GERD, Heartburn Treatment Options

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Check insulin level as high levels may trigger more acid production, Chk for H. pylori.

Treatment options, try for at least 8-12 weeks.
1. Drink 6-8oz. of water after every meal.
2. Weight Loss
3. Trail of Betaine HCL & Pepsin.
4. Make sure you have 1-2 “large” bowel movements per day
5. Take 1-2 tsp of raw liquid honey anytime you feel the discomfort of heartburn or acid reflux.
7. Raise the front of your bed by 6-7 inches. Also try to sleep on your left side.
8. Chew gum after a meal.
9. Decrease fat intake. Studies show diets high in fat may be associated with a higher risk of GERD.
10. Increase intake of fiber from whole grains and beans. Eat 30grams of fiber per day
11. Sleep several hours after eating. If you suffer from acid reflux disease, you may be going to bed too soon after your evening meal. A shorter dinner-to-bed interval is significantly associated with an increased risk of GERD. It is generally recommended that patients with GERD refrain from eating within 4 hours of going to sleep.
12. Trial of Heartburn Tx 1tsp 2-3/day alone or in combo with GI Repair Nutrients 2-3 caps TID
13. Trial of Melatonin 6mg at bedtime
14. Trial of Probiotics – Acidophilus-Bifidobacter
15. Trial of pure D-Limonene 1000mg 1 cap daily
17. Trial of 1-2 tablespoonfuls of unfiltered apple cider vinegar after meals
18. Trial of Aloe Vera juice – 2oz approximately 20 minutes before a meal. It should NOT contain any aloe latex, aloin, or aloe-emodin compounds.
19. Trial of Slippery Elm Powder – 1 tsp. In 4-6 ounces of water 2-3 x a day.
20. DGL used 3-4x a day.
21. Mastic Gum 350mg TID
22. Pancreatin/Fungal Enzyme combo, 65% decrease in sxs of dyspepsia

How to “reduce” a Hiatal Hernia - This technique may bring the bulb of the stomach that is caught in the diaphragm down below the diaphragm where it belongs. This is called reducing a Hiatal Hernia.

• Drink a tall glass of water and eat an apple to get some weight in the stomach.
• Put your forearms on top of your head.
• Jump up and down, and every time your feet hit the ground exhale sharply to raise the diaphragm a little.
**Treating GERD brings up a quandary that one often encounters in the world of medicine. That is, in many cases two diametrically opposed theories may be proposed, both of them often sounding perfectly valid and, of course, both of them having their vehement proponents. Think of the low fat versus low-carb arguments that are raging through the dietary circles of this country as an example of how two competing theories for weight loss may, at first, sound equally valid. In many cases only the actual testing of each theory will show which is the right approach.

Regarding GERD, there are also two theories which at first both sound good. Since everyone accepts the fact that it is stomach acid that causes the problem of burning, the question is why is there too much acid in the stomach? One answer could be that the person is eating too much food that "tells" the body to secrete acid. Since protein foods are what causes the stomach cells to produce acid, the therapy is simple: stop eating so much protein. Then the stimulus to produce acid will be lessened, less acid will be produced and eventually the symptoms will abate.

The competing theory states that producing acid is a natural function of the stomach in response to the eating of food—any food. In fact, the acid helps the stomach and pancreatic enzymes assume their proper form, so without stomach acid the whole digestive system is thrown off. Stomach acid is beneficial in other ways in that stomach acid kills the invading microorganisms that we inevitably ingest with our food. Stomach acid thus protects us from infections, both acute and chronic, in our GI tract.

Furthermore, the very group of people who lacks stomach acid, that is the elderly, is the group that most often suffers from GERD. So in this case, the solution is not to inhibit production by eating less protein, but rather to increase protein (and fat) consumption so as to give the acid something to do, which is to digest the protein.

