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Research Article

The prevalence of *Helicobacter pylori* infection among students of a medical college in Bangladesh

Shahida Akter[1]*, Rehana Khatun[1], Aunta Melan[1], Saimun Nahar Rumana[1], Elisha Khandker[1], Mohsina Mahmud[2], Fahmida Rahman[1], Md. Shariful Alam Jilani[1]

[1]*Department of Microbiology, Ibrahim Medical College, 1/A Ibrahim Sarani, Shegunbagicha, Dhaka, Bangladesh*
[2]*Department of Internal Medicine, Mymensingh Medical College, Mymensingh, Bangladesh*

Abstract

***Background and objectives**:* The prevalence of *Helicobacter pylori* infection differs in relation to the human population, age, living conditions, lifestyle, socioeconomic status and geographic location. The purpose of the present study was to evaluate the prevalence of *H.pylori* infection among students of Ibrahim Medical College, Dhaka, Bangladesh.

***Materials and methods**:* This cross-sectional study was conducted at the K.A. Monsur Research Laboratory, Department of Microbiology, Ibrahim Medical College. A structured questionnaire was used to collect socio-demographic information and clinical history. Blood and stool samples were collected from each participant. Serum *H. pylori* CagA IgG and *H.pylori* IgA antibodies were determined using enzyme-linked immunosorbent assay (ELISA), and *H. pylori* stool antigen (HPSAg) was detected by immunochromatographic test (ICT).

***Results**:* A total of 85 participants were enrolled in this study. The overall *H. pylori* infection rate was 69.4% by positive stool antigen test and /or the presence of *H. pylori* specific CagA IgG or IgA antibodies in serum. *H. pylori* stool antigen was detected in 9 (10.6%) individuals, of whom 8 (88.9%) were also positive for *H. pylori* specific CagA IgG and / or IgA antibodies. Among 85 participants, CagA IgG and IgA were positive in 43 (50.6%) and 46 (54.1%) students, respectively, while 31 (36.5%) were positive for both antibodies. IgA positivity rate was significantly higher ($p \leq 0.005$) in individuals who tested positive for CagA-IgG compared to those negative for CagA-IgG antibody. Gastrointestinal symptoms were reported by 17 (20.0%) participants, while 68 (80.0%) were asymptomatic. No significant difference in antibody positivity rates was observed between symptomatic and asymptomatic individuals in this study.

***Conclusion**:* The study revealed that *H. pylori* infection is common among the medical students in Bangladesh. This underscores the importance of improving awareness and early detection strategies among medical students to minimize transmission and associated health risks.

Introduction

Helicobacter pylori (*H. pylori*) is a gram-negative, spiral-shaped, microaerophilic bacterium that colonizes the human gastric mucosa. A large number of people remain asymptomatic despite being infected with *H. pylori* [1]. Once acquired, the

***Correspondence**: *Shahida Akter, Department of Microbiology. Ibrahim Medical College, 1/A Ibrahim Sarani, Shegunbagicha, Dhaka-1000, Bangladesh. Email: shahidamicro@gmail.com*

infection persists throughout life unless treated with specific antimicrobials [2]. Chronic infection by *H. pylori* is recognized as the leading cause of gastric and duodenal ulcer disease and is also associated with gastric adenocarcinoma and mucosa- associated lymphoid tissue (MALT)[3,4].

A 2017 meta-analysis found that the overall global prevalence of *H.pylori* infection was 44.3%, with a higher rate of 50.8% in developing countries compared to 34.7% in developed countries [5]. In 2024, the prevalence of *H. pylori* infection among the asymptomatic urban population of Bangladesh was reported to be 40.5% [6]. In 2022, *H. pylori*-specific IgG and IgA antibodies were detected in 64.9% and 55.1% of participants, respectively, among the asymptomatic rural population in Bangladesh [7]. *H. pylori* infection is primarily transmitted through fecal-oral, oral-oral, or gastro-oral routes, often due to poor sanitation, overcrowding, contaminated water, and low levels of education, especially in early childhood [8-10].

