

ORIGINAL ARTICLE

FREQUENCY OF *HELICOBACTER PYLORI* IN DISTAL OESOPHAGEAL MUCOSA OF PATIENTS WITH DYSPEPSIA

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Background: *Helicobacter pylori* (*H. pylori*) is one of the common causes of dyspepsia. The present study was conducted to find the frequency of *H. pylori* in the distal oesophageal mucosa of patients with dyspepsia. **Methods:** This descriptive cross sectional study was conducted in Services Hospital Lahore. History and physical examination was recorded and after informed consent oesophagoduodenoscopy of all the patients with the symptoms of dyspepsia was done. Findings were noted and gastric antral and distal oesophageal biopsies taken simultaneously. Both specimen were preserved in 10% formalin and sent for histopathological examination for the presence of *H. pylori*. **Results:** Out of the 116 patients, 16 patients were between ages 16–30, 82 patients were between ages 31–45 years and 18 were over 45 years of age. Thus percentage of subjects between 31–45 years was maximum i.e., 70.68%. Seventy-six (65.5%) of the patients were male and 40 (34.5%) were females. The *H. pylori* was found in 40 (34.5%) patients in gastric antral biopsy and it was isolated in only 14 (12.1%) patients in distal esophageal biopsies. **Conclusion:** *H. pylori* positivity was low in the distal oesophageal mucosa of patients with dyspepsia despite its presence in gastric mucosa. A close relationship could not be established between *H. pylori* in the distal oesophagus and gastric antral mucosa in dyspeptic patients. Based on these findings, it seems that there is no significant evidence for an important pathogenic role for *H. pylori* infection in the development of pathologic dyspepsia and chronic gastroesophageal reflux disease.

Keywords: *Helicobacter pylori*, dyspepsia, reflux, gastroesophageal

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INTRODUCTION

Helicobacter pylori is one of the most common human infections worldwide particularly in the developing countries. It has been established as aetiology of chronic gastritis and peptic ulcer disease, gastric adenocarcinoma and mucosal associated lymphoid tissue lymphoma (MALT).¹

Infection with *H. pylori* occurs worldwide, but the geographical prevalence varies greatly, exceeding 90% in developing countries compared to 20–50% in developed countries.² The prevalence appears to be inversely proportional to socioeconomic status.³ Colonization of *H. pylori* occurs in childhood and persists throughout life.⁴

The antral mucosa is thought to be the elective site for *H. pylori* colonization and related histological lesions. At a later stage, *H. pylori* can be detected only in the oxyntic area while the antral mucosa shows extensive metaplastic or atrophic lesions.⁵ Atrophic gastritis and intestinal metaplasia occurs predominantly at the gastric antrum and incisura with *H. pylori* infection. Antralization of the gastric incisura is a common event in *H. pylori*-infected patients, and appears to be associated with an increased risk of atrophic gastritis and intestinal metaplasia.⁶ The esophagus is a potential environment for bacterial colonization: alterations of the microenvironment, such as reflux esophagitis,

Barrett's esophagus, and esophageal carcinoma, could affect the bacterial biota. The *H. pylori* status of the gastric contents could also alter the upstream esophageal biota, due to its effects on gastric acidity and reflux.^{7,8}

In developed countries only a few studies have evaluated the colonization of *H. pylori* in the esophageal mucosa.^{9–13} *H. pylori* and many other bacterial species have been detected in the esophagus of healthy patients and patients with reflux-related diseases and Barrett's esophagus.¹⁴ The aim of this study was to determine the occurrence of *H. pylori* in the distal esophageal mucosa of dyspeptic patients in local population of Lahore by using Haematoxylin-Eosin and Giemsa staining.

MATERIAL AND METHODS

This descriptive cross-sectional study was conducted in Services Hospital, Lahore from October, 2012 to April, 2013. One hundred and sixteen Patients attending medical outpatient department for the symptoms of dyspepsia requiring upper gastrointestinal endoscopy were recruited into this study. The patients were informed about the study objectives and completed a questionnaire regarding occupation, diet, and hygiene habits. Patients who had received antibiotics, antacids, or a proton pump inhibitor in the previous month were excluded from the study. Informed consent was

obtained from each patient and all the patients underwent upper gastrointestinal endoscopy.

Two paired biopsy specimens of the gastric mucosa (antrum) and esophageal mucosa (lower third or within 2 cm of the Z-line) were collected from each patient. For each mucosa, one biopsy was immediately placed in formalin for histopathology.

All the data collected through the *pro forma* was entered and analyzed by SPSS-16. Mean and Standard deviation (SD) was used for describing continuous variables. Frequency and percentage was calculated for Qualitative data, i.e., positivity of H Pylori in both the gastric antral and distal esophageal biopsies and age ranges.

