ORIGINAL ARTICLE



# Unique features and risk factors of *Helicobacter pylori* infection at the main children's intermediate school in Rabigh, Saudi Arabia

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#### Abstract

*Background* This study was conducted to determine characters and risk factors of *Helicobacter pylori* infection and its relationship with recurrent abdominal pain and other gastrointestinal symptoms at the main children's intermediate school in Rabigh, Saudi Arabia.

*Methods* A cross-sectional study was conducted at a boys' intermediate school. A questionnaire for the gastrointestinal (GI) symptoms and relevant personal and socioeconomic risk factors related to *H. pylori* infection was distributed followed by *H. pylori* IgG antibody assay and 14C urea breath test to detect active infection.

*Results H. pylori* was diagnosed by positive urea breath test in 51.5 % of students. *H. pylori* infection was symptomatic with

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Public Health and Community Medicine, Faculty of Medicine, King Abdulaziz University, Jeddah, Saudi Arabia at least one upper GI symptom in 89.7 % of infected students which was higher than symptomatic cases reported in any other study. *H. pylori*-infected students had significantly more association with the presence of any upper GI symptom (p<0.001), recurrent abdominal pain (p<0.001), anorexia (p<0.001), nausea (p<0.026), family history of peptic disease (p<0.001), drinking desalinated municipal water (p<0.001), lower income (p=0.02), and eating outside home (p=0.003) than uninfected students. Logistic regression analysis showed that the most significant predictors of *H. pylori* infection were presence of any upper GI symptom (OR 5.3, 95 % CI 2.32– 15.71), family history of peptic disease (OR 2.2, 95 % CI 1.11–3.9), and drinking desalinated municipal water (OR 2.1, 95 % CI 1.09–3.2).

*Conclusions* This study presented unique features and risk factors of *H. pylori* infection in 12–15-year-old Saudi boys in Rabigh, and mainly supported the role of *H. pylori* in causing recurrent abdominal pain.

Keywords Children  $\cdot$  *Helicobacter pylori*  $\cdot$  Recurrent abdominal pain  $\cdot$  Saudi  $\cdot$  Urea breath test

## Introduction

*Helicobacter pylori* infection is the most common bacterial infection in humans, and it has a worldwide prevalence rate of about 50 %, with a higher prevalence in developing countries [1, 2].

*H. pylori* infection is mainly acquired during early childhood [3, 4] and causes various gastrointestinal (GI) and extragastrointestinal diseases [5]. It is recognized as a cause of gastritis and peptic ulcer disease (PUD) [6]. Chronic colonization with *H. pylori* can predispose children to significantly increased risk of gastric adenocarcinoma [7] and mucosa-associated lymphoid tissue gastric lymphoma [2]. It has been classified as a group I carcinogen by the World Health Organization [2, 8] especially in those who have harbored the organism for at least 10 years [9]. Elimination of *H. pylori* changes the natural history of PUD [10] and gastric lymphoma [11]. Thus, screening and eradication of *H. pylori* are beneficial [12].

Several risk factors have been associated with *H. pylori* infection and correlate with low socioeconomic status [13]. The role of *H. pylori* in causing specific symptoms in children such as recurrent abdominal pain (RAP) is controversial [14]. Symptoms suggestive of acute *H. pylori* infection are vague and similar to several childhood disorders [15]. However, *H. pylori* infection has been found in 60.3 % of the children with RAP who benefited from eradication therapy [16].

This infection can be diagnosed by invasive techniques requiring endoscopy and biopsy (histological examination, rapid urease test [RUT], culture) and by noninvasive tests (serology, urea breath test [UBT], *H. pylori* antigen stool specimen) [8].

Compared to noninvasive diagnostic modes, however, invasive techniques are inconvenient for patients and also have higher cost [17]. The UBT remains the best test to diagnose *H. pylori* infection [18].

Two carbon isotopes (<sup>13</sup>C and <sup>14</sup>C) are used for the UBT. Although the <sup>14</sup>C isotope is radioactive, microdose (1  $\mu$ Ci) <sup>14</sup>C has the minimal radiation of 1-day background exposure [19].

It has been shown that the *H. pylori* infection rate is very high in Saudi population. In 1990, Al Moagel et al. [20] reported that 40 % of the Saudi population in the age group of 5–10 years and 70 % of people >20 years of age had *H. pylori*, which makes it one of the highest endemic areas in the world. Also, *H. pylori* infection was found in 67 % to 87 % of Saudi children with peptic disease [21].

