Prevalence of \textit{Helicobacter pylori} Infection in Cigarette and Nargileh Smoking Males in Erbil City, Iraq

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ABSTRACT

Background: Smoking is the foremost public health problem affecting the world and it has a crucial implication in causing many common diseases due to \textit{Helicobacter pylori} infection which is globally distributed. Smoking is considered a critical risk factor that accelerates infection with this bacterium.

Objectives: The study's goal was to find out how common \textit{Helicobacter pylori} infections were among male cigarette and nargileh smokers.

Materials and methods: A case-control study was performed between August and December 2021 in Erbil Teaching Hospital in Erbil City, Kurdistan Region, Iraq. Blood samples were collected and used for detection of anti-\textit{Helicobacter pylori} IgG Ab for 80 males who were smokers and 80 who were non-smoker.

Results: The prevalence of \textit{Helicobacter pylori} positivity was 64.9% in smokers and 45.5% in nonsmokers (P-Value = 0.03). The highest percentage (54.1%) was found in the young age group (25-34) years (P-Value = 0.05), and 89.2% of \textit{Helicobacter pylori}-positive individuals exhibited stomach symptoms (P-Value = 0.01). Fifty percent of \textit{Helicobacter pylori}-infected individuals were nargileh smokers.

Conclusion: The prevalence of \textit{Helicobacter pylori} showed significant value in nargileh smoking males. Therefore, smoking was a key factor in the seroprevalence of \textit{Helicobacter pylori} and had a substantial impact on it.

Keywords: \textit{Helicobacter pylori}; IgG; Cigarette; Nargileh; Smoker.

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INTRODUCTION

Smoking is one of the foremost public health problems affecting the world and it has a crucial implication in causing many common diseases such as periodontitis, chronic obstructive pulmonary diseases, and cancer, in addition, its effects on the microbiome allow oral pathogens to grow, leading to several diseases [1].

\textit{Helicobacter pylori} is a bacterium that is in the stomach and can alter the environment around them to live. In addition, mucus protects microorganisms, and immune cells in the body can’t access them and they are not destroyed which leads to stomach problems. The majority of stomach and small intestine ulcers are caused by this bacterium [1]. Drinking contaminated water, eating unclean or poorly prepared food, sharing the residence with those infected with \textit{Helicobacter pylori}, and living in overcrowded quarters are all risk factors for bacterial infection. The illnesses are believed to travel from one person’s mouth to another, as well as from feces to the mouth, also \textit{Helicobacter pylori} has been detected in gastric refluxate, and vomitus saliva [1].

Smoking is a well-known risk factor for stomach ulcers, and both \textit{Helicobacter pylori} infection and smoking are classified as definite carcinogens [2]. \textit{Helicobacter pylori} is classified as a Class 1 carcinogen by the World Health Organization and carried by 70%-90% of the population in developing nations.
during childhood, but the prevalence is 30%-40% in developed countries [3]. Smokers are more likely than non-smokers to get *Helicobacter pylori* infections [3]. For instance, the quality of life throughout childhood has an inverse relationship with socioeconomic position and the frequency of *Helicobacter pylori* infection. This is because the likelihood of developing *Helicobacter pylori* infection is low throughout childhood and progressively increases with age, with the increase largely attributable to *Helicobacter pylori* acquisition later in life [4]. *Helicobacter pylori* is a disease that influences the relative risk of acquiring numerous upper gastrointestinal clinical problems, and testing for it should be done to discover the origin of an underlying ailment such as peptic ulcer disease or in people with familial stomach cancer [4]. The purpose of this study was to determine how frequently nargileh and cigarette smokers had *Helicobacter pylori* infection in Erbil City, Iraq.

**MATERIALS AND METHODS**

**Study Design**

A case-control study was performed between August and December 2021. Patients with non-ulcer dyspepsia were included in our research after being recommended by their physician for the *Helicobacter pylori* test. The inclusion criteria were subjected to 80 smoker’s males and 80 non-smokers attending and attending the Erbil Teaching Hospital in Erbil city, Kurdistan Region, Iraq. Blood samples were collected and prepared for the detection of anti-*Helicobacter pylori* IgG Ab in the blood (BioMaxima S.A., ul. Vetterow 5, 20-277 Lublin, Poland). Participants who consented to participate in this study were included after providing informed verbal and written consent. Participants who were less than 15 years old and those who had received treatment for *Helicobacter pylori* infection were excluded, in addition, those who declined to participate were excluded too.

