University of Thi-Qar Journal of Science (UTJsci)
E-ISSN: 2709-0256, P-ISSN: 1991-8690, Vol. 10, No. 2, Dec. 2023

Estimation of hematological parameters and lipid profile of coronary artery disease patients with *Helicobacter pylori*

1st Muntadher Abd ulateef Alhasan
Department of Biology/College of Science / University of Thi-Qar
Thi-Qar,Iraq
Email: muntadhera@sci.utq.edu.iq

2nd Manal Badi Salih
University of Thi-Qar /College of Science /Department of Biology
Iraq,Thi-Qar
Email: manalbadi_p@sci.utq.edu.iq

Received: 2023-07-05, Revised: 2023-07-17, Accepted: 2023-07-17, Published: 2023-12-22

***Abstract*— *Helicobacter pylori* infection causes generally gastric illnesses, however from the beginning of 1994, a few researchers have detailed a relationship of *H. pylori* with other systemic manifestations are outside stomach include various extra gastric appearances, for e.g., neurological, dermatological, hematologic, visual, cardiovascular, metabolic, hypersensitive, and hepatobiliary diseases. In this study, blood parameters, lipid profile, and fasting glucose were measured for patients with coronary artery disease with *H. pylori*, and the relationship of *H. pylori* with lipids, which is considered a risk factor for coronary artery disease, was measured. 150 people participated in the current study, and they were divided into three groups: The first group had arterial disease. coronary artery disease with *H. pylori* 50 people, the second group had coronary artery disease without *H. pylori* 50 people, the third group the control group 50 people. The samples were obtained from the heart center in Nasiriyah. Risk factors were taken into account: which included age, gender, smoking status, high blood pressure, and diabetes. In the first group, the number of males was 28 (56%) and females 22 (44%) with an average age of 72.60 ± 18.97, the second group had a number of males 33 (66%) and females 17 (34%) with an average age of 71.70 ± 21.06, the third group the number of males was 28 (56%) and females 22 (44%) with an average age of 55.44 ± 18.53. There were statistically significant differences (p≤0.05) in the mean lipid profile compared between the study groups. The causes of hyperlipidemia were found in patients with H. pylori who had coronary artery disease**

Keywords— *H. pylori*, coronary artery disease, lipid profile

I. INTRODUCTION

Coronary artery disease is one of the main causes of mortality in the developed countries. Diabetes, lipid profile disorders, hypertension, and smoking are the common risk factors of thermogenesis, which result in the coronary artery disease [1]. Although understanding of the relationships between the traditional risk factors for coronary artery disease has grown, the processes behind the illness's death have not been fully elucidated, therefore, it is crucial to research the connections between the disease and other risk factors, Various investigations looked into the connection between persistent infection and ischemic heart disease *H. pylori* infection has been linked in several studies to coronary heart disease risk factors [2].

A significant link between *H. pylori* infection, metabolic risk factors, atherogenesis, and cardiovascular illnesses has been documented in several research, *Helicobacter pylori* infection is still one of the most prevalent infections in the world [2-3]. Finding the pathogenicity mechanism of *H. pylori* are crucial to understanding the prevalence of cardiovascular disorders, in this regard, several of the animal model studies revealed some findings; for instance, Elizalde *et al* suggested that *H. pylori* infection resulted in platelet aggregation in rats [4]. There are numerous findings on the connection between chronic atherogenesis and *H. pylori* infection, although the mechanism is still not fully understood [5]. Changes in blood lipid levels, such as low density lipoprotein (LDL) and high density lipoprotein (HDL) cholesterol, are among the metabolic risk factors that have been linked to cardiovascular disease [6]. According to certain additional

https://doi.org/10.32792/utq/utjsci/v10i2.1073

research, *H. pylori* infection may alter blood lipid levels and heighten atherogenesis risk factors [7].

It is widely recognized that *Helicobacter pylori* may cause gastritis [8], peptic ulcer disease, and mucosa-associated lymphoid tissue lymphoma and gastric cancer [9]. There have also been theories about possible connections between *H. pylori* infection and conditions other than digestive problems, such iron deficiency anemia and idiopathic thrombocytopenic purpura [10]. Numerous studies have shown a link between cardiovascular illness, aberrant lipid levels, and seropositivity for *H. pylori*, suggesting a still-controversial causative association [11]. On the other hand, there are few instances of an association between lipids, cardiovascular disease, and an active *H. pylori* infection. Additionally, despite reports of how eliminating *H. pylori* affects lipid levels [12-14-15].

