

ORIGINAL ARTICLE

Serological and Epidemiology study of *Helicobacter pylori* infection among Dyspeptic patients in District Peshawar Pakistan

Ihteshamul Haq^{1*}, Anees Muhammad², Fazli Zahir³, Muhammad Kashif⁴, Faheem Anwar⁵,
Muhammad Saeed Akhtar⁵, Faizan Ullah⁵

¹. Graduate School of Biotechnology and Oriental Medicine Kyunghee University Suwon South Korea.

². Department of Medical Lab Technology, Medical Teaching Institution, College of Medical Technology, Bacha Khan Medical College, Pakistan

³. Institute of Biotechnology and Genetic Engineering, Agriculture University Peshawar, Pakistan

⁴. Department of Microbiology and Immunology, Comsat University Islamabad, Pakistan

⁵ Department of Genetics, Hazara University, Manshera, Pakistan

*Corresponding Author's Email: Ihteshamulhaq348@gmail.com

ABSTRACT

Helicobacter pylori (*H. pylori*) Infection is a major gastrointestinal tract pathogen that has been involved in a wide range of gastrointestinal infection, with gastritis, dyspepsia, peptic ulcer, gastrointestinal carcinoma, and lymphoma of the lymphoma associated with mucosa. The frequency of *H. pylori* infection in developing countries, *H. pylori* infection is more than in developed countries, while Pakistan has inadequate data on its frequency. The purpose of this was to examine the *H. pylori* prevalence among with dyspepsia. The data were obtained from the patients, recommended by the doctors based on their physical condition and clinical manifestation among various patients attending the Hayatabad Medical Complex (HMC), Peshawar. A total of 123 patients with dyspeptic infection irrespective of age and sex included during the study duration. A complete history of all the patients was taken and examination was performed. The upper Gastric intestine endoscopy was performed, biopsies were taken and the specimen sent for histopathology. The data were analyzed by Microsoft Excel version 2007. A total of 123 patients were studied, of which 67 were males and 56 were females aged between 12-68 years. The majority of patients were with upper abdominal pain and retrosternal burning in the third and fourth decades of life. Others seen included, in some cases, regurgitation, dyspepsia, water brash, nausea, vomiting, and hematemesis. Gastric biopsy histopathology showed gastritis associated with *H. pylori* in 45.5% (n=56) whereas 54.5% (n=67) patients had gastritis that was not associated with *H. pylori* infection. The occurrence of *H. pylori* is correlated with gastritis, and in dyspeptic patients, gastritis is not correlated with *H. pylori* being more or less comparable.

Keywords: Dyspepsia, Peptic Ulcer, *Helicobacter pylori*, G. I. Endoscopy

Received 11.05.2020

Revised 21.05.2020

Accepted 29.05.2020

How to cite this article:

I Haq, A Muhammad, F Zahir, M Kashif, F Anwar, M Saeed Akhtar, F Ullah. Serological and Epidemiology study of *Helicobacter pylori* infection among Dyspeptic patients in District Peshawar Pakistan. Adv. Biores., Vol 11 (3) May 2020: 81-85

INTRODUCTION

Helicobacter pylori (*H. pylori*) are a twist, gram-negative bacteria that has infected more than 31% individual all over the globe. In a many states, more than 51% population is infected with *H. pylori* [1]. In 1982, Marshall and Warren got Nobel Prize for the identification of helicobacter pylori bacteria. Most of the people studied helicobacter pylori bacteria and almost 29000 different article published regarding *H. pylori* infection in human. *H. pylori* bacteria play important role in different disease of stomach like , gastritis, gastric and duodenal ulcer and gastric carcinoma [2]. A Prof W. Jawroski a great clinical researcher identified the presence of coiled shaped micro-organisms like *H. pylori* about 100 years before at Krakow Jagiellonian University [3]. In 1994, *H. pylori* were categorized as class I individual carcinogen for gastric cancer by WHO [4]. In most of the cases, *H. pylori*-infected patients show no specific symptoms. It remains in the stomach of the patient asymptotically for a long period. Symptoms of infection appear after the damage of the lining of the stomach or lining of duodenum [5]. It is directly linked to the spread

of gastric and duodenal ulcers. Gastric adenocarcinoma, is a cancer of the stomach caused by *H. pylori*, is 2nd main cause of death worldwide. Studies show that *H. pylori* are found to be the main cause of gastric ulcers [6]. As the frequency of *H. pylori* different from place to place and from generation to generation therefore it is assumed that the frequency of gastric cancer is determined by ecological factors rather than genetic factors [7].