Prevalence of *H. pylori* is strongly age-dependent, rising steadily from childhood through middle age, and then declining in the very elderly due to gastric atrophy and increased antibiotic use [11]. Both Habib et al. and Rahman *et al*. reported that the highest rates of infection were observed in individuals under 30 years of age, with detection rates of 78.3% and 50%, respectively [12,13]. Accurate diagnosis and effective management of *H.pylori* infection can greatly contribute to its eradication and prevent disease complications.

Data on the prevalence of *H. pylori* infection among medical college students in Bangladesh are limited. Studying this population is particularly important because of their frequent exposure to healthcare settings, which has been implicated as a significant risk factor for acquiring *H. pylori* infection by several studies [14,15]. Liu et al. reported that the overall prevalence of *H. pylori* infection was 70.0% among medical personnel, compared to that of 44.6% among the general population in China [16]. In addition, the students often share communal living spaces, such as hostels, which may further facilitate transmission. As future healthcare providers, identifying asymptomatic carriers and associated risk factors can inform early interventions and guide targeted public health strategies for this group [17]. The present study aimed to evaluate the prevalence of *H. pylori* infection among medical students affiliated with a tertiary-level hospital in Dhaka, Bangladesh, by detecting *H. pylori* antigen in stool and, *H. pylori*-specific IgA and CagA-IgG antibodies in serum using serologic methods.

Materials and Methods

Sample collection and laboratory work were done at K. A. Monsur Research Laboratory, at the Department of Microbiology of Ibrahim Medical College, Dhaka. This cross-sectional study was conducted in 2021 among 85 fourth-year MBBS students studying at Ibrahim Medical College. Fourth-year students were selected purposively as the MBBS curriculum covers Microbiology during the fourth year. After explaining the nature and purpose of the study, all participants provided informed written consent. A structured questionnaire was used to record socio-demographic information and clinical history. The study was approved by the Institutional Ethical Committee and Research Review Board of Ibrahim Medical College. All consenting fourth-year students were included in the study, irrespective of age, gender, nationality or presence of dyspeptic symptoms. Dyspeptic symptoms were defined as having two or more of the following gastrointestinal symptoms: dyspepsia, abdominal pain, nausea, vomiting and belching [18]. Individuals without any of these symptoms were considered as asymptomatic for *H. pylori* infection. Students who had taken antibiotics, colloidal bismuth compounds, proton pump inhibitors (PPIs) or H2 blockers within four weeks prior to sample collection were excluded.

H. pylori infection was defined if an individual was found positive for *H.pylori* antigen in stool and/or anti-*H. pylori* CagA-IgG and/or anti-*H. pylori* IgA in serum using serologic methods [7]. Approximately 2.5 ml blood sample was collected from each participant. After centrifugation at 1500 rpm for 10 minutes, separated serum was stored at -20℃ and used later for detection of *H. pylori* IgA and CagA-IgG antibodies. Approximately 20-30 grams of fresh stool sample was collected from each participant in a clean, wide-mouth and screw-capped container,

