Peptic Ulcer & H. Pylori

A peptic ulcer is an ulceration/erosion in the mucosa of any part of the gut however they are most commonly present in the stomach and first part of the duodenum.

Duodenal ulcers are usually triggered by an increase in hydrochloric acid secretion in response to foods and gastrin. When there is an imbalance between acid-pepsin secretion and the amount of protective mucous and mucosal resistance, an ulcer is most likely to occur. The excess acid will flow from the sotmach to the duodenum and then damages the mucosal lining when there is poor mucous protection and not enough bicarbonate secretion to neutralize the acid. White males with blood group O and HLA-B5 are most susceptible.

Gastric ulcers appear later in life usually compared to duodenal ulcers. These too are more common in males however hyperchlorhydria is not usually the trigger. Hypochlorhydria and reduced gastric mucosa and decrease in mucous production is usually the trigger… aka and underactive stomach which is the opposite trigger for duodenal ulcers. Another trigger could be duodenal reflux of bile back into the stomach as well as decrease in pancreatic bicarbonate secretions which are triggered by smoking.

Many gastric ulcers are actually caused by taking NSAIDs which irritate the stomach causing inflammation, histamine release and excess acidity. They also increase mucosal permeability.

There is a strong relationship between a bacteria called Heliobacter pylori (H. pylori) and peptic ulcers. This microbe has an alkaline “shell” to protect it from stomach acid. It burrows under the protective mucosal layer and into the mucosa where it releases a cytotoxin that causes cell wall damage. This damage to the cell wall elicits an immune response and inflammation. it reduces gastric mucus production by goblet cells in the stomach making the stomach and duodenum more susceptible to hydrochloric acid damage. It also increases acid production and triggers acid secretion post meal. H. pylori increases risk of gastric ulcers and also duodenal ulcers up to five times. It disturbs the acid enzyme balance so much that digestion is affected and dysbiosis is created. there is a greater likelihood of developing cancer. Dental plaque is the reservoir of this and many other pathogens so very often found in the mouth however also exists as a commensal in the gut of many people.

Signs and Symptoms

Gastric ulcers can cause a gnawing, burning epigastric pain that can begin within a half hour of eating and is **not relieved by eating.**

Duodenal ulcers cause epigastric pain that can begin one to three hours after eating a meal, but is **relieved by eating.**

Development of Ulcers

H. pylori infection and NSAIDS both compromise mucosal defenses which causes mucosal damage leading to stomach ulcers. Stress is also a trigger for stomach ulcers as it elevates histamine and reduces stomach acidity.

Dietary Recommendations

- Avoid coffee, tea (also peppermint and other mint teas), stimulants and carbonated drinks
- Avoid sugar, alcohol and artificial sweeteners
- Drink lots of water
- Increase fiber consumption
- Avoid trans and hydrogenated fats
- Decrease refined and simple carbs
- Eat and alkaline diet
- Avoid food sensitivities
- Avoid inflammatory foods
- Avoid foods that irritate the stomach which would stimulate HCl secretions
- Avoid large amounts of animal protein, acidic and spicy foods
- Cabbage and potato juices contain glutamine which will help facilitate mucosal repair
- Do not consume dairy which can exacerbate the issue
- Water will dilute excess HCl

Supplements

- Probiotics (inhibit pathogens in the gut)
- L-glutamine (repair mucous lining)
- Aloe vera gel (soothe ulcerations
- Essential fatty acids (anti-inflammatory)
- Curcumin and boswellia (anti-inflammatory for ulcers)
- Manuka honey (helps regenerate goblet cells)
- Colloidal silica (controls inflammation and interferes with attachment of H, pylori to the stomach mucosa)
- Vitamins C and A, Selenium, zinc and Vitamin E (boost immunity and repair mucosal lining)
- Grapefruit seed extract and oregano oil to inhibit H. pylori
- Artemisa, barberry and pulsatilla (kill bacteria and other parasites)
- Marshmallow root and/or slippery elm (soothe and heal ulcers)
- DGL (deglycyrrhinated licorce – helps regenerate goblet cells)
- Astragalus (stimulate immune systrem)

