FREQUENCY OF HELICOBACTER PYLORI IN PATIENTS WITH GASTRIC CARCINOMA

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SUMMARY :

Purpose: *Studies concerning the etiology of gastric cancer (GC) revealed multiple carcinogenic factors that facilitate malignancy. In this study, we searched for the prevalence of Helicobacter pylori (HP) in cases of GC.* ***Methods:*** *Thirty GC patients (19 male, 11 female) and 30 healthy control subjects were included in the study.* ***Results:*** *GC patients were found to have 21 (70 %) intestinal type and 9 (30 %) diffuse type carcinoma. There was no significant difference between intestinal and diffuse type carcinoma patients with respect to gram staining and CLO (chlamidia-like organism) test for HP. CLO test was positive in 60 % and 40 % of patient group and control group, respectively, which was statistically significant. Gram staining was not significantly different for HP in either group.* ***Conclusion:*** *Our study shows correlation with the literature and HP infection increases GC incidence. But GC is a multifactorial disease and other etiological factors should not be neglected. As a result, HP may be an etiologic factor in GC but more data is needed to reach a realistic conclusion.*

Key Words: *Helicobacter Pylori, Stomach Neoplasms, Helicobacter Infections.*

INTRODUCTION

Gastric carcinoma represents the second cause of cancer mortality after lung cancer (1). Epidemiological investigations revealed a higher incidence in low socioeconomic groups (2). Environmental factors, especially nutrients, are important etiologic causes. Sanitation of nutrients leads to a dramatic decrease in the incidence of GC (3). For epidemiological purposes, Lauren classification is widely used. This classification divides GC into diffuse and intestinal types (4). For intestinal type GC, there are many hypotheses for carcinogenesis (5). The most famous of these is the study made by Correa et al. which claims an environmental agent (probably salt) causing metaplasia, dysplasia and cancer after conversion of nitrites to nitroasmines by bacteria in partial gastrectomised patients. Nevertheless, there is still some doubt about the exact mechanism of GC pathogenesis. Although salt intake carries a high risk for GC, nitrite-cancer association and bacterial overgrowth do not always exist (6). Recent studies indicated that the major cause of chronic gastritis is HP. This strong association induced many studies concerning the role of HP in GC etiopathogenesis. In prospective studies, HP positive patients had more atrophic gastritis and intestinal metaplasia (7). Epidemiological properties of HP and GC show strong correlation, such as high prevalence in low

socioeconomic levels and in developing countries (2). Another evidence is the high frequency of HP in GC patients. In a cross-sectional study, HP incidence varied between 50-100% in GC population (8). HP has affinity towards normal tissues but its affinity to metaplastic, dysplastic and cancerous tissues is low. For this reason, HP investigations should include adjacent normal tissue biopsies or if infection is being searched on the neoplastic area, diagnostic procedures other than biopsy should be considered (9). Biopsy studies only on malign tissues revealed less than 10 % incidence of HP infection (10). By which mechanism HP causes malign transformation is a question of debate. There are many assumptions for the pathogenesis. Some of them are; damage to the gastric mucosa by HP catabolic products, integration of HP DNA into host cells and causing transformation or genotoxic inflammatory response of the gastric mucosa (5) (Fig. 1). In this study, we aimed to search for possible association between gastric adenocarcinoma (not lymphomas) and HP. For this reason, we excluded gastric lymphoma cases.