II. PATIENTS AND METHODS

The current study was conducted on 100 patients with coronary artery disease, who were divided into two groups, and the third group was the control group.

The first group (G1): infected people *H. pylori* with coronary artery disease, which numbered 50 people, the second group (G2): those who did not have *H. pylori* with coronary artery disease, whose number reached 50 people, the third group (G3): the control group who did not have *H. pylori* or coronary artery disease, nor did they get infected with it Any heart disease, depending on clinical examinations and phenotypic symptoms. In the first group, the number of males was 28 (56%) and females 22 (44%) with an average age of 72.60 ± 18.97, while in the second group the number of males was 33 (66%), females 17 (34%) with an average age of 71.70 ± 21.06, while in the group Third, the number of males was 28 (56%), females 22 (44%), and the mean age was 55.44 ± 18.53.

This study was conducted at Al Nasiriyah Heart Center and consent was obtained from the patients.

Data were collected through the use of a questionnaire that included medical history, smoking status, weight, blood pressure and diabetes mellitus. Blood samples were drained from the anterior vein during morning fasting. Serum samples were separated after centrifugation. Low-density lipoprotein cholesterol was estimated using the method of. Serum high-density lipoprotein cholesterol (HDL) levels were estimated using the Randox kit. It is one of the enzymatic methods. Serum total cholesterol level was estimated using the enzymatic method by Randox kit. The level of triglycerides in blood serum was estimated using the analysis kit (kit) from the company (Randox), which is one of the enzymatic methods. Fasting blood glucose (FBS) was also measured using the hexokinase enzymatic method.

Blood test calculated by CELL-DYN Ruby Auto Analyzer. The system is used to quantify, calculate and compute hematological parameters and the measurement process for total WBC and differential WBC (neutrophils, lymphocytes, monocytes, eosinophils, basophils).

Blood pressure was measured by a trained nurse using a mercury manometer, after 5-minute resting in sitting position.

A. Statistical analysis

The data of the current study was statically analyzed using SPSS (Statistical Package of Society Science version 26) using one-way (ANOVA) for mean variation, least significant difference, independent t-test and chi square.

III. RESULTS

The current study was conducted on 100 patients who were divided into two groups, in addition to 50 people, the control group, from September 2022 to March 2023. Demographic data are shown in Table 1.

TABLE 1. DEMOGRAPHIC DATA OF THE PATIENTS AND CONTROL INVOLVED IN THE STUDY ACCORDING ON RISK FACTORS.

Group	G1 (N = 50)	G2 (N = 50)	G3 (N = 50)	*P. value*
Age in years (M± S.D)				
	72.60± 18.97	71.70± 21.06	55.44±18.53	
Gender				
Male	28 (56%)	33 (66%)	28 (56%)	0.50
Female	22 (44%)	17 (34%)	22 (44%)	
Weight (M± S.D)				
	88.64±15.88	91.94± 18.13	81.36±11.50	
Smoking				
Smoker	29 (58%)	26 (52%)	0 (0%)	0.000*
Non-smoker	21 (42%)	24 (48%)	50 (100%)	
Hypertension				
Yes	36 (72%)	21(42%)	-	0.002*
No	14 (28%)	29 (58%)	-	
Diabetes				
Yes	28 (56%)	23 (46%)	-	0.317
No	22 (44%)	27 (54%)	-	

M±S.D MEAN ± STANDARD DIVASION, P. VALUE ≤ 0.05 MEANS SIGNIFICANT

According to Table 2, there were significant differences between the three groups of average RBCs (4.84 b ± 0.97, 5.15 a ± 0.93, 4.44 c ± 0.76), respectively. While the level of Hb did not record significant differences between the first and second groups (13.21 ^{a} ± 2.17, 13.19 ^{a} ± 1.35), respectively. However, the third group had significant differences (11.93 ^{b} ± 1.73) below the level of significance $p \leq 0.05$. The level of HCT was statistically significant below $p \leq 0.05$ among the three study groups (39.42 ^{a} ± 3.98, 36.34 ^{b} ± 5.84, 33.51 ^{c} ± 5.66). While the level of PLT did not record significant differences between the first and second groups (285.70 ^{a} ± 45.35, 281.70 ^{a} ±45.33), respectively. However, the third group had significant differences (184.70 ^{b} ± 43.82) below the level of significance $p \leq 0.05$.

