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The Relationship between Polycystic Ovarian Syndrome and Helicobacter Pylori in the Mid-Euphrates Region of Iraq

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ABSTRACT

Background: *Polycystic Ovarian Syndrome (PCOS) is one of the common endocrinopathies. Infertility may result from persistent infections. Two common causes of infertility are Helicobacter pylori infection and PCOS. As far as we know, no research has connected H. pylori seropositivity to PCOS.* ***The aim****: to investigate the impact of Helicobacter pylori (H. pylori) infections on metabolic and hormonal factors, as well as long-term inflammation indicators, in patients with polycystic ovary syndrome (PCOS).* ***Materials and techniques****: We examined 40 healthy women of the same age and 60 women with PCOS. We examined sex steroid-binding globulin (SSBG), total testosterone (TTest), oestradiol, C-reactive protein (CRP), interleukin-6 (IL-6) and IgG antibodies to H. pylori. These parameters were evaluated depending on whether an H. pylori infection was present or not.* ***Results****: Depending on whether an H. pylori infection was present or not, the following outcomes were observed: In the control group, 17.5% of participants were found to have raised IgG antibodies to H. pylori, while their CRP and homocysteine levels increased and their CRP concentration decreased. H. pylori infection was much more prevalent in PCOS patients ($\chi^2 = 7.7$; $P < 0.01$).* ***Conclusions****: It has been demonstrated that PCOS patients have a higher prevalence of elevated blood CRP and IL-6 levels. The findings demonstrate a strong correlation between H. pylori infection and PCOS, which exacerbates the metabolic and hormonal issues already present and raises CRP and IL-6 levels.*

Keywords: **Hyperandrogenism, C-Reactive Protein, Interleukin-6, Polycystic Ovary Syndrome And Helicobacter Pylori Infection.**

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INTRODUCTION

Complicated multifactor known as polycystic ovarian syndrome (PCOS) are generated by combining the environment and epigenetic variables (1). More than 4,500 publications on PCOS have been published over the last five years, most of which focus on etiology and pathophysiology of the disease. Several studies have investigated hormonal and metabolic deviations associated with PCOS. It is determined that oxidative stress (2).

Pathophysiology may include stress, poor angiogenesis and the development of a persistent systemic inflammatory process (3). Recent data suggests a complicated link between PCOS, inflammation and old infectious organisms such as Helicobacter pylori (H. pylori), although the exact cause of systemic inflammation of the PCOS is not clear. About 50% of people around the world H. pylori are infected with, of which more than 70% are affected in developing countries (4). H. pylori infection is widespread in Iraq (5).

Obesity and being overweight are major risk factors for metabolic syndrome, which is prevalent worldwide, particularly in developed nations (6). Nevertheless, the question of whether obesity and H. pylori infection are related remains unanswered. Some researchers

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suggest that being overweight or obese may be associated with a higher prevalence of H. pylori colonization. However, this association has not been verified by other researchers (7). A review by Farideh et al. (8) found an increase in body weight following H. pylori eradication, indicating an inverse relationship between obesity and H. pylori infection. Meanwhile, it has been noted that when H. pylori is active and exerts a greater influence on insulin resistance (IR), the infection has a more noticeable effect on body mass index (BMI) in patients under 50 years old (9).

Polyzos et al. included nine papers in a literature analysis, which found a favourable correlation between insulin resistance and H. pylori infection (10). Other researchers have since verified these findings (11). H. pylori-induced IR in chronic inflammation may occur either directly or indirectly by activating specific signaling pathways. Interleukin-6 (IL-6), tumor necrosis factor α (TNF-α) and C-reactive protein (CRP) can all be overexpressed in chronic inflammation caused by H. pylori (12). These cytokines stimulate the JNK and IKK/NF-κB pathways, which can cause IR by either blocking the phosphorylation of insulin receptor substrate-1 on tyrosine residues or raising the phosphorylation of the insulin receptor on serine.

MATERIALS AND METHODS

Cross-sectional study: The subjects of this study were treated at private clinics located in the central and southern regions of Iraq between January 2023 and December 2024. The study was given ethical approval via the Ethical Board at Al Kufa University College (KUFA Ref No.174/01/2023). A total of 60 women diagnosed with PCOS (mean age 23.5 ± 0.3 years) were selected for the study. The control group comprised 40 healthy women with normal menstrual function who visited the clinic for a reproductive system examination prior to planning a pregnancy. A comparison of the two groups revealed no significant disparities in terms of age or body weight relative to body length.

The levels of total testosterone (T-test), estradiol (E2), and sex steroid-binding globulin (SSG) were measured in order to evaluate the hormonal condition of the individual. The level of hormone secretion in the blood serum was determined on days two and three of the menstrual cycle, irrespective of whether the cycle was self-initiated or induced. Serum C-reactive protein (CRP) levels were evaluated by the turbidimetric method using reagents from BioSystems (Spain). Serum levels of IL-6 and IgG antibodies to H. pylori were measured by an enzyme-linked immunosorbent test using Siemens kits (Germany).

