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RESEARCH ARTICLE

Significance of Stool Antigen Testing for *Helicobacter pylori* Detection in Humans

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ABSTRACT

Helicobacteriosis, a globally prevalent bacterial infection caused by *Helicobacter pylori*, affects more than half of the world's population. *H. pylori*, a Group 1 carcinogen, is a widespread pathogen associated with gastric cancer and peptic ulcers. Due to its high prevalence, carcinogenic classification, and increasing antimicrobial resistance, *H. pylori* remain a major global health concern. This study examined the frequency of *H. pylori* infection in a specific population, revealing an overall prevalence of 20.5%, with a higher infection rate among females (23.7%) than males (17.8%). The 11–20-year age group had the highest infection rate (28.6%), while the 1–10-year group had the lowest (11.5%). However, statistical analysis (Chi-square test, "P" = 0.921) indicated no significant age-related differences, suggesting a relatively uniform distribution across age groups. Similarly, rural residents exhibited a slightly higher infection rate (22.3%) than urban dwellers (18.5%). However, the difference was not statistically significant (P = 0.593), suggesting that the location of habitation had little to no influence on infection risk. Monthly variations in prevalence were noted, with peaks in September (24.3%) and December (21.6%), whereas August recorded the lowest rate (16.7%). However, statistical analysis (P = 0.982) revealed no significant seasonal trend, suggesting that temporal factors did not substantially influence infection rates in this study. Overall, the findings indicate a moderate prevalence of *H. pylori* infection with no significant variations across age, location, or season, pointing to a relatively uniform distribution within the studied population. Further investigations are warranted to elucidate the underlying risk factors and transmission dynamics.

Keywords: Detection, *Helicobacter pylori*, stool, antigen test, Erbil

INTRODUCTION

Helicobacteriosis, a globally prevalent bacterial infection caused by *Helicobacter pylori*, effects more than 50% of the world's population and has been recognized by the World Health Organization as a significance pathogen. Given its increasing frequency and the growing hazard of antimicrobial resistance, *H. pylori* has become a major focus of medical and scientific investigation. Classified as a Group 1 carcinogen, the bacterium is unequivocally related to peptic ulcer and gastric cancer disease, highlighting its significant public health problem.[1-3]

However, *H. pylori* is one of the most prevalent bacterial pathogens affecting humans. As of 2015, an assessed 4.4 billion people worldwide were infected.[4,5] However, prevalence fluctuates significantly by region, with higher infection rates detected in areas with poor hygiene and weaker economies, such as much of Asia, Africa, and South America, compared to lower rates in North America, Australia, and Western Europe.[6-8]

The worldwide prevalence of *H. pylori* infection shows marked difference, with predominantly outstanding contrasts between developed and developing countries. In low-income nations, infection rates are uncommonly high, affecting 85–95% of the population, while developed regions report pointedly lower occurrence rates of 30–50%. The primary causes of this difference remain incompletely understood but may stem from differential access to healthcare, underreporting, or other yetunidentified causative factors. The epidemiology of *H. pylori* is shaped by a complex interaction of factors, including host genetic predisposition, patterns of antibiotic use, bacterial strain virulence, and environmental influences, such as hygiene practices and socioeconomic status.[9-13]

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Even though the exact method of *H. pylori* transmission remains indistinct, several pathways have been proposed. The bacterium can spread both indirectly through environmental sources and directly person-to-person. In developed countries, person-to-person transmission seems to be the principal route. In contrast, in developing countries with suboptimal food and water hygiene, polluted food and water also play a major role in transmission. Notably, not all persons exposed to *H. pylori* develop symptoms or complications, as the infection's outcome depends on factors, such as bacterial virulence, genetic susceptibility, and host immunity.[14-17]

Several factors impact *H. pylori* transmission, including personal hygiene, bacterial strain virulence, immune response, geographic location, living conditions, and socioeconomic status.[18,19] While most infected individuals remain asymptomatic carriers, the infection shows distinguished geographic variability[20] in developed countries, infection rates rarely exceed 40%, whereas in developing countries, over 80% of the population may contract *H. pylori* during childhood.[21] Children and adolescents in developed countries show much lower infection rates than adults and the elderly.[22,23]

