Original Article

FREQUENCY OF HELICOBACTER PYLORI INFECTION AND ITS PREVALENCE ACROSS GENDER IN DISTRICT NOWSHERA KPK

Zahid Irfan Marwat[1], Muhammad Israr[2], Ejaz Afzal[3], Maliha Gul[1]

[1]Department of Biochemistry, Nowshera Medical College, Nowshera, KP, Pakistan

[2]Department of Biochemistry, Bacha Khan Medical College Mardan, KP, Pakistan

[3]Department of Anatomy, Ayub Medical College Abbottabad, KP, Pakistan

ABSTRACT

Background: *Helicobacter pylori lives in the stomach's gastric mucosa. One of the most genetically diverse bacterial species, it infects billions worldwide. There is evidence that the bacterium that causes stomach cancers, peptic ulcers, chronic gastritis, and gastric malt lymphoma has a sociodemographic and geographic pattern. Despite infections not necessarily dangerous, their frequency is higher in Africa than in other areas, according to research. The specific processes of H. pylori transmission could be clearer. However, poor hygiene, crowded living situations, unclean water, and contaminated surroundings constitute dangers. Oral-oral, fecal-oral, and person-to-person transmission are possible, with or without transitional stages, during diarrhea or vomiting. The organism's spread may also be linked to contaminated water, such as municipal tap water.*

Objectives: *To evaluate the frequency of H. pylori infection in District Nowshera and to determine the prevalence of H. Pylori across genders.*

Study design: *A Cross-sectional observational study*

Duration and place of study: *Department of Biochemistry Nowshera Medical College & Qazi Hussain Ahmed Medical Complex Nowshera from January-December 2017*

Methods: *Patients from 13 to 54 years old, both male and female, were included in this research. One hundred eight samples were taken from individuals chosen for further H. pylori research.*

Results: *The patients ranged in age from 13 to 54 years old, with a mean age of 33.54±8.496. Out of 108 patients, the gender distribution was as follows: male patients comprised 55.6% of the patient population, while female patients comprised 44.4%. Out of 108 patients, 45 had H. pylori infection, or 41.7%. In contrast, 63 patients did not have H. pylori infection, or 58.3%. The proportion of patients with H. pylori was 41.7%; the number of men with the infection was 27.8%, and the number of females was 13.7%. There were 58.3% negative instances, with 27.8% of the cases being male and 30.6% being female.*

Conclusion: *According to the study, male patients are more likely than female patients to have an H. pylori infection.*

Keywords: *Helicobacter pylori, Gastritis, Gastric and duodenal ulcers, Raynaud's syndrome, Migraine.*

INTRODUCTION

A class I carcinogenic pathogen, Helicobacter pylori is a Gram-negative bacteria.[1] Individual

Correspondence:
Dr. Zahid Irfan Marwat
Associate Professor, Department of Biochemistry, Nowshera Medical College, Nowshera.
Email: drzahidirfan@gmail.com
Cell: 0300-9112880
Date Received: April-12-2021
Date Accepted: May-24-2021
Date Revised: June-25-2021
Available Online: July-03-2021

organism is a rod with a spiral form. May show up alone or in pairs. It also has many flagella at one pole, is actively motile, and is neither spore-forming nor encapsulated. Gastritis, duodenal and stomach ulcers, gastric cancer, and mucosa-associated lymphoid tissue lymphoma are all linked to it. Over 12 years, long-term chronic gastritis linked to Helicobacter pylori is known to proceed to glandular atrophy and intestinal metaplasia (I.M.). Gastritis caused by Helicobacter pylori may cause intestinal metaplasia (I.M.) and multi-focal atrophy, which cause the antrum to enlarge into the body progressively [2].

It is well acknowledged that half of the world's population has Helicobacter pylori (H. pylori) in their stomachs. Numerous stomach conditions, such as gastritis, gastric adenocarcinoma, peptic ulcers, and gastric ulcers, are also linked to the bacterium. H. Pylori is widely present, although little is known about how it spreads. Amongst well-known mechanisms, invasive endoscopy has been shown to cause iatrogenic transmission of the pathogen, and prior research has shown a positive correlation between direct person-to-person contact and H. Pylori colonisation [3,4].

