

Evaluation of non-invasive tests for diagnosis of *Helicobacter Pylori* infection among Egyptian sewage workers

Amal Saad-Hussein, Safia Beshir* and Weam Shaheen

Environmental & Occupational Medicine Department, Division of Environmental Research, National Research Centre, El-Buhouth St, Dokki, Giza, 12622, Egypt.

ABSTRACT

Helicobacter pylori increases the risk of gastric cancer by six times. This study aims to assess the prevalence of *Helicobacter pylori* infection among sewage workers, and to evaluate Western blot and *H. pylori* (IgG & IgA) antibodies against stool antigen for diagnosis of *Helicobacter pylori* infection. Cross sectional study was carried out on 90 male sewage workers in Abo-Rawash municipal wastewater treatment plant located in Egypt, from 2017 to 2019. All participants filled in a questionnaire. *H. pylori* IgG, IgA antibodies test, and Western blot IgG test against 13 antigens of *Helicobacter pylori* and *H. pylori* antigen in stool were done. Presence of gastrointestinal symptoms was observed in 5.6% to 38.9% sewage workers. The seroprevalence of *H. pylori* IgG and IgA are 87.8% and 47.8%, respectively. Sewage workers were divided into administrators, operators and laboratory workers. The highest percent of chronic and current *Helicobacter pylori* infections were among the administrator group, and the lowest were among the laboratory workers. The prevalence of *H. pylori* antigen in stool is 46.7%. About 8.9% of the workers with positive *H. pylori* stool antigen are negative for serum *H. pylori* IgA, 4.4% are borderline and 33.3% are positive, while all the workers with positive *H. pylori* stool antigen show positive serum *H. pylori* IgG. Serum *H. pylori* IgA and IgG antibodies are significantly higher among those with positive *H. pylori* stool antigen. Workers with positive serum *H. pylori* IgG showed

higher percentage of symptoms compared to those with negative or borderline serum *H. pylori* IgG or those with positive serum *H. pylori* IgA. Serum level of *H. pylori* IgG is significantly higher among workers who have positive virulent strains except for p33. *H. Pylori* IgG assay is an excellent test that can be used to predict *H. pylori* infection. The serological investigation of antibodies to *H. pylori* using the anti-*H. pylori* enzyme-linked immunosorbent assay (IgG, IgA) and/or the anti-*H. pylori* western blot (IgG), may represent the diagnostically most-reliable and inexpensive method for the diagnosis of *H. pylori* infections.

KEYWORDS: *Helicobacter pylori*, sewage workers, *H. pylori* IgG, *H. pylori* IgA, *H. pylori* stool antigen, western blot, virulence factors.

1. INTRODUCTION

Almost, 50% of adults in developed countries and 90% in developing countries carry *Helicobacter pylori* (*H. pylori*) in their upper gastrointestinal tract making it the most prevalent infection in the world [1]. Previous studies showed that *H. pylori* is an extremely endemic disease in Egypt, especially in rural areas [2-4].

Water-borne diseases are the result of the lack of safe hygienic practices. Sewage workers are forced to work in unclean conditions as it is difficult to avoid contact with sewage [5]. Sewage pathogenic microorganisms found in sewage sludge are derived from human and animal feces [6]. Little attention is paid to the occupational risk to develop *H. pylori*

*Corresponding author: safiabeshir123@yahoo.com

infection among sewage-exposed workers and there is no existing data on the validation of non-invasive tests for the diagnosis of *H. pylori* among sewage workers in Egypt [7].

H. pylori is a gram-negative bacteria that has the ability to colonize and stay for long time in the gastric mucosa, causing chronic gastritis, duodenal and peptic ulcer, adenocarcinoma and gastric lymphoma in humans [8]. Although over 80% of cases are asymptomatic, *H. pylori* is proclaimed as a first-degree carcinogen by the World Health Organization (WHO). It is stated that *H. pylori* increases the risk of gastric cancer by six times; for this reason, an accurate and reliable method for diagnosis is required [9].

Wastewater treatment processes do not totally destroy the micro-organisms; pathogenic micro-organisms can persist in sewage [10]. Previous studies couldn't detect *H. pylori* antibodies among sewage workers, but the studied workers had different socioeconomic statuses, exposure information was not clearly defined and sample sizes used were small [11, 12].

The exact mode of transmission of *H. pylori* infection is not totally known, but person-to-person (fecal-oral or oral-oral routes) transmission is considered as the main route of transmission, followed by transmission through contaminated water and food [13]. Iatrogenic transmission is another significant mode of transmission, which occurs when tubes or endoscopes used in an infected person are used in an uninfected person [14].