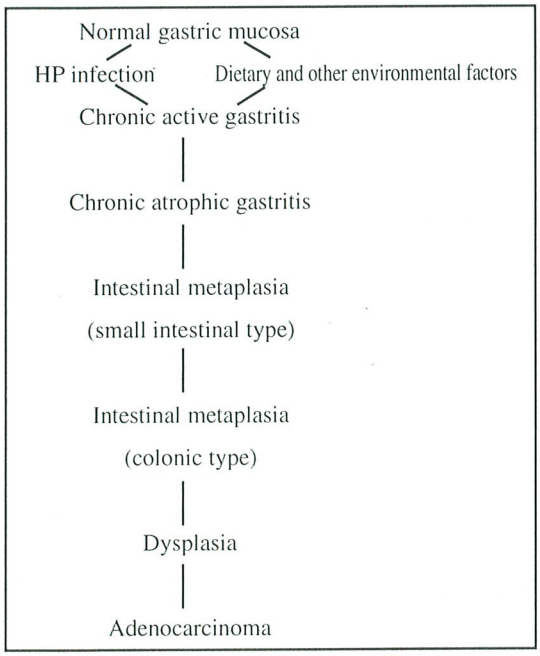

Fig. 1 : A human model of gastric carcinogenesis

MATERIAL AND METHODS

Study group consisted of 30 GC cases and 30 healthy subjects without any dyspeptic complaints. The patients did not take any anti-ulcer treatment until 8 weeks prior to the study. Pentax IPM – 300 Video endoscope was used for upper gastrointestinal investigation. In the GC group, four biopsies were taken from malign areas and preserved in 10 % formalin solution until pathologic examination. Histopathologic analysis was done by Lauren classification. For HP detection, two biopsies were taken from apparently normal mucosa from the prepyloric region. One of these biopsies was used for CLO test and the other for the gram staining. Either test result confirmed the diagnosis of HP infection. That means any positive result revealed HP infection (either CLO test or gram staining). The sterilization of the endoscopic biopsy instruments was done by 10 % succinylaldehyde and Hexanios solution (50 ml/ 5 lt) containing water applied for 30 minutes. CLO tests evaluated at 20. minute, 3. hour and 24. Hour. The results were statistically analyzed with chi-square test.

RESULTS

The patient group (GC) consisted of 11 female and 19 male patients, and the control group 10 females and 20 males. The ages were 63±22.4 (range (50 - 76) years and 53.5±31.6 (range 48 - 59), respectively. Histopathologic evaluation revealed 9 cases (30 %) of diffuse type and 21 cases (% 70) intestinal type GC. In this study, only the cases with adenocarcinoma were included, excluding other pathologic types such as MALT lymphomas. In GC patients, positive HP results with CLO test and gram staining were 18 (60 %) and 12 (40 %) respectively. In the control group, the results were 12 (40 %) and 10 (35 %), respectively (Table 1). The difference between GC and control group with CLO test was statistically significant ($p<0.05$) but with gram staining the results were similar ($p>0.05$) (Fig. 2). Neither CLO test nor gram staining revealed any difference between intestinal and diffuse type of GC ($p>0.05$).

Table 1 : Results of CLO test and gram staining in GC and control patients.

CLO test*	Gram staining	
GC	18 (60)%	12 (40%)
Control group	12 (40%)	10 (35%)
*p< 0.05		

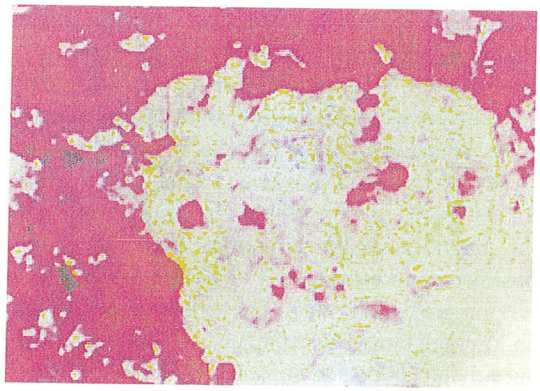

Fig. 2 : Gram staining of HP in the gastric mucosa biopsy material (100).

DISCUSSION

The role of chronic HP gastritis in the etiopathogenesis of GC is still a matter of interest. In a study conducted in Holland, HP was positive in 61 % of GC patients, but only 34 % of the control group was negative for HP (8). Another larger study performed by Tolley revealed HP prevalence 2.7 times greater in GC group in comparison with the control group (11). In the same study, no correlation was found between intestinal and diffuse types of GC for HP infection. Miscellaneous study results showed that the probability of HP infection decreases in the gastroesophageal tumors with respect to tumors of the antrum, corpus and fundus (12). In our study, HP positivity was found in 60 % of GC patients but 40 % in the control group. The results of both GC and the control group were parallel to the literature findings. We also could not find any correlation between histologic types of GC and HP positivity, as in the literature (13,14). Worldwide, studies show variable results of HP infection rate in GC patients between 19 to 94 % (15). In our country, these rates are between 40-66 % (16-18). In the light of those studies, when the rate of HP infection is about 80 % in our country (19), the finding of a relatively low HP positivity in our study may indicate the multifactorial nature of GC etiopathogenesis. In our study, controls had different demographical properties. They were from different parts of Turkey and were not a selected population. This might have an effect on our study and may be the cause of low levels of HP positivity. In our opinion, the cases should be searched for other possible factors, such as pernicious anemia, chronic atrophic gastritis, and history of gastrectomy, glandular dysplasia of the cardia, excessive nitrite and nitrate intake and corrosive injury. In spite of these factors, the role of HP in GC etiology should not be neglected. Another study concerning the possible association between HP and gastric lymphoma has revealed significant results (20). For this reason, a multidisciplinary approach is necessary. First of all, the number of GC patients in our country and the ratio of HP positivity in several groups of these patients should be carefully investigated. As a result, the question of which cases should be treated for HP arises. In our country, where the rate of HP positivity reaches 80%, such an expensive model of treatment option should be discussed. Our opinion is to select cases with high risk factors, such as atrophy, intestinal metaplasia, dysplasia etc., and to give them eradication therapy.

In conclusion, our study shows correlation with the literature. GC is a multifactorial disease and more data is needed to reach a realistic conclusion.

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