Worldwide varies kind strain of *H. pylori* look to be related to the difference in virulence, and the ensuing interplay by the host and ecological factor leads to subsequent differences in the appearance of the disease. Ethnicity, Age, geography, sex, and socioeconomic position are all factors that influence the frequency and incidence of *H. pylori* disease, *H. pylori* disease is the key public health issue in all developing countries, the greater frequency ratio of *H. pylori* disease means that every individual intervention may be required [8].

All over the world therapeutic vaccination is the only plan that would make a great variation in the frequency of *H. pylori* disease. In developing countries the prevalence ratio of *H. pylori* is higher than developed countries [9].

The frequency rate of *H. pylori* infection is different between the urban and rural populations. the major reason for the different prevalence ratio of *H. pylori* infection is differences involve socioeconomic variation among the different population. the oral-oral or fecal-oral routes is the main way of spreading of *H. pylori* infection, due to unhygienic water system, poor diet and overcrowding is the major causes of *H. pylori* infection and increases the frequency of *H. pylori* infection [10]. In 1994, WHO and International Agency for Research on Cancer (IARC) categorized that *Helicobacter pylori* as class 1 human carcinogen due to its prevalence association to gastric carcinoma [11]. In Pakistan, the frequency rate of acid peptic infection is very high due to high frequency ratio of *H. pylori* Bacterial disease among individual and the rate of the patient is growing continuously due to the lack of proper diagnosis system and the lack of proper treatment facilities in the private sector health care services. According to health experts in Pakistan if *H. pylori* infection does not treat carefully then they will chronic infection and also chances of stomach cancer and gastric carcinoma will be happened [12]. Some studies conclude that *H. pylori* infection are spreading in population through fecal-oral way but still the actual way of *H. pylori* transmission is not recognized and fecal to oral or oral to oral is the main general way of spreading at this phase [13]. Due to its remarkable difference of *H. pylori* occurrence from place to place and from one generation to the next, it has been hypothesized that the frequency of gastric cancer is determined by ecological factor rather than genetic factors [14]. In 1994, the worldwide branch of cancer research approved by WHO and confirm that *H. pylori* disease as an infection as a group 1 carcinogen and an exact reason for individual Gastric carcinoma [15].

The frequency rate of *H. pylori* is high in countries where the sanitation system is poor and the environment is unhygienic. In Bangladesh, the prevalence of *H. pylori* infection is 91% in healthy adults [16]. Habitually *H. pylori* microbes are stayed generously in the stomach mucus layer still however approximately 21% associated with the epithelial cells. Inside the Human Stomach *H. pylori* constantly infect the harsh surrounding and slowly alter the mechanism to typically colonize and patiently in this position [17].

Normally *H. pylori* bacteria live in gastric mucous layer and about 20% live in close links with epithelial cells stomach. In the human stomach where there is a harsh environment for bacterial growth, it causes infection slowly and smoothly alters the mechanism for colonization [18]. Hospital-based studies show that the prevalence of symptomatic *H. pylori* disease is very low around the globe [19]. *H. pylori* are found in 90-95% of individual of duodenal ulcer and 50-70% of individual of gastric ulcer [20]. *H. pylori* are transmitted through the oral-oral and fecal-oral route [21].

This study aimed to explore the dyspeptic infection symptom and sign of *H. pylori* among the patient of Peshawar.

MATERIAL AND METHODS

This cross-sectional study was conducted in duration of six months from March to August 2019 at Hayatabad Medical Complex, Peshawar, Khyber Pakhtunkhwa of Pakistan. The study was carried out on dyspeptic patients infected with *H. pylori* irrespective of age and gender. The history of the patient was obtained through proper channels and investigation was performed. The biopsy samples were taken from all patients and were transported for further processing to the histopathology department at HMC, Peshawar.