Laboratory tests for diagnosis of *H. pylori* gastritis are divided into invasive and non-invasive tests. Endoscopic examination and urease test are examples of invasive tests. The noninvasive tests are urea breath test, detection of *H. pylori* antibodies in the serum, detection of *H. pylori* antigens in stool and in saliva etc. [15]. Stool antigen (HpSAg) detection test is convenient for the patients and can be easily done in small laboratories. However, the accuracy of stool antigen tests is a matter of concern [16]. Detection of serum *H. pylori* IgG may play an essential role in diminishing the need for endoscopy [17]. Detection of antibodies is useful for detecting past or present exposure [18]. Serological tests are non-invasive and do not produce false negative results in patients receiving treatment (proton pump inhibitors and antibiotics) or in those with acute bleeding [19].

Previous studies showed that *H. pylori* causes diseases due to different virulence factors [20, 21]. They are divided into Type I and Type II, based on the presence and absence of cytotoxin-associated gene A (CagA), the vacuolating cytotoxin (VacA)-and iceA genes which are considered to be the recognized virulence factors of *H. pylori* [22]. CagA and VacA are the two chief *H. pylori* virulence factors among the bacterial markers. They are extremely immunogenic proteins associated with type I strains which are accountable for severe forms of gastrointestinal diseases [21].

If the decision to treat a patient for *H. pylori* infection is built only on the serological test results, tests with a higher specificity are mandatory. Western blotting may be a suitable technique because it permits direct visualization of the antibody binding to antigens highly specific for *H. pylori* [23].

This study aims to assess the prevalence of *H. pylori* infection among sewage workers, and to evaluate western blot and *H. pylori* serum antibodies (IgG & IgA) against HpSAg for diagnosis of *H. pylori* infection in these workers.

2. SUBJECTS AND METHODS

2.1. Study design and population

Cross-sectional study was carried out on 90 male sewage workers in Abo-Rawash municipal wastewater treatment plant located in Giza Governorate Egypt, from 2017 to 2019, with mean duration of employment 19.5 ± 9.4 years. The study was approved by the Medical Research Ethics Committee (National Research Centre, Cairo, Egypt) No. 17085. Written consents were signed by all the participants in the study.

2.2. Methods

1. All participants in the present study filled in a questionnaire that included questions covering demographic data (age, sex, and residence), past history of *H. pylori* infection and eradication therapy, history of oesophago-gastro-duodenoscopy, whether they had received treatment with antibiotics, proton pump inhibitors, and H2 receptor antagonists within the last four weeks, previous gastric surgery, peptic ulcer, history of gastrointestinal bleeding, and gastrointestinal symptoms (nausea, abdominal distension, heart burn, food related abdominal

- pain, digestion troubles, hematemesis, severe weight loss).
2. Blood specimens for serological testing were drawn, where 5 ml venous blood was taken and collected in dry tubes. After clotting of blood, the sera were separated by centrifugation for 10 minutes at 3000 rpm and divided into aliquots that can be refrigerated at 2–8 °C for up to seven days or frozen for up to six months till used.
 3. Serum *H. pylori* IgG & *H. pylori* IgA antibodies tests were performed for all participants using anti-*Helicobacter pylori* enzyme-linked immunosorbent assay (ELISA) (IgG and IgA) purchased from EUROIMMUN Medizinische Labordiagnostika AG (Germany). Assay values were calculated according to the manufacturer's instructions. A subject was considered as seronegative if the ratio between the absorbance reading of the patient sample and that of calibrator <0.8, borderline if the ratio is between 0.8 and 1.1 and seropositive if the ratio >1.1. The sensitivity and specificity of this anti-*Helicobacter pylori* ELISA IgG are 100% while they are 97% and 92.9%, respectively for the anti-*Helicobacter pylori* ELISA IgA.
 4. Qualitative determination of human antibodies of the immunoglobulin class IgG against *Helicobacter pylori* antigens (13 antigens) with recombinant CagA, and VacA in serum was performed by Western blot test using the commercial *Helicobacter pylori* LINE IgG immunoblot kit from EUROIMMUN Medizinische Labordiagnostika AG (Germany). A subject was considered as seropositive if he had band positions against species-specific and highly specific antigens which are UreA p29 (light urease subunit), p30 (OMP, 30 outer membrane protein, species specificity), p26 and p19 (OMP19 outer membrane protein, species specificity) and 2 other less-specific antigens (p33 and p17). Qualitative detection of *H. pylori* antibodies in human serum was performed according to the manufacturer's instructions.
 5. Samples of stools were collected in dry clean cups and stored at -20 °C until used. Detection of *H. pylori* antigen in stool (HpSAg) was performed by immune chromatographic rapid assay (CAL-TECH DIAGNOSTICS, INC. Chino, CA 91710, USA) and the results were interpreted using manufacturer's instructions.

2.3. Statistical analysis

Statistical analyses were performed using SPSS software (version 20). A two-tailed t-test was used to assess the difference in the means of the studied subgroups according to their occupation. Chi-square test for significance was used to compare proportions between qualitative parameters. The result was considered significant if the P-value was ≤ 0.05 . Receiver operating characteristic curve (ROC) was prepared to plot the sensitivity (true positive rate) against 1-specificity (false positive rate) for *H. pylori* IgA and *H. pylori* IgG versus HpsAg.