Statistical analysis

The data in the tables is shown as the arithmetic mean ± standard error of the arithmetic mean because the distribution is normal. The mean values were compared using something called Student's t-test. The differences between the groups were checked using something called the 'χ2 technique'. The results of the study were considered reliable, and the differences between the indicators were significant, with a 95% chance of making an error-free prediction ($p < 0.05$).

RESULTS

The study revealed that 28 (46.7%) women diagnosed with PCOS exhibited a positive H. pylori test result, while the control group exhibited a rate of 7 (17.5%) positive cases. In other words, the prevalence of H. pylori infection was found to be significantly higher ($p < 0.01$) among women diagnosed with PCOS compared to those classified as healthy. This finding was supported by a chi-squared test result of 7.7. Body Mass Index (BMI) was not largely different between polycystic ovarian syndrome (PCOS) diagnosed women when compared with women who formed a control group in the current study.

In the presence of study, women suffering from polycystic ovarian syndrome (PCOS), it was observed that their testosterone levels showed a significant increase ($p < 0.001$), while their estradiol levels performed a significant reduction ($p < 0.02$) compared to women without PCOS diagnosis. The results demonstrated that the bacterium caused an increase in testosterone levels and a decrease in estradiol levels (see Table 1 and Figure 1 for further details).

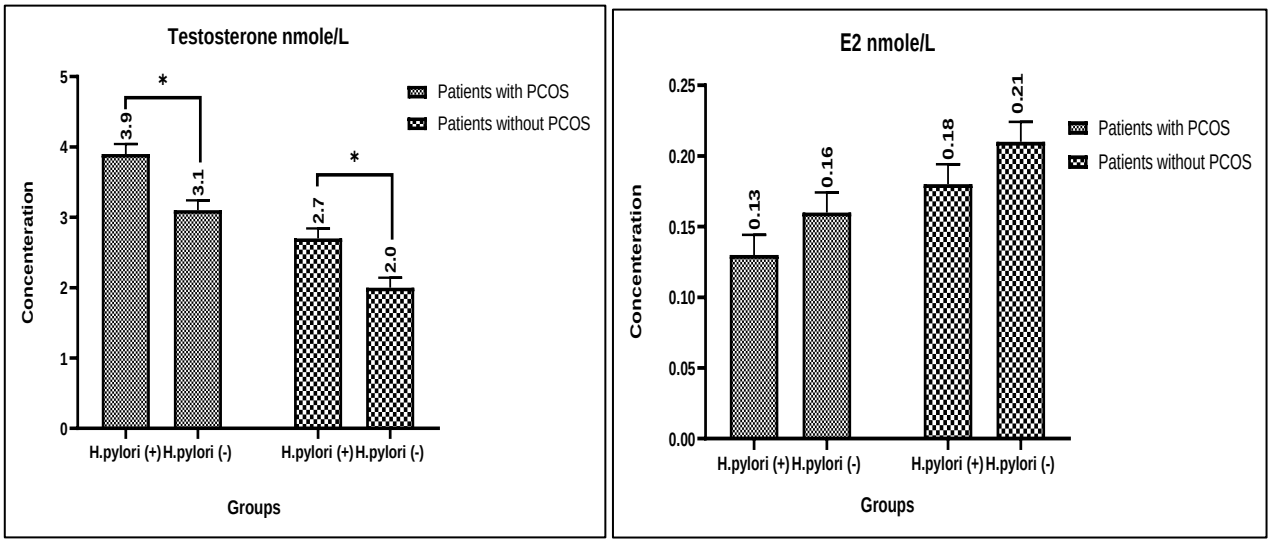

Figure 1: The impact of polycystic ovary syndrome (PCOS) and helicobacter pylori infection on hormonal levels is a subject of considerable interest.

The degree of elevation in circulating levels of serum CRP and IL-6 was found to be statistically significantly higher in the presence of PCOS in the present study, which comprised both patients with PCOS and women who constituted the comparison group. It is important to note that individuals diagnosed with polycystic ovary syndrome (PCOS) exhibit a significant ($p < 0.001$) increase in the ratio of ovaries to ovaries (OT/OS) due to an increase in the ovaries index ($p < 0.001$), suggesting a propensity for adipose tissue accumulation in the abdominal region (see Table 1 and Figure 2 for further details).

