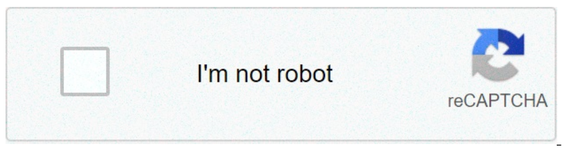

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Metronidazole for gastritis

H pylori are gram-negative bacteria that colonize and infect the stomach. The bacteria lodge within the mucous layer of the stomach along the gastric surface epithelium and the upper portions of the gastric foveolae and rarely are present in the deeper glands (see the 3 images below). Atrophic gastritis. Schematic representation of Helicobacter pylori–associated patterns of gastritis. Involvement of the corpus, fundus, and gastric antrum, with progressive development of gastric atrophy as a result of the loss of gastric glands and partial replacement of gastric glands by intestinal-type epithelium, or intestinal metaplasia (represented by the blue areas in the diagram) characterize multifocal atrophic gastritis. Individuals who develop gastric carcinoma and gastric ulcers usually present with this pattern of gastritis. Inflammation mostly limited to the antrum characterizes antral-predominant gastritis. Individuals with peptic ulcers usually develop this pattern of gastritis, and it is the most frequent pattern in the Western countries. Patterns of atrophic gastritis associated with chronic Helicobacter pylori infection and autoimmune gastritis. Atrophic gastritis. Helicobacter pylori–associated chronic active gastritis (Genta stain, 20x). Multiple organisms (brown) are observed adhering to gastric surface epithelial cells. A mononuclear lymphoplasmacytic and polymorphonuclear cell infiltrate is observed in the mucosa. The infection is usually acquired during childhood and progresses over the lifespan of the individual if left untreated. The host response to the presence of H pylori is composed of a T-lymphocytic and B-lymphocytic response, followed by infiltration of the lamina propria and gastric epithelium by polymorphonuclear leukocytes (PMNs) that eventually phagocytize the bacteria. Significant damage associated with the release of bacterial and inflammatory toxic products is inflicted on the gastric epithelial cells, resulting in increasing cell loss or gastric atrophy over time. Weck published a study supporting their hypothesis that the association between H pylori and chronic atrophic gastritis was greatly underestimated due to the clearance of the infection in advanced stages of the disease. [5] These results suggest that the association is much stronger than estimated by most epidemiologic studies to date. Another study also reported that mannan-binding lectin allele (MBL2 codon 54 B) is associated with a higher risk of developing more severe gastric mucosal atrophy in H pylori–infected Japanese patients. [6] During the process of gastric mucosal atrophy, some glandular units develop an intestinal-type epithelium, and intestinal metaplasia eventually occurs in multiple foci throughout the gastric mucosa when atrophic gastritis is fully established. Other glands are simply replaced by fibrous tissue, resulting in an expanded lamina propria. [7] Loss of gastric glands in the corpus, or corpus atrophy, reduces parietal cell numbers, which results in significant functional changes with decreased levels of acid secretion and increased gastric pH. [8] Hypochloridia or achloridia raises serum gastrin levels, thereby increasing the risk for the development of neuroendocrine tumors. [8] Studies have also reported that moderate alcohol consumption may be associated with atrophic gastritis by facilitating H pylori clearance. [9] H pylori–associated chronic gastritis progresses with 2 main topographic patterns that have different clinicopathologic consequences. The first is antral predominant gastritis. Inflammation that is mostly limited to the antrum characterizes antral predominant gastritis. Individuals with peptic ulcers usually develop this pattern of gastritis, and it is the most frequently observed pattern in Western countries. The second is multifocal atrophic gastritis. Involvement of the corpus, fundus, and gastric antrum with progressive development of gastric atrophy (ie, loss of gastric glands) and partial replacement of gastric glands by intestinal-type epithelium (intestinal metaplasia) characterize multifocal atrophic gastritis. Individuals who develop gastric carcinoma and gastric ulcers