Another urban-based study showed a high seroprevalence of *H. pylori* infection (67 %), increasing with age in a large series of outpatients, aged 2 to 82 years, suffering from GI symptoms attending large hospitals in Riyadh, Saudi Arabia [22].

The prevalence of *H. pylori* by positive UBT among an intermediate-school children in Makkah, Saudi Arabia, was 45/103 (43.7 %), and 62.9 % of these students had RAP [23]. This exploratory study was conducted to determine the characters, risk factors, and seroprevalence of *H. pylori* infection in 12–15-year-old students at the main children's intermediate school in Rabigh and to investigate the correlation between *H. pylori* infection and RAP as well as other gastrointestinal tract (GIT) symptoms.

# Methods

## Patient selection

This study was carried out at the main boys' intermediate school (12–15 years) in Rabigh city, western Saudi Arabia. Students at this age are usually reported to have high prevalence of *H. pylori* infection because *H. pylori* infection is usually acquired in early childhood [3, 4, 23]. This study was approved by the King Abdulaziz University Research Ethics Committee, and a written informed consent was obtained from fathers of all students. A questionnaire for the GI symptoms and risk factors related to *H. pylori* infection was distributed to all boys who agreed to participate in this study (included 95% of students at this intermediate school) followed by *H. pylori*-specific IgG levels assay and <sup>14</sup>C UBT testing to detect active infection.

# Exclusion criteria

Any student who received antimicrobials, antacids,  $H_2$  receptor antagonists, proton pump inhibitors, bismuth, or sucralfate within 4 weeks prior to date of entry to the study was excluded.

## Questionnaire

The questionnaire was designed to evaluate GIT symptoms and risk factors related to *H. pylori* infection. We gathered questions tested in precedent research [13, 24] and selected those most related to our study. The questions were translated from English to Arabic. We interviewed children to identify the most problematic questions to change them in the final questionnaire. An interviewer was also present to provide any needed explanation for every student while filling the questionnaire to guarantee accurate data collection. The questionnaire inquired about:

1. GIT symptoms: mainly upper GIT symptoms used as diagnostic predictors of *H. pylori* infection and disease especially recurrent abdominal pain (RAP), anorexia, nausea, vomiting, medical consultation for GIT problems, and family history of peptic disease. Children with RAP were identified according to the Apley criteria (at least three episodes of abdominal pain severe enough to affect activity over a period of at least 3 months) [23].

2. Transmission risk factors: The questions focused on the type of drinking water (desalinated or mineral water), house water supply (desalinated or public system water), personal hygiene (washing hands before eating, use of personal tableware, and washing hands after using the toilet), and education and socioeconomic levels of the family of recruited students. Subjects were also asked to provide information on domestic crowding (number of family members, number of rooms in the house, and number of siblings per room).

## Measurement of serum H. pylori IgG

The VIDAS system using the enzyme-linked fluorescent assay technique was used to determine serum IgG specific for *H. pylori* (HPY-VIDAS, bioMerieux, Marcy l'Etoile, France). The test value was interpreted as follows: negative <0.75, equivocal  $\ge 0.75$  to <1.00, and positive  $\ge 1.00$  [25].

#### Urea breath test

The <sup>14</sup>C UBT which was used only once before in Saudi children (23) was considered as the gold standard for confirming diagnosis of *H. pylori* infection in this study.

Following overnight fasting, the boys swallowed 37 kBq (1 µCi) of encapsulated <sup>14</sup>C urea (Helicap, Institute of Isotopes, Budapest, Hungary) with 25 mL of water. After 15 min, the patient breathed out into a dry cartridge (Heliprobe breath cards, Kibion AB, Uppsala, Sweden) through its mouthpiece until the color of the card indicator changed from orange to yellow, which took about 1 to 2 min. The breath samples were measured using the Heliprobe Analyzer, and the radioactivity was read after 250 s of an automated process. Finally, the test results were expressed on the analyzer in a numeric fashion (0 patient not infected, 1 borderline result, 2 patient infected), which corresponded to radioactivity as count per minute (CPM): <25 CPM patient not infected, 25-50 CPM borderline result, >50 CPM patient infected. We considered grades 0 and 1 as negative results in our study, and only samples with activities that were more than 50 CPM were regarded as positive [26].

