ABDOMINAL PAIN - GASTRITIS/DUODENITIS AND GASTROPATHY

Pathophysiology

Gastritis

Simply stated, gastritis is microscopic inflammation of the gastric mucosa. Gastric mucosa inflammation is a histologic diagnosis, and is generally either infectious or autoimmune mediated. Atrophic gastritis occurs when chronic inflammation leads to gastric gland loss and metaplastic changes. Gastric glandular loss results in an inability to secrete adequate acid, pepsin, and intrinsic factor. These changes can result in insufficient absorption of vitamins and minerals. Additionally, atrophic gastritis is associated with an increased risk of gastric adenocarcinoma, gastric carcinoid tumors, and mucosa-associated lymphoid tumor (MALT) lymphoma. Erosive gastritis occurs when inflammation results in gastric erosions or ulcers, which can subsequently lead to pain, strictures, anemia, bleeding, and/or perforation. Treatment of gastritis depends on the etiology and the type of damage that has been induced.

Gastropathy

Gastropathy, in contrast to gastritis, denotes gastric mucosal injury with minimal or no inflammation. The mechanism of injury in the setting of gastropathy usually involves disruption of the protective mucous barrier, which subsequently results in mucosal injury secondary to direct acid exposure. Gastropathy can be caused by chemical irritants (reactive gastropathy), ischemia, physical stress, or chronic vascular congestion. Diagnosis is determined through an appropriate history, endoscopic evaluation, and often histologic examination of biopsies. Treatment usually involves elimination of the offending source, gastric acid suppression, and supplementation of the protective mucous barrier.

Infectious Gastritis

H. Pylori

The most common cause of gastritis of any type is H. pylori infection. H. pylori is a small, spiral-shaped, gram negative bacillus bacteria that inhabits the mucous layer overlying the gastric mucosa. The mechanism by which H. pylori produces gastric inflammation is unclear since it does not infiltrate the gastric mucosa. Approximately half to two-thirds of the world's population has H. pylori colonization. It is most frequently acquired during childhood and, when left untreated, persists within the host for life. Transmission most frequently occurs within families and can occur via oral-oral or fecal-oral routes. Environmental sources such as contaminated water reservoirs have also been suggested; however, detailed knowledge of transmission pathways has remained elusive.

Most people with H. pylori infection never develop any symptoms. However, H. pylori can cause erosive gastritis and/or duodenitis. In fact, 90% of duodenal ulcers and 80% of gastric ulcers are caused by H. pylori infection. Ulcer disease can cause abdominal pain, nausea, bloating, loss of appetite, and/or bleeding. Bleeding can be chronic and slow resulting in chronic iron deficiency anemia, or it can be more rapid resulting in hematemesis, hematochezia,

or melena. Advanced ulcer disease can result in strictures or perforation. Additionally, individuals with H. pylori infection carry a 2 to 6-fold increased risk of developing gastric adenocarcinoma and MALT lymphoma.

There are a variety of testing methods available to diagnose H. pylori infection. The gold standard is endoscopic biopsy with histologic identification of H. pylori organisms. Histologic stains such as Warthin-Starry, Giemsa, and el-Zimaity, to name a few, can help enhance bacterial visibility. Histologic examination also offers the benefit of being able to assess the severity of gastritis, and to identify the presence of atrophic gastritis, metaplasia, adenocarcinoma, or lymphoproliferative disease. H. pylori culture is not widely utilized due to the high cost, the difficulty in achieving culture growth, and the length of time required to obtain results. The main role for H. pylori culture is to obtain antimicrobial susceptibility testing.

Noninvasive testing includes the urea breath test, serologic testing, and stool antigen testing. The urea breath test offers high sensitivity and specificity (94% and 98%, respectively) for active H. pylori infection. This represents the most widely accepted modality for confirmation of cure. The disadvantage of the urea breath test is that testing during concomitant therapy with a proton pump inhibitor (PPI) reduces test accuracy. H. pylori stool antigen is another noninvasive testing modality with a high sensitivity and specificity (both over 90%). Although PPI cessation is recommended prior to this test, PPI use has less impact on the stool antigen test than on the urea breath test. The sensitivity and specificity of serologic testing is lower than the other modalities, and IgG antibody measurements cannot differentiate between past versus active infection.

The goal of H. pylori infection treatment is eradication of the organism from the stomach, and successful treatment is defined as negative H. pylori (antigen and organism) testing greater than 4 weeks following completion of therapy. There are currently 8 therapy combinations approved by the FDA for H. pylori treatment, but there are several others which have also been used successfully. Triple therapy has the highest eradication rates and includes a PPI plus clarithromycin and either amoxicillin or metronidazole. Treatment duration is 10-14 days. Other combinations are used depending on patient tolerance, patient compliance, cost, and antimicrobial resistance patterns. Eradication rates range from 60-90%; therefore, eradication should be confirmed following completion of treatment.