TABLE 2. RED BLOOD CELLS COUNT (RBC)

Parameter / Groups	RBCs *10^6 /ml	Hb gm/dl	HCT %	PLT ×10^{3}µL
	M ± S.D			
G1	4.84 ^{b} ± 0.97	13.21 ^{a} ± 2.17	39.42 ^{a} ± 3.98	285.70 ^{a} ± 45.35
G2	5.15 ^{a} ± 0.93	13.19 ^{a} ± 1.35	36.34 ^{b} ± 5.84	281.70 ^{a} ±45.33
G3	4.44 ^{c} ± 0.76	11.93 ^{b} ± 1.73	33.51 ^{c} ± 5.66	184.70 ^{b} ± 43.82
P. Value	**0.001***	**< 0.001***	**< 0.001***	**< 0.001***
L.S.D	**0.29**	**0.59**	**1.74**	**14.97**

The results of the current study have shown that there were statistically significant differences at ($p \leq 0.05$) between the number of white blood cells in the first group, the second group, and the third group, which were 7.18 ± 1.51, 5.69 ± 1.44, 5.13 ± 0.77, respectively. The results showed that there were statistically significant differences at ($p \leq 0.05$) between the number of neutrophils between the two groups of patients, as well as between the two groups of patients compared to the control group, 4.30 ± 0.91, 3.43 ± 0.88, 3.08 ± 0.46, respectively. The results of the current study showed that there were statistically significant differences at (p 0.05) between the number of lymphocytes among the three study groups, 1.79 ± 0.37, 1.42 ± 0.36, 1.28 ± 0.19, respectively. These results also showed that there were statistically significant differences at (p 0.05) between the number of MON% in the study groups 0.49 ± 0.10, 0.39 ± 0.10, 0.35 ± 0.05, respectively. These results also showed that there were statistically significant differences at (p 0.05) between the number of EO% in the study groups 0.28 ± 0.06, 0.23 ± 0.06, 0.21 ± 0.03, respectively. These results also showed that there were statistically significant differences at (p 0.05) between the number of BASO% in the study groups 0.14 ± 0.03, 0.11 ± 0.03, 0.10 ± 0.01, respectively. as showed in Table 3

TABLE 3. WHITE BLOOD CELLS (WBC)

Parameter / Groups	G1	G2	G3	***P. Value***	**L.S.D**
WBC ×10^{3}µL M ± S.D	7.18^a ±1.51	5.69^b ±1.44	5.13^c ±0.77	**< 0.001***	**0.43**
Neutrophils ×10^{3}µL M ± S.D	4.30^a ±0.91	3.43^b ±0.88	3.08^c ±0.46	**< 0.001***	**0.26**
Lymphocyte ×10^{3}µL M ± S.D	1.79^a ±0.37	1.42^b ±0.36	1.28^c± 0.19	**< 0.001***	**0.10**
Monocyte ×10^{3}µL M ± S.D	0.49a± 0.10	0.39b± 0.10	0.35c± 0.05	**< 0.001***	**0.029**
Eosinophils ×10^{3}µL M ± S.D	0.28a± 0.06	0.23b± 0.06	0.21c± 0.03	**< 0.001***	**0.018**
Basophils ×10^{3}µL M ± S.D	0.14a ±0.03	0.11b ±0.03	0.10b ±0.01	**< 0.001***	**0.01**

The level of HDL decreased in the first group (37.24 ± 4.64) and the second (36.70 ± 7.05), compared to the third group (38.90 ± 7.99), but there were no statistically significant differences at the level ($p \leq 0.05$). The LDL level increased in the first group (150.82 ± 11.12) and the second group was (149.52 ± 20.27) compared to the third group (148.22 ± 29.26), but there were no statistically significant differences at the level ($p \leq 0.05$).The results also showed that the level of cholesterol in the blood increased at ($p \leq 0.05$) in the first two groups 263.82 ± 28.41 and the second (245.44 ± 26.7) compared to the third group (151.08 ± 36.11), and there were statistically significant differences at the level ($p \leq 0.05$).The results also showed that the level of triglycerides in the blood increased in the first two groups (223.68 ± 27.48) and the second by (256.80 ± 25.81) compared to the third group by (176.54 ± 39.67),and there are statistically significant differences at the level ($p \leq 0.05$) between the study groups. as showed in Table 4