Lifestyle Recommendations

- Drink warm water if you’re experiencing pain from an ulcer
- Reduce stress with deep breathing, mediation, hot baths, etc
- Treat dysbiosis
- Strengthen gut mucosa by following a leaky gut protocol
- Avoid cigarette smoke and other pollutants

Deeper Immunology of Ulcers and H. pylori

Goblet cells secret mucin glycoproteins and bacteria are abundantly in the outer mucous layer but the inner layer is resistant to bacteria. Plasma cells secrete IgA. As

bacteria move toward the epithelial lining of the gut, they get slowed down and killed. TH17 responses increase these epithelial antimicrobial proteins through the secretion of Interleukin-22. TH1 and TH2 responses are necessary for reducing the number of bacteria in the lamina propria. Interferon-gamma increases macrophage activation to do this. One example of this mechanism is with H. pylori infection. There is hypoacidity created by H. pylori itself and it burrows through the mucous layer and affects the parietal cells of the stomach so that they create less stomach acid. The problem then becomes that there is less mucous production as a result of less stomach acid production. This allows further penetration of H. pylori because the mechanical barrier is diminished.

Paneth cells secrete factors that help sustain and modulate the epithelial stem and progenitor cells. They are very important to sustain cells that restore epithelial integrity and are going to create repair in the epithelial lining and without these progenitor and Paneth cells, you will have trouble resealing leaky gut. Dysfunction of Paneth cell biology contributes to pathogenesis of chronic inflammatory bowel disease.

There are three phases of the migrating motor complex which is how foods, bacteria and everything else moves downwards throughout the digestive system. Phase three is the most important phase and it is useful to have it start in the stomach however if it doesn't you may end up with H. pylori. The MMC starts with an underlying slow wave (periodic depolarization). These slow waves are generated by intestinal cells of Cajal which acts as pacemakers and produce spontaneous electrical slow waves. There are 3 per minute in the stomach, 12 per min in the duodenum and 10 per min in the ileum. A contraction is achieved when a slow wave and the release of an excitatory neurotransmitter from a motor neuron of the enteric nervous system occur simultaneously. Certain foods, stress, elevated serotonin levels and other factors cause phase three of MMC to start in the duodenum rather than the stomach.

H. pylori create biofilms which make them difficult to eradicate. A study was done that showed that biofilm biomass of H. pylori was increased after treatment with clarithromycin. When H. pylori was exposed to CLR, there was a 4-fold increase at 2 days and 16-fold increase at 3 days. These bacteria are capable of expression and their efflux pumps have the ability to pump toxic elements out of the cell. If you give an antibiotic or natural antifungal and the bacteria that you are trying to kill have efflux pumps, they will pump it out of the cell.

H. pylori is highly susceptible to bismuth, a heavy metal with antimicrobial activity linked to its effect on bacterial iron uptake. However the mechanism by which bismuth is killing H. pylori is not by iron deprivation . This is important because if you have a patient who is very anemic and who needs iron, you need to know if it is OK to give iron in relation to trying to kill a microbe out of their alimentary tract. H pylori doesn't go away easily. You need to do a breath test before and after treatment and you cant assume that bismuth will wipe it away, although it is worth a shot (always work with a practitioner on this).

Proton pump inhibitors, clarithromycin and amoxicillin is the most commonly prescribed first line therapy in conventional medicine world. Levofloxacin-based therapies appear

to be useful and versatile in second and third-line therapies, with interesting results for newer generation quinolones which may partially overcome antibiotic resistance. Probiotics appear to have an effect in terms of reducing side effects and improving compliance, but data on improvement of eradication rates remain controversial. Probiotics can tune the immune response more favourable towards a Th1 response and you can assist the immune system in addressing the biofilm problem.

H. pylori is sometimes found on the calculus of teeth or plaque and it is present in the mouth sometimes. If you kill H. pylori in the mouth using a mouthrinse and periodontal (plaque removal) treatment, the gastric H. pylori gets significantly reduced.

Summary: Using Transcutaneous vagal nerve stimulation (TVNS) to improve migrating motor complex while looking into effect biofilm disrupter and antimicrobial treatments such as bismuth may be helpful in eradicating H. Pylori. Also make sure you're supporting aTh1 immune response to help push back against pathogens.