usually have this pattern of gastritis. This pattern is observed more often in developing countries and in Asia. The development of chronic atrophic gastritis limited to corpus-fundus mucosa and marked diffuse atrophy of parietal and chief cells characterize autoimmune atrophic gastritis, as shown in the following two images. Patterns of atrophic gastritis associated with chronic Helicobacter pylori infection and autoimmune gastritis. Atrophic gastritis. Intestinal metaplasia of the gastric mucosa (Genta stain, 20x). Intestinal-type epithelium with numerous goblet cells (stained blue with the Alcian blue stain) replace the gastric mucosa and represent gastric atrophy. Mild chronic inflammation is observed in the lamina propria. This pattern of atrophy is observed both in Helicobacter pylori–associated atrophic gastritis and autoimmune gastritis. Autoimmune gastritis is associated with serum antiparietal and anti-intrinsic factor antibodies that cause intrinsic factor (IF) deficiency, which, in turn, causes decreased availability of cobalamin (vitamin B-12) and, eventually, pernicious anemia in some patients. Palladino reported that methylenetetrahydrofolate reductase (MTHFR) polymorphisms may be associated with B12 deficiency and autoimmune atrophic gastritis. [10] Autoantibodies are directed against at least 3 antigens, including IF, cytoplasmic (microsomal-canalicular), and plasma membrane antigens. Two types of IF antibodies are detected (types I and II). Type I IF antibodies block the IF-cobalamin binding site, thus preventing the uptake of vitamin B12. Cell-mediated immunity also contributes to the disease. [11] T-cell lymphocytes infiltrate the gastric mucosa and contribute to the epithelial cell destruction and resulting gastric atrophy. Stummvoll reported that Th17 cells induced the most destructive disease with cellular infiltrates composed primarily of eosinophils accompanied by high levels of serum IgE. [12] Polyclonal Treg also suppresses the capacity of Th1 cells and moderately suppresses Th2 cells, but it can suppress Th17-induced disease only at early time points. The major effect of Treg was to inhibit the expansion of the effector T cells. However, effector cells isolated from protected animals were not anergic and were fully competent to proliferate and produce effector cytokines ex vivo. [12] The strong inhibitory effect of polyclonal Treg on the capacity of some types of differentiated effector cells to induce disease provides an experimental basis for the clinical use of polyclonal Treg in the treatment of autoimmune disease in humans. The above findings led to an interesting study by Huter et al, who reported that antigen-specific-induced Treg are potent suppressors of autoimmune gastritis induced by both fully differentiated Th1 and Th17 effector cells. The investigators analyzed the suppressive capacity of different types of Treg to suppress Th1- and Th17-mediated autoimmune gastritis by comparing nTreg with polyclonal TGFbeta-induced WT Treg (iTreg) or TGFbeta-induced antigen-specific TxA23 iTreg in cotransfer experiments with Th1 or Th17 TxA23 effector T cells. [13] Th1-mediated disease was prevented by cotransfer of nTreg and also antigen-specific iTreg, whereas WT iTreg did not show an effect. However, Th17-mediated disease was only suppressed by antigen-specific iTreg. Preactivation of nTreg in vitro before the transfer did not increase their suppressive activity in Th17-mediated gastritis, supporting the investigators' hypothesis. [13] Crafa P, Russo M, Miraglia C, et al. From Sidney to OLGA: an overview of atrophic gastritis. Acta Biomed. 2018 Dec 17. 89(8-S):93-9. [Medline]. Rodriguez-Castro KI, Franceschi M, Miraglia C, et al. Autoimmune diseases in autoimmune atrophic gastritis. Acta Biomed. 2018 Dec 17. 89(8-S):100-3. [Medline]. Yanaoka K, Oka M, Ohata H, et al. Eradication of Helicobacter pylori prevents cancer development in subjects with mild gastric atrophy identified by serum pepsinogen levels. Int J Cancer. 2009 Dec 1. 125(11):2697-703. [Medline]. Vannella L, Lahner E, Annibale B. Risk for gastric neoplasias in patients with chronic atrophic gastritis: a critical reappraisal. World J Gastroenterol. 2012 Mar 28. 