

Effect of cumulative time of *Helicobacter pylori* infection on gastric precancerous lesions?

Arif Mansur Coşar 

Division of Gastroenterology, Department of Internal Medicine, Karadeniz Technic University School of Medicine, Trabzon, Turkey

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Helicobacter pylori is classified as a grade 1 carcinogen affecting more than half of the world's population (1). In the pathogenesis of intestinal-type non-cardia adenocarcinoma, *H. pylori*-induced chronic gastric inflammation slowly progresses as a sequence known as Correa's cascade: non-atrophic gastritis, multifocal atrophic gastritis without intestinal metaplasia, intestinal metaplasia, dysplasia, and ultimately, cancer (2,3). Mucosal atrophy and intestinal metaplasia have been considered precancerous lesions (4). The sequential nature of gastric carcinogenesis presents an opportunity for early detection and intervention to prevent the progression of premalignant lesions. Among individuals with intestinal metaplasia (IM), the risk of progression to gastric cancer may vary by IM type (3).

Whether precancerous lesions can be reversed by *H. pylori* eradication has not been fully clarified. Some systematic reviews have reported that *H. pylori* eradication results in significant improvement in mucosal atrophy, whereas improvement in IM was not observed (5-7). However, other recent studies have reported a correlation between *H. pylori* eradication and improvement in IM as well as mucosal atrophy (4,8-10).

Most randomized trials evaluating the effect of anti-*H. pylori* therapy on the progression of precancerous lesions have been limited by short follow-up periods. In addition, the effect of the duration of *H. pylori* infection exposure on the progression of gastric lesions has not been studied in detail and for a long time. Mera et al. (3) evaluated the "effect of cumulative time of *H. pylori* infection" on the progression of gastric lesions over 16 years (during 1991-

2008). That study was based on an earlier randomized, placebo-controlled, three-way factorial trial in the Colombian Andes mountains evaluating the "effects of anti-*H. pylori* therapy with or without vitamin supplementation (ascorbic acid and/or beta-carotene) on individuals with gastric precancerous lesions" (11). They reported that 16 years of cumulative *H. pylori* exposure leads to significant progression of precancerous lesions. The benefits of *H. pylori* eradication were greater in individuals with atrophic gastritis (without IM) than in those with IM at baseline. In a Latin American population with a high gastric cancer risk, individuals with incomplete-type IM at baseline had a higher risk of progression to gastric cancer than those with only the complete type. The detrimental effect of long-term continuous *H. pylori* exposure provides additional evidence that effective anti-*H. pylori* therapy should be administered, as an adjunct to endoscopic surveillance, in individuals with IM. Individuals with incomplete-type IM should be considered to have a higher risk for gastric cancer than those with only the complete type, and they may require more frequent endoscopic surveillance and more extensive gastric mapping.

Consequently, it can be speculated that the effective treatment of patients with *H. pylori* infection and gastric malignancy risk factors, although they are asymptomatic, is very important and prophylactic.

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ORCID ID of the author: A.M.C. 0000-0002-4472-2895.

Corresponding Author: Arif Mansur Coşar; arif@doctor.com

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