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### Prevalence of *Helicobacter pylori* infections among patients referred for endoscopy at Hospital Sultan Abdul Halim

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#### ABSTRACT

**Objective:** To determine the prevalence of *Helicobacter pylori* (*H. pylori*) infection according to age, gender, ethnicity, and endoscopic finding and to study the association between smoking and alcohol consumption with *H. pylori* infection.

**Methods:** A retrospective observational study on 582 consecutive patients referred for endoscopy with *H. pylori* tested was conducted in Hospital Sultan Abdul Halim from 1st of January 2013 till 31st of December 2013. Data were analyzed using Pearson's *Chi*-square, Fisher's exact test and binary logistic regression with SPSS version 16.

**Results:** Out of the 582 patients, 74 (12.7%) were positive for *H. pylori* infection. Among those with *H. pylori* infection, 42 (56.8%) were female. Infection was highest in the age group of 51 to 60 years old, 25 (33.8%) with the mean (SD) age of 52.9 (14.9) years. From the endoscopic finding, erosions, 38 (51.4%) had the highest *H. pylori* positive cases. *H. pylori* infection was commonly found among Indians (36.3%; 41/113) followed by Chinese (17.6%, 18/102) and Malays (4.1%; 15/367) ( $P < 0.001$ ). There is no significant association between smoking and alcohol consumption with *H. pylori* infection ( $P > 0.05$ ).

**Conclusions:** The increased risk of *H. pylori* infection in Indians might be due to the unusual socio-cultural practices and difference in diet which may be responsible for the transmission of the infection. Therefore, further studies are warranted.

## 1. Introduction

*Helicobacter pylori* (*H. pylori*) is a Gram-negative bacillus that is found between the mucus layer and the mucosa of the stomach and is considered as a contributory agent for peptic ulcer disease, gastric lymphoma and gastric carcinoma[1,2]. It is the commonest chronic infections worldwide[1]. *Campylobacter*-like organism (CLO) test is a rapid urease test which is used to detect *H. pylori*[1]. CLO test measures the urease activity of *H. pylori* in the gastric mucosa[1]. It is used in conjunction with oesophago-gastric-duodeno-scopy whereby a biopsy of mucosa is taken from the stomach and placed on the kit. Lately, there has been a rise in *H. pylori* cases in Hospital Sultan Abdul Halim (HSAH). Thus, our objectives are to determine the prevalence of *H. pylori* infection according to age, ethnicity, gender and endoscopic findings and to study the association

between smoking and alcohol consumption with *H. pylori* infection.

## 2. Materials and methods

A retrospective observational study on 582 consecutive patients referred for endoscopy with *H. pylori* tested was conducted in HSAH, Sungai Petani, Kedah from 1st of January 2013 till 31st of December 2013. Those who had completed previous eradication therapy were excluded. We assessed database of the patients from a registry book in endoscopic unit and medical records. *H. pylori* infection was tested using Kimberly-Clark\* CLOtest\* Rapid Urease Test. All endoscopies were performed by experienced endoscopists using the Olympus GIF-HQ 180 video endoscope. Data were analyzed using SPSS version 16. Pearson's *Chi*-square and Fisher's exact test were used to determine the significant association between individual categorical independent factor and the outcome. Binary logistic regression was used to determine odds ratio. The protocol of this study was registered with the National Medical Research Register and approved by the Medical Research Ethics Committee, Malaysia.

## 3. Results

Out of the 582 patients, 74 (12.7%) were positive for *H. pylori*

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infection. Infection was highest in the age group of 51 to 60 years old, 25 (33.8%) (Table 1) with the mean (SD) age of 52.9 (14.9) years. Among those with *H. pylori* infection, 42 (56.8%) were female (Table 2). From the endoscopic finding, erosions, 38 (51.4%) had the highest *H. pylori* positive cases (Table 2). *H. pylori* infection was commonly found among Indians (36.3%; 41/113) followed by Chinese (17.6%, 18/102) and Malays (4.1%; 15/367) ( $P < 0.001$ ) (Table 3). There is no significant association between smoking and alcohol consumption with *H. pylori* infection ( $P > 0.05$ ) (Table 4).

**Table 1**

*H. pylori* infection rates according to age ( $n = 582$ ).  $n$  (%).

