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ORIGINAL ARTICLE

Determine the Prevalence of cagA and Baba of Helicobacter Pylori Isolated from Gastric Atrophic Patients

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ABSTRACT

Objective: The aim of this study is to determine the prevalence of cagA and babA of helicobacter pylori isolated from gastric atrophic patients.

Study Design: Descriptive/Analytical

Place and Duration: The study was conducted at Medicine/Gastroenterology department of Khyber Teaching Hospital and Peshawar Institute of Medical Sciences, Peshawar for six months duration from March 2020 to August 2020.

Methods: Total one hundred and twenty patients of both genders were presented in this study. Patients were aged between 20-80 years of age. Patients detailed demographics age, sex and body mass index were recorded after taking informed written consent. All patients of gastroduodenal disorders were undergone for isolation of bacteria by using standard techniques. Complete data was analyzed by SPSS 22.0 version.

Results: Total 50 (41.7%) patients were males and 70 (58.3%) patients were females. Mean age of the patients were 41.96 ± 16 years with mean BMI 25.24 ± 4.8 kg/m^2. Frequency of H pylori was isolated in 30 (25%) patients in which 13 patients had atrophic gastritis, 9 patients had gastric ulcer and 8 patients had acute gastritis. Prevalence of cagA gene was 16 (53.33%) and babA was 10 (33.33%) in H. pylori isolated patients. Significantly difference with p value <0.05 was observed between cagA positive strains and patients of gastric atrophic. The involvement of gastric atrophic patients was not correlated to the babA gene.

Conclusion: We concluded in this study that different cagA positive H. pylori can be retrieved from gastric atrophy patients.

Keywords: Gastric atrophy, Gastric cancer, cagA, babA, Helicobacter pylori

INTRODUCTION

An infection caused by Helicobacter pylori is one of the world's most prevalent infectious disorders. It was clear that the patient's illness and stomach pain were both very serious [1]. More than half of the world's population is infected with this pathogen. H. pylori plays a role in gastric, duodenal, and intestinal lymphoma, according to the National Cancer Institute. There have been several genes implicated in the pathophysiology of H. pylori [2-5]. There are three known factors linked with high virulence in H. pylori: the cytotoxin-associated product, the vacuoling toxin, and the adhesion protein babA2. [4]

Genetic disorders are more common than diseases that express babA. In addition, cagE gene expression has been linked to more significant clinical outcomes[5]. Epithelium (iceA) gene contains two significant allelic variations, iceA1 and iceA2. H. pylori and human epithelial cells can interact to increase the expression of Ice A1, which has been associated to peptic ulcer disease. External inflammatory protein A (oipA) expression and clinical outcomes are connected to IL-8 induction[5]. Iran has a high prevalence of H. pylori infection, but genotyping of H. pylori strains is incomplete. [6,7]

VacA and CagA, coupled with the simultaneous presence of vacAs2 and cagA, appear to be required for successful H. pylori supply in the German population[8] (triple-positive strains). BabA may potentially play a role in the etiology of severe histological alterations, such as atrophy or gut metaplasia [9]. We found no correlation between babA2 and cagA or vaca status in other research [10,11], nor did we find any correlation between babA2 with the clinical outcome. Thus, the present study aimed to compare these virulence factors of H. pylori isolated from patients in four European countries in order to further investigate the correlation between babA2, cagA, and vacAs1 genotypes and disease, and to examine the effect of genetic diversity on babA2's adhesive activity.

MATERIAL AND METHODS

This descriptive/analytical study was conducted at Medicine/Gastroenterology department of Khyber Teaching Hospital and Peshawar Institute of Medical Sciences, Peshawar for six months duration from March 2020 to August 2020and comprised of 120 patients. After taking informed written consent, detailed demographics including age, sex and body mass index were recorded. Patients who had treatment with antibiotics, H2-receptor blockers, bismuth-containing compounds, H. pylori eradication therapy protocol and those were not agreed excluded from this study.

Patients were aged between 20-80 years of age. All patients of gastroduodenal disorders were undergone for isolation of bacteria by using standard techniques. Each of the specimens was immediately put into the transport

medium of Stuart and shipped within 2 hours at 4°C to the laboratory. The specimens have been smeared on the surface with 10% horse serum, 10 mg/l vancomycin, 5 mg/ l cerebrum and 5 mg/l amphotericin B in addition to 10% horse serum and an antibiotics serum. Plates at 37°C (5 % O2, 10 % C2 and 85 % N2) were then incubated and analyzed after seven days of incubation. After this, they were incubated. Statistical analysis was measured by chi square and Fischer's test. Complete data was analyzed by SPSS 22.0 version.

