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Research Article

CHEMISTRY

Serum Adiponectin and Leptin hormone with *Helicobacter pylori* infection in Obese Patients

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KEY WORDS

H. pylori;

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ABSTRACT

Helicobacter pylori infection can cause chronic inflammation and is linked to changes in body mass index (BMI, kg/m^2). This study evaluates the association between *H. pylori* infection and overweight/obesity syndrome, in addition to its relation with adipocytokines Leptin and Adiponectin. Clear evidence indicates that Leptin hormone and Adiponectin hormone, contribute substantially to the improvement of this disease (*H. pylori* and Obesity). Furthermore, current study evaluates the relationship between serum Leptin hormone, adiponectin hormone and infection with *H. pylori* in patients with obesity A total of 50 healthy individuals and 100 patients were included in the research. Results indicated that there were 50 obese patients without infection of *H. pylori* (group 2) in addition to 50 obese patients with infection of *H. pylori* (group 3) had indicated significant change increase in serum Leptin level and significant decrease Adiponectin level comparison with 50 healthy control (group 3). Also, there was a rise in the levels of serum triglyceride and cholesterol and low HDL-c in patient groups as well as an increase in the Body Mass Index (BMI) compared to healthy group.

Introduction

The gram-negative spiral-shaped bacterium *Helicobacter pylori* (*H. pylori*) resides in the gastric mucous layer or on the surface of the stomach's epithelium. Half of all stomach cancer occurrences may be attributed to the bacterium *Helicobacter pylori*, which is also responsible for a wide range of other digestive disorders and diseases. **(Takahashi-Kanemitsu *et al.*, 2020).**

According to literature more than ninety percent of people who have duodenal ulcers and eighty percent of those who have stomach ulcers are infected with *H. pylori* **(Anand *et al.*, 1996).** Almost half of all humans have *H. pylori* in their stomachs, has recently been recognized as a threat to public health. More than 70% of the population in poor countries tests positive for *H. pylori* antibodies **(Stasi and Provan, 2008)**. Alternatively, Infection with *H. pylori* is much more common in adults than in children, starts off at a lower rate in middle age, and reaches 50% in wealthy nations **(Zhou *et al.*, 2012)**. Numerous studies conducted in Egypt confirm the widespread infections with *H. pylori* which are common in persons of all ages. People with gastrointestinal diseases have a greater prevalence of *H. pylori* in Egypt than do people who appear to be in good health **(Haggag *et al.*, 2016)**.

Obesity is the accumulation of abnormally large amounts of fat or animal tissue, which will detrimental to health. Within the previous half-century, obesity has grown into a contagious condition. Additionally, it is connected to a number of medical disorders and may lead to major complications in conditions that are persistent **(Molina and Morgan, 2021).**

Obesity is now classified methodologically use the (BMI). Even the thickness of the skin in the triceps, biceps, subscapular, and suprailiac regions can be used to evaluate obesity. Dual energy radiography absorptiometry scans can also be used to measure the amount of fat in the body **(Kozlov, 2018).**

A 167-amino acids hormone called leptin, also known as (Greek, leptos, thin), regulates how much food is consumed and how much energy is expended. Since an increase in fat mass is correlated with an increase in leptin, making leptin an indication of total fat mass, the levels of leptin in the blood and its mRNA expression in adipose tissue are directly related to the obesity degree **(Tsai, 2017).** *H. pylori* infection significantly increased gastric leptin expression, its elimination resulted in a reduction in gastric leptin expression without a corresponding drop in blood

leptin levels **(Chuang *et al.*, 2009).** The idea that the *H. pylori* strain is responsible for regulating the bacterial connection to the production of these hormones is gaining support **(Isomoto *et al.*, 2005).**

Adiponectin, a hormone secreted by adipocytes, is a well-known homeostatic factor involved in maintaining appropriate glucose homeostasis, lipid metabolism, and insulin sensitivity. Adiponectin has been linked to anti-inflammatory, anti-fibrotic, and antioxidant properties **(Nguyen, 2020)**. It is unclear how HP infection affects serum adiponectin. There was no discernible difference in adiponectin levels between HP-positive and HP-negative people; however, **Ando *et al.*, (2013)** did observe that adiponectin levels increased when HP infection was treated.

Material and Methods

Human subjects:

Venous blood samples were taken from 100 obese patients with and without infection of *H. pylori* with age above 18 years old in Malawy General Hospital, Menya Governorate during the period from September 2021 to March 2022. All participates gave an informed consent after illustrating the nature of study. An official permission was obtained from Mallawi General Hospital. The study was reviewed and approved by the committee of local institution of ethics of Pharmacy Faculty, Menya University (No. MPEC230201).