Age group (year)	<i>H. pylori</i> positive	<i>H. pylori</i> negative	Total
11–20	2 (0.4)	13 (2.2)	15 (2.6)
21–30	3 (0.5)	37 (6.4)	40 (6.9)
31–40	11 (1.9)	49 (8.4)	60 (10.3)
41–50	11 (1.9)	80 (13.7)	91 (15.6)
51–60	25 (4.3)	117 (20.1)	142 (24.4)
61–70	12 (2.1)	119 (20.4)	131 (22.5)
71–100	10 (1.7)	93 (16.0)	103 (17.7)

**Table 2**

*H. pylori* infection rates according to gender and endoscopic findings ( $n = 582$ ).  $n$  (%).

Variables	<i>H. pylori</i> positive	<i>H. pylori</i> negative	$P$ value
Gender			0.151*
Male	32 (5.5)	265 (45.5)	
Female	42 (7.2)	243 (41.8)	
Endoscopic findings			0.684*
Non-erosions	36 (6.2)	260 (44.7)	
Erosions	38 (6.5)	248 (42.6)	

\*: Pearson *Chi*-square.

**Table 3**

Ethnicity distribution for *H. pylori* positive ( $n = 74$ ).

Race	<i>H. pylori</i> positive $n$ (%)	Prevalence (%)	$P$ value	Odds ratio (95% CI)
Malay	15 (20.3)	4.1 (15/367)	0.000*	Reference group
Chinese	18 (24.3)	17.6 (18/102)		5.03 (2.43–10.39)
Indian	41 (55.4)	36.3 (41/113)		13.36 (7.02–25.43)

\*: Pearson *Chi*-square. CI: Confidence interval.

**Table 4**

Association between smoking behavior and alcohol consumption with *H. pylori* positive ( $n = 74$ ).

Variables	<i>H. pylori</i> positive $n$ (%)	$P$ value	Odds ratio (95% CI)
Smoking status			1.01 (0.52–1.95)
Smoker	12 (16.2)	0.987*	
Non-smoker	62 (83.8)		
Alcohol status			1.55 (0.66–3.66)
Alcoholic	7 (9.5)	0.318**	
Non-alcoholic	67 (90.5)		

\*: Pearson *Chi*-square; \*\*: Fisher's exact test.

## 4. Discussion

The prevalence of *H. Pylori* infection for patients underwent endoscopy at HSAH was 12.7% (74/582). A recent study carried out by Yu *et al.* reported a comparable overall prevalence of 14.7% for *H. Pylori* infection at their centre, Serdang Hospital, Malaysia[3]. In the study of Sasidharan *et al.*, the prevalence was 30.4% (597/1965) [4]. They conducted their study 4 years ago at the same district as our study but at Hospital Sungai Petani which is currently known as Klinik Kesihatan Bandar Sungai Petani. Increased awareness and eradication, improved hygiene, better living environment and healthier diet might be the factors for the declined prevalence. Nakajima *et al.* observed a decreasing pattern in the prevalence of *H. Pylori* infection, peptic ulcer and stomach cancer at their setting from 1988 to 2005[5]. This reducing trend of prevalence was also reported by Chong *et al.* over a period of 5 years in Brunei[6].

In our study, infection was highest in the age group of 51 to 60 years old. In contrary, Tanih *et al.* and Chieng *et al.* reported the highest number of patients with *H. pylori* infection were between 31 and 50 years old in Eastern Cape Province and Serdang Hospital, respectively[3,7]. Some studies found that the prevalence of *H. pylori* infection increases with age[4,6,8]. However, few studies did not find any association between age and *H. pylori* infection[9,10].

In our study and in Kaur and Naing's, there was no significant association between gender and *H. pylori* infection[10]. The similarities might be due to comparable population because both studies were done in Northeastern Peninsular Malaysia. Also, a number of other studies failed to link gender to the likelihood of *H. pylori* infection[3,8,11–14]. According to Brown, improper sanitation practices, low social class and crowded or high-density living conditions seem to be linked to a higher prevalence of *H. pylori* infection which explains why the majority of the studies including ours fail to determine the association between gender and the infection[15]. In general, the most likely mode of transmission is from person to person, by either the oral-oral route (through vomitus or possibly saliva) or the fecal-oral route[15]. Waterborne transmission, probably due to fecal contamination, may be an important cause of the infection, especially in parts of the world where untreated water is common[15].