The groundbreaking detection that *H. pylori* is the root cause of gastritis and peptic ulcers is attributed to the original work of Dr. Barry Marshall and Dr. Robin Warren. In a daring act of scientific opinion, Marshall intentionally consumed a live culture of *H. pylori* isolated from a patient, and soon established acute gastritis. At a time when the health institution definitely supposed that stress and lifestyle factors were to responsibility, Marshall and Warren challenged traditional wisdom. This audacious self-experiment supported certain proof of the bacterium's role in disease, crushing long-held medical beliefs and shifting the thoughtful and treatment of gastric conditions.[24,25]

Their pioneering work faced important doubt, sparking strong discussion inside the scientific community over whether *H. pylori*, rather than other factors, was actually accountable for peptic ulcers. However, Marshall and Warren finally altered gastroenterology, proving that ulcers and chronic inflammation were mainly bacterial in origin. Their scientific work contributions earned them the 2005 Nobel Prize in Physiology or Medicine, strengthening their heritage in medical history.[26,27]

For decades, the human stomach was considered a sterile environment, as its harsh acidic conditions were supposed to be unfavorable to microbial life. This long-held belief was inverted in 1982 when Barry Marshall and Robin Warren made a groundbreaking discovery: They identified *H. pylori* in gastric biopsies, revealing its essential role in gastritis and peptic ulcer disease.[28-31]

A critical survival strategy employed by *H. pylori* in the harsh acidic milieu of the stomach is its robust production of urease. This enzyme facilitates the breakdown of urea into ammonia, which counteracts gastric acid, thereby creating a more hospitable niche for bacterial colonization and long-term persistence.[32,33]

Today, their findings remain a cornerstone of modern gastroenterology, as evidenced by contemporary research. Further studies continue to reinforce their conclusions, underscoring the enduring impact of their discovery on clinical practice and ulcer treatment worldwide.[34,35]

H. pylori is a Gram-negative, microaerophilic bacterium linking to the *Helicobacteraceae* family. Naturally showing an S-shaped or curved morphology, it ranges from 0.5 to 3 μm in length and mostly colonizes the gastric mucus layer. A hallmark of *H. pylori* is its characteristic unipolar flagellar bundle, composed of 5–7 sheathed flagella, each measuring about 3 μm in length and ending in prominent bulb-like structures. These flagella, which are critical for pathogenicity and motility, are formed by the copolymerization of two flagellin proteins, FlaA and FlaB.[36,37]

The prevalence of *H. pylori* infection in Iraq has been rising, with most cases acquired during early childhood. Given this trend, the aims of the study include: (1) Detect the frequency of *H. pylori* in human stool samples from Erbil Governorate, (2) Study its occurrence across different age groups and residential areas, and (3) Evaluate infection rates over the study period. Furthermore, epidemiological understandings into *H. pylori* can advise public health strategies to control transmission, avoid new infections, and support therapeutic efforts to eliminate the bacterium.

MATERIALS AND METHODS

Collection of Samples

A total of 215 stool samples (118 from males and 97 from females) were collected from several private medical laboratories in Erbil City between July and December 2024. Each specimen was obtained in a clean, dry, sterile container following the protocol described by.[38] Relevant patient information accompanying the samples, including age, sex, and place of residence, was recorded for epidemiological analysis.

Preparation of Samples

All reagents and biological specimens were equilibrated to ambient room temperature before analytical procedures to ensure consistency and accuracy of results. The sample bottles were opened and using the applicator stick attached to the bottle cap, a small portion of stool (5–6 mm in diameter; approximately 100–200 mg or 0.1–0.2 g) was transferred into the bottle containing specimen preparation buffer. The applicator sticks were then returned to their respective bottles, matched to their assigned identification numbers, and the caps were tightened securely. The stool samples were mixed thoroughly with the buffer by gently shaking the bottles for several seconds to ensure homogeneity.[39]

Testing of the Samples

Test cards were taken out of their sealed foil pouches; samples were thoroughly mixed by shaking the bottles before initiating the test procedure. While holding each bottle in a vertical position over the sample well of the test card, approximately three drops (120–150 μL) of the diluted stool sample were dispensed into the sample hole. The results were interpreted within 10–15 min, with strongly positive samples occasionally producing visible results in a shorter time.

