

PROFESSIONAL PAPER

Helicobacter Pylori Infection And Precancerous Lesions of The Stomach

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Introduction: Chronic atrophic gastritis, intestinal metaplasia, hereditary non-polyposos colon cancer, gastric dysplasia, gastric adenoma, Barrett esophagitis and familiar adenomatous polyposis are confirmed precancerous lesions of the stomach. Most of these conditions are correlated with long-term infections with *Helicobacter pylori*. **Material and method:** Patients which were included in our study underwent gastro endoscopy with multiple biopsies from antrum and corpus ventricle, also urease test and histopathological examination, using special coloring for *Helicobacter pylori*. **Results:** 802 patients entered this study, of which 369 female and 483 male. Among female patients 56.4% resulted *Helicobacter pylori* positive, whereas among male patients this was at a rate of 62.6%. The most affected age was 40-49 years, in which group *Helicobacter pylori* infection was 64.2%. In each precancerous lesion positivity of *Helicobacter pylori* infection was very high.–in patients with intestinal metaplasia: 71.7%, with gastric dysplasia: 71.4%, with gastric ulcer: 68.4%, with atrophic gastritis: 66.0% and with Barrett esophagitis: 55.0%. **Discussion:** The main purpose of this study was to determine the percentage of *Helicobacter pylori* infection among patients with precancerous lesions, which resulted to be very high. The highest percentage of infection resulted in patients with intestinal metaplasia (71.7%). **Conclusions:** Precancerous lesions of stomach are associated with high percentage of *Helicobacter pylori* infection. This confirms once more the importance of *Helicobacter pylori* eradication in early stages and patient's surveillance. **KEY WORDS: PRECANCEROUS LESION, HELICOBACTER PYLORI INFECTION**

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1. INTRODUCTION

A precancerous lesion is a histopathological abnormality in which cancer is more likely to occur than in its apparently normal counterpart (1).

As described in the literature, confirmed precancerous lesions of the stomach are: chronic atrophic gastritis, intestinal metaplasia, hereditary non-polyposis colon cancer, *gastric dysplasia*, *gastric adenoma*, *Barrett esophagitis* and *familiar adenomatous polyposis* (the last four lesions are indications for patient surveillance) (2,3).

Helicobacter pylori is the causative

agent of most peptic ulcers and other serious outcomes such as atrophic gastritis, intestinal metaplasia, dysplasia and

gastric cancer are correlated with long-term infections (4,5). When antibodies to *H. pylori* in stored serum from patients were measured, it was found that persons with previous *H. pylori* infection (seropositive) had a significantly greater frequency of gastric cancer; although such studies have to be corroborated and carefully interpreted, *H. pylori* may represent another possible factor in the development of gastric cancer(3).

2. MATERIAL AND METHOD

802 patients with disorders of the upper gastrointestinal tract were examined. All these patients underwent gastro endoscopy with multiple biopsies from antrum and corpus ventricle. Urease test of each patient was read after 30, 60, 120 minutes and after 24 hours. Histopathological examination was made by the pathologist, using special coloring for *Helicobacter pylori*.

3. RESULTS

802 patients entered this study, of which 369 female and 483 male. Among female patients 56.4% resulted *Helicobacter pylori* positive, whereas among male patients 62.6%. The most affected age was 40-49 years, in which group *Helicobacter pylori* infection was 64.2%. In each precancerous lesion positivity

	H.Pylori				Total
	Positive		Negative		
	N	%	N	%	
Total	479	59.7	323	40.3	802
Intestinal metaplasia	33	71.7	13	28.3	46
Gastric dysplasia	5	71.4	2	28.6	7
Gastric ulcer	39	68.4	18	31.6	57
Atrophic gastritis	93	66.0	48	34.0	141
Barret esophagitis	6	50.0	6	50.0	12

TABLE 1. Prevalence of *H. pylori* according to disease

of *Helicobacter pylori* infection is very high. The highest percentage of infection resulted in patients with intestinal metaplasia (71.7%). Among our patients with gastric dysplasia, 71.4% were *Helicobacter pylori* positive, with gastric ulcer 68.4, with atrophic gastritis 66.0, and among those with Barret esophagitis – 50.0%.

