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Research Article

HELICOBACTER PYLORI INFECTION IN PATIENTS WITH GASTRIC CARCINOMA

¹Dr. Muhammad Adnan Bawany, ²Dr. Kiran Hafeez, ^{3*}Dr. Majid Ali Soomro,
³Dr. Alveena Batool Syed, ⁴Dr. Hamid Nawaz Ali Memon and ³Dr. Ali Raza Shaikh

^{1,2}Isra University Hospital Hyderabad, Sindh, Pakistan

³Liaquat University of Medical and Health Sciences – LUMHS Jamshoro

⁴Zulekha Hospital, Dubai United Arab Emirates

Abstract:

Objective: To determine the frequency of *Helicobacter pylori* in patients with gastric cancer.

Patients and Methods: The two year cross section study screened fifty cases of carcinoma stomach for *H. pylori* infection. The inclusion criteria were patients with carcinoma stomach confirmed by endoscopy and histopathology while the exclusion criteria patients received anti *H pylori* treatment within 6 months and the patients not willing/refuse to give consent for the study. All patients who presented with upper GI symptoms and suspected to have carcinoma stomach investigated in the form of complete blood count, ultrasound and upper gastrointestinal endoscopy. On endoscopy patient who had growth in the stomach biopsy was taken from and around the growth and subjected to Rapid urease test and histopathologic examination along with serology test for antibodies to *H. pylori*. The frequency / percentages (%) and means \pm SD computed for study variables.

Results: During two year study period total fifty patients were explored for *H. pylori* infection. The frequency for male and female population was 32 (64%) and 18 (36%) with mean \pm SD for age of male and female individuals was 55.83 ± 9.62 and 58.21 ± 8.72 respectively. The *H. Pylori* infection was positive in 30 (60%) patients while the growth exists in antrum 25 (50%), antrum and body 10 (20%), body 08 (16%), body and fundus 04 (8%), fundus 03 (6%) while regarding the histopathology well differentiated carcinoma 07 (14%), moderately differentiated carcinoma 10 (20%), poorly differentiated carcinoma 33 (66%).

Conclusion: The existence of *H. pylori* observed in gastric carcinoma cases may lead to a spread of *H. pylori* in histologic sections.

Keywords: *Helicobacter pylori*, Gastric Carcinoma, Malignancy and Stomach.

Corresponding author:

* Dr. Majid Ali Soomro,

Email: zulfikar229@hotmail.com

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INTRODUCTION:

Around the world, particularly in Asia and Eastern Europe, gastric disease remains a main source of malignant growth demise. Many hazard factors for gastric disease have been distinguished, a large portion of which are identified with way of life and dietary propensities, for example, high salt admission, liquor utilization and tobacco smoking [1]. A higher danger of advancement of gastric malignancy has been accounted for in patients with positive serologic tests for H pylori [2]. Studies have been done to demonstrate the relationship of H pylori contamination with carcinoma stomach [3]. This investigation is a push to demonstrate a relationship of H pylori as a cancer-causing agent and the seriousness of harm it is related with. ELISA and RUT are utilized to analyze H pylori disease. For a more drawn out timeframe relationship between H. pylori and GC was easily proven wrong. A study from Japan on 1526 patients gives a reasonable proof that H. pylori contamination is altogether connected with danger of creating gastric cancer [4]. Confirmation that H. pylori has an effect on beginning periods of gastric carcinogenesis is shown by randomized planned investigations which demonstrates relationship between H. pylori annihilation and decrease of premalignant tumors [5]. The wide geological and worldly varieties in gastric malignant growth frequency show the key job of ecological components. It has been suggested that disease with H. pylori is one of these components. H. pylori is perceived as a reason for constant gastritis, which might be viewed as the initial phase in a grouping of changes to the gastric mucosa perhaps

bringing about malignancy [6]. In this manner, the present examination investigated the presence of H. pylori disease in people with gastric malignant growth at tertiary consideration healing center.