Methods

The Research study included 150 participants; 50 obese patients without infection with *H. pylori* and 50 obese patients with *H. pylori* infection and 50 controls (healthy). Standardized techniques were used to measure both height and weight. The body mass index (BMI) was determined by dividing the weight in kilograms by the square of the height in meters, with 1 kg deducted to account for clothes. The subjects were divided into three groups based on their BMIs: healthy weight (BMI 25), overweight (BMI 25–30), and obesity (BMI > 30 kg/m^2). All participants were subjected to full history taking, and Laboratory investigations included: *H. Pylori* Ag. *H. pylori* Ab. Techniques for diagnosing *H. pylori* were divided into two categories: invasive testing and noninvasive testing. Included in non-invasive diagnostics are stool antigen detection and serological methods for *H. pylori* antibody identification. The isolation of serum. after an overnight fast, participants had one blood samples taken in plain tube (4ml each). After 30 minutes at room temperature of incubation, blood in clear tubes will be centrifuged at 4,000 rpm. At the lab visit, patients' body parameters were measured.

Liver enzymes (ALT, AST). Kidney function (Blood Urea, Serum Creatinine). Lipid Profile (Cholesterol, Triglycerides, HDL-c, LDL-c). Enzyme-linked immunosorbent assay (ELISA) technique (serum Lipten and Adiponectin Hormones).

Analysis of statistics

All statistical computations were performed using Microsoft Excel (Microsoft Corporation, New York, USA) and SPSS version 20 for Windows (SPSS Inc., Chicago, IL, USA). Results were presented in a mean and standard error (SE) format. The quantitative variables' means, standard deviations, and ranges were given as descriptions. The analysis of variance was used to compare quantitative data acquired from the same group at different dates. The LSD was used for a post hoc analysis when there was a significant difference between the groups. To ascertain the degree of interdependence between variables, a simple linear correlation study was performed using the statistical programme MedCalc (Ostend, Belgium). Statistical significance was determined using the P 0.05 threshold.

Results

Table (1) showed clinical and biochemical parameters covered in the studied groups (Age, Gender, BMI, CHOL, TGs, HDL-c, LDL-c). There is a statistically significant diffrence in Age and body mass index between the groups,but not in age. CHOL, TGs and LDL-c were signifcantly increased in group 2 and group 3 as compared to control group. While HDL-c was signifcantly decreased in group 2 and group 3 as compared to control group.

Table (2) indicated lipten and Adiponectin concentrations in studied groups. There is a statistically significant diffrence in lipten and Adiponectin between the studied groups. Lipten was signifcantly increased in group 2 and group 3 as compared to control group. While Adiponectin was signifcantly decreased in group 2 and group 3 as compared to control group. **Fig. (1)** showed that obese groups with and without *H. pylori* infection had significantly higher leptin levels than control group. On the other hand, **Fig. (2)** indicated that obese groups with and without *H. pylori* infection had significantly lower Adiponectin levels than control group.

Table (3 and 4) showed that leptin was a significant positively correlated to BMI and Adiponectin was negatively correlated to BMI in Obese pateints Without *H.Pylori* infection group 2 . In addition, **Table (5 and 6)** showed that leptin showed a significant positive correlation with BMI, whreas

Adiponectin was negatively correlated to BMI in Obese pateints with *H. pylori* infection group 3.

Table (1): Clinical and biochemical parameters among the studied groups

Clinical and biochemicals parameters	Statistics parameters	Group I (n =50)	Group II (n =50)	Group III (n =50)	P value
Age (years)	Mean ± SE	31.24 ± 2.59	48.44 ± 2.2	49.64 ± 2.3	<0.001*
	Range	19 – 59	30 – 68	23 – 70	
Gender	Male	18 (36%)	14 (28%)	16 (32%)	0.832
	Female	32 (64%)	36 (72%)	34 (68%)	
BMI (Kg/m^2)	Mean ± SE	23.73 ± 0.48	33.19 ± 0.6	34.76 ± 0.55	<0.001*
	Range	20.28 – 27.22	30.0 – 40.0	30.0 – 40.0	
CHOL (mg/dL)	Mean ± SE	137.52 ± 4.05	211.32 ± 11.42	191.00 ± 9.34	<0.001*
	Range	100 – 203	100 – 289	100 – 278	
TGs (mg/dL)	Mean ± SE	66.66 ± 4.24	137.12 ± 7.44	124.04 ± 6.08	<0.001*
	Range	41 – 132	65 – 188	62 – 180	
HDL (mg/dL)	Mean ± SE	48.14 ± 1.64	36.64 ± 1.56	39.04 ± 1.40	<0.001*
	Range	35 – 62	27 – 52	30 – 53	
LDL (mg/dL)	Mean ± SE	70.01 ± 4.08	147.36 ± 11.49	126.90 ± 9.50	<0.001*