#### Statistical analysis

The qualitative data were presented in the form of numbers and percentages. Chi-square was used to compare between qualitative data of two groups. Yates correction was used when appropriate. Odds ratios (ORs) and 95 % confidence intervals (CIs) were calculated to estimate the risk. Significance was considered when *p*-value was less than 0.05. The most significant predictors by univariate analysis were chosen to perform multiple forward conditional logistic regressions for adjustment of the risk and estimation of the overall predictability of the significant predictors of *H. pylori* infection in this model. Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy for positive specific *H. pylori* IgG serology compared to the gold standard <sup>14</sup>C UBT were calculated. Pearson correlation was used to study the relation between number of GIT symptoms and *H. pylori* IgG antibody titer.

## Results

A total of 132 intermediate-school boys between 12 and 15 years, with a mean age of  $14.3\pm1.4$  years were enrolled. The prevalence of active (not past) *H. pylori* infection in the recruited students by positive 14C UBT was 68/132 (51.5).

Forty-three students (32.6 %) were completely asymptomatic, and the other 89 students (67.4 %) had at least one GIT symptom. *H. pylori* infection was symptomatic with at least one upper GIT symptom in 89.7 % of infected students. *H. pylori* infection was asymptomatic in 10.3 % of infected students.

The seroprevalence of *H. pylori* IgG antibodies among all students and among actively infected students was 70/132 (53 %) and 59/68 (86.8 %), respectively. The sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy for specific *H. pylori* IgG antibodies in relation to the gold standard <sup>14</sup>C UBT were 86.8 %, 82.8 %, 84.3 %, 85.5 % and 84.8 %, respectively.

*H. pylori*-infected students had significantly more presence of any upper GIT symptom (OR 11.2, CI 4.12–31.67, p<0.001), RAP (OR 4.13, CI 1.87–9.21, p<0.001), anorexia (OR 3.72, CI 1.68–8.31, p<0.001), nausea (OR 2.68, CI 1.13–6.43, p=0.026), and family history of peptic disease (OR 11.2, CI 4.12–31.67, p<0.001) than uninfected students. Table 1 presents the comparison of clinical data between children with and without *H. pylori* infection.

Detailed analysis of upper GIT symptoms could reveal that the most frequent upper GIT symptoms in *H. pylori*-infected students were RAP followed by anorexia and then nausea whether isolated or combined with other upper GIT symptoms and were more common than in uninfected students. Out of 42 *H. pylori*-infected students with RAP, 8 (19 %) had isolated RAP, 23 (54.8 %) had RAP combined with other upper GIT symptoms mainly anorexia and nausea, and 11 (26.2 %) had RAP associated with other lower GIT symptoms. Table 2 shows the frequency of upper GIT symptoms in children with and without *H. pylori* infection.

Risk factors for *H. pylori* infection as parental education level, personal hygienic measures, and domestic crowding in the families of recruited students were not significantly different between infected and uninfected students. Drinking desalinated municipal water, eating outside home, and low income were significantly more prevalent among *H. pylori*-infected students than uninfected students. Table 3 shows the

Variable	<i>H. pylori</i> -infected students <sup>a</sup> ( $n=68$ )		H. pylor students	<i>i</i> -uninfected ( <i>n</i> =64)	ORs for positive <i>H. pylori</i> (95 % CIs)	<i>p</i> -value <sup>b</sup>
	n	%	n	%		
Recurrent abdominal epigastric pain	42	61.8	18	28.1	4.13 (1.87–9.21)	< 0.001
Anorexia	39	57.4	17	26.6	3.72 (1.68-8.31)	< 0.001
Nausea	26	38.2	12	18.8	2.68 (1.13-6.43)	0.026
Vomiting	15	22.1	7	10.9	2.3 (0.8-6.83)	0.13
Flatulence	7	10.3	6	9.4	1.1 (0.31-4.01)	0.90
Diarrhea	13	19.1	11	17.2	1.14 (0.43-3.02)	0.95
Presence of any upper GIT symptom	61	89.7	28	43.8	11.2 (4.12–31.67)	< 0.001
Medical consultation for upper GIT symptoms	17	25	11	17.2	1.61 (0.64-4.1)	0.32
Family history of peptic disease	25	36.8	5	7.8	6.86 (2.25–22.36)	< 0.001

GIT gastrointestinal tract

<sup>a</sup> Active infection of *H. pylori* was considered according to positive urea breath test (the golden standard test)

<sup>b</sup> Chi-square test

comparison of risk factors between children with and without *H. pylori* infection.

