

Controlling GERD Without the Use of Drugs

Acid reflux, which is often referred to as gastroesophageal reflux disease (GERD) when symptoms become chronic, can be tough to control, especially after using a proton pump inhibitor (PPI), and then trying to wean off the drug. This is due to the unfortunate reality that PPIs actually make acid reflux problems worse. The details of exactly how PPIs make acid reflux worse will be explored later in this article. And techniques that can be used to successfully wean off PPIs will also be discussed, because this class of drugs can be very difficult to stop using after they have been used for more than just a few weeks.

Medical researchers continue to discover additional ways in which PPIs are not only harmful, but can be downright dangerous for long-term health. What's surprising is that physicians continue to prescribe these drugs as if they were the greatest thing since sliced bread.

GERD is commonly associated with microscopic colitis and other IBDs.

Because of compromised digestion and related issues caused by inflammation, microscopic colitis (MC) is commonly associated with heartburn, acid reflux, or GERD. As a result of that association many MC patients have to deal with the problem until they are able to get their MC symptoms under control. But for many patients there is a catch — it may be impossible to get MC under control if their doctor has persuaded them to take a PPI to treat the acid reflux problem.

PPIs have been documented to cause many health problems, but possibly their cruelest side effect is the significantly increased risk of triggering MC. And the effect is so powerful that as long as a PPI is being used, treatments directed at controlling the patient's MC symptoms will typically be unsuccessful. In fact, drug-induced MC in general tends to frustrate attempts to control MC symptoms in almost every instance. The bottom line is that in cases where MC is either drug-induced or symptoms are perpetuated by the use of a drug, in almost every instance the use of the drug must be halted before the MC symptoms can be successfully controlled. And here's where an even more challenging dilemma comes into play — discontinuing the use of a PPI can be extremely difficult because when the treatment is ended, not only do the reflux symptoms return, but they are almost always worse than they were originally.

PPIs are rather insidious drugs. They trick users into believing that they are helping, by masking the effects of refluxed acid so that the acid cannot be detected. It will not be detected because it no longer burns the lining of the esophagus. Patients think that the reflux is being prevented, but instead they are only unable to feel it because it no longer burns. Because they can't feel the symptoms, they are happy campers— at least until the time comes to try to wean off the drug, or until some other health problem develops as a result of one of the numerous side effects of PPIs.

PPIs adversely affect digestion.

To understand how that can happen, let's consider what PPIs actually do in the body. PPIs are primarily prescribed to lower stomach acidity. And they are very effective at lowering gastric acid production. In fact, it could be argued that they are far too effective because they typically lower stomach acidity in most cases to less than 10 % of normal levels. [Reference 1](#) At that excessively-weakened acid level the stomach can no longer kill most food-borne bacteria, nor can it properly digest food. This implies that as food continues its journey through the gastrointestinal tract it will continue to be in-

completely digested at every subsequent step in the process. So it's not surprising that PPIs cause so many adverse health problems. These problems range from allowing food-borne bacteria to survive their trip through the stomach (because of the absence of an adequate acidity level that would normally kill them) to drastically reduced nutrient absorption (because of poor digestion) leading to osteoporosis and other adverse effects of inadequate absorption of vitamins and minerals.

Have you ever wondered why so many more people are getting sick from eating vegetables and other foods that contain E. coli, Listeria, or other pathogenic bacteria these days, and why the infections seem to cause so many fatalities these days? Back in the "good old days" such events were uncommon and if people did get sick, they rarely died as a result. But that was back before such a huge percentage of the population began to regularly use PPIs, and immune system suppressants, and other iatrogenic drugs. And unfortunately another major problem that is typically overlooked by physicians is the fact that PPIs do not treat the cause of acid reflux or GERD, they only treat the symptoms, and in doing so they actually intensify the cause of the problem.

PPIs cause physical changes over time that actually increase the risk of acid reflux.