*Statistically significant as p ≤0.05; BMI , Body Mass Index CHOL, cholesterol , TGs, Triglycerides

Table (2): Comparison of Leptin and Adiponectin concentration (ng/dL) among the studied groups

Leptin and Adiponectin	Statistics parameters	Group I (n =50)	Group II (n =50)	Group III (n =50)	P value
Lipten (ng/dL)	**Mean ± SE**	10.21 ± 0.02	28.42 ± 1.4	26.92 ± 1.21	<0.001*
	Range	10 – 10.3	17.2 – 39.5	15.1 – 35.1	
Adiponectin (ng/dL)	**Mean ± SE**	15.99 ± 0.47	8.04 ± 0.83	8.42 ± 0.8	<0.001*

*Statistically significant as p ≤0.05; BMI , Body Mass Index CHOL, cholesterol , TGs,Triglycerides

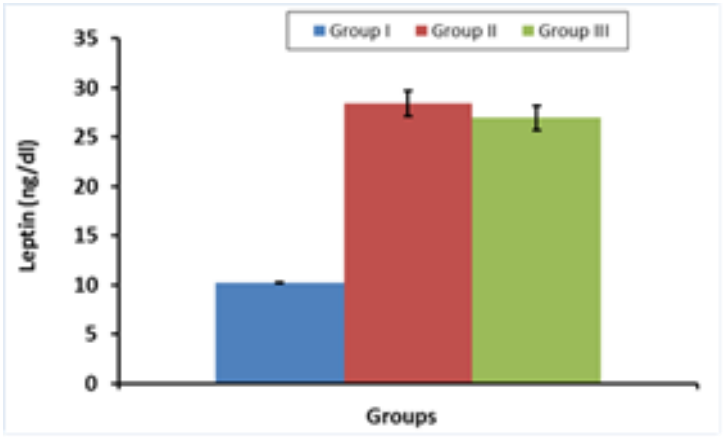

Fig. (1): Leptin hormone (ng/dL) of Group I: healthy controls; Group II: Obese + *H.pylori* negative infection; Group III: Obese *H.pylori* positive infection

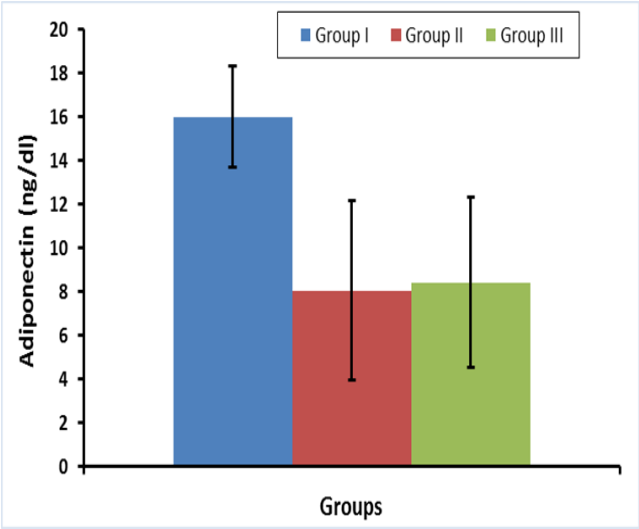

Fig. (2): Adiponectin hormone (ng/dL) of Group I: healthy controls;Group II: Obese + *H.pylori* negative infection; Group III: Obese + *H.pylori* positive infection

Table (3): Correlation between Leptin and BMI in Obese Pateints without *H.pylori* infection

Correlations		
	Leptin	
	R	**P=value**
BMI	0.764	0.001*

*** Statistically significant as p ≤0.05**

Table (4): Correlation between Adiponectin and BMI in Obese Pateints without *H. pylori* infection

Correlations		
	Adiponectin	
	R	**P= value**
BMI	-0.700	0.001*

*** Statistically significant as p ≤0.05**

Table (5): Correlation between Leptin and BMI in Obese Pateints with *H. pylori* infection

Correlations		
	Leptin	
	R	**P= value**
BMI	0.815	0.001*

*** Statistically significant as p ≤0.05**

Table (6): Correlation between Adiponectin and BMI in Obese Pateints with *H. pylori* infection

Correlations		
	Adiponectin	
	R	**P= value**
BMI	-0.715	.001*

*** Statistically significant as p ≤0.05**

Discussion

Bacterium known as *H. pylori* has been linked to gastric cancer and ulcer illness. The latter is a common type of cancer that accounts for 4% of all cancer-related deaths worldwide **(Sharndama and Mba, 2022)**.