PATIENTS AND METHODS:

The two year cross section study screened fifty cases of carcinoma stomach for H. pylori infection. The inclusion criteria were patients with carcinoma stomach confirmed by endoscopy and histopathology while the exclusion criteria patients received anti H pylori treatment within 6 months and the patients not willing/ refuse to give consent for the study. All patients who presented with upper GI symptoms and suspected to have carcinoma stomach investigated in the form of complete blood count, ultrasound and upper gastrointestinal endoscopy. On endoscopy patient who had growth in the stomach biopsy was taken from and around the growth and subjected to Rapid urease test and histopathologic examination along with serology test for antibodies to H. pylori. The data was collected on proforma while the frequency and percentages was calculated for qualitative and quantitative variables.

RESULTS:

During two year study period total fifty patients were explored for H. pylori infection. The frequency for male and female population was 32 (64%) and 18 (36%) with mean \pm SD for age of male and female individuals was 55.83 ± 9.62 and 58.21 ± 8.72 respectively. The demographical and clinical profile of study population is presented in Table 1.

TABLE 1: THE DEMOGRAPHICAL AND CLINICAL PROFILE OF STUDY POPULATION

| Parameter | Frequency (N=50) | Percentage (%) |
|-------------------------------------|------------------|----------------|
| AGE (yrs) | | |
| 20-29 | 01 | 2 |
| 30-39 | 02 | 4 |
| 40-49 | 09 | 18 |
| 50-59 | 11 | 22 |
| 60-69 | 12 | 24 |
| 70+ | 15 | 30 |
| | | |
| GENDER | | |
| Male | 32 | 64 |
| Female | 18 | 36 |
| | | |
| SITE OF GROWTH | | |
| Antrum | 25 | 50 |
| Antrum and body | 10 | 20 |
| Body | 08 | 16 |
| Body and fundus | 04 | 8 |
| Fundus | 03 | 6 |
| | | |
| H. PYLORI INFECTION | | |
| Positive | 30 | 60 |
| Negative | 20 | 40 |
| | | |
| HISTOPATHOLOGY | | |
| Well differentiated carcinoma | 07 | 14 |
| Moderately differentiated carcinoma | 10 | 20 |
| Poorly differentiated carcinoma | 33 | 66 |

DISCUSSION:

H. pylori disease is a standout amongst the most widely recognized contaminations in people, the quantity of instances of gastric malignancy is relatively little [7]. This has incited the scan for cofactors for movement from *H. pylori* disease to gastric malignant growth [8]. *H. pylori* is lost from the stomach in the precancerous phases of the infection originates from endoscopic investigations. Karnes et al [9] watched essentially bring down predominance of *H. pylori* (33%) by histological analysis than by serology (86%) in subjects with atrophic body gastritis. In an endoscopic overview in Italy, Farinati et al [10] found that pervasiveness of disease expanded with age however turned out to be fundamentally lower with the movement of gastric harm. The thickness of colonization pursued a similar pattern. A similar wonder has been seen in an extensive endoscopic examination in Venezuela, in which histological finding of *H. pylori* estimated on a four-point scale (negative, hard to discover, simple to discover, and bottomless) was adversely corresponded with level of decay, intestinal metaplasia, and dysplasia [11]. Since the revelation of *H. pylori* in the human stomach, disease by these microscopic organisms has been appeared to be firmly connected with gastric injuries, including ceaseless atrophic gastritis, intestinal metaplasia, and gastric malignant growth. Epidemiological investigations, in mix with results from creature models, affirm that annihilation of *H. pylori* successfully forestalls gastric carcinogenesis and mellow gastritis without extreme decay or intestinal metaplasia [12]. Notwithstanding, bacterial destruction raises the issue of relapse of gastric dysplasia (intramucosal adenocarcinoma), which may be underdiagnosed as a recovering organ. Just by exact judgments the chemopreventive methodologies and *H. pylori* annihilation gastric malignant growth can be cured.

CONCLUSION:

The existence of *H. pylori* observed in gastric carcinoma cases may lead to a spread of *H. pylori* in histologic sections and might describe the variation in *H. pylori* infection rates reported for patients with gastric cancer.

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