Eradication of Helicobacter pylori infection-related diseases.

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Background
According to the National Cancer Institute (NCI) (NCI, 2016) [1], Helicobacter pylori, or H. pylori, is a spiral-shaped bacterium that grows in the mucus layer that coats the inside of the human stomach. Although H. pylori infection does not cause illness in most infected people, it is a major risk factor for peptic ulcer disease and is responsible for the majority of ulcers of the stomach and upper small intestine. Other associated diseases include gastritis and gastrointestinal bleeding. It has been increasingly accepted that colonization of the stomach with H. pylori, is an important cause of gastric cancer and of gastric mucosa-associated lymphoid tissue (MALT) lymphoma (NCI, 2016). Infection with H. pylori is also associated with an increased risk of esophageal adenocarcinoma, (NCI, 2016) most likely from increased acid reflux causing Barrett's Esophagus, a precancerous state.

Management Problems of Chronic H. pylori Infection: Eradication versus Treatment
Eradication is defined as the presence of negative tests for H. pylori 4 weeks or longer after the end of antimicrobial therapy [2]. The tests for detection of H. pylori infection include non-invasive tests such as urea breath test and stool H. pylori antigen test. Invasive tests include serum H. pylori IgG antibody and biopsy of the gastric mucosa by esophagogastroduodenoscopy (EGD). Sampling errors in gastric biopsy can be associated with false negative results. False-negative results may also be observed on non-invasive tests in patients who are taking PPIs, bismuth, or antibiotics and in the setting of active peptic ulcer bleeding [3]. Serum IgG antibody titer is a good indicator of persistent infection. Since H. pylori is a chronic infective condition, the use of IgG antibody as a marker of H. pylori chronic disease is useful to monitor disease activity. We aim to define eradication of H. pylori infection based on the serum IgG titers. According to current practice, it is assumed that treatment is completed after a standard two-week course of antibiotics (Amoxicillin, Clarithromycin, with or without the use of Metronidazole) combined with a protein pump inhibitor (Omeprazole). Despite a completion of a two-week course of therapy, some patients may continue to exhibit recurrent symptoms of H. pylori infection, probably indicating an ongoing low grade infection. Our experience shows that a re-challenge treatment with the same regimen decreases IgG antibody titers and was associated with an improvement in symptoms. It was noted that a complete resolution of the symptoms is associated with the normalization of the IgG antibody titers. This is a better indicator of eradication of H. pylori infection. Hence, a follow-up laboratory testing is necessary to monitor antibody titers.

If the titers are positive, then the initial two-week treatment is considered inadequate for the eradication of H. pylori infection. A re-challenge with additional courses of treatment needs to be continued as long as the IgG antibody remains positive. In that way, H. pylori eradication can be achieved.

The current practice standard recommends against treatment of asymptomatic patients with H. pylori infection detected by serum IgG antibody testing. These patients are known to have an increased risk of gastric cancer [4]. Therefore, both asymptomatic and symptomatic patients need to be treated completely. The patients in this retrospective analysis tested negative for urea breath test, stool H. pylori antigen and gastric mucosa biopsy, but tested positive for IgG antibody titers. Asymptomatic patients reported subjective improvement after initial course of treatment. This indicates that being asymptomatic is not a reliable marker for withholding therapy. From our experience, serum IgG antibody to H. pylori antigen is more reliable than other tests to monitor eradication of H. pylori infection. Further studies need to be performed to have a better understanding of eradication of chronic H. pylori infection, a leading cause of gastric cancer in developing and undeveloped countries in the world.

References

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