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Original Article

Association of Smoking with the Severity of *H. pylori* with the Extent of Its Impact on Blood Parameters

Abed, A. K[1 *], Yahya Al-Ma'amouri, M[2], Abdulkareem Salman, M[3]

1. Genetic, College of Science, University of Diyala, Diyala, Iraq
2. Institute of Medical Technology Al-Mansur, Middle Technical University, Baghdad, Iraq
3. Molecular Cytology and Cell Culture, College of Medicine, University of Diyala, Diyala, Iraq

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Corresponding Author: aidaa41mijbel@yahoo.com

Abstract

H. pylori infection is considered a major global gastric disorder. Furthermore, the spread of *H. pylori* infection and its effect on systemic disorders and blood is not fully understood. On the other hand, the high percentage of smokers and their impacts on the health system have become a significant concern. Therefore, the current study aimed to compare the *H. pylori* infection in smokers and non-smokers and their effects on hematological parameters.190 patients participated and were divided into two groups; 95 were smokers and 95 were non-smokers. The *Helicobacter pylori* detector instrument showed that the participants were infected. Vein blood was collected to check hematological parameters via a fully automatic hematological analyzer (DIAGON Ltd.-D-Cell 60). The recorded data showed that the highest percentage of infected patients was 26–45 years in both smokers and non-smokers ($P \leq 0.05$). Furthermore, depending on the residence, our study revealed that the urban cases were the highest percentage compared to rural cases ($P \leq 0.05$). The hematological parameters showed that RBCs, Hb, PCV, MCV, MCH, and MCHC were significantly higher in smokers compared with non-smokers ($P \leq 0.05$).

Keywords: Smoking, *H. pylori*, Blood parameters

1. Introduction

H. pylori is a gastric pathogenic bacteria that commonly infects the human gastric mucosa. *H. pylori* has been classified as the primary etiological factor of chronic gastritis and is considered to infect half of the world (1). It is a type of gram-negative bacteria with a spiral form and can grow in the upper digestive tract (2). *H. pylori* can increase clinical gastrointestinal disorders involving gastric lymphoma, peptic ulcer disease, chronic urticarial, Raynaud phenomenon, iron deficiency anemia, chronic headache, and infant death syndrome, which are associated with *H. pylori* (3).

Recent studies have shown that *H. pylori* are related to diseases such as primary immune thrombocytopenia (ITP), ischemic cerebrovascular disease, iron deficiency anemia (IDA), and lymphoma (4). Studies found that *H. pylori* attract lymphocytes and neutrophils released in the stomach. Therefore, the substances released by neutrophils will stimulate mucosal inflammation, causing gastritis. So, neutrophils, macrophages, and lymphocytes can penetrate the gastric mucosa and several cytokine signals, causing a subclinical, low-grade inflammation. For this reason, *H. pylori* are associated with chronic gastritis, peptic ulcers, gastric lymphoma, and gastric cancer (5).

Other studies found that interleukin-6 was significantly increased in *H. pylori* patients (6), as it is known that interleukin-6 secretes numerous cells involved in lymphocytes, monocytes, mesangial cells,

and endothelial cells. Furthermore, an epidemiological study found a link between the serum interleukin-6 level and the severity of coronary artery disease (7).

Because of this, researchers confirmed that the existence of *H. pylori* plays a vital role in other extra gastroenterological diseases. For instance, cardiovascular diseases also stimulate systemic inflammation because of an increase in IL-6 (7). On the other hand, smoking has become one of the significant risks of developing some types of cancer worldwide (8). An epidemiological study found that about 19% of the evaluated smoking-attributable disability-adjusted life-years were because of cancer. The study reported that gastric cancer had been linked to smoking (9).

Despite smoking being responsible for many carcinogenic components (10), there is still a proof point that smoking increases the risk of cancer by indirectly increasing the prevalence of *H. pylori* infection (11). Furthermore, studies have shown that tobacco smoke contains many compounds involving carcinogens. One of them is N-nitroso compounds, which are associated with gastric carcinogenesis (12). Because of this, tobacco has been classified as a high-risk factor related to developing preneoplastic gastric lesions (13).

Studies also showed that people with *H. pylori* smoke, increasing their risk of intestinal metaplasia and gastric cancer, which is the most important risk factor for gastric cancer (14, 15). It can be explained that the association of smoking with increased virulence of *H. pylori* by increasing oxidative stress, which leads to a weakening of immunity and thus increases the virulence of *H. pylori* infection (10). Furthermore, some studies have reported that smoking impedes the successful eradication of *H. pylori* (16, 17). Some researchers differed in their evaluation of the importance of the association between smoking and *H. pylori* (18).