Experts performed the endoscopy by inserting the thin and a flexible tube along with camera and light through the mouth and reached to stomach (sometimes to the upper intestine). A small endoscopic device was inserted for the collection sample (tissue) from the lining of the stomach (sometimes upper

intestine). The obtained tissue sample was placed in a petri dish and was culture in appropriate media for the identification and isolation of *H. pylori*.

All the collected data were analyzed by Microsoft word software 2007.

RESULTS

A total of 123 individuals with dyspeptic symptoms were studied in which 54.5% (n=67) were male and 45.5% (n=56) were female patients as shown in table no.1. The proportion rate of *H. pylori* is recorded differently in several studies as well as with various socio-economical and environmental agents [22]. The origin and genetic diversity of *H. pylori* strains are the broad factor that evaluate the consequence of *H. pylori* disease in several cases (23). The typical progression of *H. pylori* infections are many including gastritis, atrophy, dysplasia, and intestinal metaplasia that resulting in carcinoma of mucosa layer. Gastritis is predominantly developed in approximately 90% of infected population with *H. pylori* while intestinal metaplasia and gastric atrophy seen more often in patients infected with *H. pylori* [24]. About 25% cases of dyspepsia has been reported from western countries including the United States [25]. A report shows that the *H. pylori* infection significantly increase with the age [26].

A significant association of factors did not identify due to the negative association of various factors with *H. pylori* infection [27]. The *H. pylori*-infected cases along with dyspepsia may lead to ulcer diseases [28]. The incidence of dyspeptic *H. pylori* infection was 44.2% reported by researchers [29]. A study conducted by O'Connor et al. shows the prevalence rate of *H. pylori* infection is 40% [30]. Another studied shows the prevalence rate of *H. pylori* infection is 42.5% [31].

Table No.1: Gender Base distribution of patients

Parameters	Male	Female	Total
Number of cases	67	56	123
Percentage	54.5%	45.5%	100%

The minimum age was 12 years whereas the maximum was found 68 years. The suspected patients were categorized in various age groups. The highest number of cases were observed in age of 21-30 years with 30.9% (n=38), followed by age 31-40 years (21.1%), 11-20 years (14.6%), greater than 60 years (13.8%) and 13% in the age of 41-50 years whereas least cases were found in the age of 51-60 years with 6.5% (n=08) as shown in table no.2.

Table No.2: Age-wise distribution of patients

Age Group	Number of patients % (n)
11-20 years	14.6 (18)
21- 30 year	30.9 (38)
31-40 years	21.1 (26)
41-50 years	13.0 (16)
51-60 year	06.5 (08)
> 60 years	13.8 (17)

Gastric biopsy histopathology revealed gastritis associated with *H.pylori* in 56 (45.5%) patients, while 67 (54.5%) patients had gastritis that was not associated with *H. pylori*.

A study conducted in Karachi Pakistan by Khalid shows the prevalence rate of *H. pylori* infection is 47.6% which is comparatively lower than those from western Europe and North America [32]. Another study conducted by Asaka on 2455 patients concluded that gastritis and intestinal metaplasia are strongly linked with *Helicobacter pylori* [33].

Different patients infected with *H. pylori* complaint about various signs and symptoms associated with *H. pylori* infection. The upper abdomen pain was found in 86% of patients whereas retrosternal burning was observed in 91% of patients. Other symptoms noted were regurgitation, nausea, and vomiting whereas some patients also complaint about haematemesis as shown in table no.3. Abdominal pain, retrosternal burning, regurgitation, nausea, vomiting, and haematemesis was 86%, 91%, 63%, 57%, 33%, and 5% respectively, were reported in the present study. A study conducted by Asaka et al., [29] on 2455 patients revealed that gastritis and intestinal metaplasia are strongly linked with *Helicobacter pylori*.

Table No.3: Several symptoms obtained from *H. pylori*-infected patients

Different Symptom <i>H. pylori</i>	Patient percentage (%)
Pain upper abdomen	86.0
Retrosternal Burning	91.0
Regurgitation	63.12
Nausea	57.51
Vomiting	33.32
Haematemesis	5.23

CONCLUSION

In our study, 45.5% of patients were *H. pylori*-infected, and the trend of *H. pylori* infection is similar to that reported in the previous and available literature. Moreover, the studied patients in present belong to a younger age group. The incidence of *H. pylori*-associated with gastritis while on the other hand, gastritis not linked with *H. pylori*.