**Methods**

The qualitative membrane-based immunoassay known as the *Helicobacter pylori* IgG Ab test (rapid cassette test) is used to find *Helicobacter pylori* antibodies in serum or plasma. When the specimen was put in the specimen well of the cassette, the test’s *Helicobacter pylori* antigen-coated particles caused the specimen to respond. A colorful line emerged in the test line region to indicate a positive result if the samples had *Helicobacter pylori* antibodies. If the samples didn’t contain *Helicobacter pylori* antibodies a colorful line indicating a negative result has not appeared. A colorful line consistently emerged in the control line region as a procedural check to make sure the correct amount of material has been provided and membrane wicking has taken place; including socio-demographic information (age, educational level, family history, gastritis symptoms) and health-related behaviors (cigarette and nargileh smoking).

**Ethical Considerations**

The Ethics Committee of the Scientific Research (ECSR RN:6;2,6,2021) got approval to conduct the research after outlining the procedures in the research process and explaining them to the committee of Erbil Technical Health and Medical College at Erbil Polytechnic University, Kurdistan Region, Iraq.

### Statistical Analysis

SPSS (Statistical Package for Social Science) version 25 was used. Data were summarized as frequency, mean ± SD, and percentages. The Chi-square test was used to compare qualitative variables. A P-value < 0.05 was considered a statistically significant difference.

### RESULTS

We looked at the data of 160 non-ulcer dyspepsia male patients. Eighty patients were smokers and 80 were nonsmokers. The mean age of the participants was 27.21 ± 7.093, ranging from 15-54 years old.

In the present study, the prevalence of *Helicobacter pylori* positivity was 64.9% in smokers and 45.5% in nonsmokers. Additionally, there was a statistically significant difference in the frequencies of *Helicobacter pylori* positivity between smokers and non-smokers (P-Value = 0.03) (Table 1).

In our study, although there were no significant differences (P-Value = 0.7) between the two types of smoking we investigated that the prevalence of *Helicobacter pylori* was more common in nargileh smoking males than in cigarettes (Table 2).

### Table 1. Distribution of *Helicobacter pylori* seropositivity in smoker and non-smoker patients (n=160). *

<table>
<thead>
<tr>
<th><em>Helicobacter pylori</em> infection</th>
<th>Smoker Number(%)</th>
<th>Nonsmoker Number(%)</th>
<th>Total Number(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>24(64.9%)</td>
<td>13(35.1%)</td>
<td>37(100%)</td>
</tr>
<tr>
<td>Negative</td>
<td>56(45.5%)</td>
<td>67(54.5%)</td>
<td>123(100%)</td>
</tr>
<tr>
<td>Total</td>
<td>80(50%)</td>
<td>80(50%)</td>
<td>160(100%)</td>
</tr>
</tbody>
</table>

* P-value=0.03

### Table 2. Distribution of *Helicobacter pylori* seropositivity concerning types of smoking (n=80). *

<table>
<thead>
<tr>
<th><em>Helicobacter pylori</em></th>
<th>Cigarette Number(%)</th>
<th>Nargileh Number(%)</th>
<th>Both Number(%)</th>
<th>Total Number(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>5(20.8%)</td>
<td>12(50%)</td>
<td>7(29.2%)</td>
<td>24(19.2%)</td>
</tr>
<tr>
<td>Negative</td>
<td>9(16.1%)</td>
<td>26(46.4%)</td>
<td>21(37.5%)</td>
<td>56(44.8%)</td>
</tr>
<tr>
<td>Total</td>
<td>14(17.5%)</td>
<td>38(47.5%)</td>
<td>28(35%)</td>
<td>80(100%)</td>
</tr>
</tbody>
</table>

* P-value=0.7

### Table 3. Distribution of *Helicobacter pylori* seropositivity according to ages. *

<table>
<thead>
<tr>
<th>Ages group (years)</th>
<th>Smoker Number(%)</th>
<th>Non-smoker Number(%)</th>
<th>Total Number(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-24</td>
<td>7(29.2%)</td>
<td>3(23.1%)</td>
<td>10(27%)</td>
</tr>
<tr>
<td>25-34</td>
<td>11(45.8%)</td>
<td>9(69.2%)</td>
<td>20(54.1%)</td>
</tr>
<tr>
<td>35-44</td>
<td>4(16.7%)</td>
<td>1(7.7%)</td>
<td>5(13.5%)</td>
</tr>
<tr>
<td>45-54</td>
<td>2(8.3%)</td>
<td>0(0%)</td>
<td>2(5.4%)</td>
</tr>
<tr>
<td>Total</td>
<td>24(100%)</td>
<td>13(100%)</td>
<td>37(100%)</td>
</tr>
</tbody>
</table>

* P-value=0.05
According to the results of our study, among 37 participants that were infected with *Helicobacter pylori*, we found that the prevalence of infection was more common in the age group 25-34 years, followed by 15-24 years old (P-Value = 0.05) (Table 3).

As shown in Table 4 there were significant differences in the prevalence of *Helicobacter pylori* seropositivity among 160 smoker and non-smoker males according to the level of education, gastric symptoms, and family history of infection (P-Value = 0.01), whereas, the economic status of participants had no impact on the prevalence of *Helicobacter pylori* infection (P-Value = 0.2).