18(12):1279-85. [Medline]. [Full Text]. Weck MN, Gao L, Brenner H. Helicobacter pylori infection and chronic atrophic gastritis: associations according to severity of disease. Epidemiology. 2009 Jul. 20(4):569-74. [Medline]. Tahara T, Shibata T, Wang FY, et al. Mannan-binding lectin B allele is associated with a risk of developing more severe gastric mucosal atrophy in Helicobacter pylori-infected Japanese patients. Eur J Gastroenterol Hepatol. 2009 Jul. 21(7):781-6. [Medline]. Gao L, Weck MN, Nieters A, Brenner H. Inverse association between a pro-inflammatory genetic profile and Helicobacter pylori seropositivity among patients with chronic atrophic gastritis: enhanced elimination of the infection during disease progression?. Eur J Cancer. 2009 Nov. 45(16):2860-6. [Medline]. Rodriguez-Castro KI, Franceschi M, Noto A, et al. Clinical manifestations of chronic atrophic gastritis. Acta Biomed. 2018 Dec 17. 89(8-S):88-92. [Medline]. Gao L, Weck MN, Stegmaier C, Rothenbacher D, Brenner H. Alcohol consumption and chronic atrophic gastritis: Population-based study among 9,444 older adults from Germany. Int J Cancer. 2009 Jun 2. 125(12):2918-22. [Medline]. [Full Text]. Palladino M, Chiusolo P, Reddiconto G, et al. MTHFR polymorphisms involved in vitamin B12 deficiency associated with atrophic gastritis. Biochem Genet. 2009 Oct. 47(9-10):645-50. [Medline]. Lahner E, Norman GL, Severi C, et al. Reassessment of intrinsic factor and parietal cell autoantibodies in atrophic gastritis with respect to cobalamin deficiency. Am J Gastroenterol. 2009 Aug. 104(8):2071-9. [Medline]. Stummvoll GH, DiPaolo RJ, Huter EN, et al. Th1, Th2, and Th17 effector T cell-induced autoimmune gastritis differs in pathological pattern and in susceptibility to suppression by regulatory T cells. J Immunol. 2008 Aug 1. 181(3):1908-16. [Medline]. [Full Text]. Huter EN, Stummvoll GH, DiPaolo RJ, Glass DD, Shevach EM. Pre-differentiated Th1 and Th17 effector T cells in autoimmune gastritis: Ag-specific regulatory T cells are more potent suppressors than polyclonal regulatory T cells. Int Immunopharmacol. 2009 May. 9(5):540-5. [Medline]. Yagi K, Nakamura A, Sekine A, Graham D. Features of the atrophic corpus mucosa in three cases of autoimmune gastritis revealed by magnifying endoscopy. Case Report Med. 2012. 2012:368160. [Medline]. [Full Text]. Zhang Y, Weck MN, Schottker B, et al. Gastric parietal cell antibodies, Helicobacter pylori infection, and chronic atrophic gastritis: evidence from a large population-based study in Germany. Cancer Epidemiol Biomarkers Prev. 2013 May. 22(5):821-6. [Medline]. Chen WC, Warner RRP, Harpaz N, Zhu H, Roayaie S, Kim MK. Gastric neuroendocrine tumor and duodenal gastrinoma with chronic autoimmune atrophic gastritis. Pancreas. 2019 Jan. 48(1):131-4. [Medline]. Massironi S, Cavalcoli F, Rossi RE, et al. Chronic autoimmune atrophic gastritis associated with primary hyperparathyroidism: a transversal prospective study. Eur J Endocrinol. 2013 May. 168(5):755-61. [Medline]. [Guideline] Early DS, Lightdale JR, Vargo JJ 2nd, et al, for the ASGE Standards of Practice Committee. Guidelines for sedation and anesthesia in GI endoscopy. Gastrointest Endosc. 2018 Feb. 87(2):327-37. [Medline]. Zoalfaghari A, Aletaha N, Roushan N, Taslimi R, Foroutan H, Faridnia B. Accuracy of pepsinogens for early diagnosis of atrophic gastritis and gastric cancer in Iranian population. Med J Islam Repub Iran. 2014. 28:150. [Medline]. Zagari RM, Rabitti S, Greenwood DC, Eusebi LH, Vestito A, Bazzoli F. Systematic review with meta-analysis: diagnostic performance of the combination of pepsinogen, gastrin-17 and anti-Helicobacter pylori antibodies serum assays for the diagnosis of atrophic gastritis. Aliment Pharmacol Ther. 2017 Oct. 46(7):657-67. [Medline]. Pittman ME, Voltaggio L, Bhaijee F, Robertson SA, Montgomery EA. Autoimmune metaplastic atrophic gastritis: recognizing precursor lesions for appropriate patient evaluation. Am J Surg Pathol. 2015 Dec. 39(12):1611-20. [Medline]. Capella C, Fiocca R, Cornaggia M. Autoimmune gastritis. Graham DY, Genta RM, Dixon MF, eds. Gastritis. Philadelphia, Pa: Lippincott Williams; 1999. 79-96. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process-- First American Cancer Society Award Lecture on Cancer Epidemiology and Prevention. Cancer Res. 1992 Dec 15. 