Which reasoning is correct?
A recent study done by Professor Yancy and his team at the gastroenterology department at Duke University examined this very question. The article was published in *Alternative Therapies* Nov/Dec 2001, Vol. 7 No. 6 under the title “Improvement of Gastroesophageal Reflux Disease After Initiation of a Low-Carbohydrate Diet: Five Brief Case Reports.” In this study, the Duke researchers took on people very much like yourself. They were mostly diabetic patients, often with a host of other medical problems. Furthermore, they were described as patients who had failed all other conventional therapies. In other words these were their most refractory patients with GERD.

Much to their amazement they report that in spite of continuing to smoke, drink coffee, and other GERD-unfriendly habits, in each case the symptoms of GERD were completely eliminated within one week of adopting a very low-carbohydrate diet (about 20 grams per day.) The patients were able to stop all antacids and prescription stomach medicines and this improvement continued even after they liberalized their carbohydrate intake to a more tolerable 70 gram per day. The researchers were unable to definitively say why this had occurred but they postulated that the lower-carb intake influenced the activity of various hormones that open and close the value between the esophagus and the stomach. By the way, this therapy is particularly appropriate for a diabetic, for it stabilizes the blood sugar (although you still need to carefully monitor your blood sugar, as you know.)

To address the question of the long term effects of taking antacid drugs, the main problem is simply that our stomach acid in not only necessary for protein digestion, but it protects us against a variety of gastrointestinal infections. Long term blocking of this acid is a very poor strategy indeed.

I have used this low-carbohydrate approach for the treatment of GERD for many years and with many patients. I can report that it is one of the most effective interventions that I use. It is not unusual for people to report relief even within a few days. There is no longer any doubt in my mind as to which of the above theories in correct.

**About the Author** - Tom Cowan, MD is a physician in private practice in San Francisco, California. He is the author of *The Fourfold Path to Healing*. Visit his website at [http://www.fourfoldhealing.com](http://www.fourfoldhealing.com).
Melatonin & GERD References

Mechanisms of esophageal protection, gastroprotection and ulcer healing by melatonin. implications for the therapeutic use of melatonin in gastroesophageal reflux disease (GERD) and peptic ulcer disease.
Brzozowska I, Strzalka M, Drozdowicz D, Konturek SJ, Brzozowski T.  
Author information
Abstract
Melatonin is a potent reactive oxygen metabolite scavenger and antioxidant that has been shown to influence many physiological functions of the gastrointestinal (GI) tract including secretion, motility, digestion and absorption of nutrients. The role of melatonin in gastroduodenal defense and ulcer healing has been the subject of recent investigations. Melatonin produced in the GI mucosa plays an important role in protection against noxious agents thus contributing to the maintenance of GI integrity and to esophageal protection, gastroprotection and ulcer healing. This review was designed to summarize the involvement of melatonin, conventionally considered as a major hormone of the pineal gland, in the maintenance of gastric mucosal integrity, gastroprotection, ulcer healing and intestinal disorders. Melatonin was originally shown to attenuate gastric mucosal lesions but controversy exists in the literature as to whether melatonin derived from the pineal gland, considered as the major source of this indole, or rather gastrointestinal melatonin plays predominant role in gastroprotection. Intragastric and central administration of exogenous melatonin and L-tryptophan, this indoleamine precursor, affords protection against gastric hemorrhagic damage caused by the exposure of gastric mucosa to variety of non-topical and topical ulcerogens such as stress, ethanol and ischemia-reperfusion. The speed of ulcer healing in experimental animals and humans is accelerated by melatonin. This indoleamine could be also effective against the esophageal lesions provoked by reflux esophagitis in animal models and prevents the incidence of GERD in humans. The melatonin-induced gastroprotection is accompanied by an increase in gastric blood flow, plasma melatonin concentration, enhancement in mucosal generation of PGE2, luminal NO content and plasma gastrin levels. Melatonin scavenges reactive oxygen metabolites, exerts anti-oxidizing and anti-inflammatory actions and inhibits the formation of metalloproteinases-3 and -9; both implicated in the pathogenesis of gastrointestinal injury and formation of gastric ulcers. Blockade of MT2 receptors by luzindole, significantly attenuated melatonin- and L-tryptophan-induced protection and increased the speed of ulcer healing and these effects were accompanied by an increase in the GBF and luminal content of NO suggesting that melatonin exhibits gastroprotection and hyperemia via activation of MT2 receptors and release of NO. The accumulated evidence indicates that the melatonin-induced gastroprotection and the enhancement in healing rate of gastric ulcers may involve the gastroprotective factors derived from the activation of PG/COX and NO/NOS systems as well as gastrin which also was shown to exhibit protective and trophic effects in the upper GI tract. Interestingly, pinealectomy, which suppressed plasma melatonin levels, markedly exacerbated gastric lesions induced by topical and non-topical ulcerogens and these effects are counteracted by a concurrent supplementation with melatonin. Evidence is provided that exogenous melatonin and that converted from its precursor, L-tryptophan, attenuates acute gastric lesions and accelerates ulcer healing via interaction with MT2 receptors due to an enhancement of gastric microcirculation, probably mediated by NO and PG derived from NOS and COX-1 and COX-2 overexpression and activity. The pineal gland plays an important role in the limitation of gastric mucosal injury and the acceleration of ulcer healing via releasing endogenous melatonin, which attenuates oxidative stress and exerts anti-inflammatory actions and anti-inflammatory actions.