3. RESULTS

Mean age of the workers was 47.5 ± 9.83 years. The mean duration period of employment was 19.5 ± 9.4 years. Sewage workers (90) were classified according to their occupation as administrators (N = 12), operators (N = 68), and laboratory workers (N = 10).

Table 1 shows that among the studied workers, 18/90 complained of nausea, 28/90 complained of abdominal distension, 13/90 complained of severe weight loss, 27/90 complained of digestion troubles, 15/90 complained of food-related abdominal pain, 5/90 complained of hematemesis, 35/90 complained of heartburn, and 9/90 complained of peptic ulcer. Presence of gastrointestinal symptoms was observed for 5.6 to 38.9% sewage workers.

Table 2 shows that the seroprevalence of *H. pylori* IgG and *H. pylori* IgA antibodies among sewage workers are 87.8% (79/90) and 47.8% (43/90), respectively.

Figure 1 shows that the highest percent of chronic (*H. pylori* IgG) and current (*H. pylori* IgA) infections are among the administrators, and the lowest are among the laboratory workers.

The prevalence of *H. pylori* antigen in stool (HpSAg) among the examined sewage workers is 46.7% (42/90). About 8.9% of those HpSAg-positive workers showed negative serum *H. pylori* IgA, 4.4% were borderline and 33.3% were positive. All the workers with positive HpSAg showed positive serum *H. pylori* IgG antibodies.

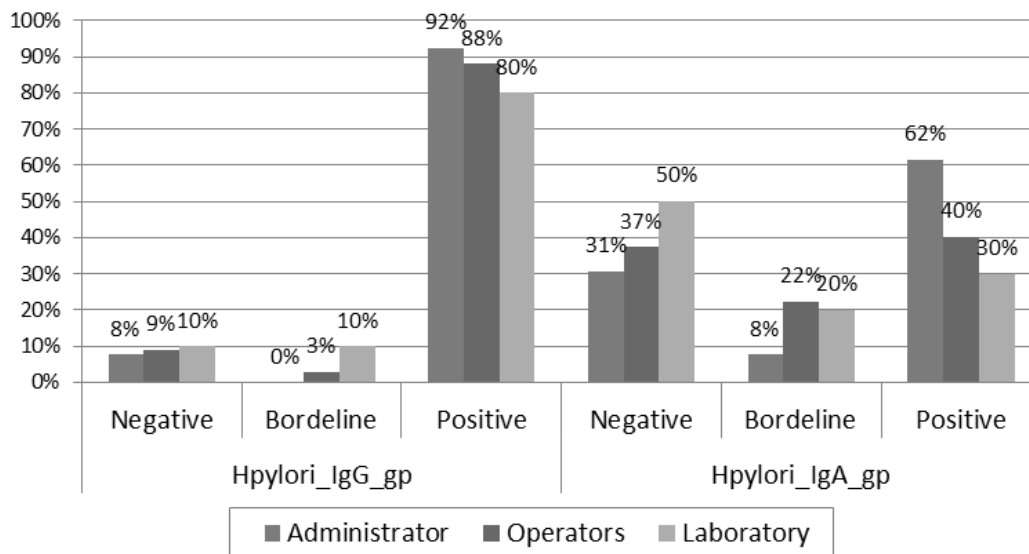
Table 3 shows that the mean of *H. pylori* antibodies (IgA and IgG) is significantly higher ($p < 0.01$) among the sewage worker group having positive *H. pylori* stool antigen.

Table 1. Prevalence of the gastrointestinal symptoms among sewage workers according to their occupation.

	Occupation			Chi-square p-value
	Administrators (N = 12)	Operators (N = 68)	Laboratory workers (N = 10)	
Nausea	4 (33%)	13 (19%)	1 (10%)	NS
Abdominal distension	3 (25%)	21 (31%)	4 (40%)	NS
Severe weight loss	2 (17%)	11 (16%)	0 (0%)	NS
Digestion troubles	5 (42%)	18 (27%)	4 (40%)	NS
Food-related abdominal pain	2 (17%)	12 (18%)	1 (10%)	NS
Hematemesis	1 (8%)	2 (3%)	2 (20%)	NS
Heartburn	5 (42%)	24 (36%)	6 (60%)	NS
Peptic ulcer	1 (8%)	7 (10%)	1 (10%)	NS

Table 2. *H. pylori* IgG and *H. pylori* IgA antibodies' prevalence among sewage workers.

	Negative	Borderline	Positive
<i>H. pylori</i> IgG antibodies (N = 90)	9 (10.0%)	2 (2.2%)	79 (87.8%)
<i>H. pylori</i> IgA antibodies (N = 90)	35 (38.9%)	12 (13.3%)	43 (47.8%)

**Figure 1.** Prevalence of antibodies to *H. pylori* among sewage workers classified according to their occupation.**Table 3.** Mean and SD of *H. pylori* antibodies among sewage workers in relation to *H. pylori* stool antigen.