and tested for *H. pylori* stool antigen within 6 hours of collection. Stool antigen was detected by immune chromatography using ABON one strip *H.pylori* antigen ICT test device (Inverness Medical Innovation Hong Kong Ltd., Hong Kong). Approximately 50 mg of stool was obtained from at least three different areas of each stool specimen. The stool was then mixed with supplied extraction buffer solution using a vortex mixer, and centrifuged at 4000 rpm for 5 minutes. After centrifugation, two drops of supernatant were transferred into the sample well of the test device and kept at room temperature for 10 minutes. The result was then recorded. A positive result was indicated by the presence of purple-pink line along with the control line. When only the control line appeared, the result was considered negative. If no control line appeared, the result was termed as invalid. Serum anti-*H. pylori* CagA-IgG and anti-*H.pylori* IgA antibodies were determined by quantitative enzyme-linked immune sorbent assay (ELISA) using commercial kits namely CagA IgG ELISA and *Helicobacter pylori* IgA ELISA (DRG International Inc., USA), respectively. The tests were performed and interpreted according to the manufacturer's instructions. The present study did not evaluate the sensitivity and specificity of the test methods. However, the manufacturer (DRG International Inc., USA) reported that the sensitivity and specificity of both ELISA kits are greater than 90% for detecting *H. pylori*-specific antibodies. This is comparable to previously reported results for other *H. pylori* ELISAs, which demonstrated a sensitivity of 97.6% and a specificity of 90.5%. [19]. Participants who were positive for *H. pylori* stool antigen were treated with a proton pump inhibitor (PPI) and the two antibiotics, amoxicillin and metronidazole, for 14 days to eradicate *H. pylori* infection [20,21]. Statistical analyses were performed using Statistical Product and Service Solutions (SPSS), version 20. Categorical values between two groups were compared using chi-square test. Differences were considered statistically significant at $p \leq 0.05$.

Result

A total of 85participants were enrolled in this study, with a mean age of 22.01 (SD ±1.14) years. Of them, 29 (34.11%) were male and 56 (65.88%) were female. All participants came from middle- or upper-class backgrounds with the majority having graduate parents (87.1% of fathers, and 71.8% of mothers). All subjects reported practicing hand hygiene and drinking safe water. Among 85 participants tested, 59 (69.4%) were positive for *H. pylori* infection either by positive stool antigen test or by the presence of serum *H. pylori*-specific CagA-IgG or IgA antibodies.

Among 85 individuals tested, 9 (10.6%) were positive for *H. pylori* stool antigen, of whom 8 were also positive for *H. pylori*-specific CagA-IgG and/or IgA antibodies. Out of 76 stool antigen-negative cases, 50 demonstrated positive result for CagA-IgG and/or IgA antibodies. There was no significant association between stool antigen positivity and presence of *H. pylori*-specific antibodies among the study population. Overall, 58 (68.2%) participants tested positive for *H. pylori* infection using antibody-based methods. (Table-1).

Table-1: *Comparison of H. pylori stool antigen with the presence of serum anti-H. pylori CagA-IgG and anti-H. pylori IgA antibodies*

Stool antigen test	**Anti *H. pylori***		
	CagA-IgG positive n(%)	**IgA positive n (%)**	**CagA-IgG and/or IgA positive n (%)**
Positive (n=09)	06(66.6)	07 (77.7)	08(88.9)
Negative (n=76)	37(48.6)	39 (51.3)	50 (65.8)
Total (N=85)	43(50.5)	46 (54.1)	58(68.2)

Note: p=0.483, p=0.170 and p=0.261 respectively compared for CagA-IgG, IgA and CagA-IgG and/or IgA between stool antigen positive and negative cases. p value was calculated using chi square test.

Table-2: *Comparison of serum anti-H. pylori CagA-IgG with anti-H. pylori IgA of the study population (N=85)*

Anti-*H. pylori* CagA-IgG	Anti-*H. pylori* IgA	
	Positive n (%)	Negative n (%)
Positive (n=43)	31 (72.0)	12 (27.9)
Negative (n=42)	15 (35.7)	27(64.2)
Total (N= 85)	46 (54.1)	39(45.8)

Note: p≤0.005, when compared between serum anti-H. pylori CagA-IgG and anti-H. pylori gA result of the study population using chi square test.