RESULTS

Total 50 (41.7%) patients were males and 70 (59.3%) patients were females. Mean age of the patients were 41.96 ± 16 years with mean BMI 25.24 ± 4.8 kg/m^2. Frequency of H pylori was isolated in 30 (25%) patients (Table 1)

Table 1: Baseline detailed demographics on enrolled cases

Variables	Frequency (n=120)	% age
Sex		
Male	50	41.7
Female	70	58.3
Mean age (yrs)	41.96 ± 16	
Mean BMI (kg/m$^{2)}$	25.24 ± 4.8	
Prevalence of H pylori		
Yes	30	25
No	90	75

Among 30 (25%) patients of H pylori isolated, 13 patients had atrophic gastritis, 9 patients had gastric ulcer and 8 patients had acute gastritis. (Table 2)

Table 2: Distribution of Helicobacter pylori isolated patients with respect to disease

Variables	Frequency (n=30)	%age
Atrophic gastritis	13	43.33
Gastric ulcer	9	30
Acute gastritis	8	26.7

Prevalence of cagA gene was 16 (53.33%) and babA was 10 (33.33%) in H. pylori isolated patients. Significantly difference with p value <0.05 was observed between cagA positive strains and patients of gastric atrophic. The involvement of gastric atrophic patients was not correlated to the babA gene. (table 3)

Table 3: Prevalence of cagA and babA of helicobacter pylori isolated

Variables	Frequency (n=30)	%age
cagA gene	16	53.33
babA gene	10	33.33
Others	4	13.33
Total	30	100

DISCUSSION

It is well known that H. pylori colonizes more than half of the world's human population. [12] Previous studies have documented that H. pylori isolates obtained from various parts of the world vary in the genotype and frequency of the vacAcagA gene. These differences of genotype influence the clinical presentation in patients infected with H. pylori. In many East Asian countries the presence of cagA is variable from a minimum of 50% in some Middle East[9] to a maximum of 99% [13, 14] CagA-positive H. pylori strains were found in 53.33% less than the European and Northern American studies published (74% to 88%). [15-17] In some trials, cagA has been suggested to be a useful marker for the most virulent strains of peptic ulcer, atrophic gastritis and adenocarcinoma. [18]

In this analysis, the prevailing rates for BabA were 33.3%, down from 57% in Colombia, and 73.7% in Costa Rica, 97.4% in Chilie and 96.8% in Japan. [19-21] The rate of incidence of babA2 was 71.6 percent in a report from Isfahan, Iran. They indicated that genotype and clinical results have no relationship (gastritis and PUD)[22]. BabA2, which is curiously unconnected to clinical results, is the majority of H. pylori strains in asia [23]. A total of 120 patients, 58.3% of the patients in our sample, and the rest 41.7% of males, were presented. The patients' mean age was 41.96 ± 16 years, the average BMI was 25,24 ± 4,8 kg/m2. In 30 (25 percent) patients the frequency of H pylori has been isolated. These findings were similar to previous research. [24, 25] In our sample 13 patients with astrophic gastritis, 9 patients with a gastric ulcer, 8 acute gastritis, out of 30 (25 per cent) isolated patients with H pylori. These demonstrated similarities with SafarnejadS etal's previous research in 2020. [24]Dabiri et al. indicated that caga and cagE status or vacA genotypes had no clear correlation to clinical results. In nonulcer dyspepsia, the oipA-positive strains were more common than in peptic ulcers. [6]

HomB as an external membrane protein is extremely critical for adherence to the gastric epithelium in infection and an increase in H. pylori colonization. A statistically significant association was observed in this analysis between homB and aspA ($p < 0.0001$) and babA ($p< 0.05$). On the other hand, according to this report, there was no important relationship between homB and sabA. [26] H. pylori has a large number of various adhesive components for binding carbohydrates. In primary colonization of H. pylori, the sabA has a crucial and significant role. H. pylori sabA proteins are also taking part in stable infections and development of chronic inflammation which directs to tissue damage. [27] The report is in progress. A diversity gene is H. pylori sabA. The sabA gene is linked to various stomach conditionssuch as 100% in gastric cancer, 86.7% in gastric ulcer, and 83.3% in gastritis and duodenal ulcer. [28]

CONCLUSION

We concluded in this study that different cagA positive H. pylori can be retrieved from gastric atrophy patients.

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