limiting PPI use to 20 mg daily for 4 to 8 weeks.

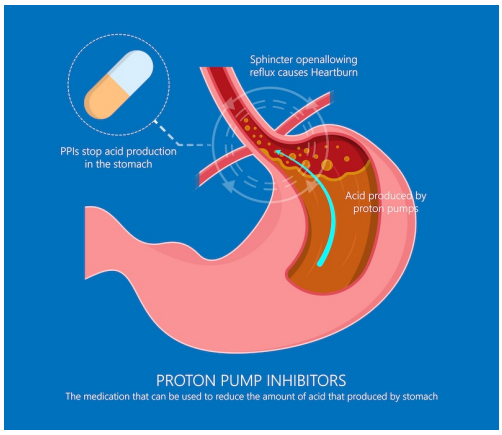
But both doctors and patients often ignore that recommendation, and some patients take PPIs for years. Part of the problem is that when a patient tries to wean off a PPI, they typically experience a rebound effect of hypersecretion of stomach acid, and this tends to be painful, because of the fact that the PPI never stopped the reflux, it only neutralized the acid. So the prompt return of symptoms causes the patient to resume the PPI treatment. And the longer PPIs are used, the more likely the rebound effect of symptoms is likely to be worse than it was before the PPI treatment was originally initiated. This tends to become a vicious cycle, because the longer a PPI is used, the weaker the LES becomes, further reducing the likelihood of ever weaning off the drugs.

PPI use (especially long-term use) often leads to these deficiencies:

- Vitamin B12 — stomach acid helps release B12 from food proteins
- Iron — stomach acid promotes the conversion of ferric iron (Fe^{3+}) to the more easily absorbed form known as ferrous iron (Fe^{2+}), and PPIs may reduce non-heme iron absorption
- Calcium, particularly the carbonate form, which requires acid for optimal absorption, possibly contributing to osteoporosis risk with long-term PPI use
- Magnesium — even the FDA warns about the risk of developing hypomagnesemia from PPI use

And since stomach acid serves as a barrier to ingested pathogens, reduced stomach acid can promote bacterial overgrowth (SIBO) and alter the gut microbiota composition, which can affect nutrient metabolism and immune system function. Additionally, PPIs have been shown to cause gastroparesis in some individuals, contributing to unpleasant gastrointestinal symptoms.

So what's the take away for us as MC patients?



While PPIs remain the mainstay of the medical approach to GERD treatment, we know that although they're able to suppress stomach acid, they definitely don't stop the reflux, itself, and published medical research shows that they typically weaken the LES over time. This sets up a vicious cycle in which the underlying reflux continues or worsens, while digestive capacity is diminished due to hypochlorhydria. For MC patients who have been prescribed PPIs before their MC was diagnosed, this can present a major dilemma.

GERD is so commonly associated with MC, that many of us get caught in the PPI trap before we realize the risks associated with PPI treatment. For some of us, our MC may have even been triggered by PPI use. In any case, if we find ourselves dependent on the continuing use of PPIs, it's very unlikely that we will be able to control our MC by making the proper diet changes as long as we continue to take a PPI.

Here's why:

Incomplete digestion is the hallmark of MC, and the primary reason why we have increased intestinal permeability (leaky gut) that results in the development of food sensitivities and chronic inflammation caused by our immune system reactions against the antibodies that are triggered by those antigens (food

sensitivities). MC patients have compromised digestion primarily because of the inadequate production of digestive enzymes, so the last thing we need is low stomach acid to further compromise our ability to digest food, and increase the bloating, malabsorption, and partially digested food issues that contribute to diarrhea.

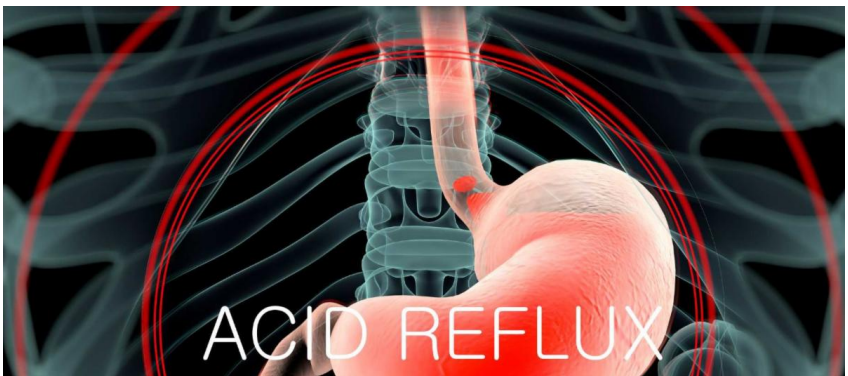
> **Long-term PPI use interfers with digestion.**