Correlation studies found insignificant positive correlation between number of symptoms and *H. pylori* IgG antibody titer in *H. pylori*-infected students (r=0.111, p=0.204). predictability of this model for *H. pylori* infection was 76.7 %, Table 4.

# Discussion

Logistic regression analysis showed that the most significant predictors of *H. pylori* infection were the presence of any upper GIT symptom, positive family history of peptic disease, and drinking desalinated municipal water. The overall The prevalence of *H. pylori* infection in 51.5 % in this study was closely similar to *H. pylori* prevalence of 54.7 % reported among school students of the same age in Taiwan [27], but higher than *H. pylori* prevalence in many parts of the world

Table 2	Frequency of upper
gastroint	estinal tract symptoms
in childre	en with and without
H. pylor	<i>i</i> infection

*GIT* gastrointestinal tract, *RAP* recurrent abdominal epigastric pain

<sup>a</sup> Active infection of *H. pylori* was considered according to positive urea breath test (the golden standard test)

Variable	H. pylori students <sup>a</sup>		<i>H. pylori</i> -uninfected students $(n=64)$	
	п	%	п	%
One upper GIT symptom	17	25	11	17.2
Isolated RAP	8	11.8	5	7.8
Isolated anorexia	5	7.4	3	4.7
Isolated nausea	4	4.7 0	0 0	0 0
Isolated vomiting	0			
Two upper GIT symptoms	14	20.6	3	4.7
RAP and anorexia	6	8.8	1	1.6
RAP and nausea	2	2.9	0	0
RAP and vomiting	1	14.7	0	0
Anorexia and nausea	3	4.4	1	1.6
Nausea and vomiting	2	2.9	1	1.6
Three upper GIT symptoms	13	16.2	4	6.3
RAP, anorexia, and nausea	6	8.8	2	3.1
RAP, anorexia, and vomiting	4	4.7	2	3.1
Anorexia, nausea, and vomiting	3	4.4	0	0
Four upper GIT symptoms				
RAP, anorexia, nausea, and vomiting	4	4.7	2	3.1

Table 3	Comparison	of risk factors	s between children	with and	l without <i>H. pyl</i>	ori infection
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Risk factor	<i>H. pylori</i> infected students <sup>a</sup> ( $n=68$ )		<i>H. pylori</i> uninfected students $(n=64)$		ORs for <i>H. pylori</i> infection (95 % CIs)	p-value <sup>b</sup>
	n	%	n	%		
Mother level of education						
Less than secondary school education	41	60.3	39	61	0.97 (0.46-2.08)	0.91
Father level of education						
Less than secondary school education	33	48.5	32	50	0.94 (0.45-1.98)	0.99
Income less than 3,000 Saudi $RS = US$ \$800	30	44.1	15	23.4	2.58 (1.14-5.87)	0.02
Eating outside home	37	54.4	18	28.1	3.05 (1.39-6.73)	0.003
Drinking desalinated municipal water	48	70.6	21	32.8	4.91 (2.21–11.05)	< 0.001
Residential public water supply use for non-drinking purposes	12	17.6	4	6.3	3.21 (0.89–12.64)	0.08
No use of personal tableware	43	63.2	35	54.7	1.43 (0.67–3.09)	0.41
No hand washing before eating	6	8.8	7	10.9	0.79 (0.22-2.81)	0.63
No hand washing after toilet	5	7.4	3	4.7	1.61 (0.32-8.9)	0.71
Family members ≥4	64	94.1	60	93.8	1.07 (0.21–5.37)	0.60
Number of rooms $\leq 3$	10	14.7	5	7.8	2.03 (0.59-7.35)	0.32
Number of persons/room $\geq 2$	28	41.2	17	26.6	1.94 (0.87-4.32)	0.11

<sup>a</sup> Active infection of *H. pylori* was considered according to positive urea breath test (the golden standard test)

<sup>b</sup> Chi-square test

where several studies have shown that the prevalence of *H. pylori* infection is decreasing to about 30 % in adults and children especially in developed countries [18, 28, 29]. In this study, the prevalence of *H. pylori* infection seems to be decreasing if compared to the previously reported higher prevalence in Saudi children with peptic disease [22, 30].