Furthermore, a high incidence rate among people connected to hospitals and illness clustering within families provide an appropriate source for infection transmission from person to person. Various bodily fluids, including saliva, gastric juice, vomitus, and feces, have been suggested as potential transmission means [5]. Several studies comparing the spread of illness from sick mothers to young children revealed the frequency of infection in newborns under two years old [6, 7].

Water contamination is a significant contributing element to the spread of infections. Water may be a significant and overlooked source of Helicobacter transmission because of fecal contamination. The seroprevalence in children aged 11-15 years in recent research conducted in Karachi on seemingly healthy youngsters was 54%.[8]Sero-positivity rose in low-middle socioeconomic positions and with age.

Although the precise process is unknown, it most likely includes host and bacterial components. There is a modest bacterial invasion of the surface of the epithelial cells. Mucosal cells may be harmed by toxins, lipopolysaccharides, and urease activity's ammonia production, which can potentially cause direct cell injury.

While H. Pylori colonization may not cause illness in and of itself, it can raise the risk of some gastrointestinal disorders, including stomach cancer and peptic ulcers.

A pattern of gastritis includes intestinal metaplasia, atrophy, and chronic inflammation in cases of pangastritis. It may also result in increased acid production, which can cause duodenal ulcers in cases of gastric metaplasia.[9]Risk factors for H. Pylori infection include being born or living in a developing country, having a low socioeconomic status, having little education, living in close quarters, having contaminated food

and water, and, if an intubation tube is not cleaned properly, exposing the patient's stomach contents [10, 11].

It has been shown that 90% of people in underdeveloped nations and 50% of individuals in industrialized nations have H. pylori-positive serum antibodies.[12] Childhood is the most hazardous time to acquire H. pylori, particularly in poor nations and locations with high population density and low socioeconomic status. Thirteen The population in developing nations is more vulnerable because of their low socioeconomic status. Poor hygienic conditions, population growth, consumption of uncooked foods, such as those bought from street vendors, and contaminated water supplies are among the risk factors that are becoming more prevalent.

The risk of H. pylori, which is prevalent in South Asian nations, particularly Pakistan, is increased by tobacco usage. Although the findings of this research are inconsistent, some have shown that coffee has the opposite effect of protecting against infection while alcohol does. According to epidemiological research, children have a greater incidence of H. pylori infection than adults. This difference may be caused by younger people's need for more personal cleanliness standards [14, 15].

As of right now, research has shown how H. pylori infection affects the emergence of extra-alimentary illnesses such as migraines, dermatological conditions, iron deficient anemia, cardiovascular disease, myocardial infarction, and various autoimmune diseases.[16] Nonetheless, most people have no symptoms from the infection and only a small percentage of those with H pylori experience stomach problems. This demonstrates that it is influenced by a few other variables, including the host's genetic predisposition and the genotype of the H pylori strains.

The stomach and duodenal bacteria H. pylori cause the most prevalent chronic bacterial infection in humans. H. pylori damages the linings of the stomach and duodenum in several ways. H. pylori creates ammonia to control pH, which is harmful to epithelial cells. This resulted in specific enzymes, including phospholipases, which harm epithelial cells, break tight junctions, promote inflammation, and eventually cause death. Proteases that vacuolate cytotoxin A (VacA).[17, 18] The stomach lining was colonized by H. pylori, which led to chronic gastritis (inflammation of the stomach lining at the site of infection). With a pH of less

than[4], the stomach is one of the most hostile habitats in the human body, and H. pylori is a bacteria with an incredible capacity to thrive there. The stomach's acidity is one of the body's defenses against foodborne microorganisms. Few organisms can survive in such an acidic environment. H. pylori, however, has a few "tricks" that nature has given it to help it survive in such a harsh environment.