Therefore, this study was designed to investigate the association between smoking and *H. pylori* infection and their effect on blood parameters.

2. Materials and Methods

2.1. Participants

This study started from November 2020 to February 2021. It included 190 patients suffering from *H. pylori*, 95 of whom were smokers infected by *H. pylori*, and the other 95 patients were non-smokers also infected by *H. pylori*. All participants attended the private Baqubah Medical Care Center, Digestive System Lobby.

2.2. Exclusion Criteria and Study Design

Patients who had diabetes, high blood pressure, heart failure, asthma, kidney disease, liver disease, a lack of vitamin B12, or a previous infection with *H. pylori* were not allowed to take part in the study.

Firstly, all patients have been confirmed to be infected by *H. pylori* for the first time via (urease breathing test, by *Helicobacter Pylori* detector instrument (headway /HUBT-20P *Helicobacter Pylori* detector, Germany). According to the company's recommended kit procedure, the patient must have eaten a meal before two hours of the examination. Then, the patient takes one oral tablet related to the *H. pylori* diagnosis and waits for 20 minutes. After that, the patient will be examined by taking a deep inhale from the nose and exhaling deeply from the mouth, and the process is repeated several times to ensure accurate results.

2.3. Blood Sampling

4 ml of vein blood were collected from each patient and put in Ethylene diamine tetra-acetic acid (EDTA) tubes with constant stirring. Then all blood samples were examined via an instrument called the complete blood cell count DIAGON Ltd.-D-Cell 60 fully automatic haematological analyzer.

2.4. Statistical Design

The current study's data was analyzed using the Chi-square (X^2) test to compare percentages. Also measured the sensitivity and specificity of mean platelet volume (MPV). The T-test was used to describe numerical data was used to compare two numeric variables, while the F test (ANOVA) was used to compare three numeric

variables or more. A level of significance of α=0.05 was applied to the test. (SPSS v.22 and Excel 2013) analyze current data.

3. Results

In this study, 190 people with *Helicobacter pylori* infection were split into two groups: 95 people who smoked and 95 people who didn't smoke. The recorded data showed that the highest percentage of age for infected patients was between 26-45 years in both patients, smokers and non-smokers ($P \leq 0.05$). Furthermore, depending on the residence, our study revealed that the urban cases were the highest percentage of rural ($P \leq 0.05$). The haematological parameters showed that RBCs, Hb, PCV, MCV, MCH, and MCHC were significantly higher in smokers compared with non-smokers ($P \leq 0.05$) (Table 1).

Interestingly the recorded data showed that the smokers who are infected by *H. pylori* appearing an inadequate response to WBCs. Especially in neutrophils, eosinophils, and monocyte, where the Eosinophil and monocyte have noted that significantly lower than the patients who are non-smokers, while the Lymphocytes have recorded a significantly higher response in smokers than non-smokers patients ($P \leq 0.01$). As shown in table 2.

Table 1. Distribution and percentage of anthropometric characters of patients

			Groups		Total
			***H. pylori* non-smokers N=95**	***H. pylori* smokers N=95**	
Age	15-25	N	13	15	28
		%	12.35%	14.25%	14.73%
	26-35	N	34	41	75
		%	32.3%	38.95%	39.47%
	36-45	N	36	39	75
		%	34.2%	37.05%	39.47%
	46-55	N	12	0	12
		%	11.4%	0%	6.31%
Residence	Urban	N	60	81	141
		%	63.16%	85.26%	74.21%
	Rural	N	35	14	49
		%	36.84%	14.73%	25.79%

Table 2. Comparison of hematological parameters between smokers and non-smokers who are infected by *H. pylori*

Parameters	***H. pylori* in smokers N=95**	***H. pylori* in Non-smokers N=95**	***P*-value**
RBCs,c/mm3	5101±1280	4630±1370	0.015*
Hb g/dl	15.98±2.1	13.05±2.45	0.001*
PCV %	46.77±4.02	39.15±5.33	0.001*
MCV, fl	90.7±1.38	82.6±4.63	0.001*
MCH, Pg	27.5±1.55	26.20±1.68	0.001*
MCHC, g/dl	35.27±1.27	34.15±1.68	0.001*
WBCs, c/mm3	6848±1894	6950±1748	0.7
Neutrophil	57.93±8.5	60.77±10.5	0.042*
Eosinophil	5.53±1.8	5.81±2.9	0.001*
Monocytes	5.1±1.72	5.9±1.6	0.001*
Lymphocyte	29.89±5.79	27.59±6.8	0.013*

4. Discussion

Numerous studies have revealed that smoking has a negative effect on haematological parameters (8). Besides that, *H. pylori* infection has become one of the leading causes of morbidity and mortality worldwide (19). According to WHO reports, smoking and *H. pylori* have been considered significant health complications worldwide. According to the WHO, smoking kills four million people each year (20). In addition, *H. pylori* causes infection in more than 50% of people worldwide (19).