Among 85 enrolled students, anti-*H. pylori* CagA-IgG and IgA antibodies were detected in 43 (50.6%) and 46 (54.1%) individuals, respectively. Both antibodies were detected in 31 cases. IgA positivity rate was significantly higher (p≤0.005) in individuals who tested positive for CagA-IgG compared to those who were negative for Cag-IgG antibody. (Table-2)

Table-3 shows that, out of 85 participants, 17 (20.0%) complained of gastrointestinal symptoms whereas 68 (80.0%) were asymptomatic. No significant difference was observed in antibody positivity rates between symptomatic and asymptomatic individuals in this study.

Table-4 shows that, out of 85 participants, 17 (20.0%) complained of gastrointestinal symptoms whereas 68 (80.0%) were asymptomatic. A statistically significant association was found between stool antigen positivity and the presence of symptoms among the study population.

Table-3: *The relationship between serum anti-H. pylori CagA-IgG and anti-H.pylori IgA antibodies among symptomatic and asymptomatic cases.*

Symptoms	Anti-*H.pylori* CagA-IgG positive n (%)	Anti-H. *Pylori* IgA positive n (%)
Symptomatic (n=17)	07 (41.2)	10 (58.8)
Asymptomatic (n=68)	36 (52.9)	36 (52.9)
Total (N=85)	43 (50.5)	46(54.1)

Note: p=0.276, p=0.437 respectively compared with the presence of serum anti H. pylori CagA-IgG and anti H. pylori IgA antibodies of symptomatic and asymptomatic cases. p-value was calculated using chi square test.

Table-4: *Comparison of H. pylori stool antigen with the symptomatic and asymptomatic cases*

Symptoms	Stool antigen positive	Negative	Total
Symptomatic (n=17)	06 (35.2)	11 (64.8)	17
Asymptomatic (n=68)	03(4.4)	65(95.6)	68
Total (N=85)	09 (10.6)	76 (89.4)	85

p ≤ 0.05, p-value was calculated using chi square test.

Discussion

It is widely recognized that *H. pylori* is associated not only with peptic ulcer disease but also with gastric carcinoma and MALT lymphoma [22]. Several studies have shown that the prevalence of *H. pylori* among medical personnel tends to be higher than that in general population, one reason being their frequent exposure to hospital settings [23-25]. The present study aimed to evaluate prevalence of *H. pylori* infection among fourth-year MBBS students studying at Ibrahim Medical College, Dhaka.

In this study, an individual was considered positive for *H. pylori* infection based on a positive stool antigen test and/or the presence of *H. pylori*-specific CagA-IgG and/or IgA antibodies in serum. Overall, 69.4% of the study population tested positive for *H. pylori* infection in this study. However, an overall detection rate of 79.5% was observed in a previous study conducted in Bangladesh among asymptomatic rural children and adolescents [7]. The comparatively lower detection rate in this study may be attributed to better hygienic practices among medical students, who predominantly came from higher educational and socio-economic backgrounds [26].

Approximately 10.6% of individuals demonstrated a positive stool antigen test in the current study. Detection of *H. pylori* antigen in stool indicates active infection [27]. Rajan et al. found a stool antigen positivity rate of 8.4% in a hospital-based study in Singapore, which is consistent with this finding [28]. In contrast, Mazumder et al. detected stool antigen in 24.9% of enrolled children and adolescents in a rural area of Bangladesh [7]. This discrepancy may be attributed to lower hygienic practices among children compared to the adult subjects in the current study, as well as differences in socio-economic status and availability of sanitation facilities.

The prevalence of *H. pylori*-specific antibodies was reported as 55.8% using immunochromatography among students at a medical university in Iraq, compared to an overall 68.2% antibody positivity rate observed in the present study [29]. This discrepancy may be due to the higher sensitivity of ELISA-based assays in contrast to ICT.

Although 8 of the 9 participants who were positive for *H. pylori* stool antigen also tested positive for CagA-IgG and/or IgA antibodies, the association was not statistically significant, likely due to the small number of stool antigen-positive cases.