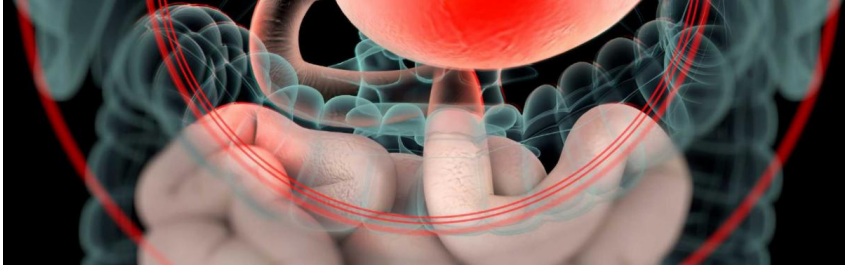
> **Nutrient deficiencies are common.**

Malabsorption is the primary cause of weight loss for MC patients.

Nutrient malabsorption is often the biggest problem some of us face when our MC is active, as it causes rapid weight loss for many of us, and a general decline in our health — especially our immune system's ability to perform its duty of protecting us from disease and healing the damage to our body. The decline in the functional ability of our immune system results in retarded healing and the development of additional autoimmune diseases for some of us. MC depletes vitamin D, vitamin B12, magnesium, calcium, and iron (although for most of us, supplementation with only vitamin D and magnesium is needed, and this is to strengthen the immune system). Only in cases involving long-term reactions should supplementation with B12, calcium, and iron normally be considered.

Notably, the long-term use of a PPI is associated with very similar nutrient malabsorption (B12, magnesium, calcium, and iron). So clearly, taking a PPI significantly amplifies the problems we already have with digestion and nutrient malabsorption. As difficult as it can be to coax MC into remission under ideal conditions, trying to reach remission while having to contend with the additional negative effects of PPIs, can make achieving remission an impossible goal.





For us, lifestyle changes appear to be the only practical way to control GERD.
In essence, GERD is not resolved with a pill. Managing it successfully requires a holistic approach that integrates lifestyle modifications, consideration of comorbidities (like asthma and postnasal drip), and if medications are used at all (to treat GERD), they should be used very carefully. Potentially, newer agents such as P-CABs, may offer benefits over PPIs. But for MC patients in particular, overreliance on acid-suppressing drugs may worsen gut health and lead to a cascade of complications.

So if we have reflux (or GERD) problems, we need to:

- Avoid eating at least two hours before bedtime
- Elevate the head of the bed a few inches with a brick or something similar under the legs
- Avoid trigger foods during the day, such as fried, spicy, caffeine, or alcohol, for example
- Never sleep on our right side because this places the LES below the stomach

If GERD is already a problem, then weaning off a PPI is going to take time, and is rarely easy, but it can be done with patience and perseverance, if we follow the suggestions listed above, and take an H2 blocker as needed. Initially, we may need to take a relatively high dose, but as time passes that should become less necessary so that we'll be able to wean down and eventually do without the H2 blocker.

Our pharmacist is usually a great source of information for details on dosing, unless we happen to be lucky enough to have a GP who recognizes the risks of using PPIs, and the advantages of using H2 blockers to wean off of PPIs. Prescription H2 blockers are stronger than over-the-counter versions, and may be needed in some cases. For many of us, an over-the-counter H2 blocker will adequately suppress the acidity, without weakening our LES, so that the LES can slowly regain its strength. After several months (or so) it will regain enough of its strength to stop the reflux, and eventually, we'll even be able to safely sleep on our right side after our LES completely regains its strength.

Check Out Wayne's Blog

He posts a new blog about once a week. Here are the titles of the most recent. Click the button at the top of the newsletter to access.

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BENEFITS OF EXTRA VITAMIN D DURING PREGNANCY

MICROSCOPIC COLITIS AND PREGNANCY

NEW RESEARCH DISPUTES THAT ANTIBIOTICS CAN CAUSE MC

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