Melatonin for the treatment of gastroesophageal reflux disease.
Werbach MR. 
Abstract
The enterochromaffin cells of the gastrointestinal (GI) tract secrete 400 times as much melatonin as the pineal gland; therefore, it is not surprising that research is finding that this indole plays an important role in GI functioning. In animal studies, it protects against GI ulcerations, and randomized clinical trials suggest its efficacy in treating functional dyspepsia and irritable bowel syndrome. Melatonin administration has been shown to protect against esophageal lesions in animals. Moreover, in a randomized, single-blind clinical trial of subjects with gastroesophageal reflux disease (GERD), the combination of melatonin with other natural supplements was found to be superior to omeprazole, a proton pump inhibitor (PPI). Its administration as a
Regression of gastroesophageal reflux disease symptoms using dietary supplementation with melatonin, vitamins and aminoacids: comparison with omeprazole.


Abstract

The prevalence of gastroesophageal reflux disease (GERD) is increasing. GERD is a chronic disease and its treatment is problematic. It may present with various symptoms including heartburn, regurgitation, dysphagia, coughing, hoarseness or chest pain. The aim of this study was to investigate if a dietary supplementation containing: melatonin, L-tryptophan, vitamin B6, folic acid, vitamin B12, methionine and betaine would help patients with GERD, and to compare the preparation with 20 mg omeprazole. Melatonin has known inhibitory activities on gastric acid secretion and nitric oxide biosynthesis. Nitric oxide has an important role in the transient lower esophageal sphincter relaxation (TLESR), which is a major mechanism of reflux in patients with GERD. Others biocompounds of the formula display anti-inflammatory and analgesic effects. A single blind randomized study was performed in which 176 patients underwent treatment using the supplement cited above (group A) and 175 received treatment of 20 mg omeprazole (group B). Symptoms were recorded in a diary and changes in severity of symptoms noted. All patients of the group A (100%) reported a complete regression of symptoms after 40 days of treatment. On the other hand, 115 subjects (65.7%) of the group B wished to substitute a natural treatment because of the risk of side effects. There was statistically significant difference between the groups (P < 0.05). This formulation promotes regression of GERD symptoms with no significant side effects.