Interpretation of Results

A distinct pink band appearing in the test line region, along with a pink line in the control line region, indicates a positive result. In contrast, a negative result is characterized by the absence of a line in the test line region, while a clear pink line remains visible in the control line region.[40] Statistical Analyses

The Chi-square (χ^2) test was used to determine whether there was a statistically significant association between definite variables- age groups, sex, habitation, and the prevalence of *H. pylori* infection. The test compares the observed frequencies in each category with the frequencies expected if there were no association.

RESULTS

According to the *H. pylori* antigen test, the overall frequency of *H. pylori* infection was 20.5% [Table 1]. The prevalence rate was higher in females (23.7%) than in males (17.8%). Chi-square analysis indicated no significant difference in the prevalence of *H. pylori* antigen between participants from rural areas (22.3%) and urban areas (18.5%) (χ^2 = 0.29, df = 1, P = 0.593).

The relationship between the age groups and frequency was illustrated in Figure 1. The age group 11–20 years had the highest frequency of *H. pylori* antigen (28.6%), followed by the age group 61–70 years (22.2%). The age range of 1–10 years old had the lowest prevalence (11.5%). The statistical analysis (Chi-square test) showed no statistically significant difference in *H. pylori* antibody prevalence among the different age groups (χ^2 = 2.58, df = 7, P = 0.921, χ^2 = 2.58, df = 7, P = 0.921). Since $P > 0.05$, there is.

Chi-square analysis showed no significant variation in *H. pylori* antibody prevalence in the tested groups (χ^2 = 2.58, df = 7, P = 0.921). The prevalence ranged from 11.5% in the 1–10-year group to 28.6% in the 11–20-year group, with no consistent trend across ages.

Prevalence of *H. pylori* antigen in humans according to habitation site.

As shown in Table 2, the prevalence of *H. pylori* antigen was higher among participants residing in rural areas (22.3%) compared with those living in urban areas (18.5%).

Chi-square analysis indicated no significant difference in the prevalence of *H. pylori* antigen between participants from rural areas (22.3%) and urban areas (18.5%) (χ^2 = 0.29, df = 1, P = 0.593). This suggests that the habitation site had no detectable influence on infection rates in the study population.

Relationship between month and prevalence of *H. pylori* antigen during the study period The *H. pylori* antigen prevalence changed over the research months, as indicated in Figure 2. August had the lowest prevalence (16.7%), while September had the highest (24.3%), and December had the second peak (21.6%).

Chi-square analysis revealed no significant variation in the monthly prevalence of *H. pylori* antigen (χ^2 = 0.72, df = 5, P = 0.982). Prevalence ranged from 16.7% in August to 24.3% in September, with no consistent seasonal pattern. These results suggest that the month of sampling did not influence infection rates in this population.

Table 1: Prevalence of *Helicobacter pylori* antigen in human samples according to sex

Sex	No. of samples	Positive No. (%)	Negative No. (%)
Male	118	21 (17.8)	97 (82.2)
Female	97	23 (23.7)	74 (76.3)
Total	215	44 (20.5)	171 (79.5)
Chi-square analysis		χ^2=0.81, df=1	P=0.368 (NS)

Table 2: Prevalence of *Helicobacter pylori* antigen in Human according to the habitation site

Habitation site	No. of samples examined	Positive samples No%	Negative samples No%	Statistical results
Urban area	103	19 (18.5)	84 (81.5)	
Rural area	112	25 (22.3)	87 (77.7)	
Total	215	44 (20.5)	171 (79.5)	χ^2=0.29, df=1, P=0.593