In our study, IgA positivity rate was significantly higher in individuals who tested positive for CagA-IgG antibody compared to those who were negative for CagA-IgG which corroborates the finding of Rautelin et al. (2000), who theorized that CagA- positive infections may induce a markedly higher IgA response than CagA-negative infections. CagA is an immunodominant protein of *H. pylori*, which is associated with cytoskeletal rearrangements and morphological changes in the host cell [30-33]. Previous research suggests that CagA-positive *H. pylori* strains are more likely to induce gastric inflammation and the subsequent development of peptic ulcer disease and gastric cancer compared to infections with CagA-negative strains [34-37].

In the present study, 31 (36.5%) participants tested positive for both CagA-IgG and IgA antibodies. Rautelin et al. observed that two-thirds of the subjects demonstrating both CagA-IgG and IgA antibodies had more severe gastric inflammation and were probably at higher risk for severe long-term sequelae [33]. In this study, antibody positivity did not differ significantly between participants with and without gastrointestinal symptoms, which is consistent with the findings of several studies conducted in Bangladesh and other Asian countries [38-41].

The current study showed that a large proportion of the study population demonstrated both IgA and CagA-IgG classes of *H. pylori*-specific antibodies. The simultaneous presence of these antibodies is important, regardless of symptom status, as it increases the risk of complications such as peptic ulcer disease and gastric carcinoma.

Stool antigen (HpSA) positivity was observed in35.2%ofsymptomatic individuals. Detection of *H. pylori* antigen (HpSA) in stool among symptomatic individuals indicates an active infection. Patients having two or more gastrointestinal symptoms were more likely to demonstrate a positive stool antigen test which is consistent with the findings of other studies conducted in Bangladesh and other Asian countries [27,42,43].

The organism is primarily transmitted through contaminated water and food, as well as direct person-to-person contact. Therefore, raising awareness among medical students is essential to help reduce the transmission. A limitation of our study was that only fourth-year MBBS students were included. However, it is fundamental to conduct large-scale studies which not only investigate the prevalence of the *H. pylori* among medical students but also thoroughly evaluate the determinants contributing to its transmission.

Conclusion

The study revealed that *H. pylori* infection is highly prevalent among medical students in Bangladesh. Given the risk of transmission and potential ling-term complications, it is essential to increase awareness and implement early detection strategies in this population. Further large-scale studies are required to assess the prevalence across different groups and to identify the key determinants contributing to infection and transmission.

Conflict of interest

The authors declare that there is no conflict of interest.

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Author contributions

Authors' contributions SA: sample/data collection, laboratory work, data entry and analysis and manuscript writing; RK: data collection, laboratory work. AM: Data entry and analysis, editing of manuscript.SN and EK sample/data collection; SPSS: data entry; MM: data collection, data entry. FR: sample/data collection, laboratory work, data entry and analysis; MSAJ: Idea generation, study design, data analysis.