52(24):6735-40. [Medline]. Dixon MF, Genta RM, Yardley JH. Classification and grading of gastritis. The updated Sydney System. International Workshop on the Histopathology of Gastritis, Houston 1994. Am J Surg Pathol. 1996 Oct. 20(10):1161-81. [Medline]. Dore MP, Leandro G, Realdi G, Sepulveda AR, Graham DY. Effect of pretreatment antibiotic resistance to metronidazole and clarithromycin on outcome of Helicobacter pylori therapy: a meta-analytical approach. Dig Dis Sci. 2000 Jan. 45(1):68-76. [Medline]. Franceschi F, Genta RM, Sepulveda AR. Gastric mucosa: long-term outcome after cure of Helicobacter pylori infection. J Gastroenterol. 2002. 37 Suppl 13:17-23. [Medline]. Gao L, Weck MN, Raum E, et al. Sibship size, Helicobacter pylori infection and chronic atrophic gastritis: a population-based study among 9444 older adults from Germany. Int J Epidemiol. 2010 Feb. 39(1):129-34. [Medline]. Graham DY. Therapy of Helicobacter pylori: current status and issues. Gastroenterology. 2000 Feb. 118(2 Suppl 1):S2-8. [Medline]. Graham DY, Belson G, Abudayyeh S, et al. Twice daily (mid-day and evening) quadruple therapy for H. pylori infection in the United States. Dig Liver Dis. 2004 Jun. 36(6):384-7. [Medline]. Hershko C, Hoffbrand AV, Keret D, et al. Role of autoimmune gastritis, Helicobacter pylori and celiac disease in refractory or unexplained iron deficiency anemia. Haematologica. 2005 May. 90(5):585-95. [Medline]. Inoue T, Uedo N, Ishihara R, et al. Autofluorescence imaging videoendoscopy in the diagnosis of chronic atrophic fundal gastritis. J Gastroenterol. 2010 Jan. 45(1):45-51. [Medline]. Konturek PC, Konturek SJ, Brzozowski T. Helicobacter pylori infection in gastric cancerogenesis. J Physiol Pharmacol. 2009 Sep. 60(3):3-21. [Medline]. Krasinskas AM, Abraham SC, Metz DC, et al. Oxyntic mucosa pseudopolyps: a presentation of atrophic autoimmune gastritis. Am J Surg Pathol. 2003 Feb. 27(2):236-41. [Medline]. Laiyemo AO, Kamangar F, Marcus PM, et al. Atrophic gastritis and the risk of incident colorectal cancer. Cancer Causes Control. 2010 Jan. 21(1):163-70. [Medline]. Leung WK, Kim JJ, Kim JG. Microsatellite instability in gastric intestinal metaplasia in patients with and without gastric cancer. Am J Pathol. 2000 Feb. 156(2):537-43. [Medline]. Malfertheiner P, Megraud F, O'Morain C, et al. Current concepts in the management of Helicobacter pylori infection--the Maastricht 2-2000 Consensus Report. Aliment Pharmacol Ther. 2002 Feb. 16(2):167-80. [Medline]. Rugge M, Genta RM. Staging and grading of chronic gastritis. Hum Pathol. 2005 Mar. 36(3):228-33. [Medline]. Shin CM, Kim N, Lee HS, et al. Validation of diagnostic tests for Helicobacter pylori with regard to grade of atrophic gastritis and/or intestinal metaplasia. Helicobacter. 2009 Dec. 14(6):512-9. [Medline]. Sipponen P, Harkonen M, Alanko A, et al. Diagnosis of atrophic gastritis from a serum sample. Clin Lab. 2002. 48(9-10):505-15. [Medline]. Vaananen H, Vauhkonen M, Helske T, et al. Non-endoscopic diagnosis of atrophic gastritis with a blood test. Correlation between gastric histology and serum levels of gastrin-17 and pepsinogen I: a multicentre study. Eur J Gastroenterol Hepatol. 2003 Aug. 15(8):885-91. [Medline]. Whittingham S, Mackay IR. Autoimmune gastritis: historical antecedents, outstanding discoveries, and unresolved problems. Int Rev Immunol. 2005 Jan-Apr. 24(1-2):1-29. [Medline]. Parsons BN, Ijaz UZ, D'Amore R, et al. Comparison of the human gastric microbiota in hypochlorhydric states arising as a result of Helicobacter pylori-induced atrophic gastritis, autoimmune atrophic gastritis and proton pump inhibitor use. PLoS Pathog. 2017 Nov. 13(11):e1006653. [Medline]. Moss SF. The clinical evidence linking Helicobacter pylori to gastric cancer. Cell Mol Gastroenterol Hepatol. 2017 Mar. 3(2):183-91. [Medline]. Ikeda F, Shikata K, Hata J, et al. Combination of Helicobacter pylori antibody and serum pepsinogen as a good predictive tool of gastric cancer incidence: 20-year prospective data from the Hisayama Study. J Epidemiol. 2016 Dec 5. 26(12):629-36. [Medline]. Kono S, Gotoda T, Yoshida S, et al. Can endoscopic atrophy predict histological atrophy? Historical study in United Kingdom and Japan. World J Gastroenterol. 2015 Dec 14. 21(46):13113-23. [Medline].

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