		<i>H. pylori</i> IgA (Mean ± SD)	t-test	P value	<i>H. pylori</i> IgG (Mean ± SD)	t-test	P value
HpSA	Negative (48)	0.9 ± 0.6	4.1	0.01*	2.4 ± 1.2	7.9	0.01*
	Positive (42)	1.5 ± 0.8			4.3 ± 1.0		

*p < 0.01.

Figure 2 shows that serum *H. pylori* IgA can be used to predict the *H. pylori* infection as its sensitivity is about 80%. Therefore, *H. pylori* IgG detection is an excellent test to predict *H. pylori* infection as its sensitivity is more than 90% (Figure 3).

Table 4 shows that workers with positive serum *H. pylori* IgG antibodies show higher percentage of symptoms compared to those with negative or borderline serum *H. pylori* IgG antibodies or those with positive serum *H. pylori* IgA antibodies.

H. pylori Western blot line IgG test was done to all sewage workers who had positive *H. pylori* stool antigens (42) to detect the virulent strains of *H. pylori*.

Figure 4 shows the distribution of the serum anti-*H. pylori* line IgG against the virulent antigens, among the sewage worker group with positive *H. pylori* stool antigen (HpSAg). Table 5 shows that the mean of serum *H. pylori* IgG is significantly

higher ($p < 0.01$) among the sewage workers who had positive virulent strains expect for P33.

4. DISCUSSION

Our study was done on workers employed at the municipal wastewater treatment plant in (Giza Governorate, Egypt) Abo-Rawash. The occurrence of gastrointestinal symptoms was between 5.6 to 38.9% among the studied sewage workers. Abdominal symptoms among sewage workers may be related to the exposure to gram-negative bacteria endotoxins [24]. A study conducted for 60 workers working in the sewage collection system in Mansoura Governorate, Egypt showed prevalence of abdominal symptoms: heart burn 35%, colic 11.6%, changes in bowel habits 1.7%, and distension 8.3% [25]. Also, in another study conducted on sewage workers from different sectors in Alexandria Governorate, Egypt, heartburn was significantly

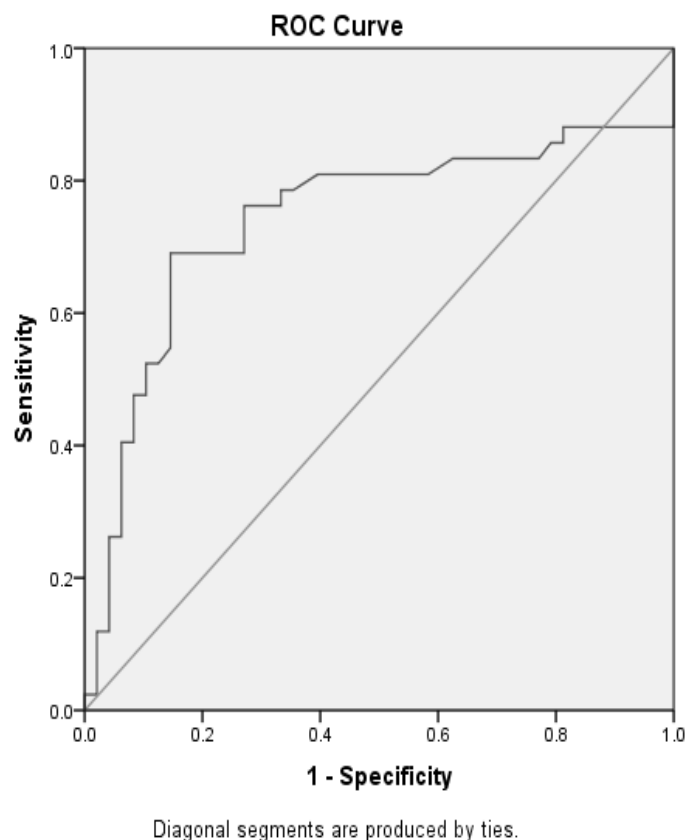


Figure 2. Receiver operating characteristic curve (ROC) curve for *H. pylori* IgA versus HpsAg. The optimal sensitivity and specificity of *H. pylori* IgA are 80% and 56%, respectively and the area under curve (AUC) is 0.741 (95% CI; 0.627–0.854).