In more recent studies, the prevalence of *H. pylori* infection by positive UBT among the intermediate-school children in Makkah, Saudi Arabia, was 45/103 (43.7 %) [23], and *H. pylori* infection was documented in more than 40 % of both symptomatic and asymptomatic children at Jeddah Clinic Hospital, Saudi Arabia, from January 2009 to December 2010 [31]. The prevalence of *H. pylori* in our study was relatively higher than *H. pylori* prevalence detected in the previously mentioned two studies carried out on children of approximately the same age as our students.

In addition, the seroprevalence of *H. pylori* IgG antibodies among our students (53 %) was lower than the 67 % seroprevalence recorded in an urban-based study among a large series of patients, aged 2 to 82 years suffering from gastrointestinal symptoms attending large hospital in Riyadh, Saudi Arabia [22]. However, the recorded seroprevalence in our study was higher than the prevalence in a group of randomly selected 1,200 adolescents, 16-18-year-old students from three regions around Saudi Arabia which was found to be 47 % [32].

In the current study, *H. pylori* infection was symptomatic with at least one upper GIT symptom in 89.7 % of infected students, which was higher than the percentage of symptomatic *H. pylori* cases recorded in other studies to range from 40 % to 53 % (24, 27).

*H. pylori* infection was significantly associated with higher frequency of upper GIT symptoms (RAP, anorexia, nausea) and family history of peptic disease (Table 1). Similarly, other studies showed that symptoms of *H. pylori*-related peptic ulcer disease were nonspecific in children and may include epigastric pain, nausea and/or vomiting, and anorexia [2]. In another recent study of school children between the age of 6 and 15 years from Sardinia, Italy, nausea/vomiting (OR=2.2, 95 % CI=1.2–5.1) was significantly associated with *H. pylori* infection [33].

In this study, detailed analysis of upper GIT symptoms could reveal that the most frequent upper GIT symptoms in *H. pylori*-infected students were RAP (found in 61.8 % of

**Table 4**Multiple logistic regression analysis for prediction of*H. pylori* infection

Variable	Adjusted ORs, 95 % CIs	<i>p</i> -value
Presence of any upper gastrointestinal tract symptom	5.3 (2.32–15.71)	< 0.001
Family history of peptic disease	2.2 (1.11–3.9)	< 0.001
Drinking desalinated municipal water	2.1 (1.09–3.2)	0.02

infected students) followed by anorexia and then nausea whether they were isolated or combined with other upper GIT symptoms and which were more common than in uninfected students. These results were emphasized when multiple logistic regression analysis (Table 4) showed that the most significant independent predictor or association with H. pylori infection was the presence of any upper GIT symptom (OR 5.3, 95 % CI 2.32–15.71, and p<0.001). These results were also consistent with other studies [16, 30, 31] which found that H. pylori infection was more common in patients suffering from epigastric pain. Similarly, Telmesani [23] reported that 62.9 % of intermediate- and secondary-school children in Makkah, Saudi Arabia, had RAP. The odd ratio of RAP were 10.40 (95 % CI 1.75-11.73) for the intermediate-school students. Thus, there was a significant relation between H. pylori infection and RAP among intermediate-school students of the same age as our studied students.

Although it was observed that children from families with a higher crowding index had a higher risk of being colonized with *H. pylori* [24], family members greater than or equal to four and number of persons/room greater than or equal to two were only associated with small risk of *H. pylori* infection in our study.

Eating outside home commonly seen in adolescents in Saudi Arabia as well as in other countries was significantly associated with risk of *H. pylori* infection (OR 3.05, CI 1.39–6.73, p=0.003) in our students consistent with other studies which showed that the transmission of *H. pylori* also takes place through the consumption of food prepared under unhygienic conditions [24]. Moreover, low income was found to be significantly associated with increased risk of *H. pylori* infection (OR 2.58, CI 1.14–5.87, p=0.02) which was demonstrated in other studies where *H. pylori* infection was correlated with low socioeconomic status [1, 13].