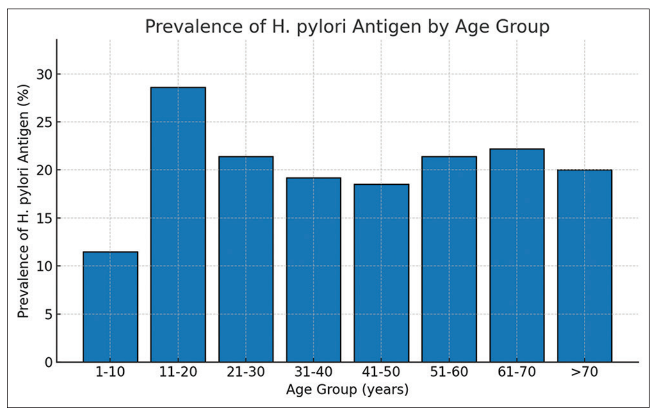

Figure 1: Prevalence of *Helicobacter pylori* antibody in human according to age

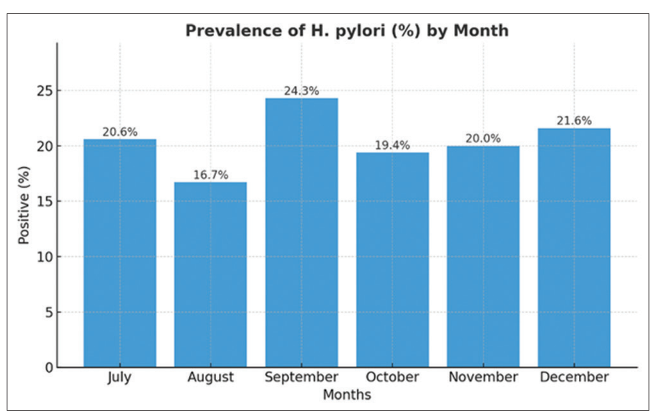

Figure 2: Relationship between months and prevalence of *Helicobacter pylori* antigen

DISCUSSION

H. pylori is a highly sophisticated and globally prevalent bacterium. The exact timing of its acquisition remains unclear; however, epidemiological evidence suggests that infection predominantly occurs in children under the age of five, indicating that most individuals contract *H. pylori* during early childhood.[41]

Extensive research has established a strong association between *H. pylori* infection and gastrointestinal disorders. Notably, 90% of duodenal ulcers and 70% of gastric ulcers are linked to *H. pylori*, making it a leading cause of peptic ulcer disease and gastric cancer. The bacterium's survival in the highly acidic stomach environment is facilitated by its production of urease, an enzyme that hydrolyzes urea into carbon dioxide and ammonia. This biochemical activity not only neutralizes gastric acid but also contributes to clinical manifestations, such as halitosis (bad breath) and excessive belching.[42,43]

Non-invasive diagnostic methods play a crucial role in assessing *H. pylori* infection. Among these, the stool antigen test (SAT) stands out as a cost-effective, highly sensitive, and easily applicable rapid diagnostic tool. Due to its reliability and simplicity, the SAT is widely utilized worldwide for detecting *H. pylori.*[44]

In the present study, the prevalence of *H. pylori* infection was found to be 20.5% [Table 1], which is higher than the 13.1% reported by Namakin and Nejad[45] in a study involving 282 Iranian students. Their research identified significant correlations between *H. pylori* colonization and factors, such as sex, duration of breastfeeding, and family crowding, though no significant association was found with age.

Conversely, our findings indicate a lower prevalence compared to studies conducted in other regions. For instance, research in Makkah, Saudi Arabia, reported a 27.4% infection rate using the urea breath test[46] Similarly, a study in Turkey by Elmas and Akçam[47] involving 1,737 cases found that 29% were *H. pylori*-positive, with infection rates increasing with age: 14.5% (0–6 years), 26.9% (7–12 years), and 33.6% (13–18 years).