RESULTS

One-hundred-sixteen patients who had symptoms of gastro esophageal reflux disease (GERD) and dyspepsia were selected according to selection criteria from medical outpatient department of Services Hospital, Lahore. Age ranged from 17–59 years with a mean of 39.31±8.48 years as shown in table-1. 16 patients were between ages 16–30, 82 patients were between ages 31–45 and 18 were over 45 years of age. Thus percentage of subjects between 31–45years was maximum, i.e., 70.68%. Seventy-six (65.5%) patients were male and 40 (34.5%) were females. Table-2 shows comparasion of age groups and *H. pylori* presence in gastric antrum. Out of 16 patients who were between 16–30 years of age range, 5 patients had positive biopsy for *H. pylori* in gastric antrum. Moreover, amongst 82 patients between ages of 31–4 years, 23 biopsies were positive for *H. pylori* infection and out of 18 patients who were more than 45 years of age, 12 patients had positive biopsy results for *H. pylori* infection in gastric antrum. Table-3 shows comparasion of age groups and *H. pylori* presence in distal esophagus. Out of 16 patients who were between 16–30 years of age range, 1 patient had positive biopsy for *H. pylori* in distal esophageal biopsy. Moreover, amongst 82 patients between ages of 31–4 years, 9 biopsies were positive for *H. pylori* infection and out of 18 patients who were more than 45 years of age 4 patients had positive biopsy results for *H. pylori* infection in distal esophageal mucosa.

Table-1: Distribution according to age n=116

| Age | Frequency | Percentage |
|-------|-----------|------------|
| 16–30 | 16 | 13.79 |
| 31–45 | 82 | 70.68 |
| >45 | 18 | 15.51 |

Table-2: Cross tabulation of age groups and *H. pylori* presence in gastric antrum

| Age range | Gastric Antrum | | Total |
|-----------|----------------|----|-------|
| | Yes | No | |
| 16–30 | 5 | 11 | 16 |
| 31–45 | 23 | 59 | 82 |
| >45 | 12 | 6 | 18 |

Table-3: Cross tabulation of age groups and *H. pylori* presence in Distal oesophagus

| Age range | Distal oesophagus | | Total |
|-----------|-------------------|----|-------|
| | Yes | No | |
| 16–30 | 1 | 15 | 16 |
| 31–45 | 9 | 73 | 82 |
| >45 | 4 | 14 | 18 |

DISCUSSION

The association of *H. pylori* infection in oesophageal mucosa was not found to be high or related in dyspeptic patients despite its presence in gastric mucosa. Several studies have been conducted and yielded mixed results. There is no significant evidence for an important pathogenic role for *H. pylori* infection in the development of pathologic chronic gastroesophageal reflux, erosive esophagitis or Barrett's oesophagus in accordance with my study results.¹⁵ The presence of gastric type mucosa within the oesophagus is a prerequisite for *H. pylori* colonization, and that *H. pylori* may contribute to the severity of inflammation in Barrett's epithelium.¹⁶ Helicobacter pylori play no role in the pathogenesis of gastroesophageal reflux disease or its complications.¹⁷ This study has shown that *H. pylori* did not colonise oesophagus in patients of esophagitis or patients of non-ulcer dyspepsia.¹⁸ The persistence of inflammation can induce other changes such as basal hyperplasia and dysplasia.¹⁹ *H. pylori* and many other bacterial species have been detected in theoesophagus of healthy patients¹⁰ and patients with reflux-related diseases¹⁴ and Barrett's esophagus^{16,20}. It is interesting to note that *H. pylori* eradication in dyspeptic patients may lead to an increased frequency of histopathological esophagitis.²¹ Most of the studies yielded these results except a very recent study conducted in Venezuela. *H. pylori* found in the gastroesophageal mucosa of dyspeptic patients by PCR and FISH was 86.1%, while it was detected in only one mucosa in 91% of the patients. Finally, this is the first report of the occurrence of *H. pylori* in the oesophageal mucosa of dyspeptic patients in the developing world. These results demonstrate the high prevalence of *H. pylori* in the oesophagus, and its presence was correlated with signs of inflammation; however, this association deserves further investigation to determine whether *H. pylori* is the direct cause as a result of gastric infection or whether it is the presence of other pathogen species producing opportunistic infections.²² This study has interesting conclusion with respect to the above mentioned study conducted in Venezuela recently. There could be many reasons for these discrepancies. First of all the technique used in the international study to isolate *H. pylori* in esophageal mucosa is state of the art and has better yield. Secondly the sample size of that study is larger. Moreover it could be possible that certain epidemiologic factors could come into play and *H. pylori* growth in the oesophageal mucosa could be area dependent.

Irrespective of the reason, it is quite clear from this study that further research in this area is required to get the clearer picture of *H. pylori* presence in oesophageal mucosa of dyspeptic patients and this could modify the future prospective regarding the management of dyspepsia.

CONCLUSION

H. pylori positivity was low in the distal oesophageal mucosa of patients with dyspepsia despite its presence in gastric mucosa. A close relationship could not be established between *H. pylori* in the distal esophagus and gastric antral mucosa in dyspeptic patients. Based on these findings, it seems that there is no significant evidence for an important pathogenic role for *H. pylori* infection in the development of pathologic dyspepsia and chronic gastroesophageal reflux disease.

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