References

1. Cover TL, Blaser MJ. *Helicobacter pylori* in health and disease. *Gastroenterology*. 2009; **136**(6): 1863-1873. doi:10.1053/j.gastro.2009.01.073.
2. Miranda AC, Machado RS, Silva EM, Kawakami E. Seroprevalence of *Helicobacter pylori* infection among children of low socioeconomic level in São Paulo. *Sao Paulo Med J*. 2010; **128**(4): 187-191. doi:10.1590/s1516-31802010000400002.
3. Goodwin CS, Worsley BW. Microbiology of *Helicobacter pylori*. *Gastroenterol Clin North Am*. 1993; **22**(1): 5-19.
4. Sachs G, Scott DR, Wen Y. Gastric infection by *Helicobacter pylori*. *Curr Gastroenterol Rep*. 2011; **13**(6): 540-546. doi:10.1007/s11894-011-0226-4.
5. Hooi JKY, Lai WY, Ng WK, Suen MMY, Underwood FE, Tanyingoh D, et al. Global prevalence of *Helicobacter pylori*infection: systematic review and meta-analysis. *Gastroenterology*. 2017; **153**(2): 420-429. doi:10.1053/j.gastro.2017.04.022.
6. Jarin I, Ahmed T, Nahar J J. Understanding *Helicobacter pylori* Infection Prevalence and Associated Factors in slum and swampy areas of Narayanganj, Bangladesh: Findings from Medical Camps.
7. Mazumder S, Rahman F, Akter F, Khatun R, Akter S, Saha SP, et al. Asymptomatic *Helicobacter pylori* infection among rural children and adolescents in Bangladesh. *IMC J Med Sci*. 2022; **16**(2): 007. doi: https://doi.org/10.55010/imcjms.16.017.
8. Windle HJ, Kelleher D, Crabtree JE. Childhood *Helicobacter pylori* infection and growth impairment in developing countries: a vicious cycle? *Pediatrics*. 2007; **119**(3): e754-9. doi:10.1542/peds.2006-2196.
9. Nicoline FT, Ndip RN. A South African perspective on *Helicobacter pylori*: prevalence, epidemiology and antimicrobial chemotherapy. *Afr J Microbiol*. 2013; **7**(21): 2430-7. doi:10.5897/AJMR2013.5594.
10. Ahmed KS, Khan AA, Ahmed I, Tiwari SK, Habeeb MA, Ali SM, et al. Prevalence study to elucidate the transmission pathways of *Helicobacter pylori* at oral and gastroduodenal sites of a South Indian population. *Singap Med J*. 2006; **47**(4): 291–6.
11. Wang X, Shu X, Li Q, LiY, Chen Z, Wang Y, et al. Prevalence and risk factors of *Helicobacter pylori* infection in Wuwei, a high-risk area for gastric cancer in northwest China: an all-ages population-based cross-sectional study. *Helicobacter*. 2021; **26**(4): e12810. doi:10.1111/hel.12810.
12. Habib AM, Alam MJ, Rudra B, Quader A, Al-Forkan M. Analysis of *Helicobacter pylori*

prevalence in Chittagong, Bangladesh, based on PCR and CLO test. *Microbiol Insights*. 2016; **9**: 47-50. doi:10.4137/MBI.S39858.