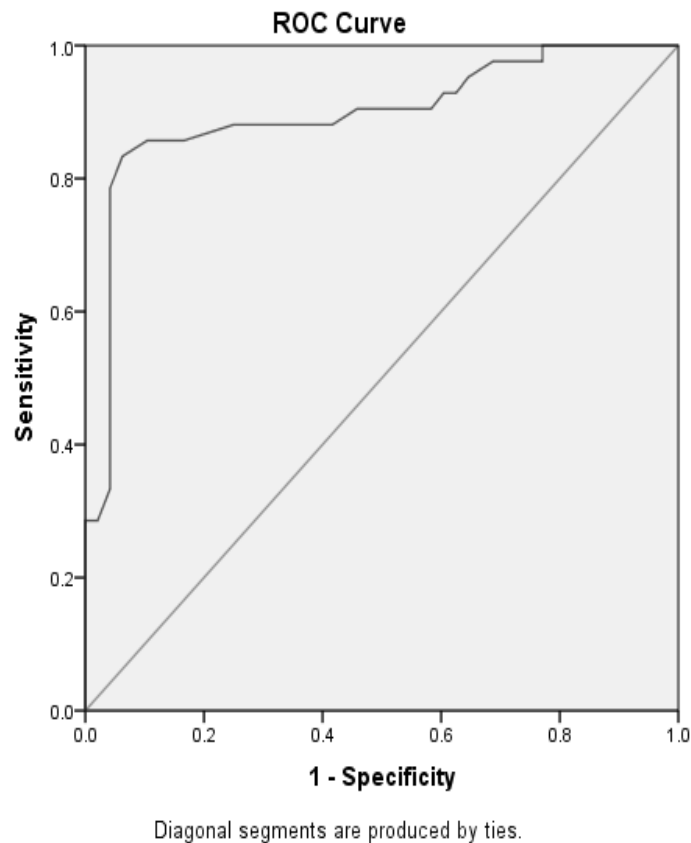


Figure 3. Receiver operating characteristic curve (ROC) curve for *H. pylori* IgG versus HpsAg. The optimal sensitivity and specificity of the *H. pylori* IgG are 100% and 81%, respectively and area under curve (AUC) is 0.896 (95% CI; 0.825–0.968).

Table 4. Percentage of occurrence of gastrointestinal symptoms and *H. pylori* antibodies (IgG & IgA) in sewage workers.

	<i>H. pylori</i> IgG Ab			<i>H. pylori</i> IgA Ab		
	Negative	Borderline	Positive	Negative	Borderline	Positive
Nausea (18)	3 (17%)	1 (6%)	14 (78%)	6 (33%)	6 (33%)	6 (33%)
Abdominal distension (28)	5 (18%)	1 (4%)	22 (79%)	13 (46%)	4 (14%)	11 (39%)
Severe weight loss (13)	2 (15%)	0 (0%)	11 (85%)	7 (54%)	1 (8%)	5 (38%)
Digestion troubles (27)	2 (7%)	1 (4%)	24 (89%)	12 (44%)	5 (19%)	10 (37%)
Food-related abdominal pain (15)	3 (20%)	0 (0%)	12 (80%)	7 (47%)	2 (13%)	6 (40%)
Hematemesis (5)	0 (0%)	0 (0%)	5 (100%)	3 (60%)	1 (20%)	1 (20%)
Heartburn (35)	3 (9%)	1 (3%)	31 (89%)	12 (34%)	7 (20%)	16 (46%)
Peptic ulcer (9)	1 (11%)	1 (11%)	7 (78%)	6 (67%)	0 (0%)	3 (33%)

higher in sewage workers (43.3%) compared with that in the unexposed subjects [7]. A preliminary study performed among 404 Belgian sewage-exposed and non-exposed employees, showed higher

prevalence of gastrointestinal symptoms, and peptic ulcer among sewage-exposed workers than among non-sewage-exposed employees [26]. Another Belgian study done on 317 sewage employees showed that

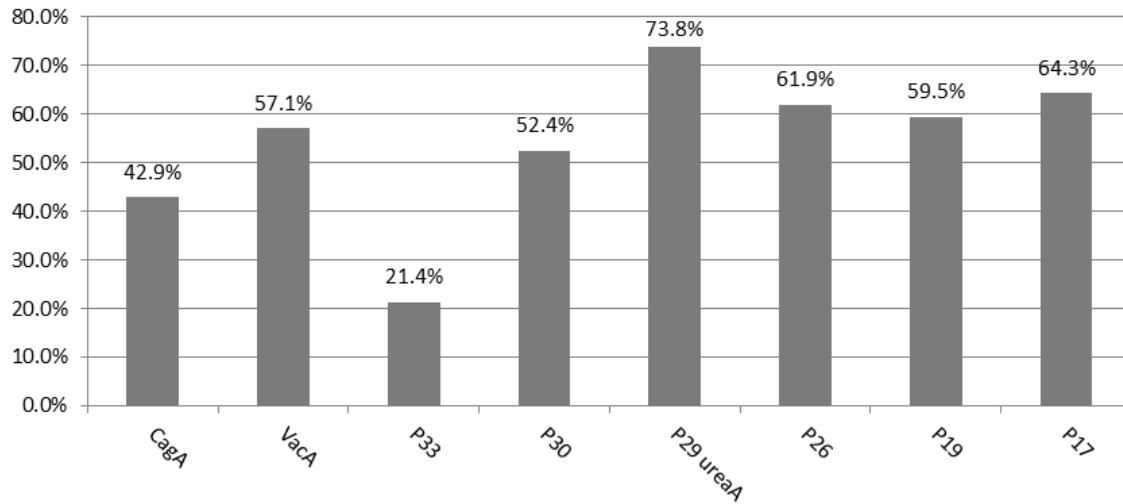


Figure 4. Distribution of the percent of positive anti-*H. pylori* line IgG against the virulent antigens, among the sewage worker group with positive *H. pylori* stool antigen (HpSAg).