Previous studies showed that smoking increased WBCs, including neutrophils, monocytes, and lymphocytes, the same for RBCs, hemoglobin, and mean corpuscular volume (21). On the other hand, it was found that most haematological changes are linked to *H. pylori* infection (22).

Furthermore, studies have shown that *H. pylori* alters RBC parameters through various mechanisms because *H. pylori* consumes iron (23). The results of our study are in agreement with previously published studies.

On the contrary, the results of the current study found that the levels of iron and red blood cells in smokers were normal without a decrease due to the fact that the increase in red blood cells and iron was caused by smoking, which is compatible with other studies related to the impact of smoking on blood (8), despite the decrease in the absorption of secondary iron in the stomach in the case of *H. pylori* infection.

These results are similar to or consistent with the recent studies that linked smoking with pylori infection, where they noted that *H. pylori* infection was mild in smokers despite the increased acidity in the stomach caused by *H. pylori* (24).

Our current study results indicated that most *H. pylori* infections targeted persons between the ages of 25-46.

Interestingly, most of the infected persons were from urban areas, which are supposed to present the requirements of a clean environment; this could be due to the neglect of the health, service, and environmental aspects, which led to high pollution rates and the deterioration of the health status of the city.

Moreover, according to a questionnaire, most patients who participated in our research preferred eating food from restaurants and fast food. Due to health neglect and contaminated food, the *H. pylori* infection will undoubtedly increase.

After performing a blood analysis for each participating patient, we found that the haematological indicators in smokers, which are red blood cells, haemoglobin, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration, mean corpuscular volume, and packed cell volume, were significantly higher than the haematological indicators of non-smoker patients. Our results are entirely consistent with other explanations that link the increase in red blood cell markers and what is associated with them with an increase in daily tobacco consumption (8, 24), where we found that all of the smoker participants were smoking at least 35 cigarettes per day, which is considered a vast number of cigarettes smoked per day.

As for non-smoking patients, the decrease in the indicators of blood cells and their components is caused by the consumption of high iron ions by pylori bacteria. On the other hand, we found fluctuations in white blood cell indicators during infection with *H. pylori*. Smokers' WBCs were lower. While neutrophils, monocytes, and eosinophils were significantly lower in smokers than in nonsmokers (*P*-value 0.005). At the same time, the lymphocytes in the smoker patients were significantly higher than in the non-smoker patients. According to other studies, this increase of some white blood cells is a normal response due to stressors, where numerous studies concluded that each pull of tobacco smoke contains more than 1000 radical organics, which leads to stimulating the cytokines and increasing white blood cells (24). Alternatively, infections like *H. pylori* stimulate the immune system, especially white blood cells.

The same applies to the red blood cells, where these cells are affected by carbon monoxide and thus lead to the formation of carboxyhemoglobin in the red blood cells, subsequently increasing the number of red blood cells in the body (25).

According to the results obtained, smoking has a negative relationship with *H. pylori* and a positive relationship with the human body in the event of an infection with *H. pylori*. However, it does not prevent gastrointestinal ulcers from forming. Therefore, lifestyle factors such as smoking do not lead to failure to eliminate *H. pylori* but will help reduce the severity of the infection.

Moreover, smoking is one of the factors stimulating the oxidative stress of a healthy body, despite what we found in our study of a positive attitude toward smoking in the case of *H. pylori* infection, which led to some blood parameters remaining high or normal levels compared with non-smoker patients. This can be explained by the possibility that some blood parameters may rise before infection with pylori infection. After *H. pylori* infection, the proportions of some blood parameters began to decrease until they reached their normal states.

Authors' Contribution

Study concept and design: A. K. A.
Acquisition of data: M. Y. A.
Analysis and interpretation of data: M. A. S.
Drafting of the manuscript: M. Y. A.
Critical revision of the manuscript for important intellectual content: A. K. A.
Statistical analysis: A. K. A.
Administrative, technical, and material support: A. K. A.

Ethics

The current study received critical ethical approval after being presented to the Scientific Research Ethics Committee at the College of Medicine and the University of Diyala before we began collecting samples and asking patients questionnaire questions.

Conflict of Interest

The authors declare that they have no conflict of interest.

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