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Gastritis

Gastritis is a general term for a group of conditions in which the lining of the stomach is inflamed. Normally, the gastric mucosal barrier that protects the gastric cells from the low pH environment of the stomach lumen is made up of a relatively impermeable layer of epithelial cells. These cells are bound together by tight junctions and are covered by a protective mucus layer. The epithelial cells of the gastric mucosa also have a rapid turnover rate which helps minimize erosion. When this epithelial layer gets disrupted in some way, gastritis can result. There are both acute and chronic forms of gastritis.

Acute Gastritis

Acute gastritis is characterized by an acute inflammatory process in the mucosa of the stomach. It is usually transient but may involve bleeding into the mucosa and sloughing off of the superficial mucosal cells. Acute gastritis may be characterized as erosive, which is where damaged areas have disrupted or missing mucosal cells.

Common causes of acute gastritis include NSAID use, alcohol, bacterial toxins, corticosteroids, serious illness, trauma, and kidney failure. Treatment of cancer with chemotherapy drugs and gastric radiation are also causes of acute gastritis. Kidney failure results in high blood urea levels and spontaneous bleeding in mucosal membranes like the GI is common. However, it is not known exactly what in the blood contributes to the spontaneous bleeding. It has been suggested that the excessive amounts of urea affect platelets in a way that prevents their formation of a platelet plug, but studies refute that the urea is the cause. It may be something else building up in the blood that is causing the platelet dysfunction.

NSAIDs are generally weak organic acids that are protonated in the low pH of the stomach secretions. In this uncharged protonated form, they can cross mucosal cell membranes in the stomach. Once inside the more neutral pH of the cytoplasm, they lose their proton. In their now ionized form, they are trapped in the mucosal cell where they proceed to inhibit the COX enzymes. COX enzymes play a critical role in the production of prostaglandins. In the stomach mucosa, prostaglandins are important for paracrine signaling that maintain a thick and healthy layer of mucus on the surface of the cells. Because NSAIDs inhibit COX and thus prostaglandins, they cause an overall decrease in mucous production. The deficiency of mucus caused by the NSAIDS results in a thinner and less effective mucus layer, leaving the mucosal cells more vulnerable to injury by the actions of acid and pepsin.

Chronic Gastritis

Chronic gastritis occurs when the gastric mucosa is irritated over a long period of time. It is less likely to present with erosions than acute gastritis. However, the stomach lining is still inflamed and over time there can be a loss of mucosal cells which negatively affects the mucus protective layer. This may lead to an increased risk of developing acute gastritis symptoms and erosions. We will discuss two types or causes of chronic gastritis: an autoimmune disorder and gastritis caused by a *H. pylori* infection.

Chronic Autoimmune Gastritis

Chronic gastritis can be caused by an autoimmune disorder where autoantibodies attack gastric and parietal cells that produce intrinsic factor. The body and fundus portions of the stomach are usually affected by chronic autoimmune

gastritis. Due to lack of vitamin B-12 absorption from decreased intrinsic factor production, chronic autoimmune gastritis can lead to megaloblastic anemia. Lack of vitamin B-12 can also lead to atrophic glossitis where the tongue becomes smooth and beefy red. This is because vitamin B-12 is important for cell division, so if it is low it is harder to replace epithelial cells. This decreased division can lead to a weakened area where epithelial cells experience a lot of friction like the tongue. As a result, the tongue can become irritated, swollen, and red. Clinical manifestations of chronic autoimmune gastritis are related to anemia from the lack of vitamin B-12 and other malabsorptive problems.

Helicobacter Pylori Gastritis

Watch the video Helicobacter Pylori and Ulcers

***Helicobacter pylori* gastritis** is a chronic inflammatory disease of the antrum and body of the stomach caused by the bacteria *Helicobacter pylori*. *H. pylori* is responsible for the majority of gastritis cases and stomach ulcers that are diagnosed. *H. pylori* is transferred by the oral/oral route or oral/fecal route. *H. pylori* is a gram-negative rod with 4-6 flagella that infects the mucus secreting cells of the stomach and duodenum. *H. pylori* escapes the very acidic environment of the stomach juices by using its powerful flagella to push it into the mucus that lines the stomach. This mucus helps buffer and protect the healthy stomach cells from acid damage, and it does the same for *H. pylori*. The bacteria also produces an enzyme called **urease** that catalyzes the small amounts of urea always found in stomach juices into CO_2 and ammonia (NH3). NH_3 helps neutralize the acid in the area surrounding the *H. pylori* because it will take on a hydrogen to become NH_4^+. This area's higher pH creates an environment where the bacteria can more readily proliferate and allow some of the bacteria to even enter the epithelial cells.

Several substances are produced by *H. pylori* that cause damage to the gastric epithelial lining. A few of these substances include ammonia, mucinase, and protease, which are all toxic to epithelial cells. Vacuolating cytotoxin A (VacA) is also made by the bacteria and disrupts tight junctions and causes apoptosis of epithelial cells. Cytotoxin associate gene CagA is a carcinogen released by *H. pylori* that causes inflammation. Altogether, these toxic substances damage and kill epithelial cells, leading to ulcers.

Some cruciferous vegetables like Brussel sprouts, cauliflower, and broccoli are believed to inhibit the growth of *H. pylori*. This is because these vegetables contain a chemical called sulforaphane that has been shown to have bactericidal effects on *H. pylori*. The exact mechanism of how this compound affects *H. pylori* is not known. Studies investigating how eating these vegetables might impact *H. pylori* gastritis show that cruciferous vegetables likely decrease *H. pylori* numbers and virulence, but do not eradicate the organism like an antibiotic treatment would.

The carbon urea breath test can be used in the diagnosis of *H. pylori*. For this test, the patient drinks radio labeled ^{13}C urea. *H. pylori* in the digestive tract will then break down the urea using urease and make CO_2 that contains the ^{13}C from the urea. The patient will breathe the CO_2 out and the ^{13}C can be detected using breath analyzers. Other diagnostic tools to determine if an individual has a *H. pylori* infection include testing the blood for *H. pylori* antibodies and testing the urine or stool for *H. pylori* antigens. The most accurate diagnostic method for an *H. pylori* infection is to take a biopsy from two different locations of the stomach and culture the possible bacteria or do a urease test on the specimens to determine if *H. pylori* is present.

Prevpac is a common treatment used to eradicate *H. pylori*. It contains 2 antibiotics to kill the bacteria and a proton pump inhibitor to decrease acid production and create an environment more conducive to healing. Bismuth is another medication that can be used as a treatment. It prevents the binding of *H. pylori* to epithelial cells and causes the lysis/death of *H. pylori*.

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