Table 5. Mean of serum *H. pylori* IgG antibodies against the *H. pylori* virulent antigens among the sewage worker group with positive HpSAg.

		<i>H. pylori</i> IgG Ab. (mean±SD)	t-test	P-value
CagA	Negative (24)	4.1 ± 1.2	2.6	0.04*
	Positive (18)	4.7 ± 0.6		
VacA	Negative (18)	3.8 ± 1.2	3.3	0.002**
	Positive (24)	4.8 ± 0.6		
P33	Negative (33)	4.2 ± 1.1	1.4	0.15
	Positive (9)	4.8 ± 0.7		
P30	Negative (20)	4.0 ± 1.3	2.2	0.03*
	Positive (22)	4.7 ± 0.5		
P29ureaA	Negative (11)	3.1 ± 1.0	6.9	0.001**
	Positive (31)	4.8 ± 0.6		
P26	Negative (16)	3.7 ± 1.3	3.7	0.001**
	Positive (26)	4.8 ± 0.6		
P19	Negative (17)	3.7 ± 1.3	3.5	0.001**
	Positive (25)	4.8 ± 0.6		
P17	Negative (15)	3.5 ± 1.2	4.3	0.001**
	Positive (27)	4.8 ± 0.6		

*(p < 0.05), ** (p < 0.01).

the prevalence of gastrointestinal symptoms (stomach ache, abdominal pain, dyspepsia) was higher among the sewage workers (37%) than among the control group [24].

H. pylori infection can be diagnosed by invasive and non-invasive tests. The stool antigen test is used to detect *H. pylori* antigens in stools. It is a reliable test for diagnosis of the *H. pylori* infection and

confirmation of its cure after treatment, as it avoids detection of previous *H. pylori* infection [27].

The present study shows that 46.7% (42/90) of workers were positive for *H. pylori* stool antigen. The prevalence of positive HpSAg was 31.2% in a previous study conducted on sewage workers from different sectors in Alexandria Governorate, Egypt [6] and 56.7% in a study conducted on sewage workers in Mansoura Governorate, Egypt sewage treatment plant [7].

IgM antibodies first develop among *H. pylori*-infected persons. Then IgG and IgA antibodies develop systematically and locally in the gastric mucosa and their level can be maintained for months or years. *H. pylori* first causes acute gastritis which then turns into chronic gastritis slowly through 20-40 years [8]. IgA-positive results correlated with the activity of gastritis. It is formed locally, but not identified in the serum in every case. However, an increase in IgG serum level can continue for many years and is an indication of ongoing infection. Elevated IgG serum level is considered to be a marker for chronic infection [28].

In our study 87.8% of the studied group showed serum positive *H. pylori* IgG antibodies while 47.8% showed serum positive *H. pylori* IgA antibodies. Meanwhile 40% of the workers (36/90) had both *H. pylori* IgA & IgG antibodies. These findings agreed with those of a study done to detect *H. pylori* infection in adult Turkish dyspeptic patients which showed that *H. pylori* seropositivity was 82.1% for IgG and 48.2% for IgA antibodies [29]. A previous study conducted among 317 Belgian sewage workers found that IgG seroprevalence among sewage workers was 16.7% [24].

Moreover, our study shows that 42/90 subjects showed both positive HpsAg and serum *H. pylori* IgG antibodies, while 30/42 subjects showed both positive HpsAg and serum *H. pylori* IgA antibodies. Gosciniak (1997) stated that there is considerable evidence that *H. pylori* infection leads to elevation of *H. pylori* antibodies level in serum [30].

In the present study mean serum levels of *H. pylori* IgG & IgA are significantly higher among those with positive HpSA, 4.3 ± 1.0 and 1.5 ± 0.8 , respectively, than those with negative HpSA, 2.4 ± 1.2 and 0.9 ± 0.6 , respectively. Similar results were observed in a previous study done on *H. pylori*-infected and

non- infected patients, where serum *H. pylori* IgG and IgA levels were higher in *H. pylori*-positive patients, 1.45 ± 0.17 and 1.13 ± 0.10 , respectively, compared to those in *H. pylori*-negative subjects, 0.56 ± 0.14 and 0.59 ± 0.1 , respectively [30].