Let's explore the details of how PPIs cause the very symptoms that they are prescribed to treat. This class of drugs actually increases the risk of acid reflux by virtue of their side effect of progressively weakening the lower esophageal sphincter (LES), and as the LES loses its clinching strength, this increases the likelihood that it may allow stomach contents to backflow into the esophagus. It appears that no research has ever been done to specifically document the details of exactly what happens to the LES clinching strength in the human body as a result of using PPIs. But fortunately research data are available describing what happens when the LES of a laboratory rat is exposed to a PPI. Yurtsever et al. (2011) wrote: [Reference 2](#)

***CONCLUSIONS:** Omeprazole, lansoprazole and pantoprazole had a suppressor effect on lower oesophageal sphincter contractions.*

So just as we would expect (based on the observation that stopping a PPI treatment results in symptoms that tend to be worse than they were before the treatment), the clinching strength of the rat LES is reduced when exposed to a PPI. There is no reason to doubt that the same effect would be seen in humans if the research were done using human subjects. But since most research is funded by the pharmaceutical companies and they are not known for funding research that is not likely to result in increased sales of drugs, that research is very unlikely to ever be done. PPIs are a huge cash cow for the pharmaceutical industry. And in the meantime physicians continue to ignore these research data, and sales of PPIs continue to make drug companies rich at the expense of the health of anyone who chooses to use them.

So it's no wonder then that when a PPI is used for a while and then discontinued, after the acidity recovers to a more normal level, the LES is incapable of clinching tightly enough to reliably prevent stomach acid from refluxing into the esophagus. As long as treatment with a PPI is continued, the increased reflux typically goes unnoticed, because the PPI effectively suppresses the acidity of the stomach (so that the refluxed stomach contents will not burn the lining of the esophagus). But after the PPI treatment is ended, and the effects of the drug wear off, stomach acidity returns to a more normal level and the symptoms become painfully obvious. Furthermore, the evidence demonstrated by this re-

search also implies that the strength of the LES can never increase as long as a PPI is being used — it can only decrease. This situation can definitely make weaning off a PPI a very difficult task unless one goes about it methodically.

Published medical research verifies the dark side of taking PPIs.

A few paragraphs back a reference was made to PPIs as "iatrogenic" drugs. While this might seem a harsh indictment, it's based on solid research that verified by means of a randomized, double-blind, placebo-controlled trial, that even many normal people who have no symptoms of acid reflux will develop acid reflux symptoms after using a PPI for as little as 8 weeks. In the abstract of an article, Reimer, Sondergaard, Hilsted, and Bytzer (2009) concluded that: [Reference 3](#), [Reference 4](#)

CONCLUSIONS: PPI therapy for 8 weeks induces acid-related symptoms in healthy volunteers after withdrawal. This study indicates unrecognized aspects of PPI withdrawal and supports the hypothesis that RAHS has clinical implications. (p. 80)

In that quote "RAHS" stands for rebound acid hypersecretion, a phrase that implies significantly increased acid production resulting from a rebound effect caused by withdrawing from the drug. Any drug that causes the very symptoms that it is prescribed to treat is clearly iatrogenic.

In 2013, researchers published data showing that in some cases PPIs can cause serious kidney damage (Sampathkumar, Ramalingam, Prabakar, and Abraham, 2013). [Reference 5](#) And now that the problem has been documented, reported cases appear to be on the increase (Charnow, 2014, June 25). [Reference 6](#)

But possibly the most worrisome aspect of using a PPI is the evidence that long term use of PPIs may significantly increase the risk of developing Alzheimer's disease and other forms of age-related dementia. [Reference 7](#), [Reference 8](#)

Certain treatment choices can make weaning off a PPI much easier.

Now that the risks of using PPIs are understood, it's time to consider ways for those who are using any of these drugs to discontinue their use while minimizing the risk of triggering the known rebound effect (RAHS). PPIs typically remain effective (for suppressing gastric acid production) for approximately 3 days. So a few days after a PPI treatment is discontinued, most of the medication's efficacy will have ended so that the LES will once again be free to begin its long journey back to normalcy as it attempts to regain its strength. But the tricky part is avoiding the dreaded rebound effect typically caused by ending the PPI treatment. As soon as the effects of the medication begin to wear off, the symptoms will almost surely return, and they will probably be worse than they were before the treatment began.