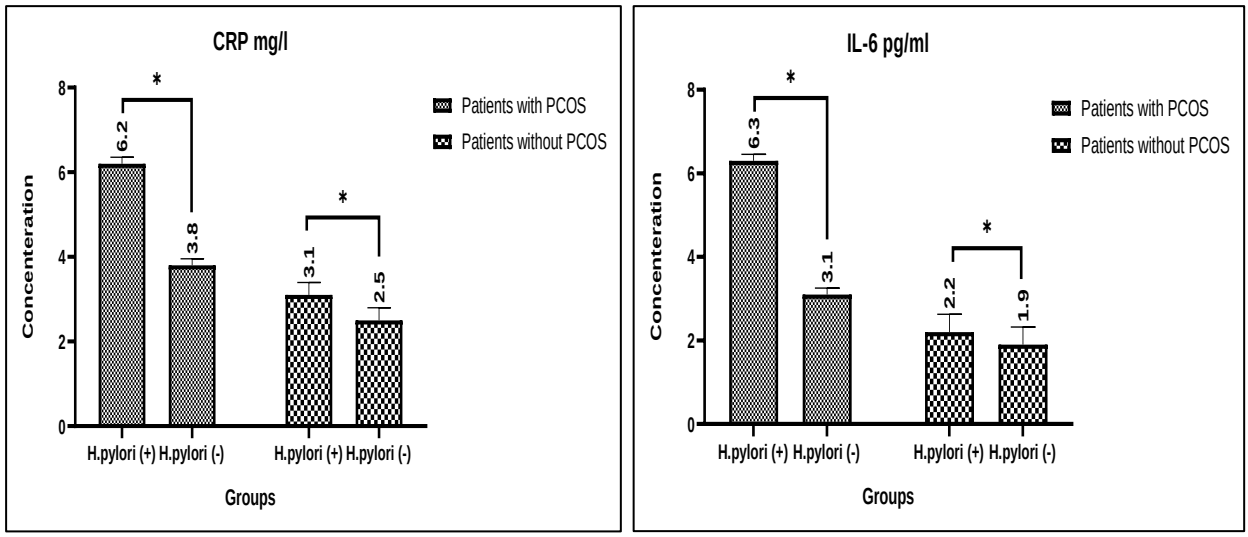

Figure 2: The impact of polycystic ovary syndrome (PCOS) and helicobacter pylori infection on hormonal levels is a subject of considerable interest.

Table 1: The impact of polycystic ovary syndrome (PCOS) and helicobacter pylori infection on hormonal levels is a subject of considerable interest.

Indicators	Patients with PCOS (n = 60)		Patients without PCOS (n = 40)	
	***H.pylori (+)* (n = 28)**	***H.pylori (–)* (n = 32)**	***H.pylori (+)* (n = 7)**	***H.pylori (–)* (n = 33)**
Age (years)	23.5 ± 0.3	23.3 ± 0.3	23.9 ± 0.2	23.8 ± 0.2
BMI (kg/m2)	25.1 ± 0.5	26.9 ± 0.3	22.8 ± 0.5	24.9 ± 0.3
Testosterone nmole/L	3.9 ± 0.1	3.1 ± 0.12	2.7 ± 0.1	2.0 ± 0.1
E_2 nmole/L	0.13 ± 0.01	0.18 ± 0.01	0.16 ± 0.01	0.21 ± 0.01
CRP, mg/l	6.2 ± 0.11	3.8 ± 0.1	3.1 ± 0.21	2.5 ± 0.2
IL-6 pg/ml	6.3 ± 0.11	3.1 ± 0.1	2.2 ± 0.3	1.9 ± 0.2

DISCUSSION

The study established that there was an increase in antibodies to H. pylori in women diagnosed with PCOS in comparison with the control group. The prevalence of H. pylori infection was observed to be lower in the study population than in healthy women, a finding that is consistent with the existing literature on the subject (13).

H. pylori infection is another controversial issue that deserves further study. Currently, H. pylori is considered a causative agent of local inflammation in the gastric and duodenal mucosa, as well as a contributor to systemic inflammation through its impact on metabolic pathways (14). During the study, it was found that H. pylori infection in young women is accompanied by an increase in serum CRP and IL-6 levels.

H. pylori infection is associated with the development of various gastrointestinal diseases, including chronic gastritis, peptic ulcer disease, and gastric malignancies. The infection triggers a chronic inflammatory response in the mucous membranes of the stomach and intestines (15). However, the effects of this localized inflammation are not limited to the digestive tract; they can also extend to tissues or organs outside the intestine (16). Many papers on extra-gastroduodenal symptoms of H. pylori infection have been written recently including hematological, cardiovascular, neurological, and allergy illnesses (17). While the available data does not provide evidence of H. pylori's role in most of these diseases, their potential relationship cannot be ruled out. For instance, numerous epidemiological studies have confirmed a link between H. pylori infection and metabolic syndrome.

Daniel et al. demonstrated that metabolic syndrome was more prevalent among individuals infected with H. pylori than among uninfected patients (18). In this context, it has been suggested that H. pylori infection may cause various endocrine disorders, such as obesity, insulin resistance and dyslipidaemia (19).

This unequivocally shows that PCOS is a disorder linked to a considerable rise in inflammatory markers CRP and IL-6. PCOS-related illnesses such dyslipidaemia and excess adipose tissue (20) and raised CRP and IL-6 aggravate the pathogenic processes linked with PCOS. This rise is thereby related to these factors. This study implies that the degree of hyperandrogenism in PCOS patients corresponds with changes in blood CRP and IL-6 levels. Given that the combination of these pathogenic diseases aggravates metabolic and hormonal problems in PCOS and stimulates the development of chronic inflammatory markers, further research on the link between H. pylori and PCOS is justified.

COCLUSIONS

Our study has demonstrated that PCOS patients have a higher prevalence of elevated blood CRP and IL-6 levels. The findings demonstrate a strong correlation between H. pylori infection and PCOS, which exacerbates the metabolic and hormonal issues already present and raises CRP and IL-6 levels.

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