In the current study workers with positive serum *H. pylori* IgG antibodies show higher percentage of symptoms compared to those with negative or borderline serum *H. pylori* IgG antibodies or those with positive *H. pylori* IgA antibodies. In contrast, a previous study which was conducted among 317 Belgian sewage workers found no significant difference between subjects with positive *H. pylori* IgG and negative *H. pylori* IgG regarding gastrointestinal symptoms [24].

No single test can be considered as the gold standard for the diagnosis of *H. pylori* infection. Advantages and disadvantages must be taken in consideration when choosing the test [31].

Infection with *H. pylori* induces local and systemic immune responses. Western blot can detect presence of antibodies against *H. pylori* antigens. It is less probable to give false positive results as it can efficiently differentiate *H. pylori* antibodies from other antibodies. In addition, it can detect antibodies to *H. pylori* virulence factors such as CagA and VacA [32].

In the present study, 42.9% from those with positive *H. pylori* stool antigen, showed CagA-positive *H. pylori* antibodies. Some studies done on dyspeptic individuals found that 50% of patients with *H. pylori* infection had CagA-positive serology [29, 32]. These results were different from those of a previous study done on Egyptian population, where CagA-positive strains were not common among the studied group [33]. In the current study, mean serum level of *H. pylori* IgG is significantly higher in sewage workers who were CagA-positive compared to CagA-negative workers. Results of a previous study done on a group of 191 patients showed reactivity of CagA with serum IgG from *H. pylori* -positive patients [34].

Vac A is the second most important toxin among the *H. pylori*'s virulence factors. It causes injury to stomach cells, which induces host cell vacuolation and, lastly, cell death. The present study shows that 57.1% showed seropositive Vac A. Results of Rashed *et al.*, (2016) showed seropositive VacA (40%) in

H. pylori-infected patients [32]. In the current study, *H. pylori* IgG was observed to be significantly higher among the sewage workers who had positive Vac A virulent strain compared to Vac A-negative workers. Results of a previous study done on a group of 191 patients, showed reactivity of VacA with serum IgG from *H. pylori*-positive patients [34].

P29 urea A, p26, p17 anti- *H. pylori* Western blot of IgG antibodies showed highest seropositivity in the present study (73.8%, 61.9%, 64.3%, respectively). A previous study done on adult Turkish dyspeptic patients found that p29 urea A, p26, and p17 seropositivity were 80.4%, 41.1%, and 35.7%, respectively [29]. Also, in another study done on a group of *H. pylori*-infected patients, highest seropositivity (> 80%) was observed for the antigens p19, p26, and p29 [34]. Moreover, our study shows that the mean serum level of *H. pylori* IgG antibodies is significantly higher in those with seropositive P29 urea A, p26 and p17 compared to those with negative results.

In the present study sensitivity and specificity for serum *H. pylori* IgG are 100% and 81%, respectively, compared to 81% and 56%, respectively for serum *H. pylori* IgA. Similar results were found in a previous study in which sensitivity and specificity were 98% and 88%, respectively for serum *H. pylori* IgG and 79% and 82%, respectively for serum *H. pylori* IgA [30]. Also, in a previous study which was done to evaluate *H. pylori* IgG and IgA, sensitivity and specificity of serum *H. pylori* IgG was 87.6% and 61.0%, respectively, and for serum *H. pylori* IgA was 73.8% and 48.8%, respectively [35]. Our study confirmed that serum *H. pylori* IgG detection is an excellent test to predict *H. pylori* infection as its sensitivity is more than 90%.

5. CONCLUSION

Seroprevalence of *H. pylori* IgG and IgA antibodies are 87.8% and 47.8%, respectively in Abo-Rawash municipal wastewater treatment plant sewage workers. Presence of gastrointestinal symptoms is between 5.6 and 38.9%. Gastrointestinal symptoms are associated with positive *H. pylori* serology status. HpSA detection can be used as screening test for *H. pylori* infection. The serological investigation of antibodies to *H. pylori* using the

method for the diagnosis of *H. pylori* infection. Moreover, only the patients with positive anti-*H. pylori* Western blot (IgG) must take eradication treatment.

RECOMMENDATION

Surveillance of the sewage workers complaining of heartburn and/or epigastric pain, through serological investigation of antibodies to *H. pylori* to start the eradication treatment. Further studies need to be done on a larger scale.

LIMITATION OF THE STUDY

The study was done on a small scale.