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www.jpp.krakow.pl Review article P.C. KONTUREK1, T. BRZOZOWSKI2, S.J. KONTUREK2

STRESS AND THE GUT: PATHOPHYSIOLOGY, CLINICAL CONSEQUENCES, DIAGNOSTIC APPROACH AND TREATMENT OPTIONS

Stress, which is defined as an acute threat to homeostasis, shows both short- and long-term effects on the functions of the gastrointestinal tract. Exposure to stress results in alterations of the brain-gut interactions ("brain-gut axis") ultimately leading to the development of a broad array of gastrointestinal disorders including inflammatory bowel disease (IBD), irritable bowel syndrome (IBS) and other functional gastrointestinal diseases, food antigen-related adverse responses, peptic ulcer and gastroesophageal reflux disease (GERD). The major effects of stress on gut physiology include: 1) alterations in gastrointestinal motility; 2) increase in visceral perception; 3) changes in gastrointestinal secretion; 4) increase in intestinal permeability; 5) negative effects on regenerative capacity of gastrointestinal mucosa and mucosal blood flow; and 6) negative effects on intestinal microbiota. Mast cells (MC) are important effectors of brain-gut axis that translate the stress signals into the release of a wide range of neurotransmitters and proinflammatory cytokines, which may profoundly affect the gastrointestinal physiology. IBS represents the most important gastrointestinal disorder in humans, and is characterized by chronic or recurrent pain associated with altered bowel motility. The diagnostic testing for IBS patients include routine blood tests, stool tests, celiac disease serology, abdominal sonography, breath testing to rule out carbohydrate (lactose, fructose, etc.) intolerance and small intestinal bacterial overgrowth. Colonoscopy is recommended if alarming symptoms are present or to obtain colonic biopsies especially in patients with diarrhoea predominant IBS. The management of IBS is based on a multifactorial approach and includes pharmacotherapy targeted against the predominant symptom, behavioural and psychological treatment, dietary alterations, education, reassurance and effective patient physician relationship. When evaluating for the stress-induced condition in the upper GI tract, the diagnostic testing includes mainly blood
tests and gastroscopy to rule out GERD and peptic ulcer disease. The therapy for these conditions is mainly based on the inhibition of gastric acid by proton pump inhibitors and eradication of *Helicobacter pylori*-infection. Additionally, melatonin an important mediator of brain gut axis has been shown to exhibit important protective effects against stress-induced lesions in the gastrointestinal tract. Finally, probiotics may profoundly affect the brain-gut interactions ("microbiome-gut-brain axis") and attenuate the development of stress-induced disorders in both the upper and lower gastrointestinal tract. Further studies on the brain-gut axis are needed to open new therapeutic avenues in the future.

The potential therapeutic effect of melatonin in gastro-esophageal reflux disease.
BMC Gastroenterology 2010, 10:7
HYDROCHLORIC ACID SUPPLEMENTATION

Hydrochloric acid is a necessary part of digestion in the stomach. As we age the secretion of acid by the stomach can go down. One way of testing to see if more hydrochloric acid is needed by the stomach is to give a trial and see if digestion or intestinal symptoms improve. Hydrochloric acid supplementation is usually a very safe treatment when done under appropriate medical supervision. However, hydrochloric acid should never be used at the same time as aspirin, Butazolidin, Inodcin, Motrin or any other anti-inflammatory medication. These medications themselves can cause stomach bleeding and ulcers, as can steroid drugs. Using hydrochloric acid increases the risk of bleeding and ulcers so it must be used under medical supervision.

PROCEDURE:

1. On day one, start with one capsule (8 grains) of hydrochloric acid as Betaine HCL./Pepsin. Ideally, take it at the start of the meal. (NOTE: this means a full meal, not a snack) If you forget to take it at the beginning of the meal it can be taken during or right at the end of the meal.
2. Observe for any side effects, which may include stomach discomfort, pain, burning or additional gas. If these are not present proceed to step 3. If side effects occur (usually heartburn), take an antacid, drink some fluid or eat more food and the heartburn or discomfort will probably subside. Wait 2-3 days and try it once again. If you have the same reaction don’t take it any longer.
3. If you tolerate 1 capsule of Betaine HCL/Pepsin at the start of each meal then stay at this dose until your next appointment. A change in dosing schedule will be discussed at that time.