Helicobacter pylori-related Chronic Gastritis

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Chronic gastritis is one of inflammatory diseases on gastric mucosa caused by various etiologies. One of the most important causes of gastritis which recently has been studied is infection by a microorganism called Helicobacter pylori.1-3 It is a gram-negative microaerophilic microorganism that can inhabit area between mucus and gastric mucosa and protected from gastric acid. It has 6 flagella and produces enzymes such as oxidase, catalase, protease and urease which may damage the gastric mucosa. Helicobacter pylori infection may be transmitted among patients through oral to oral or fecal to oral transmission. Although there is no evidence nowadays, but it is assumed that contaminated food, drinks and utilities may be one of other route of transmission. The presence of Helicobacter pylori infection may be determined either by non-invasive tests (serologic, Urea Breath Test, stool antigen Helicobacter pylori test) or invasive tests through upper gastrointestinal endoscopy (microorganism culture, pathology anatomy, urease test and PCR). Clinical manifestations that generally may be found in patients with Helicobacter pylori-related chronic gastritis are similar to patients with dyspepsia i.e. epigastric pain and discomfort, bloating, belching, early satiety, anorexia, nausea and vomiting which may occur for long period of time. Actually, such infection of Helicobacter pylori may also cause gastric/duodenal ulcer, cancer and lymphoma in addition to the chronic gastritis. Inflammatory response to such bacterial infection stimulates the G cells in the antrum to secrete the hormone gastrin. Gastrin stimulates the parietal cells in the corpus to secrete more gastric acid. Chronic increased gastrin serum level causes the number of parietal cells to increase which eventually enhances the gastric acid secretion. The increased acid damages the duodenum and resulting in ulceration. In contrast, gastric ulcers are frequently associated with normal or even reduced gastric acid production which indicates that there is a defect in defense mechanism of gastric mucosa. In these patients with chronic gastritis, Helicobacter pylori can colonize the gastric corpus, where the parietal cells are located. However, chronic inflammation caused by the bacteria brings further reduction of acid production and produces atrophy of the gastric mucosa, which may lead to gastric ulcer and increases the risk for stomach cancer.3-5 One of the laboratory tests that has been studied and can detect the presence of gastric mucosa atrophy non-invasively includes the gastrin, pepsinogen I, pepsinogen II level and pepsinogen I/II ratio. Indeed, the results worldwide on such tests are still controversial and the sensitivity/specificity are not so high. However, when in combination with endoscopy, it may become a very good screening tool for detecting gastric atrophy. Arinton, in the article of his study found that gastrin serum level and PGI/PGII ratio can be utilized as a biomarker for diagnosing chronic gastritis caused by Helicobacter pylori infection. Gastrin level of >5.89 pmol/L and PGI/PGII ratio ≤13.6 can be used as a biomarker on the presence of gastric atrophy, intestinal metaplasia, and gastric cancer. As other studies in the world, the study also has some limitations, i.e. it could not control the consumed drugs and eating habit; whereas, the results actually depend greatly on cessation of PPI/H2RA drug consumption and certain food which may interrupt the study results. Other limitation of the study is that it did not evaluate the virulence factors (vac A, Cag A, etc), cytokines, gastric CA and NO level, which the positive results are correlated to more severe condition of gastric defect.
REFERENCES