Obesity is defined as a body mass index (BMI) of 30 or more, that has been linked with a higher risk of acquiring diabetes mellitus, cardiovascular disease, elevated levels of both blood pressure and cholesterol For the last 50 years, this public health catastrophe has become much worse **(Masuda and Yamashita, 2017)**.

Thjodleifsson *et al.*, (2008) have discovered *H. pylori* infection in their study of 985 Swedish participants (mean age 42 years). Additionally, two studies from Taiwan and China, found that patients who were overweight and obese people were more likely to have *H. pylori*. than those who have normal weight **(Xu *et al.*, 2019; Zhang *et al.*, 2015)**. Researchers found that the rates of *H. pylori* infection among normal-weight, overweight, and obese people all rose over time with 37.36 percent, 41.8 percent, and 45.77 percent, respectively **(Zhang *et al.*, 2015)**. Other cross-sectional research, however, has not discovered a connection between infection with *H. pylori* and the likelihood of becoming overweight **(Kyriazanos *et al.*, 2002; Cho *et al.*, 2005; Ioannou *et al.*, 2005; Méndez-Sánchez *et al.*, 2007).** According to other research *H. pylori* colonization was not linked with BMI in Japanese and Greek participants **(Archimandritis *et al.*, 2003; KAWANO *et al.*, 2001; Kyriazanos *et al.*, 2002*)***. Studies conducted in the United States found no relationship between *H. pylori* and overweight/obesity **(Cho *et al.*, 2005; Ioannou *et al.*, 2005)** . Patients under 50 years old who have *H. pylori* infection may have a higher risk of obesity than those who do not have this infection **(Chen *et al.*, 2018)**.

Ashraf *et al.*, (2018) study showed that obese patients were younger and more probable to be female than non-obese patients . This finding is consistent with our findings, which showed that there were more females than males in all groups. Body mass index (BMI) is one of the most generally recognized indicators of obesity; in the current study, patient groups' BMIs were considerably higher than those of the healthy controls.

One of the most used methods of measuring obesity is body mass index (BMI); in the current study, patient groups' BMIs were considerably higher than those of the healthy controls. This study, was discovered that, across all

groups, obesity as measured by BMI exhibited a clear positive link with LDL-c and TGs levels and a HDL-c strongly negatively correlated, similar result was mentioned with **(Jain, 2022; Van Hemelrijck *et al.*, 2018)**. They found lipid profile and BMI-related statistically. These results may potentially be due to increased levels of TGs synthesis substrates from increased free fatty acid (FFA) mobilization from adipose tissue in obese individuals **(Hussain *et al.*, 2019)**. Additionally, the liver secretes more LDL-c than usual, and TG-rich lipoproteins are removed slowly.

Close results were previously reported **(Hussain *et al.*, 2019; Milyani and Al-Agha, 2019).** Current results were in agreement with the results of **(Barbalho *et al.*, 2019)** who observed a link between dyslipidemia, atherogenic indices, and the development of metabolic syndrome and both HDL-c and non-HDL-c. **Otsuka *et al.*, (2016)** concluded that high serum levels of LDL-c, TC and non HDL-c were linked with an elevated risk of hypertension. The data of the present study showed a significant elevation in serum TC, TGs, and LDL-c with both patient groups, and on the contrary, our results exhibited a significant inhibition in serum HDL-c with both patient groups. Also, the results showed that serum leptin levels in both patient groups had significantly increased. Current results were almost consistent with **(Tsai, 2017)** who found that elevated serum leptin levels were linked to the growth of the metabolic syndrome, diabetes mellitus, hypertension, and a number of cardiovascular illnesses. **Kasai *et al.*, (2016)** hypothesized that leptin secretion, adipose tissue mass, and gut mucosal damage have a complicated interaction, and BMI and plasma leptin levels had a positive relationship**.**