On the other hand, Moon *et al*.[48] found that among 318 seropositive subjects, 256 (80.5%) tested positive in stool examinations. Similarly, El-Shabrawi *et al*.[49] reported that in Egypt, the *H. pylori* SAT was positive in 34 out of 38 patients (89.5%). The observed variations in *H. pylori* infection rates may be attributed to differences in socioeconomic status, hygiene standards, and drinking water sources. Additional contributing factors include poor social and economic development, low education levels, inadequate childhood hygiene practices, household overcrowding, lack of proper sewage disposal systems during early life, and improper food handling.

Furthermore, as shown in Table 1, the highest prevalence rate of *H. pylori* antigens was observed in females (23.7%), whereas the lowest rate was found in males (17.8%). These findings contrast with those of Qiao *et al.*,[50] who reported an overall infection rate of 49.59%, with males at 49.74% and females at 49.3%.

As illustrated in Figure 1, the frequency of *H. pylori* antigen varied across age groups, with the highest prevalence observed in the 11–20-year cohort (28.6%), followed by the 61–70-year group (22.2%). In contrast, the lowest infection rate was found in children aged 1–10 years (11.5%). However, Chi-square analysis revealed no statistically significant differences in *H. pylori* seropositivity among age groups (χ^2 = 2.58, df = 7, "*P*" = 0.921), as "*P*" > 0.05. These findings align with a study by Elmas and Akçam[47] in Turkey, which reported an overall *H. pylori* positivity rate of 29% (504/1737 cases), with prevalence increasing with age: 14.5% (0–6 years), 26.9% (7–12 years), and 33.6% (13–18 years).

Geographic disparities were evident, with rural participants exhibiting a higher *H. pylori* antigen prevalence (22.3%) than urban residents (18.5%) [Table 2]. This trend was corroborated by Almashhadany *et al*.[15] in Yemen, where stool antigen testing (HpSA) identified a 19.3% positivity rate (89/460 specimens). Notably, rural populations had a higher infection rate (20.9%) than urban dwellers (14.7%), and females showed slightly greater susceptibility (21.9%) compared to males (17.2%).

A study by AL-Sinani *et al*.[51] in Oman revealed that the prevalence of *H. pylori* in children increased significantly with age, rising from 7% in those under 5 years to 33% in the 5–10-year age group. Similarly, Awuku *et al*.[52] reported an overall *H. pylori* infection rate of 14.2% among children in Ghana, with the lowest prevalence (11.9%) observed in adolescents aged 14–16 years. Notably, the infection was more common in females (16.8%) than in males (10.7%), yielding a female-to-male ratio of 1.3:1.

The high prevalence of *H. pylori* in adulthood may stem from early-life exposure due to risk factors, such as poor sanitation, inadequate hygiene, and genetic predisposition. Overcrowding, contaminated water, and food further contribute to transmission, particularly in children. These findings align with epidemiological studies published earlier this decade.

As illustrated in Figure 2, the monthly distribution of *H. pylori* antigen prevalence varied, with the lowest rate (16.7%) occurring in August and the highest (24.3%) in September, followed by a secondary peak (21.6%) in December. Conversely, Almashhadany and Mayass[39] reported February as the peak month for *H. pylori* antigen detection (40.0%), with prevalence declining progressively thereafter.

CONCLUSION

Although *H. pylori* infection remains a significant global health concern, this study found no notable correlations with age, geographic location, or seasonal variation within the examined population. These results underscore the importance of exploring alternative risk factors, such as overcrowding, socioeconomic disparities, educational background, dietary habits, sanitary conditions, and the prevalence of immigrant children from adjacent towns, as well as other potential contributors, including poor sanitation, inadequate hygiene, and genetic susceptibility. Understanding these factors is critical to elucidating transmission dynamics and informing targeted prevention measures. Given *H. pylori*'s classification as a Group 1 carcinogen, sustained surveillance and proactive public health initiatives are imperative to mitigate its global impact. Addressing these gaps through multidisciplinary research and evidence-based interventions will be crucial in reducing the persistent burden of *H. pylori*-related diseases.

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