

## COMMENTED SUMMARIES FROM CURRENT MEDICAL LITERATURE

### HELICOBACTER PYLORI INFECTION AND THE DEVELOPMENT OF GASTRIC CANCER

**Summary:** Although many studies have found an association between *Helicobacter pylori* infection and the development of gastric cancer, many aspects of this relation remain uncertain. We prospectively studied 1526 Japanese patients who had duodenal ulcers, gastric ulcers, gastric hyperplasia, or nonulcer dyspepsia at the time of enrollment; 1246 had *H. pylori* infection and 280 did not. The mean follow-up was 7.8 years (range, 1.0 to 10.6). Patients underwent endoscopy with biopsy at enrollment and then between one and three years after enrollment. *H. pylori* infection was assessed by histologic examination, serologic testing, and rapid urease test and was defined by a positive result on any of these tests. Gastric cancers developed in 36 (2.9%) of the infected and none of the uninfected patients. There were 23 intestinal-type and 13 diffuse-type cancers. Among the patients with *H. pylori* infection, those with severe gastric atrophy, corpus-predominant gastritis, and intestinal metaplasia were at significantly higher risk for gastric cancer. We detected gastric cancers in 21 (4.7%) of the 445 patients with nonulcer dyspepsia, 10 (3.4%) of the 297 with gastric ulcers, 5 (2.2%) of the 229 with gastric hyperplastic polyps, and none of the 275 with duodenal ulcers. Gastric cancer develops in persons infected with *H. pylori* but not in uninfected persons. Those with histologic findings of severe gastric atrophy, corpus-predominant gastritis, or intestinal metaplasia are at increased risk. Persons with *H. pylori* infection and nonulcer dyspepsia, gastric ulcers, or gastric hyperplastic polyps are also at risk, but those with duodenal ulcers are not.

**Comment:** The incidence of gastric adenocarcinoma has decreased in western industrialized countries during the past half a century.<sup>1</sup> A striking feature of this decline is a concomitant rise in the incidence of gastric cardia cancer<sup>2</sup>, which is now becoming more common than adenocarcinoma of the distal esophagus. In developing countries the overall incidence of gastric cancer is increasing and projections indicate that the annual number of new cases will increase significantly during the next few decades as a result of population aging.<sup>2</sup>

Initial reports<sup>3</sup> attribute only 60% of cases of gastric cancer to *H. pylori* (HP) infection, but in this study 3% of patients with HP infection 5% and about of those presenting with nonulcer dyspepsia (NUD) developed stomach cancer during a period of 7.8 years. According to this study, the treatment of NUD patients with HP infection may prevent the development of gastric cancer, because none of the subjects without HP infection developed the malignancy.

In developing countries like Iran where gastric cancer is the most common lethal malignancy and 90% of population more than 40 years of age have HP infection and about 40% of them have atrophic gastritis, the role of HP in the initiation of carcinogenesis process as suggested by Correa<sup>4</sup> is again reiterated. HP may be responsible for the majority of stomach cancers. Worldwide, stomach cancer is thought to be responsible for more than one million deaths annually. The majority of these deaths can be prevented if we can prevent or treat HP infection at an appropriate time. It is now the time to revamp the strategy of HP eradication in NUD subjects.

Therefore, it is imperative that strategies for HP eradication in NUD subjects to be reviewed and and redefined. Close attention should be paid to the findings of research conducted to determine genetic and environmental factors responsible for gastric carcinogenesis. Such studies may help better define the subgroups of HP-infected subjects in whom HP eradication is more cost-effective in preventing stomach cancer. Development of a safe and effective HP vaccine is probably the best and most cost-effective way of gastric cancer prevention.

## Commented Summaries

### References:

1. Parkin DM, Laara E, Muir CS. Estimates of the worldwide frequency of sixteen major cancers in 1980. *Int Cancer*. 1988; **41**: 184-97.
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4. Correa P. A human model of gastric pathogenesis. *Cancer Res*. 1988; **48**: 3554-60.

**Author:** Reza Malekzadeh MD, Digestive Disease Research Center, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

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