Therefore if a PPI has been used for months or years, stopping cold turkey is not likely to be a practical option. When trying to wean off a PPI, initially using a PPI on alternate days may work, but if symptoms seem persistent, H2 blockers can be used in place of a PPI on an every-other-day basis. An H2 blocker will lower stomach acidity almost as effectively as a PPI and this will help protect the esophagus, while still avoiding most of the side effect risks of PPIs. And it's much easier to wean off

an H2 blocker, than a PPI. H2 blockers are only effective for several hours however, so they may need to be taken as needed, such as before each meal. After a few days it should be possible to skip 2 days between PPIs and eventually wean off them completely, relying on H2 blockers, as needed to prevent symptoms, and then wean off the H2 blockers. Examples of H2 blockers include:

Famotidine (Pepcid AC, Pepcid Oral)

Cimetidine (Tagamet, Tagamet HB)

Ranitidine (Zantac, Zantac 75, Zantac Efferdose, Zantac injection, and Zantac Syrup)

Nizatidine Capsules (Axid AR, Axid Capsules, Nizatidine Capsules)

One option to consider is taking a vitamin D supplement, or increasing the dose if vitamin D is already being supplemented. Research shows that GERD is commonly associated with a vitamin D deficiency. The food sensitivities commonly associated with MC not only increase the risk of developing GERD, but they also prevent the absorption of many nutrients, vitamins, and minerals, including calcium, vitamin D, and magnesium. It is well known that IBDs are associated with vitamin D deficiency. In addition, corticosteroids are known to deplete vitamin D, and corticosteroids are commonly prescribed to treat MC. The combination of all these possibilities provides the opportunity for a triple whammy against the vitamin D reserves of many MC patients.

The average person uses at least 5,000 IU of vitamin D each day, according to the Vitamin D Council. [Reference 9](#) So it is very likely that most MC patients use significantly more.. Once they become deficient, they need even more vitamin D on a daily basis. Some patients find that taking a significant daily dose (up to 10,000 IU) can help to reduce acid reflux problems. This dosing level is safe for a few weeks or more, but before continuing for a much longer period it's always a good idea to test one's vitamin D level to make sure that it remains in the normal range.

Similarly, research shows that most Americans are magnesium deficient. [Reference 10](#) And many medications, especially antibiotics and PPIs, are known to deplete magnesium. The diarrhea associated with IBDs also depletes magnesium, so most people who have MC are very likely to be magnesium deficient. Because magnesium is an important electrolyte it has many important functions in the body. But the reason why a magnesium deficiency can cause GERD is because a magnesium deficiency tends to cause muscle weakness, and the LES is a muscle. Furthermore a magnesium deficiency can cause muscle cramps, and this would clearly play havoc with the functioning of the LES. So supplementation with magnesium can reduce the likelihood of having acid reflux in many cases. Topical magnesium in the form of oil sprays, foot soaks in Epsom salts, and even adding Epsom salts to bath water, can be very effective if done regularly. If oral supplements are used, chelated magnesium (magnesium glycinate) is the form least likely to cause diarrhea. There are many forms of oral magnesium supplements, and many of the cheaper forms (such as magnesium oxide for example) are poorly absorbed and can cause diarrhea.

And remember that adopting a low-carb diet can be very effective at resolving acid reflux or heartburn issues. [Reference 11](#) Many alternative medicine practitioners have successfully used a low-carb diet to resolve reflux and heartburn problems for many of their patients.

There is another trick that may be helpful for weaning off PPIs. Research shows that melatonin is effective for treating GERD. [Reference 12](#) Based on research using 3 mg of melatonin taken at bedtime, Kandil, Mousa, El-Gendy, and Abbas (2010) wrote:

Conclusion

The present study showed that oral melatonin is a promising therapeutic agent for the treatment of GERD. It is an effective line of treatment in relieving epigastric pain and heartburn. However, further studies are required to confirm the efficacy and long-term safety of melatonin before being recommended for routine clinical use.

Interestingly, the researchers noted that baseline evaluations showed that the GERD patients in the study had significantly lower levels of melatonin, when compared with the control subjects (who did not have GERD). And this deficiency of melatonin in GERD patients had been previously documented by Klupińska et al (2006). [Reference 13](#) Furthermore, Klupińska et al (2006) concluded that adequate amounts of melatonin would probably prevent the development of peptic ulcers:

The findings of this study support the notion that melatonin exerts beneficial influences on the upper digestive tract. It is likely that high or relatively correct secretion of melatonin is sufficient to prevent peptic changes in esophageal and duodenal mucosa.