### DISCUSSION

Smoking has been linked to the development of *Helicobacter pylori* infection, its increased persistence, and the reduced efficacy of its eradication. Smokers may have a higher risk of infection, which might raise their chance of developing stomach cancer [5].

In our study, the prevalence of *Helicobacter pylori* was 64.9% in male smokers. This result agrees with other studies that found that 63.64% and 71.4% of subjects that had a history of cigarette smoking were found to have *Helicobacter pylori* and, according to their statistical analysis, discovered a relationship between cigarette smoking and an increased frequency of *Helicobacter pylori* infection (P-Value, 0.0001) [2, 3, 6]. As a result, smoking has a major role in the development of peptic ulcer disease and dyspeptic symptoms. Based on the finding of the urea breath test, the prevalence of *Helicobacter pylori* in the case group is determined to be 68.4% [7]. According to the findings, 75.2% of the human blood samples tested had antibodies to *Helicobacter pylori* [8]. Another study revealed that by using various techniques, the prevalence of *Helicobacter pylori* in Iraqi patients ranged from 47.8 to 70.4% [9]. According to the result of another study, 86 individuals (78.2%) had rapid urease tests that revealed *Helicobacter pylori* infection [10]. A total of 53.3% of 240 people with gastroduodenal diseases were infected with *Helicobacter pylori*, according to another study [11].

It should be emphasized that the rate of *Helicobacter pylori* infection in smokers was much greater than in non-smokers [12]. Strong interactions between cigarette smoking and *Helicobacter pylori* were discovered [13]. Tobacco and alcohol diminish mucus production and mucosal blood flow in the gastric mucosa, reducing the gastric mucosa’s defensive function and rendering it prone to *Helicobacter pylori* invasion [14]. Since smoking affects the immune system and makes a person more likely to get *Helicobacter pylori*, it is a major cause of peptic ulcer disease and dyspeptic symptoms [14].

An association between *Helicobacter pylori* and the type of smoking from the results in Table 2 that there was no significant relationship between them, although that 50% of patients who had *Helicobacter pylori* infection were nargileh smokers and from our results, we concluded that H. pylori infection spreads from person to person via the oral route. It was found that smokers had a slightly higher rate of *Helicobacter pylori* infection, but this was not statistically significant [15].

In this study, the age distribution of *Helicobacter pylori* infection revealed a rising trend (54.1%) in younger age groups (25-34 years old) and a decreasing trend in older age groups. This observation was similar to the observations made by Priyadarshini et al. (2018) and Abdulrahman et al. (2022) who revealed that younger peopole (2040 years old) had greater rates of *Helicobacter pylori* infection than older people, and they discovered that infections were more common in young and middle age groups (2550 years) than in other age groups, as approved by WHO [15]. In addition, similar findings to the results of a study done by Lim et al., (2018) who said that seropositivity to *Helicobacter pylori* increased with age, then declined somewhat as people got older (P-Value < 0.001)[16]. A prior study found that *Helicobacter pylori* infection was present in 23.4% of patients in the age group of 20- 29 [17].

The relationship between the level of education and *Helicobacter pylori* infection was significant, the result agrees with results of other studies that low levels of education, especially inadequate health education and a proclivity to live in an environment that encourages fecal contamination of food and water, have both been linked to an increased risk of chronic *Helicobacter pylori* infection [3, 15]. Statistical significance differences were only identified in respondents with low and

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**Table 4.** Demographic characteristics of *Helicobacter pylori* seropositivity in 160 smokers and male non-smoker