ACKNOWLEDGMENT

I am extremely thankful to Dr. Ayaz Ahmad Chairman Department of Biotechnology Abdul Wali Khan University, Mardan, Pakistan.

REFERENCES

- Go MF. (2002). Review article: natural history and epidemiology of *Helicobacter pylori* infection. *Aliment Pharmacol Ther.* 16:3–15.
- Saima Choudhry, MateenIzhar, Ayyaz Ali, M.Idrees Khan (2011). Correlation of *Helicobacter Pylori* in dental plaque and gastric mucosa of dyspeptic patient. University of Punjab,QuaidAzam campus Lahore,Pakistan.
- Tummuru MK, Cover TL, Blaser MJ. (1993). Cloning and expression of a high-molecular-mass major antigen of *Helicobacter pylori*: evidence of linkage to cytotoxin production. *Infect Immun*;61(5):1799–809.
- Salih BA, Abasiyanik MF, Bayyurt N, Sander E. (2007). *H. pylori* infection and other risk factors associated with peptic ulcers in Turkish patients: a retrospective study. *World J Gastroenterol.* Jun 21; 13(23):3245-8. DOI: 10.3748/wjg.v13.i23.3245. PMID: 17589905; PMCID: PMC4436612.
- Moss SF, Malfertheiner P. (2007). *Helicobacter*, and gastric malignancies. *Helicobacter.* 12:23–30.
- Mbulaiteye SM, Hisada M, El-Omar EM. (2009). *Helicobacter pylori*-associated global gastric cancer burden. *Front Biosci.*; 14:1490–504.
- Khan A, Farooqui A, Raza Y, et al. (2012). Prevalence, diversity and disease association of *Helicobacter pylori* in dyspeptic patients from Pakistan. *J Infect Dev Ctries.* 7: 220-8.
- World Gastroenterology Organization Global Guideline: *Helicobacter pylori* in Developing Countries. *Journal of Digestive Diseases* 2011; 12; 319–326 DOI: 10.1111/j.1751-2980.2011.00529.x
- Rasheed F. Ahmad T, Bilal R (2011) frequency of *Helicobacter pylori* infection using 13 C –UBT in asymptomatic individual of barakaho islamabad pakistan, *journal of the college of physician and surgeons pakistan* 6:379-381
- Correlation of *Helicobacter pylori* in dental plaque and gastric mucosa of dyspeptic patient saima choudry, mateen izhar ,ayyaz ali, M.iddress khan 2011 univesty of punjab quaid azam campus lahore Pakistan. *International Journal of Applied Research* Volume # 3, 2014, Page 68 of 84
- Fuchs CS, Mayor RJ. (1995). Gastric carcinoma. *N Engl J Med.* 333:32-41.
- World Health Organization.(1994), International Agency for Research on Cancer, IARC. Monograph on the evaluation of carcinogenic risks to human: Schistosomes, Liver flukes and *Helicobacter pylori*. Vol. 61. Lyon France: IARC p. 177-240.
- Ertem, D., H. Harmanci and E. Pehlivanoglu, (2003). *Helicobacter pylori* infection in Turkish preschool and school children: role of socioeconomic factors and breast feeding. *Turkish J. Pediatr.*, 45: 114–22
- Roosendaal R, Kuipers EJ, Buitenwerf J, van Uffelen C, Meuwissen SGM, van Kamp GJ, Vandenbroucke-Grauls CMJE. (1997). *Helicobacter pylori* and the birth cohort effect: evidence of a continuous decrease of infection rates in childhood. *Am J Gastroenterol*; 92: 1480– 82.
- Klein PD, Graham DY, Gaillour A, Opekun AR, Smith EO. (1991). Water source as risk factor for *Helicobacter pylori* infection in Peruvian children. *Gastrointestinal Physiology Working Group. Lancet.* 337:1503–6.
- Hessey SJ, Spencer J., Wyatt JI, Sobala G., Rathbone BJ, Axon AT, Dixon MR., (1990). Bacterial adhesion and disease activity in *Helicobacter* associated chronic gastritis,*GUT*; 31(2); 134-8.
- Ugwu N, Ugwuja E, Ejikeme B, Obeka N. *Helicobacter pylori* seropostivity in Nigerians with Type 2 Diabetes mellitus. *Internet J Trop Med.* 2008;4:32-6.
- Kuipers, E. J., J. C. Thijs, and H. P. Festen. (1995). The prevalence of *Helicobacter pylori* in peptic ulcer disease. *Aliment. Pharmacol. Ther.* 9(Suppl. 2):59–69.