For such a risky class of drugs, PPIs are enormously overprescribed.

Why most physicians are so quick to prescribe a PPI for virtually any patient who complains of acid reflux or heartburn symptoms is somewhat of a mystery. In most cases those symptoms are due to too little stomach acid, not too much. As we get older, our stomach tends to produce less gastric acid, not more. But physicians rarely order any tests to determine gastric acid levels. Instead they follow the recommendations of drug reps and reach for the prescription pad and add yet another patient to the long list of victims who should never have been prescribed a PPI in the first place.

Many people have found that the old home remedy for acid reflux or heartburn based on 2 or 3 teaspoons of apple cider vinegar in an 8-ounce glass of water before meals (or as needed) can be very helpful. But the fact that this helps suggests that more acid is needed, not less, which is prima facie evidence that PPIs are counterproductive.

Additional helpful tips for preventing acid reflux and indigestion.

Most people are already aware that for preventing acid reflux problems while sleeping, it helps to avoid eating for several hours before bedtime. And foods known to cause reflux problems may need to be avoided at all times when treating a persistent GERD problem, especially during the recovery period while trying to wean off a PPI treatment regimen. Examples of such foods include chocolate, alcohol, coffee, tomatoes, peppermint, and possibly others in some cases. These foods should definitely be avoided for several hours before bedtime, because they can almost guarantee reflux problems when lying down.

One very important trick to help prevent acid reflux or GERD issues while sleeping is to never lie on one's right side, because that position places much of the stomach higher than the LES, which will allow acidic stomach contents to backflow into the esophagus if the LES fails to clench tightly enough at all times. This is very important, especially if an antacid or H₂ blocker (or PPI) is being used. Some people find that elevating the head of their bed a few inches by placing the corresponding legs of the bed on blocks, can also help to prevent acid reflux problems.

Additional information can be found in the references listed below:

Reference 1. Study: Heartburn drugs can cause more heartburn

<http://www.npr.org/templates/story/story.php?storyId=112564382>

2. Proton pump inhibitors omeprazole, lansoprazole and pantoprazole induce relaxation in the rat lower oesophageal sphincter.

<http://www.ncbi.nlm.nih.gov/pubmed/21899545>

3. Proton-pump inhibitor therapy induces acid-related symptoms in healthy volunteers after withdrawal of therapy

<http://download.journals.elsevierhealth.com/pdfs/journals/0016-5085/PIIS0016508509005228.pdf>

4. Evidence that proton-pump inhibitor therapy induces the symptoms it is used to treat

<http://www.gastrojournal.org/article/S0016-5085%2809%2900780-X/fulltext>

5. Acute interstitial nephritis due to proton pump inhibitors.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3741979/>

6. PPI-Induced Acute Interstitial Nephritis May Be On the Rise

<http://www.renalandurologynews.com/acute-kidney-injury/ppi-induced-acute-interstitial-nephritis-may-be-on-the-rise/article/357629/>

7. Proton pump inhibitors: predisposers to Alzheimer disease?

<http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2710.2009.01100.x/full>

8. Risk of dementia in elderly patients with the use of proton pump inhibitors

<http://link.springer.com/article/10.1007%2Fs00406-014-0554-0>

9. Why does the Vitamin D Council recommend 5,000 IU/day?

<https://www.vitamincouncil.org/blog/why-does-the-vitamin-d-council-recommend-5000-iuday/>

10 GERD and heartburn: Medication reduces magnesium stores

<https://www.vitamincouncil.org/blog/gerd-and-heartburn-medication-reduces-magnesium-stores/>

11. Heartburn cured

<https://proteinpower.com/drmike/2005/11/25/heartburn-cured/>

12. The Potential Therapeutic Effect of Melatonin in Gastro-esophageal Reflux Disease

<http://bmcgastroenterol.biomedcentral.com/articles/10.1186/1471-230X-10-7>

13. Nocturnal secretion of melatonin in patients with upper digestive tract disorders.

<https://www.ncbi.nlm.nih.gov/pubmed/17218759?dopt=Abstract>