Other Infectious Gastritis Agents

Other types of infectious agents that can cause gastritis are quite uncommon and/or are only seen in special patient populations such as severely immunocompromised patients. Bacterial, mycobacterial, viral, fungal, and parasitic agents have been noted to cause gastritis. Phlegmonous gastritis is an acute, life-threatening infection of the gastric submucosa that can occur in the immunocompromised, the elderly, and alcoholics. The most common organisms are alpha hemolytic streptococci and Clostridium species. Mortality is high and surgical source control may be necessary.

Cytomegalovirus (CMV) is the most common viral etiology for gastritis. This infection typically occurs in immunocompromised patients but has been seen in elderly immunocompetent patients. Diagnosis is established with endoscopic biopsies showing viral inclusions. Treatment for CMV gastritis is with ganciclovir.

Autoimmune Gastritis

In patients with autoimmune gastritis, antibodies to the parietal cells and/or intrinsic factor induce a chronic atrophic gastritis. Chronic atrophic gastritis is a gastric glandular atrophy that results in an inability to secrete adequate acid, pepsin, and intrinsic factor. Patients often remain asymptomatic for many years until parietal cell mass has decreased to the point that achlorhydria and hypergastrinemia result. Subsequent symptoms develop secondary to B12 deficient pernicious anemia and/or iron deficiency. Symptoms are related to hematologic, neurologic, and/or gastrointestinal disturbances resulting from B12 deficiency. These symptoms can include fatigue, dizziness, anorexia, weight loss, sore tongue, confusion, memory loss, irritability, paresthesias, numbness, weakness, and ataxia. Patients with autoimmune gastritis often suffer from other endocrine-related autoimmune disorders. Autoimmune gastritis is rarely erosive.

The diagnostic approach should include endoscopic evaluation with biopsies, blood work to demonstrate autoimmune antibodies, hematologic studies, and blood work to evaluate for B12 and iron deficiency. Treatment centers on replacement of essential vitamins and minerals to avoid downstream effects of vitamin and mineral deficiencies.

Patients suffering from autoimmune gastritis are at increased risk of gastric carcinoid and gastric adenocarcinoma. Depending on the severity of disease, patients may require endoscopic surveillance.

Reactive Gastropathy

Reactive or chemical gastropathy involves disruption of the protective mucous barrier. The most common causes of mucous barrier disruption are non-steroidal anti-inflammatory drugs (NSAIDs) and bile reflux. NSAIDs disrupt the protective barrier through a mechanism of reduced prostaglandin synthesis. NSAID use is the second most common cause of ulcer disease following H. pylori infection. Diagnosis is achieved through an appropriate medication use history and endoscopic evaluation including biopsies. Treatment involves elimination of the offending agent, acid suppression with PPI therapy, and the use of sucralfate.

The pathogenesis of bile reflux gastropathy involves alkaline duodenal contents disrupting the gastric mucous barrier. Once this barrier has been disrupted, further gastric injury occurs through acid exposure. Patients with a distal gastrectomy and Billroth I or II reconstruction are the most likely to develop bile reflux gastropathy; however, this can also occur in patients with normal gastrointestinal anatomy. Severe or refractory erosive disease may require operative diversion of biliopancreatic secretions by utilizing a roux-en-y reconstruction.

Ischemic Gastropathy

Critically ill patients are at risk of developing ischemic gastropathy. The pathogenesis is due to inadequate gastric mucosal blood flow during intense physiologic stress such as severe trauma, head injury, burns, or sepsis. This has previously been termed “stress gastritis”. Similar to reactive gastropathy, there is a subsequent disruption of the protective mucous barrier making the gastric mucosa susceptible to acid induced damage. This can lead to bleeding erosions or ulcers. Fortunately, these physiologic stress-induced injuries are less common in today’s critically ill patients. Prevention of ischemic gastropathy has been attributed to a combination of improved critical care that emphasizes appropriate tissue perfusion and oxygenation as well as acid suppression. However, there is concern that aggressive acid suppression in this patient population increases the risk of ventilator associated pneumonia and aspiration pneumonia.

Vascular Gastropathy

The most common type of vascular gastropathy is portal hypertensive gastropathy that results for cirrhosis. This type of gastropathy can result in acute or chronic bleeding. Diagnosis is made with endoscopic evaluation which shows a reticular mucosal pattern throughout the stomach. There may also be vascular ectasias. Treatment is targeted at reducing portal hypertension and maintaining the protective mucous barrier. Beta-blockers may be used to reduce portal pressures. Alternatively, transjugular intrahepatic portosystemic shunt (TIPS) is effective for directly treating the underlying problem of portal hypertension. Bleeding ectatic vessels may be treated with endoscopic application of argon plasma coagulation (APC).

Author

Andrew S. Kastenmeier, MD, FACS
Froedtert Hospital and Medical College of Wisconsin, Milwaukee, WI