KRESSER INSTITUTE FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Gut Treatment Protocols: GERD, IBD & IBS, Part 1

Hey, everyone, in this presentation we're going to talk about specific treatment protocols for GERD; inflammatory bowel disease, or IBD; and irritable bowel syndrome, or IBS. So far, we've been talking about the treatment of the pathologies that lead to gut dysfunction, including SIBO, dysbiosis, fungal overgrowth, pathogens like bacteria and parasites. Now we're going to talk more about treatment of particular gut syndromes, diseases, and even symptoms.

Now obviously, the most important focus in these conditions is to address all of the underlying pathologies that we've reviewed so far, so you always want to make sure that you do that first or at least as part of your treatment plan. For example, if a patient has an ulcer and it's caused by H. pylori, obviously treating H. pylori is the first step, and it's going to be necessary for long-term resolution. Same thing if the patient has irritable bowel syndrome that is caused by SIBO. But in functional medicine, although we always want to focus on the root or the underlying cause, it's also true that sometimes we have to simultaneously address the branch, or the symptoms or expression of that underlying cause, and this is true for a couple of different reasons. Number one, sometimes we just need to give our patient more immediate relief than they can get in the longer-term process of addressing the underlying cause, if it's going to take a while. In other situations, it's not always possible to entirely address the underlying cause, so, for example, if the underlying cause is autoimmunity, there are a lot of things we can do to balance and regulate the immune system, but at least at present, we don't necessarily know how to cure autoimmunity in every situation, so we may need to take actions that help to ameliorate the symptoms and help the patient to live with the imbalance that they have if it's going to be relatively long term.

So let's start with acid reflux, GERD, and ulcer, since we were just talking about H. pylori most recently. I've written and spoken extensively on reflux and GERD. There's a free e-book on my website that we've included in the supplemental materials for this week, with a lot of good background info for the stuff that we're going to be talking about in this section, so I'm not going to rehash it all here. I'm sure many of you have already read it, but if you haven't, please download it and read it. I'm going to review the important highlights and treatment recommendations, along with some updates to what I've written in the past.

As I'm sure you know, conventional wisdom on heartburn and GERD is that it's caused by too much stomach acid. Never mind the fact that no scientists in the research community really believe that, and the research community as a whole moved on from that theory many years ago, if they ever even believed it at all. But it's still prevalent in the mainstream media and the general public's mind. If you ask kind of the average person on the street, that's what they'll tell you. There's very little evidence that GERD or heartburn is caused by excess acid production, and in fact, in an editorial on the treatment of GERD in the ***Journal of Gastroenterology***, the authors said treating

gastroesophageal reflux disease with profound acid inhibition will never be ideal because acid secretion is not the primary underlying defect.

So if it's not excess acid production that caused GERD, what is the primary defect? The accepted theory is that GERD is caused by malfunction of the lower esophageal sphincter, or LES, which allows acid to reflux into the esophagus. Of course, this leads to the next question, which is what's causing the LES malfunction in the first place? Again, according to current thinking, the answer is overeating, obesity, bending down after eating, lying down after eating, these kinds of mechanical changes, and consuming spicy or fatty foods. Now I don't disagree that these may play a role in some cases, but I don't think they're the primary cause.

Instead, I agree with microbiologist Dr. Norm Robillard's theory, which is that carbohydrate malabsorption leads to bacterial overgrowth, which in turn causes an increase in gas production and intra-abdominal pressure and leads to a malfunction of the lower esophageal sphincter. If we continue to look deeper, though, what might be causing the carbohydrate malabsorption in the first place, and are there any other causes of bacterial overgrowth that may precede carbohydrate malabsorption? I believe that cause, in many cases, is low stomach acid, which is known as hypochlorhydria.

One of the chief roles of stomach acid is to inhibit bacterial overgrowth. At a pH of three or less, which is the normal pH of the stomach, most bacteria can't survive for more than 15 minutes, but when stomach acid is insufficient and the pH of the stomach rises above five, bacteria begin to thrive. The gastro knockout mouse, which is incapable of producing stomach acid, suffers from severe bacterial overgrowth as well as inflammation, damage, and precancerous polyps in its intestines. It's also well-documented that acid-suppressing drugs promote bacterial overgrowth. Long-term use of Prilosec, one of the most potent acid-suppressing drugs, reduces the secretion of hydrochloric acid in the stomach to near zero. Stomach acid supports the digestion and absorption of carbohydrates by stimulating the release of pancreatic enzymes into the small intestine. If the pH of the stomach is too high due to insufficient stomach acid, the pancreatic enzymes will not be secreted and the carbohydrates will not be broken down properly. The fermentation of carbohydrates that haven't been digested properly is what produces gas. The resulting gas increases intra-abdominal pressure, which is the driving force behind reflux and GERD.