13. Rhaman MM, Rahman F, Mazumder S, Sayeed MA, Haq JA. *Helicobacter pylori* infection in asymptomatic rural Bangladeshi population. *IMC J Med Sci*. 2021; **15**(1): 41-6. doi:10.3329/imcjms.v15i1.54201.
14. Lin SK, Lambert JR, Schembri MA, Nicholson L, Korman MG. *Helicobacter pylori* prevalence in endoscopy and medical staff. *J Gastroen Hepatol*. 1994; **9**(4): 319-324. doi:10.1111/j.1440-1746.1994.tb01249.x.
15. Mastromarino P, Conti C, Donato K, Strappini PM, Cattaruzza MS, Orsi GB.Does hospital work constitute a risk factor for *Helicobacter pylori* infection? *J Hosp Infect*. 2005; **60**(3): 261–268. doi: 10.1016/j.jhin.2004.12.019.
16. Jahan H, Chowdhury OA, Uddin MJ. *Helicobacter pylori* infection on medical students: A study on MAG Osmani Medical College, Bangladesh. *African Journal of Virology.* 2013; **7**(2): 001-005.
17. Naser NKAA, Bashir MBM, Ali ASMA. Prevalence and associated risk factors of *Helicobacter pylori* infection among medical students at Shendi University, Sudan. *BMC Gastroenterol.* 2025; **25**(1): 466. doi:10.1186/s12876-025-04074-9.
18. Ferdaus SJ, Paul SK, Nasreen SA, Haque N, Sadekuzzaman M, Karim MR, et al. The prevalence, risk factors, and antimicrobial resistance determinants of *Helicobacter pylori* detected in dyspeptic patients in North–Central Bangladesh. *Infect Dis Rep*. 2024; **16**(2): 181-8. doi:10.3390/idr16020014.
19. Tshibangu-Kabamba E, Phuc BH, Tuan VP, Fauzia KA, Kabongo-Tshibaka A, Kayiba NK, et al. Assessment of the diagnostic accuracy and relevance of a novel ELISA system developed for seroepidemiologic surveys of *Helicobacter pylori* infection in African settings. *PLoS Negl Trop Dis*. 2021; **15**(9): e0009763. doi:10.1371/journal.pntd.0009763.
20. Khurana R, Fischbach L, Chiba N, Van Zanten SV, Sherman PM, George BA, et al. Meta-analysis: *Helicobacter pylori* eradication treatment efficacy in children. *Aliment Pharmacol Ther*. 2007; **25**(5): 523-536. doi:10.1111/j.1365-2036.2006.03236.x.
21. de Boer WA, Tytgat GN. Regular review: treatment of *Helicobacter pylori* infection. *BMJ*. 2000; **320**(7226): 31-34. doi:10.1136/bmj.320.7226.31.
22. Yi M, Chen S, Yi X, Zhang F, Zhou X, Zeng M, et al. *Helicobacter pylori* infection process: from the molecular world to clinical treatment. *Front Microbiol*. 2025; **16**: 1541140. doi:10.3389/fmicb.2025.1541140.
23. Braden B, Duan LP, Caspary WF, Lembcke B. Endoscopy is not a risk factor for *Helicobacter pylori* infection--but medical practice is. *Gastrointest Endosc.* 1997; **46**(4): 305-310. doi:10.1016/s0016-5107(97)70115-9.
24. Liu WZ, Xiao SD, Jiang SJ, Li RR, Pang ZJ. Seroprevalence of *Helicobacter pylori* infection in medical staff in Shanghai. *Scand J Gastroenterol.* 1996; **31**(8): 749-752. doi:10.3109/00365529609010346.
25. Nishikawa J, Kawai H, Takahashi A, Seki T, Yoshikawa N, Akita Y, et al. Seroprevalence of immunoglobulin G antibodies against *Helicobacter pylori* among endoscopy personnel in Japan. *Gastrointest Endosc.* 1998; **48**(3): 237-243. doi:10.1016/s0016-5107(98)70184-1.
26. Almadi MA, Aljebreen AM, Tounesi FA, Abdo AA. *Helicobacter pylori* prevalence among medical students in a high endemic area. *Saudi Med J*. 2007; **28**(6): 896-898.
27. Al Ofairi BA, Saeed MK, Al-Qubaty M, Abdulkareem AM, Al-Jahrani MA. Diagnostic value of IgG antibody and stool antigen tests for chronic *Helicobacter pylori* infections in Ibb Governorate, Yemen. *Sci Rep*. 2024; **14**(1): 7536. doi:10.1038/s41598-024-58165-w.
28. Rajan C, Chiou FK, Ho CWW. Prevalence, Management, and Outcomes of Non-invasive *Helicobacter pylori* testing in children at a tertiary paediatric hospital in Singapore. *Pediatr Gastroenterol Hepatol Nutr*. 2024; **27**(6): 336–344. doi:10.5223/pghn.2024.27.6.336.

29. Hussen BM, Qader SS, Ahmed HF, Ahmed SH. Ahmed. The prevalence of *Helicobacter pylori* among university students in Iraq. *Indian Journal of Science and Technology.* 2013; **6**(8): 1-5. doi: 10.17485/ijst/2013/v6i8.4.

30. Segal ED, Cha J, Lo J, Falkow S, Tompkins LS. Altered states: involvement of phosphorylated CagA in the induction of host cell growth changes by *Helicobacter pylori*. *Proc Natl Acad Sci U S A*. 1999; **96**(25): 14559-14564. doi:10.1073/pnas.96.25.14559.