FUNDING

The present study was funded by the Scientific Research Academy.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

REFERENCES

1. Zamani, M., Ebrahimitabar, F. and Zamani, V. 2018, *Aliment. Pharmacol. Ther.*, 47, 876.
2. Rocha, M., Avenaud, P., Ménard, A., Bail, B., Balabaud, C., Bioulac, P., Queiroz, D. M. and Mégraud, F. 2005, *Gut.*, 3, 401.
3. Mohammad, M. A., Hussein, L., Coward, A. and Jackson, S. J. 2008, *Pub. Health Nutri.*, 3, 236.
4. Ragheb, M. M., Awad, M. M. E., Tag Eldeen, L. E. and Dosoki, T. M. 2012, *J. Advan. Res.*, 3, 293.
5. Strunz, E. C., Addiss, D. G., Stocks, M. E., Ogden, S., Utzinger, J. and Freeman, M. C. 2014, *PLoS. Med.*, 11(3), e1001620.
6. Hassanein, F. I., Masoud, I. M. and Shehata, I. A. 2019, *P.U.J.*, 12, 138.
7. Agha, S., Foad, M. F., Awadalla, N. J. and Saady, N. 2013, *Afr. J. of Path. and Microb.*, 2, Article ID 235847.
8. Alim, A., Ataş, M., Güneş, T., Özkan, S. and Dündar, N. 2010, *Basic. Clin. Sci.*, 1(4), 70.
9. Ahmed, S. A. and Al Shammari, H. N. 2015, *Inter. J. Immunol.*, 3, 26.
10. Thorn, J. and Kerekes, E. 2001, *Am. J. Ind. Med.*, 40(2), 179.

11. Friis, L., Engstrand, L. and Edling, C. 1996, *Scand. J. Work. Environ. Health*, 22(5), 368.
12. Jeggli, S., Steiner, D. and Joller, H. 2004, *Occup. Environ. Med.*, 61(7), 627.
13. Sethi, A., Chaudhuri, M., Kelly, L. and Hopman W. 2013, *Can. Fam. Physician.*, 59, e187.
14. Rastogi, M., Rastogi, D. and Singh, S. H. 2014, *Biomed. Res.*, 25(4), 122.
15. Malfërtheiner, P., Megraud, F., O'Morain, C., Hungin, A. P., Jones, R., Axon, A., Graham, D. Y. and Tytgat, G. 2002, *Aliment. Pharmacol. Ther.*, 16, 180.
16. Gisbert, J. P. and Pajares, J. M. 2004, *Helicobacter*, 9, 368.
17. Jais, M. and Barua, S. 2004, *J. Commun. Dis.*, 36, 135.
18. Burucoa, C., Delchier, J. C., Courillon-Mallet, A., de Korwin, J. D., Mégraud, F., Zerbib, F., Raymond, J. and Fauchère, J. L. 2013, *Helicobacter*, 18, 169.
19. Braden, B. 2012, *B.M.J.*, 344, e828.
20. Rudi, J., Kolb, C., Maiwald, M., Kuck, D., Sieg, A., Galle, P. R. and Stremmel, W. 1998, *J. Clin. Microbiol.*, 4, 948.
21. Farshad, S., Japoni, A. and Kalani, M. 2009, *Hormozgan. Med. J.*, 13, 87.
22. Yamaoka, Y., Kodama, T., Gutierrez, O., Kim, J. G., Kashima, K. and Graham, D. Y. 1999, *J. Clin. Microbiol.*, 7, 2279.
23. Lepper, P. L., Mo'ricke, A., Vogt, K., Bode, G. and Trautmann. 2004, *Clin. and Diag. Lab. Immun.*, 11(3), 576.
24. Hooste, W. V., Charlier, A., Rotsaert, P., Bulterys, S., Moens, G., Sprundel, M. V. and Schryver, A. D. 2010, *Occup. Environ. Med.*, 67, 97.
25. Foad, M. F. and Awadalla, N. J. 2011, *Egypt. J. Occup. Med.*, 35(1), 81.
26. De Schryver, A., Bulterys, S. and Rotsaert, P. 2003, *International Congress on Occupational Health (ICOH)*, Iguassu Falls, Brazil.
27. Omosor, K. L., Omosor, O. H., Adejumo, B. L. G., Ibeh, I. N. and Dimkpa, U. 2018, *J. Mol. Microbiol.*, 2, 3.
28. Fox, J. G. and Megraud, F. 2009, *Manual of Clinical Microbiology*. 9th ed., 1, 962.
29. Yilmaz, O., Şen, N., Küpelioğlu, A. A. and Şimşek, I. 2006, *World J. Gastroenterol*, 12(33), 5378.
30. Gosciniak, G. 1997, *Zbl. Bakt.*, 286, 502.
31. Miftahussurur, M. and Yamaoka, Y. 2016, *Biomed. Research. Int.*, 4819423
32. Rashed, M. E., Amer, M. M., Elnakeeb, M. and Omer, W. S. 2016, *Int. J. Appl. Sci. Biotechnol.*, 4(3), 358.
33. El Dine, S. S., Mubarak, M., Salama, R., El Raziky, M. and El Sherbiny, E. 2008, *Res. J. Med. Med. Sci.*, 2, 123.
34. Reynders, M. B., Deyi, V. Y., Dahma, H., Scheper, T., Hanke, M., Decolvenaer, M., and Dediste, A. 2012, *FEMS Immunol. Med. Microbiol.*, 64, 363.
35. She, R. C., Wilson, A. R. and Litwin, C. M. 2009, *Clin. and Vac. Immun.*, 16(8), 1255.