The findings of significantly higher frequency of peptic disease in the families of *H. pylori*-infected students were also evident in other studies [34]. In addition, prevalence of H. pylori infection was higher in family members of infected patients. A common source of infection could not be excluded, but facts suggest that person-to-person transmission occurs, especially from mother to child [35]. In Saudi Arabia, it was demonstrated that intrafamilial clustering of H. pylori infection occurred in a similar pattern to that described in the developed countries and living conditions and social conditions can lead to person to person transmission of H. pylori infection [36]. Considering the known possibility of intrafamilial clustering of H. pylori infection, the high prevalence detected in our studied students and increasing prevalence of *H. pylori* infection with advancing of age, the general overall prevalence of H. pylori infection may be expected to be also high in our locality.

Significantly higher risk of *H. pylori* infection was associated with drinking desalinated municipal water. In Rabigh, desalinated water is sea water which is transported in vehicles

from stations to houses. It is poured in underground tanks then elevated by electrical water pumps to tanks on the roof to be let down to the tubes in the house. The significant association between the use of desalinated water and *H. pylori* infection revealed in the present study may be explained by the lack of regular cleaning of the underground water tanks in most houses due to technical difficulties. Similarly, in another study, the consumption of municipal tap water rather than boiled or filtered water had a high impact in the transmission of *H. pylori* [37].

In the current study, the sensitivity of *H. pylori* IgG of 86.8 % was slightly lower than that reported in other studies [38]. Although serologic assays using validated IgG antibodies detection may be helpful for screening children for presence of *H. pylori* in areas of high prevalence, they do not predict active infection or assess the success of antimicrobial eradication therapy [2]. Our recorded relatively low sensitivity of *H. pylori* IgG necessitate the use of the more sensitive and specific UBT for better detection of *H. pylori* active infection in our locality. *H. pylori* infection may occur frequently but rarely produce detectable antibodies especially in children. For clinical or epidemiological investigations, serology should not be used as the sole method for detecting *H. pylori* infection [39].

In addition, the relatively low sensitivity of *H. pylori* IgG assay in this study may be related to more frequent upper GIT symptoms especially RAP in most of our studied students indicating active and not past *H. pylori* infection, so IgG antibodies may have not been formed yet in few (13.2 %) of them. Also, this can explain the finding of insignificant positive correlation between number of GIT symptoms and *H. pylori* IgG antibody titer in actively infected students in the current study.

In conclusion, this study revealed that in our locality, the prevalence of *H. pylori* infection in intermediate-school boys was relatively still high particularly if compared to the trend of decreasing *H. pylori* infection in developed countries. Considering the known possibility of intrafamilial clustering of *H. pylori* infection, the high prevalence detected in our students, and the increasing prevalence of *H. pylori* infection with advancing of age, the general overall prevalence of *H. pylori* infection may be expected to be also high enough to be responsible for an important public health problem in our locality.

This study presented important unique features and risk factors of *H. pylori* infection in 12–15-year-old boys, which can guide future required studies in our locality as the percentage of symptomatic infected students with at least one upper GIT symptom was higher than symptomatic cases reported in any other study and infected students had significantly higher RAP, anorexia, and nausea than uninfected students. This study supported the role of *H. pylori* infection in causing RAP in contrast to some other studies which found

a controversial role for *H. pylori* and RAP. The most significant predictors of *H. pylori* infection were the presence of any upper GIT symptom, positive family history of peptic disease, and drinking desalinated municipal water with an overall predictability of this model for *H. pylori* infection approaching 77 %.

This study emphasized the role of using the more sensitive and specific <sup>14</sup>C UBT in diagnosis of *H. pylori* infection in a locality with relatively low sensitivity of *H. pylori* IgG serology, and this is important in clinical practice to diagnose active *H. pylori* infection in the absence of positive IgG serology.

*H. pylori* infection can be a re-emerging serious infection in our locality as well as in any other area of the world with the same risk factors, so we must be ready to tackle it with effective and more powerful preventive measures.

It is recognized that this study had some limitations as it was a cross-sectional and not randomly sampled study, it was carried out on boys because schools in Saudi Arabia are either for boys or girls, and we did not have a female team work.

Large random epidemiological community-based studies for *H. pylori* infection among the different age groups in our locality may provide further insights.

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**Conflict of interest** HSH, MAH, HAM, EMA, TFH, and BSE all declare that they have no conflict of interest.