The bacteria creates compounds that neutralize acids, creating a cloud of protection that keeps it mobile inside the stomach until it locates a place to settle. Furthermore, H. pylori can stick to the mucosa, the mucous layer under which the acidity is far less strong, circumventing the stomach's natural mucus barrier to protect itself from the acidity. Therefore, H. pylori can enter stomach areas where the environment is less hostile and release chemicals that neutralize acidity.[19, 20]

The systems that protect the mucous membranes of the stomach and duodenum are overwhelmed by stomach acid and the digesting enzyme pepsin as a result of inflammation. This leads to ulcers in the stomach and duodenum. One of the bacteria that causes stomach ulcers in humans is H. pylori.

The stomach's acidity influences the H. pylori colonization site, determining the ulcer's location. H. pylori may also colonize the remainder of the stomach in individuals who produce normal or decreased acid levels.

We know that H. pylori is present in most people, but only a small percentage of them go on to develop stomach cancer. Consequently, we can say that although H. pylori raises the risk of cancer, it is not the only reason. As a result, not every patient with Helicobacter pylori should get therapy. Patients should only be concerned about the presence of asymptomatic H. pylori if they have a family history of stomach cancer. In these situations, studies on the bacterium and therapy are recommended to remove it, even when the patient shows no symptoms.

There is a connection between H. pylori and the development of MALT, or stomach adenocarcinoma. Because of the close association, antibiotics are used to treat the tumor, and eliminating the bacteria will cure the malignancy. Infection with Helicobacter pylori is increasingly acknowledged as a global issue. It is the most frequent cause of persistent gastritis and has a high correlation with both stomach cancer and peptic ulcer disease. A thorough grasp of the epidemiology of H. pylori infection is crucial to unraveling the pathophysiological mystery and may pave the way for improved infection treatment and prognosis.

Globally, the prevalence of H. pylori and related illnesses has been wildly inconsistent. In developed nations, the incidence of H. pylori infection is often low, but the incidence of stomach cancer is very high. However, certain nations—especially those in Asia—have lower incidences of stomach cancer than others with high H. pylori prevalence rates. In less developed Asian nations like Bangladesh, India, Pakistan, and Thailand, the prevalence of H. pylori infection is higher, and it is acquired at a younger age than in more developed Asian nations like China and Japan. In contrast to China and Japan, the occurrence of stomach cancer is very rare in Thailand, Bangladesh, Pakistan, and India.[21]

Diagnosing an H. Pylori infection is possible using persistent or non-persistent methods. Upper gastrointestinal endoscopy is part of persistent procedures; OGD aids in H. Pylori detection through histology, culture, or urea tests, each of which has advantages and disadvantages of its own, but the advantages outweigh the disadvantages. It plays a significant role in diagnosis by facilitating imaging, photography, ultrasonography, and biopsies of questionable cases. Physicians may learn much about inflammatory, atrophic, and metaplastic stomach lesions in patients with and without H. Pylori via this whole process.[22] Among the persistent techniques is immunochromatographic technology or ICT.

MATERIALS AND METHODS

This cross-sectional observational research randomly sampled District Nowshera residents from January to December 2017. Over 108 samples from the Qazi Hussain Ahmed Medical Complex in Nowshera were sent to the biochemistry laboratory of Nowshera Medical College for H. pylori investigation.

The H. pylori serum test can screen many people fast. Immune responses generally precede clinical indications, making it ideal for early H. pylori identification. Diagnostically, high blood levels of H. pylori antibodies indicate type B silent gastritis. The SD BIOLINE H. pylori membrane strip features H. pylori capture antigen pre-coated on the test band. H. pylori

antigen colloid gold conjugate and blood sample form a visible line down the membrane chromatographically to the test region (T) as the antigen-antibody-antigen gold particle complex develops with great sensitivity and specificity.

The H. pylori test quickly qualitatively identifies all H. pylori-specific antibodies (IgG, IgM, IgA, etc.) in human blood or plasma. These measures were taken. The recommendations were followed exactly for accurate results. The test device was placed on a dry, flat surface after being removed from the foil. The test device had 10μl of serum or plasma in the sample well (S). After adding three drops of 110μl assay diluents, the timer was set. Immediately after the exam began, purple appeared on the central result display. The test results were assessed after 10 minutes. To prevent erroneous findings, the data were not examined beyond 10 minutes. Only one purple band (the C band) in the result window indicated 58.3% unsatisfactory results. First, the T and C color bands within the result window indicate an excellent result, 41.7%.