19. Ernst, F. D., E. J. Kuipers, A. Heijens, R. Sarwari, J. Stoof, C. W. Penn, J. G. Kusters, and A. H. van Vliet. (2005). The nickel-responsive regulator NikR controls activation and repression of gene transcription in *Helicobacter pylori*. *Infect. Immun.* 73:7252–7258.
20. Sipponen, P., K. Varis, O. Frañki, U. M. Korri, K. Seppälä, and M. Siurala. (1990). Cumulative 10-year risk of symptomatic duodenal and gastric ulcer in patients with or without chronic gastritis. *Scand. J. Gastroenterol.* 25:966– 973.
21. Allaker, R. P., K. A. Young, J. M. Hardie, P. Domizio, and N. J. Meadows. 2002. Prevalence of *Helicobacter pylori* at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. *J. Med. Microbiol.* 51:312–317.
22. Chen SY, Liu TS, Fan XM, Dong L, Fang GT, Tu CT, et al. (2005). Epidemiological study of *Helicobacter pylori* infection and its risk factors in Shanghai. *Zhonghua Yi Xue Za Zhi* ;85:802–806
23. Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, Yamakido M et al. (2001). *Helicobacter pylori* infection and the development of gastric cancer. *N Engl J Med*;345(11):784–9.
24. Khalid Mehmood, Abdul Aleem Awan, Naveed Muhammad, Fariha Hasan, Abdul Nadir. *Helicobacter pylori*, (2014). *Histopathology, Dyspepsia, Gastritis*, Pakistan J Ayub Med Coll Abbottabad 2014;26(2):182
25. Ertem, D., H. Harmanci and E. Pehlivanoglu, 2003. *Helicobacter pylori* infection in Turkish preschool and school children: role of socioeconomic factors and breast feeding. *Turkish J. Pediatr.*, 45: 114–22
26. Klein PD, Opekun AR, Smith EO, Graham DY, Gaillour A.(1991). Water source as risk factor for *Helicobacter pylori* infection in Peruvian children. *Lancet*;337:1503-1506.
27. Bures J, Kopacova M, Koupil I, Vorisek V, Rejchrt S, Beranek M, et al. (2006). Epidemiology of *Helicobacter pylori* infection in the Czech Republic. *Helicobacter*;11:56–65
28. O'Connor HJ. (1999). *Helicobacter pylori* and Gastro-oesophageal disease: Clinical implication and management. *Alimen Pharmacol Therap*; 13: 117-27.
29. Asaka M. (2001). Atrophic Gastritis and intestinal metaplasia in Japan: The result of a large multicentre study of *Helicobacter pylori*. *Gut* ; 6: 294-9.
30. Tytgat GN. (1996). Current indication for *Helicobacter pylori* eradication therapy. *Scand J Gastroenterol*: 215 (Suppl): 70-3
31. Weston AP. (2000). Prospective evaluation of prevalence of gastric *Helicobacter pylori* infection in patients with Gastro-oesophageal disease. *Am J Gastroenterol*; 95: 387-94.
32. Malik KA, Shaikh RA, Rehman S. (2007). Prevalence of *Helicobacter Pylori* in dyspeptic patients at Liyari General Hospital. *Pak J Surg*.23:33-5.
33. Schwizer W.(2001). *Helicobacter pylori* and symptomatic response of Gastro-oesophageal disease. *Lancet* ; 357: 1738-4.

Copyright: © 2020 Society of Education. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.