31. Asahi M, Azuma T, Ito S, Ito Y, Suto H, Nagai Y, et al. *Helicobacter pylori*CagA protein can be tyrosine phosphorylated in gastric epithelial cells. *J Exp Med.* 2000; **191**(4): 593-602. doi:10.1084/jem.191.4.593.

32. Odenbreit S, Piils J, Sedlmaier B, Gerland E, Fischer W, Haas R. Translocation of *Helicobacter pylori*CagA into gastric epithelial cells by type IV secretion. *Science.* 2000; **287**(5457): 1497-1500. doi:10.1126/science.287.5457.1497.

33. Stein M, Rappuoli R, Covacci A. Tyrosine phosphorylation of the *Helicobacter pylori*CagA antigen after cag-driven host cell translocation. *Proc Natl Acad Sci U S A.* 2000; **97**(3): 1263-1268. doi:10.1073/pnas.97.3.1263.

34. Crabtree JE, Taylor JD, Wyatt JI, Heatley RV, Shallcross TM, Tompkins DS, et al. Mucosal IgA recognition of *Helicobacter pylori* 120 kDa protein, peptic ulceration, and gastric pathology. *Lancet*. 1991; **338**(8763): 332-335. doi:10.1016/0140-6736(91)90477-7.

35. Peek RM, Miller GG, Tham KT, Perez-Perez GI, Zhao X, Atherton JC, et al. Heightened inflammatory response and cytokine expression in vivo to cagA+ *Helicobacter pylori* strains. *Lab Invest*. 1995; **73**(6): 760-70.

36. Parsonnet J, Friedman GD, Orentreich N, Vogelman H. Risk for gastric cancer in people with CagA positive or CagA negative *Helicobacter pylori* infection. *Gut.* 1997; **40**(3): 297-301. doi:10.1136/gut.40.3.297.

37. Blaser MJ, Perez-Perez GI, Kleanthouse H, Cover T, Peek R, Chyou P, et al. Infection with *Helicobacter pylori* strains possessing cagA is associated with an increased risk of developing adenocarcinoma of the stomach. *Cancer Res.* 1995; **55**(10): 2111-2115.

38. Perveen I, Saha M, Hasan MQ. Is there decreasing prevalence of *Helicobacter Pylori*infection in patients with dyspepsia? *Journal of Chittagong Medical College Teachers' Association.* 2022; **33**(1): 97-102. doi:10.3329/jcmcta.v33i1.67265.

39. Jenks PJ, Mégraud F, Labigne A. Clinical outcome after infection with *Helicobacter pylori* does not appear to be reliably predicted by the presence of any of the genes of the cag pathogenicity island. *Gut*. 1998; **43**(6): 752-758. doi:10.1136/gut.43.6.752.

40. Hua J, Zheng PY, Yeoh KG, Ho B. The status of the cagA gene does not predict *Helicobacter pylori*-associated peptic ulcer disease in Singapore. *Microbios*. 2000; **102**(402): 113-120.

41. Yang JC, Wang TH, Wang HJ, Kuo CH, Wang JT, Wang WC. Genetic analysis of the cytotoxin-associated gene and the vacuolating toxin gene in *Helicobacter pylori* strains isolated from Taiwanese patients. *Am J Gastroenterol*. 1997; **92**(8): 1316-1321.

42. Sultana A, Ahmed S, Ahmed EU, Nuruddin Chowdhury AF, Kalam A, Rahman A et al. Stool Antigen Test is Effective and Sensitive for Detecting *Helicobacter Pylori* Infection in Bangladeshi Peptic Ulcer Patients. *medRxiv*. 2021: 2021-10.

43. Ebar MH, Ahmed MO. Frequency of *H. pylori* Infection among Patients with Gastrointestinal Symptoms Attending Somali Sudanese Specialized Hospital (SSSH), Mogadishu, Somalia. *Asian journal of medicine and health*. 2023; **21**(8): 27-31.

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