RESULTS

Almost 108 samples from chosen patients at Qazi Hussain Ahmed Medical Complex Nowshera were taken to the Department of Biochemistry Laboratory of Nowshera Medical College to test for H. pylori. The average patient age was 33.54±8.496 years, ranging from 13 to 54 years. As stated in Table 1, 55.6% of 108 patients were male and 44.4% female.

Table 2 shows that 45 of 108 patients had H. pylori, 41.7%, whereas 63 had no infection, 58.3%. The frequency distribution of gender with H. pylori showed 27.8% men and 13.7% females, totaling 41.7%. Table 3 shows that 27.8% of the 58.3% negative cases were male and 30.6% female.

DISCUSSION

Significant evidence for early-life acquisition of H. pylori primary infection has been reported in both industrialized and developing nations. [23, 24] Nonetheless, the method of H. pylori transmission is still up for debate. Age, gender, unsatisfactory living circumstances in the home, and low socioeconomic level have all been linked to an increased risk of H. pylori infection in children, according to earlier research.[25]

Males had greater levels of H. pylori colonization than females. However, the difference was not statisti-

Table 1: Frequency of Gender of the Patients

	Frequency	Percent
Male	60	55.6
Female	48	44.4
Total	108	100.0

Table 2: Frequency of H. pylori Patients

	Frequency	Percent
Positive	45	41.7
Negative	63	58.3
Total	108	100.0

Table 3: Frequency distribution of gender with H. Pylori

		H. Pylori		Total
		Positive	Negative	
Marital status	Male	30	30	60
		27.8%	27.8%	55.6%
Total	Female	15	33	48
		13.9%	30.6%	44.4%
Total		45	63	108
		41.7%	58.3%	100.0%

cally significant (P = 0.039). This outcome is consistent with other investigations' findings[26], which showed that men had a greater prevalence rate than women. Because H. pylori infections are more common in men than women, this difference might be related to young boys' better cleanliness practices than young girls.

The patients ranged in age from 13 to 54 years old, with a mean age of 33.54±8.496. Out of 108 patients, the gender distribution was as follows: male patients comprised 55.6% of the patient population, while female patients comprised 44.4%. Out of 108 patients, 45 had H. pylori infection, or 41.7%.

In contrast, 63 patients did not have H. pylori infection, or 58.3%. The proportion of patients with H. pylori was 41.7%; the number of men with the infection was 27.8%, and the number of females was 13.7%. There were 58.3% negative instances, with 27.8% of the cases being male and 30.6% being female.

CONCLUSION

According to the study, male patients had a greater prevalence of H. pylori infection than female

patients, consistent with the illness's overall prevalence. The increased percentage of male patients in this research might be attributed to younger guys' worse cleanliness than younger girls'.

REFERENCES

1. Smoot DT. How does Helicobacter pylori cause mucosal damage? Direct mechanisms.Gastroenterology1997; 113 (6 Suppl): 31–4;50. http://www.ncbi.nlm.nih.gov/pubmed/16367902

2. Dumrese C, Slomianka L, Ziegler The secreted Helicobacter cysteine-rich protein HYPERLINK "http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2764743" HYPERLINK "http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2764743" causes adherence of human monocytes and differentiation into a macrophage-like phenotype. FEBS Letters 2009; 583 (10): 1637–43.

3. Shiotani A, Graham D. Pathogenesis and therapy of gastric and duodenal ulcer disease. Med. Clin. North Am.2002;86 (6): 1447–66, viii.

4. Dixon MF. Patterns of inflammation linked to ulcer disease. Baillieres Best Pract Res ClinGastroenterol2000;14 (1): 27–40.

5. Anon: NIH consensus conference. In peptic ulcer disease. NIH consensus development panel on helicobacter pylori in peptic ulcer disease. JAMA 1994, 272:65–9.