**Ethics statement** The study was performed in a manner to conform to the Helsinki Declaration of 1975, as revised in 2000 and 2008 concerning Human and Animal Rights, and the authors followed the policy concerning informed consent as shown on Springer.com.

#### References

- Blanchard SS, Czinn SJ. Peptic ulcer disease in children. *In:* Kliegman RM, Stanton BF, St. Geme JW, Schor NF, Behrman RE, (eds). Nelson Text Book of Pediatrics. 19th ed. Philadelphia: Elsevier Saunders; 2011. pp. 4638–44.
- Ertem D. Clinical practice: *Helicobacter pylori* infection in childhood. Eur J Pediatr. 2013;172:1427–34.
- Malaty HM, El-Kasabany A, Graham DY, et al. Age at acquisition of *Helicobacter pylori* infection: a follow-up study from infancy to adulthood. Lancet. 2002;359:931–5.
- Perez-Perez GI, Rothenbacher D, Brenner H. Epidemiology of *Helicobacter pylori* infection. Helicobacter. 2004;9:1–6.
- Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of *Helicobacter* pylori infection. Clin Microbiol Rev. 2006;19:449–90.
- Paptheodoridis GV, Sougioultzis S, Archimandritis AJ. Effects of Helicobacter pylori and nonsteroidal anti-inflammatory drugs on

peptic ulcer disease: a systematic review. Clin Gastroenterol Hepatol. 2006;4:130-42.

- Nomura AM, Pérez-Pérez GI, Lee J, Stemmermann G, Blaser MJ. Relation between *Helicobacter pylori* cagA status and risk of peptic ulcer disease. Am J Epidemiol. 2002;155:1054–9.
- Sabbi T. Short review about *Helicobacter pylori* infection in pediatric age: epidemiological and clinical findings, diagnosis, therapy and role of probiotics. Pediatr Med Chir. 2011;33:221–6.
- Mera R, Fontham ET, Bravo LE, et al. Long term follow-up of patients treated for *Helicobacter pylori* infection. Gut. 2005;54: 1536–40.
- Leodolter A, Kulig M, Brasch H, Meyer-Sabellek W, Willich SN, Malfertheiner P. A meta-analysis comparing eradication, healing and relapse rates in patients with *Helicobacter pylori*-associated gastric or duodenal ulcer. Aliment Pharmacol Ther. 2001;15:1949–58.
- Montalban C, Norman F. Treatment of gastric mucosa-associated lymphoid tissue lymphoma: *Helicobacter pylori* eradication and beyond. Expert Rev Anticancer Ther. 2006;6:361–71.
- Pakodi F, Abdel-Salam OM, Debreceni A, Mózsik G. *Helicobacter pylori*: one bacterium and a broad spectrum of human disease! An overview. J Physiol Paris. 2000;94:139–52.
- Yucel T, Aygin D, Sen S, Yucel O. The prevalence of *Helicobacter* pylori and related factors among university students in Turkey. Jpn J Infect Dis. 2008;61:179–83.
- Crone J, Gold BD. *Helicobacter pylori* infection in pediatrics. Helicobacter. 2004;9:49–56.
- Czinn SJ. *Helicobacter pylori* infection: detection, investigation, and management. J Pediatr. 2005;146:S21–6.
- Ozen H, Dinler G, Akyön Y, Koçak N, Yüce A, Gürakan F. *Helicobacter pylori* infection and recurrent abdominal pain in Turkish children. Helicobacter. 2001;6:234–8.
- Vakil N, Rhew D, Soll A, Ofman JJ. The cost effectiveness of diagnostic testing strategies for *Helicobacter pylori*. Am J Gastroenterol. 2000;95:1691–8.
- Tonkic A, Tonkic M, Lehours P, Mégraud F. Epidemiology and diagnosis of *Helicobacter pylori* infection. Helicobacter. 2012;17: 1–8.
- Leide-Svegborn S, Stenström K, Olofsson M, et al. Biokinetics and radiation doses for carbon-14 urea in adults and children undergoing the *Helicobacter pylori* breath test. Eur J Nucl Med. 1999;26:573– 80.
- al-Moagel MA, Evans DG, Abdulghani ME, et al. Prevalence of *Helicobacter pylori* (formerly Campylobacter) infection in Saudi Arabia, and comparison of those with and without upper gastrointestinal symptoms. Am J Gastroenterol. 1990;85:944–8.
- El Mouazan MI, Abdullah AM. Peptic ulcer disease in children and adolescents. J Trop Pediatr. 2004;50:328–30.
- Marie MA. Seroprevalence of *Helicobacter pylori* infection in large series of patients in an urban area of Saudi Arabia. Korean J Gastroenterol. 2008;52:226–9.
- Telmesani AM. *Helicobacter pylori*: prevalence and relationship with abdominal pain in school children in Makkah City, western Saudi Arabia. Saudi J Gastroenterol. 2009;15:100–3.
- Goldman C, Barrado A, Janjetic M, et al. Factors associated with H. pylori epidemiology in symptomatic children in Buenos Aires, Argentina. World J Gastroenterol. 2006;12:5384–8.
- Zhubi B, Baruti-Gafurri Z, Mekaj Y, et al. Helicobacter pylori infection according to ABO blood group among blood donors in Kosovo. J Health Sci. 2011;1:83–9.
- Mansour-Ghanaei F, Sanaei O, Joukar F. Clinical validation of an office-based C-UBT (Heliprobe) for H. pylori diagnosis in Iranian dyspeptic patients. Gastroenterol Res Pract. 2011;2011:930941.
- Chi H, Bair MJ, Wu MS, Chiu NC, Hsiao YC, Chang KY. Prevalence of *Helicobacter pylori* infection in high-school students on Lanyu Island, Taiwan: risk factor analysis and effect on growth. J Formos Med Assoc. 2009;108:929–36.