4. DISCUSSION

Among our patients with atrophic gastritis, percentage of *Helicobacter pylori* infection was 66(%), whereas in those with intestinal metaplasia of stomach, *Helicobacter pylori* infection was 71.7%. Chronic atrophic gastritis and intestinal metaplasia are frequently considered as precancerous lesions (6). *H.pylori* may cause a gastritis that might progress to chronic atrophic gastritis, a possible precursor lesion for gastric cancer, when antibodies to *H.pylori* in stored serum from patients were measured; it was found that persons with previous *H.pylori* infection (seropositive) had a significantly greater frequency of gastric cancer (3). *H.pylori* is a Gram-negative microaerophilic bacterium that typically establishes a lifelong infection in humans after acquisition during childhood (10). Virtually all hosts develop gastritis but only a small subset progress to chronic atrophic gastritis (ChAG), a condition characterized by loss of acid-producing parietal cells. ChAG is, in certain instances, an antecedent to gastric adenocarcinoma (11). Intracellular *H.pylori* has also been detected by transmission electron microscopy in preneoplastic and neoplastic gastric epithelium recovered by endoscopic biopsy of the stomachs of infected humans (12). Although typically viewed as an extracellular pathogen, a "liaison" between *H.pylori* and GEPs (gastric epithelial progenitors) may have implications for tumor genesis. Gastric stem cells are long-lived. The cancer-stem cell hy-

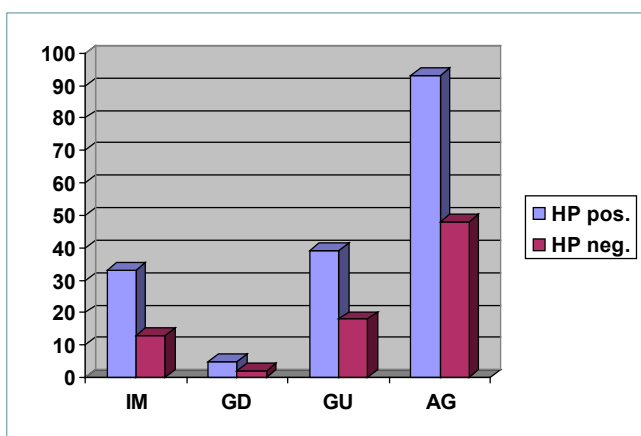


FIGURE 1. Prevalence of *Helicobacter pylori* according to disease

pothesis argues for a stem-cell origin of many tumor types (13). The concept that a bacterium, classified as a class I carcinogen (14) can adapt to an intracellular stem cell habitat suggests a potentially novel form of initiation of tumor genesis and begs the question of how bacterial and host cells establish and coevolve their relationship (15).

Intestinal metaplasia in general is not precancerous, but certain subtypes of this histologic phenomenon may be associated with gastric cancer and more likely to be precancerous (3). It is also to be noted that studies indicate that intestinal metaplasia is primarily associated with the intestinal type of gastric cancer, whereas no definite relationship has been demonstrated with the diffuse type of gastric cancer (7). Intestinal metaplasia is a precancerous condition and dysplasia is a precancerous lesion; the former is a clinical state associated with significantly increased risk of cancer, whereas a precancerous lesion is a histopathological abnormality in which cancer is more likely to occur than in its apparently normal counterpart (1). In the stomach, intestinal type metaplasia is most common; this occurs as a result of *Helicobacter pylori* infection, bile reflux (8) or can be induced experimentally by irradiation (9).

Our patients with gastric dysplasia were infected with *H.pylori* in 71.4% of cases. When mild dysplasia is encountered, follow-up biopsies and examinations are recommended at intervals; if severe dysplasia is identified, more frequent follow-up is warranted (3).

Gastric ulcer is related with *H.pylori* infection in 68.4% of our patients. Re-

ferring to the literature, this association is 73% of a total of 320 patients harboring this organism in antral specimens (3). *H.pylori* generally resides in the mucous coat above the apical surface of the epithelium, but some mucosal invasion occurs. The organism itself, factors released from the organism, and the resulting inflammatory response must influence pathogenesis, damaging epithelial cells and retarding repair of acid/peptic activity-induced injury. Candidate factors include adhesion molecules, lipopolysaccharides, chemoaxins and cytotoxins (3).

5. CONCLUSION

Every patient suspected for a precancerous lesion at endoscopic examination, should undergo multiple biopsies and histopathological examination. Follow-up biopsies and examinations are recommended at intervals, according to the results for each patient.

The importance of *Helicobacter pylori* eradication is once more confirmed by the high percentage of infection among our patients with precancerous lesions.

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