Indians and Chinese were more likely to be infected with *H. pylori* in comparison to Malays which is consistent with the findings by Kaur and Naing[10]. Chinese were 5 times more likely to be infected with *H. pylori* in our study and in Kaur and Naing's[10]. In comparison to Malays, both our study and Kaur and Naing's demonstrated that Indians were more likely to be infected with *H. pylori* with the odds ratio of 13.36 (95% CI 7.02–25.43) and 5.67 (95% CI 1.93–16.34), respectively[10]. A similar trend was observed in various studies carried out in Malaysia and other neighbouring countries such as Brunei with higher proportion of Indians having *H. pylori* infection in comparison to other races[2,4,6,16]. Increased risk among Indians in acquiring *H. pylori* infection as compared to other races is probably due to the different socio-economic status, cultures, hygiene and diet. It was believed that the prohibition of smoking and alcohol intake by Islam religion could lead to lower prevalence of *H. pylori* infection among Malays[3,4]. However, our study failed to demonstrate any significant association between these two behaviours and *H. pylori* infection. Yu *et al.* pointed out that diet consisting of herbs and chillies which are potential gastroprotective might be the cause of lower prevalence of *H. pylori* infection among Malays[3]. Lee *et al.* looked into the dietary factors which might contribute to lower *H. pylori* infection prevalence among Malays in Northeastern Peninsular Malaysia[17]. They have successfully identified three dietary practices which could prevent *H. pylori* infection. The potential prophylactic agents include frequent consumption of "pegaga", tea and "budu"[17]. Asiaticoside in "pegaga" (*Centella asiatica*) can reduce inflammation, oxidation and ulcer occurrence[17]. Tea contains catechins with antibacterial properties[17]. "Budu" is a local fermented anchovy sauce which may inhibit the growth of *H. pylori*[17].

From the endoscopic finding, erosions (defined as esophagitis/gastritis/duodenitis) had the highest *H. pylori* positive cases, similar to the results reported by Chong *et al.*[6]. Also, erosions were more common in *H. pylori* infected patients in Serdang Hospital[3]. *H. pylori* works by damaging the mucous shield or lining that coats the stomach and duodenum, which leads to gastritis and duodenitis[3].

In our study, there was no significant association between smoking habits and *H. pylori* infection. Many studies have also failed to establish any association between smoking and *H. pylori* infection[8,9,14,18–20]. However, few other studies managed to link

smoking to an increased risk of *H. pylori* infection[11,21]. Smoking may create a suitable environment for *H. pylori* colonisation by disrupting the gastric protective mechanisms, gastrointestinal motility and host immunity[22].

No significant association between alcohol consumption and *H. pylori* infection was found in our study in line with few other studies[8,14,18,19]. However, one study discovered that heavy alcohol consumption and drinking wine are protective against *H. pylori* infection[23]. It is believed that alcohol might have antimicrobial activities resulting in inhibition of *H. pylori*[24]. On the other hand, Zhang *et al.* discovered that alcohol intake could increase the risk of getting *H. pylori* infection by 9 folds (95% CI 1.05–77.98)[9]. Heavy alcohol intake might damage the gastrointestinal mucosa, which increases bacterial adhesion and colonisation[9].

Overall, all of these inconsistent results on the association between smoking and alcohol behaviour with *H. pylori* infection might be confounded by other factors such as different types of cigarettes or alcohol, amount of cigarettes or alcohol consumption, education levels and socio-economic status.

There are some limitations in our study. This was a retrospective review of endoscopic records. Some records have to be excluded due to incomplete data. For example, the CLO test result wasn't reported. Also, CLO test was not done for all subjects who underwent endoscopy at our setting. Other possible confounding factors such as household incomes, socio-economic status, education level and diet were unable to be traced from our records. Therefore, the association of these factors with *H. pylori* infection was unable to be determined. However, the results that we obtained were consistent with many studies conducted at other places.

The increased risk of *H. pylori* infection in Indians might be due to the unusual socio-cultural practices and difference in diet which may be responsible for the transmission of the infection. Therefore, further studies are warranted.

### Conflict of interest statement

We declare that we have no conflict of interest.

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