Variations in leptin and TNF- α levels in the stomach mucosa brought on by infection with *H. pylori* can affect changes in body weight **(Polyzos *et al.*, 2011; Roper *et al.*, 2008).** Convincing findings from recent cross-sectional studies showed a connection between *H. pylori* infection and BMI **(Wu *et al.*, 2005 and Zhang *et al.*, 2015).** There is a prediction that chronic inflammation and insulin resistance brought on by H. pylori colonization would result in a change in BMI and that cytokines (C-reactive protein and TNF) and adipokines would also play a role (adiponectin and leptin) **(Chen *et al.*, 2018).** Infection with *H. pylori* significantly raised the expression of gastric leptin, and although blood levels of leptin did not alter significantly after infection with *H. pylori* was treated, gastric leptin expression did decrease

following *H. pylori* removal **(Chuang *et al.*, 2009).**

The relationship between serum adiponectin and infection with *H. pylori* has not been extensively studied. Adiponectin levels in the serum were not significantly different between HP positive and negative individuals, but after HP elimination, they rose **(Ando *et al.*, 2013)**. This study shows a reduction in serum level of adiponectin in all groups, that in agree with study conducted with **Torisu *et al.*, (2009)**. It showed that there was no discernible difference between those who tested negative or positive for *H. pylori* and those who had atrophic gastritis in terms of the level of circulating adiponectin.

Alkhalidy *et al.*, (2018) have demonstrated that adiponectin has a significant part in gastritis, particularly when *H. pylori* infection is present. Leptin levels increased and adiponectin levels decreased in obese patients, suggesting leptin resistance and adiponectin deficit. In these circumstances, elevated leptin levels may also increase the production of cytokines that promote inflammation, such as IL-6, TNF-a and lead to insulin resistance **(Abdel-Moneim *et al.*, 2019)**.

Conclusion

Current study suggests that Leptin and Adiponectin concentrations in obese individuals dissimilar to those in people with normal BMI. There is no discernible a distinction between adipocytokine levels (including Adiponectin and leptin) between people with and without *H. pylori*.

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هرمون الليبتن والاديبونكتن وعلاقتهم بعدوى الميكروب الحلزونى في مرضي السمنه

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[2]قسم المعامل، مستشفي ملوى التخصصي ، المنيا، مصر.
[3]قسم الباطنه والامراض المعديه.كليه الطب جامعه الازهر، اسيوط، مصر.

تسبب عدوى الملوية البوابية التهابًا مزمنًا وترتبط بتغيرات مؤشر كتلة الجسم (مؤشر كتلة الجسم ، كجم / م^2). تقييم العلاقه بين العدوى بجرثومة المعدة ومرض السمنه وذلك عن طريق قياس انزيمات الكبد والكلى وتحليل الدهون الكاملة والهرمونات المرتبطه بالسمنة (الليبتن والاديبونكتن) فى مرضى غير مصابون بعدوى جرثومة المعدة وآخرون مصابون بعدوى جرثومة المعدة ومدى انتشار جرثومة المعدة فى جميعهم.

ولقد تم فحص ١٥٠ مشارك منهم ٥٠ أصحاء كمجموعه ضابطه و١٠٠ من المرضي وتم تقسيمهم إلى ٥٠ مريض سمنة غير مصاب بعدوى جرثومة المعدة و٥٠ مريض سمنة مصاب بعدوى جرثومة المعدة فى جميعهم تم قياس مستوى هرمون الليبتين وهرمون اديبونيكتين وكذلك الاجسام المضادة لجرثومة المعدة والانتى جين الخاص بها فى البراز.

أوضحت هذه الدراسة وجود ارتفاع ملحوظ فى كل من الكلستيرول الكلي والكلستيرول الضار والأجسام المضادة لجرثومة المعدة، وهرمون النحافه (الليبتن) وكذلك نقص فى كل من الكلستيرول النافع وهرمون (الاديبونكتين) فى مرضى السمنه اثناء الإصابه بعدوى جرثومة المعدة وكذلك نفس القياسات فى المرضي المصابين وجدت في المرضي غير المصابين بعدوى جرثومة المعدة.

في الختام ، تشير البيانات التي تم جمعها ودراستها إلى أن تركيزات اللبتين والأديبونكتين في الأفراد الذين يعانون من السمنة تختلف عن تلك الموجودة في الأشخاص ذوي مؤشر كتلة الجسم الطبيعي. لا يوجد تمييز واضح بين مستويات (Adiponectin و leptin) بين الأشخاص الذين يعانون من جرثومة المعدة والاشخاص الذين لايعانون من الاصابه بجرثومه المعدة.