6. Sipponen P: Gastric cancer: a long-term consequence of infection?ScandGastroenterol1994, 29:24–7.

7. Mera R, Fontham ET, Bravo L.E., Bravo JC, Piazuelo MB, Camargo MC, Correa P: Long-term follow-up of patients treated for infection. Gut 2005; 54:1536–40.

8. Tytgat G. Endoscopic transmission of Helicobacter pylori. Aliment PharmacolTher1995;9:105.

9. Guimaraes N, Azevedo N, Figueiredo C, Keevil C, Vieira M. A multi-route way of transmission for Helicobacter pylori. Helicobacter. 2007; 12: 456.

10. Brown LM. Helicobacter pylori: epidemiology and routes of transmission. Epidemiol Rev 2000;22: 283.

11. Baldassarre ME, Monno R, Laforgia N, Fumarola L, Fanelli M, Sgobba C, et al. The source of Helicobacter pylori infection in the neonatal period. J Perinat Med 2009; 37: 288-92.

12. Yucel O, Sayan A, Yildiz M. The factors associated with asymptomatic carriage of Helicobacter pylori in children and their mothers living in three socioeconomic settings. Jpn J Infect Dis. 2009;62: 120-24

13. Kusters JG, Around H.M. and Ernst JK., Pathogenesis of Helicobacter Pylori infection. ClinMicrob Review 2006;19: 449-90.

14. Jafri W, Yakoob J, Abid S, Siddiqui S, Awan S, Nizami S.Q.: Helicobacter pylori infection in children: population-based age-specific prevalence and risk factors in a developing country. Acta Paediatr 2010; 99:279–82

15. Marshal BJ Warren JR. Unidentified curved bacilli in the stomach of patients with Gastritis and Peptic ulceration. The Lancet .1984; 1 (8390): 1311-5.

16. DalchierJC.Gastrointestinal manifestation of Helicobacter Pylori infection in adults: from Gastritis to Gastric Cancer. Presse Med. 2008; (3):519-24.

17. F Mégraud. H pylori antibiotic resistance: prevalence, importance, and advances in testing. Gut. 2004;53(9):1374-84.

18. John Del Valle. Peptic Ulcer disease and related disorders. In: Braunwald. E , FauciA.S ,Kasperd, Hauser SL, Longo DL, Jameson JL,editors. Harrison's Principles of Internal Medicine 15th ed. New York McGraw-Hill 2001; 1649 -65.

19. Wu TC, Chen LK, Hwang SJ. Sero-prevalence of Helicobacter pylori in school-aged Chinese in Taipei City and the relationship between ABO blood groups. World JGastroenterol, 2003;9: 1752-5.

20. Moayyedi P, Axon AT, Feltbolwer R. Relation of adult lifestyle and socioeconomic factors to the prevalence of Helicobacter pylori infection. Int J Epidemiol, 2002; 31:624-31.

21. Zhong C, Li NK, Bi JW. Sodium intake, salt taste, and gastric cancer risk according to helicobacter pylori infection, smoking, histological type, and tumor site in China. Asian Pac J Cancer Prev, 2012;13:2481-4.

22. Ahmed KS, Khan AA, Ahmed I, Tiwari SK, Habeeb A, Ahi J.D., et al. Impact of household hygiene and water source on the prevalence and transmission of H. pylori: a South Indian perspective. Singapore Medical Journal, 2007;48 (6):543-9.

23. Alborzia A, Soltania J, Pourabbasa B, Oboodia B, Haghighatb M, Hayatia M. Prevalence of Helicobacter pylori infection in children (South of Iran). Diagnostic Microbiology and Infectious Diseases, 2006; 54:259-61.

24. Bardhan KP. Epidemiological features of Helicobacter pylori infection in developing countries. Clinical Infectious Diseases, 1997;25:973-8.

25. Ndip NR, Malange EA, Akoachere TFJ, MacKay GW, Titanji KPV, Weaver TL. Helicobacter pylori antigens in the feces of asymptomatic children in the Buea and Limbe health districts of Cameroon: A pilot study. Tropical Medicine and International Health, 2004; 9(9):1036-40.