<table>
<thead>
<tr>
<th>Variables</th>
<th><em>Helicobacter pylori</em> Positive</th>
<th><em>Helicobacter pylori</em> Negative</th>
<th>Total</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number (%)</td>
<td>Number (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illiterate</td>
<td>5 (13.5%)</td>
<td>3 (2.4%)</td>
<td>8 (5%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Primary school</td>
<td>5 (13.5%)</td>
<td>7 (5.7%)</td>
<td>12 (7.5%)</td>
<td></td>
</tr>
<tr>
<td>Secondary school</td>
<td>5 (13.5%)</td>
<td>26 (21%)</td>
<td>31 (19.4%)</td>
<td></td>
</tr>
<tr>
<td>College or higher</td>
<td>22 (59.5%)</td>
<td>87 (70.7%)</td>
<td>109 (68%)</td>
<td></td>
</tr>
<tr>
<td>Economic status</td>
<td></td>
<td></td>
<td></td>
<td>0.2</td>
</tr>
<tr>
<td>Low</td>
<td>6 (16.2%)</td>
<td>13 (10.6%)</td>
<td>19 (11.9%)</td>
<td></td>
</tr>
<tr>
<td>Middle</td>
<td>31 (83.8%)</td>
<td>102 (82.9%)</td>
<td>133 (83.1%)</td>
<td></td>
</tr>
<tr>
<td>Good</td>
<td>0 (0.0%)</td>
<td>8 (6.5%)</td>
<td>8 (5%)</td>
<td></td>
</tr>
<tr>
<td>Gastric symptoms</td>
<td></td>
<td></td>
<td></td>
<td>0.01</td>
</tr>
<tr>
<td>With symptoms</td>
<td>33 (89.2%)</td>
<td>44 (35.8%)</td>
<td>77 (48.1%)</td>
<td></td>
</tr>
<tr>
<td>Without symptoms</td>
<td>4 (10.8%)</td>
<td>79 (64.2%)</td>
<td>83 (51.9%)</td>
<td></td>
</tr>
<tr>
<td>Family history of infection</td>
<td></td>
<td></td>
<td></td>
<td>0.01</td>
</tr>
<tr>
<td>Yes</td>
<td>25 (67.6%)</td>
<td>33 (26.8%)</td>
<td>58 (36.3%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>12 (32.4%)</td>
<td>90 (73.2%)</td>
<td>102 (63.7%)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>37 (100%)</td>
<td>123 (%)</td>
<td>100 (%)</td>
<td>0.03</td>
</tr>
</tbody>
</table>
medium educational levels, according to some studies [16].

It’s very interesting to look at the additive discriminative power of multiple risk factors and as seen in Table 4. Individuals from poor and middle socioeconomic backgrounds exhibited high Helicobacter pylori prevalence rates, although the differences were not significant. Poor socioeconomic status, a lack of education, and genetic factors all contribute to higher rates of Helicobacter pylori colonisation [15].

A study found that participants with lower (low and medium) household income levels had a larger percentage of Helicobacter pylori seropositivity than those with higher income levels and that only those with medium income levels reached statistical significance [16]. 87.83% of people in lower socioeconomic groups and 76.4% of those in middle socioeconomic classes had higher rates of Helicobacter pylori infection as compared to people in higher socioeconomic classes [3]. Based on all of these data, we indicated that lower and middle socioeconomic class significantly increased the probability of Helicobacter pylori infection and were a significant risk factor. Individuals from lower socioeconomic classes are more likely to have inadequate health knowledge, poor environmental sanitation, congestion, and a greater proclivity to live in a setting that predisposes to fecal contamination of food and water [3].

In the current study, we looked at how smoking status and Helicobacter pylori positivity can interact, and we discovered that 89.2% of patients with Helicobacter pylori positivity experience gastrointestinal symptoms, with significant variations. The persistence of the Helicobacter pylori infection has been linked to heartburn, epigastric pain, belching, and dyspepsia. Smoking increases the production of stomach acid and weakens the mucosal barrier, making it a significant risk factor for peptic ulcer disease. Furthermore, Helicobacter pylori infection and smoking together enhance a patient’s chance of developing an ulcer. Infection by Helicobacter pylori boosts pepsinogen production while lowering mucus production [18].

As represented in Table 4 in our study, in general practice, patients discovered a substantial link between Helicobacter pylori infection and family history, with 67.6% of patients having a history of the illness. The results of the study showed that 80.39% of the people who were seropositive for Helicobacter pylori had a family history of the disease, and the difference was significant (P-Value = 0.001[19].

Owing to the study period during the COVID-19 pandemic, only 160 participants gave consent to enrol in the study. This sample size is considered a small one if one compares it with the sample sizes of other similar studies. Therefore, this is considered a limitation of the present study.

CONCLUSION

Our study led us to the conclusion that smoking was a significant contributing factor to the seroprevalence of Helicobacter pylori and that it significantly influenced that seroprevalence. This may be due to hygienic habits involving the transmission of bacteria through the saliva of smokers. Regarding the level of education, socioeconomic status, and family history, all these factors increase the chance of transmission due to improper hygiene and health care. Age shows a significant effect, and the plurality of infection is manifested in the young and middle age groups rather than in older age groups. The prevalence of Helicobacter pylori shows significant value in nargile smoking patients and this shows that the transmission of Helicobacter pylori is spread from person to person by oral route.

ETHICAL DECLARATIONS

Acknowledgements

None.

Ethics Approval and Consent to Participate

Written approval had been gained from the Ethics Committee of the Scientific Research of Erbil Technical Health and Medical College at Erbil Polytechnic University, Kurdistan Region, Iraq. Study data/information was used for the research purpose only. Informed consents from every participant was taken.

Consent for Publication

Not applicable (no individual personal data included).

Availability of Data and Material

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that there is no conflict of interest.

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Authors’ Contributions

All the authors listed have made a substantial, direct, and intellectual contribution to the work, and approved it for publication.

REFERENCES