- Elitsur Y, Dementieva Y, Rewalt M, Lawrence Z. *Helicobacter pylori* infection rate decreases in symptomatic children: a retrospective analysis of 13 years (1993-2005) from a gastroenterology clinic in West Virginia. J Clin Gastroenterol. 2009;43:147–51.
- 29. Llanes R, Millán LM, Escobar MP, et al. Low prevalence of *Helicobacter pylori* among symptomatic children from a hospital in Havana, Cuba. J Trop Pediatr. 2012;58:231–4.
- El-Mouzan MI, Abdullah AM, Al-Mofleh IA. Gastritis in Saudi Arab children. Saudi Med J. 2005;26:576–9.
- Mansour MM, Al Hadidi KM, Omar MA. *Helicobacter pylori* and recurrent abdominal pain in children: is there any relation? Trop Gastroenterol. 2012;33:55–61.
- 32. Al Faleh FZ, Ali S, Aljebreen AM, et al. Seroprevalence rates of *Helicobacter pylori* and viral hepatitis A among adolescents in three regions of the Kingdom of Saudi Arabia: is there any correlation? Helicobacter. 2010;15:532–7.
- Dore MP, Fanciulli G, Tomasi PA, et al. Gastrointestinal symptoms and *Helicobacter pylori* infection in school-age children residing in Porto Torres, Sardinia, Italy. Helicobacter. 2012;17:369–73.

- Nijevitch AA, Shcherbakov PL. *Helicobacter pylori* and gastrointestinal symptoms in school children in Russia. J Gastroenterol Hepatol. 2004;19:490–6.
- Escobar ML, Kawakami E. Evidence of mother-child transmission of Helicobacter pylori infection. Arq Gastroenterol. 2004;41:239–44.
- Al-Knawy BA, Ahmed ME, Mirdad S, ElMekki A, Al-Ammari O. Intrafamilial clustering of *Helicobacter pylori* infection in Saudi Arabia. Can J Gastroenterol. 2000;14:772–4.
- Ahmed KS, Khan AA, Ahmed I, et al. Prevalence study to elucidate the transmission pathways of *Helicobacter pylori* at oral and gastroduodenal sites of a South Indian population. Singapore Med J. 2006;47:291–6.
- Logan RP, Walker MM. ABC of the upper gastrointestinal tract: epidemiology and diagnosis of *Helicobacter pylori* infection. BMJ. 2001;323:920–2.
- Nurgalieva Z, Goodman KJ, Phillips CV, Fischbach L, de la Rosa JM, Gold BD. Correspondence between *Helicobacter pylori* antibodies and urea breath test results in a US-Mexico birth cohort. Paediatr Perinat Epidemiol. 2008;22:302–12.