Original Article

Association between Helicobacter pylori infection & atrophic gastritis

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Summary:

Background: *Helicobacter pylori (H. pylori) infection is associated with gastritis and may induce atrophic gastritis have specific circulating immunoglobulin G (IgG) antibodies.*

Aim of the study: *To confirm the correlation between Helicobacter pylori infection and gastric atrophy.*

Patient and Method: *A study was conducted in the period between December 2005 and March 2006 on 25 patients with atrophic gastritis attending Gastroenterology and Hepatology Teaching Hospital in Baghdad, and 25 healthy volunteers who agreed to donate blood. Sera were tested for H. pylori IgG Ab by ELISA test.*

Results and Conclusions: *detection of H. pylori IgG Ab were applied to each individual, showed that (92 %) of patients with atrophic gastritis had positive H. pylori IgG Ab were as only 4 (16 %) of normal healthy individuals had positive H. pylori IgG Ab.*

Key Words: *H. pylori and Chronic atrophic gastritis.*

Introduction:

Helicobacter pylori infection is associated with gastritis, peptic ulcer disease, and gastric malignancies *(1)*. In about one-third or up to one-half of those infected with *H. pylori*, gastritis proceeds to atrophic gastritis, resulting in a loss of mucosal glands, decreased helicobacter colonization and, when affecting the corpus mucosa, decreased secretion of pepsinogen I (PGI) *(2&3)*. It is postulated that *H. pylori* infection may induce atrophic gastritis, which results in a less acidic gastric refluxate, and *H. pylori* may also neutralize gastric acid by producing urease, independent of the presence or absence of gastric atrophy *(4)*. Most helicobacter-infected subjects have specific circulating immunoglobulin G (IgG) antibodies. Patients with atrophic corpus gastritis often have positive helicobacter serology, although microscopic examination *(5&6)*, culture of biopsy samples, and even the urea breath test remain helicobacter negative *(7)*. These particular patients may still be infected, as shown by rapidly falling antibody titers after therapy *(8)*. Enzyme immunoassay (EIA) is the most commonly used serological method for detecting antibodies to *H. pylori*. The best commercial kits have shown sensitivities and specificities of 90% to in excess of 95% *(9&10)*. This study focuses on the presence of *H. pylori* IgG Ab in patients with atrophic gastritis as a risk factor for gastric cancer.

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Patients & Methods:

A study was conducted in the period between December 2005 and March 2006, to compare the results of detecting *H. pylori* IgG Ab in the serum of the following groups:

1-Twenty-five patients attending Gastroenterology and Hepatology Teaching Hospital in Baghdad, who had undergone gastroscopy due to clinical indications and the histopathological examination reveals the presence of atrophic gastritis.

2-Twenty-five healthy volunteers who agreed to donate blood.

Fasting for 10 hours is recommended prior to blood sampling. Blood sample were taken from each individual, sera were tested for *H. pylori* IgG Ab by using commericially available ELISA (Biohit Plc).

Results:

The results presented in this section were based on the analysis of Twenty-five patients with atrophic gastritis, with age ranged between (40-69) years old with a female to male ratio 2.1. Table 1 shows the age and sex distribution of patient group.

Table 1-The age and sex distribution of patient group

Age	Male No. (%)	Female No. (%)	Female:male ratio
40-49 years	3 (12)	5 (20)	1.6
50-59 years	4 (16)	9 (35)	2.2
60-69 years	1 (4)	3 (12)	3.1
Total	8 (32)	17 (68)	2.1

Table 2 shows that 23 (92 %) out of Twenty-five patients with atrophic gastritis had positive *H. pylori* IgG Ab were as only 4 (16 %) of normal healthy individuals show positive *H. pylori* IgG Ab.

Table 2- Percentages of *H. pylori* IgG Ab

Groups	*H. pylori* IgG Ab positive No. (%)	*H. pylori* IgG Ab negative No. (%)	Total
Patient	23 (92)	2 (8)	25
Control	4 (16)	21 (84)	25

Discussion

A large amount of epidemiological evidence has accumulated indicating a significant relationship between *Helicobacter pylori* infection and chronic gastritis (11&12), the World Health Organization/International Agency for Research on Cancer concluded that `Hp is a definite carcinogen' based on the epidemiological findings (13). Hp infection almost always results in chronic antral gastritis (14&15). Until recently, it has been considered that Hp infection causes atrophic gastritis followed by development of intestinal metaplasia and well differentiated adenocarcinomas (16). Since Tomb *et al.* elucidated the complete genome sequence of the Hp in 1997 (17), it is likely that a fuller understanding will be generated in the near future. Concerning the host interaction, it was demonstrated that T helper 1 cellular immune responses contribute to *Helicobacter*-associated gastritis in mice (18) and man (19), and D'Elios *et al.* showed that Hp-specific T helper 1 effectors may play a role in peptic ulcers in humans (20). In our study, the titers of anti-*Helicobacter pylori* antibodies of the patient group were higher than in control group and the positive results among healthy individuals could be due to asymptomatic carriers of *Helicobacter pylori*. So we think it is appropriate that titers were evaluated within each group, but not in general, because different treatments may be indicated accordingly.

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