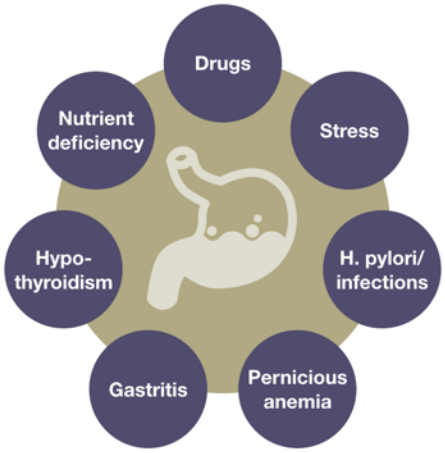
Dr. Robillard also argues that if gas is produced by microbial fermentation of carbohydrates and that caused acid reflux, you might expect that reflux could be treated by either, one, reducing bacterial overgrowth, or two, reducing carbohydrate intake. There are a few studies showing efficacy of a very-low-carb diet for treating GERD, which I mentioned in my e-book. Also, I published an article in 2010 advising the treatment of GERD with a very-low-carb diet and betaine hydrochloric acid, and as of late 2015, there were over 900 comments, many of them from people who experience relief and resolution even after taking PPIs for decades. You can really see that people have had some incredible transformations if you scan through the comments on that blog post. In addition, over several years of treating patients with GERD in my clinic, it's very common for them to have SIBO, and treating SIBO will often resolve the problem.

GERD/heartburn algorithm

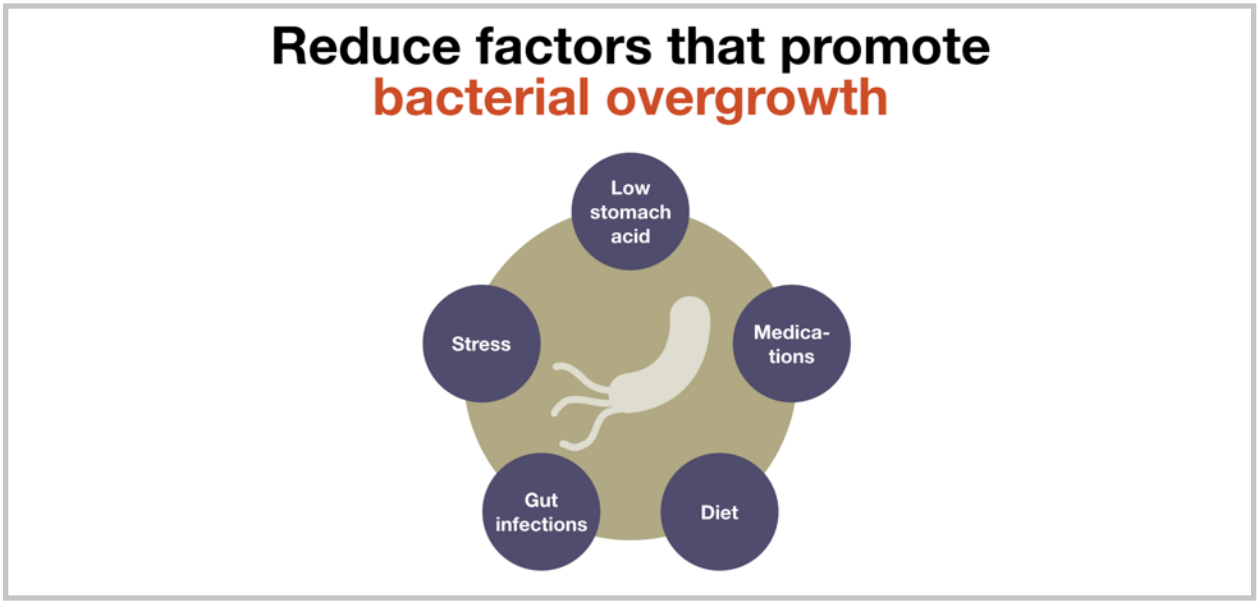
1. Reduce factors that promote **bacterial overgrowth** and **low stomach acid**
2. Replace stomach acid, enzymes and nutrients that **aid digestion** and are **necessary for health**
3. Restore **beneficial bacteria** and a **healthy mucosal lining** in the gut

With this in mind, here's an algorithm for how we approach GERD and heartburn treatment. Number one, we reduce factors that promote bacterial overgrowth and low stomach acid. Number two, we replace stomach acid, enzymes, and nutrients that aid digestion and are necessary for health. And number three, we restore beneficial bacteria and a healthy mucosal lining in the gut. So let's look at each of these in more detail.

Reduce factors that promote low stomach acid



Reducing factors that promote low stomach acid includes several different steps. So we want to address things like nutrient deficiencies; there are certain nutrients that are required to produce stomach acid, like niacin, chloride, sodium, potassium, zinc, and iodine. Of course, PPIs and other acid-stopping drugs profoundly reduce the production of stomach acid, and eventually it's a goal to get patients off of those, although you can't usually do that right away, and you have to be careful with how you do it. Chronic stress suppresses stomach acid production. H. pylori and other pathogens can suppress stomach acid production. Pernicious anemia, which involves an autoimmune attack, often against the parietal cells, which secrete stomach acid, can cause hypochlorhydria. Hypothyroidism can cause low stomach acid, and gastritis, or inflammation of the stomach, can cause it.



We also want to reduce factors that cause bacterial overgrowth, so this means testing and treating for SIBO, addressing the risk factors for low stomach acid that we just mentioned, reducing use of antibiotics when they're not necessary, and birth control pills, addressing diet, and then treating gut infections. And then you would also want to work with the patient to reduce consumption of carbohydrates and/or poorly absorbed carbohydrates if SIBO or hypochlorhydria is present. We talked about most of these steps already, and we'll be talking about others later in the unit, so let's move on to discuss the role of acid-suppressing drugs.