TABLE 4. SERUM LIPID PROFILE OF THE STUDY GROUP

Parameter / Group	HDL-C (mg/dL)	LDL-C (mg/dL)	Total-C (mg/dL)	Tri (mg/dL)
	M ± S.D			
G1	37.24 ^{a} ±4.64	150.82^a ±11.12	263.82^a ±28.41	223.68 ^{b} ± 27.48
G2	36.70 ^{a} ±7.05	149.52 ^{a} ± 20.27	245.44^b ±26.7	256.80 ^{a} ± 25.81
G3	38.90 ^{a} ±7.99	148.22^a ± 29.26	151.08^c ±36.11	176.54 ^{c} ± 39.67
P. Value	**0.236**	**0.834**	**0.001***	**0.001***
LSD	**2.25**	**7.19**	**10.25**	**10.55**

Determine the FBS of all samples of the study aggregates as mentioned in the table 5. The results of the current study showed that there were significant differences between the study groups. There is an increase in the FBS level of the first group 116.46 ± 24.79 compared to the second group 115.92 ± 11.75, but there are no significant differences at the level ($p \leq 0.05$). While compared to the third group (104.30 ± 11.78), there are statistically significant differences at the level ($p \leq 0.05$).

TABLE 5. FASTING BLOOD SUGAR OF THE STUDY GROUP

Parameter / Group	**FBS mg/dL M ± S.D**
G1	116.46^a±24.79
G2	115.92^a±11.75
G3	104.30^b±11.78
P. Value	**0.001***
LSD	**5.75**

IV. DISCUSSION

Many factors related to red blood cells are associated with coronary artery disease, including levels of hemoglobin, hematocrit, and platelets. However, there was

insufficient data on this association between cardiovascular disease and coronary artery disease [15].

Red blood cells decreased in the first group compared to the second group, while both groups recorded a significant increase compared to the third group. The decrease in red blood cells may be due to iron deficiency resulting from *H. pylori* infection, because the process of *H. pylori* infection reduces the absorption of iron in the body [16], as well as leads to deficiency Vitamin B12, which in turn contributes to a decrease in the number of red blood cells [17]. However, there is not enough data evidence to indicate an association between the number of red blood cells and cardiovascular disease [15] .

Through the results and based on previous studies, there is an important relationship between hemoglobin and some risk factors for heart diseases. The high level of hemoglobin may be an independent risk factor, but its effect may be secondary because it is directly related to other factors [18-19].

As for the hematocrit, it was found to have increased in the first and second groups compared to the third group, as these results agreed with previous studies that indicated a relationship between hematocrit and cardiovascular diseases, myocardial infarction and coronary artery disease [20]. Consistent with the findings of previous studies, our results indicate that elevated HCT is associated with several CVD risk factors, including cigarette smoking, blood pressure, and total cholesterol [21].

Increase in the number of platelets in the first and second groups compared to the control group, there was no significant difference between the first and second groups, as infection with *H. pylori* had no effect on the number of platelets [22]. Where the increase in the first and second groups compared to the control group may be due to risk factors such as smoking, obesity and high blood pressure.

There is a study that proved the involvement of platelets in heart diseases, especially myocardial infarction and coronary artery disease, as a study found that coronary artery obstruction in a high percentage of patients is due to a clot that causes the formation of an obstructive plaque in the coronary artery [23].

Hypertension is one of the risk factors that have a strong association with an increase in the number of platelets, as persistent pressure disorders cause metabolic disorders and functional changes, and thus cause changes in the vascular endothelium, platelets, and an increase in blood plasma, which is considered one of the substances that contribute to coagulation, which can be linked to the development of heart diseases especially coronary artery disease [24-25].

White blood cells(WBCs) are blood parameters by which we can diagnose many different diseases and health conditions. WBCs protect the body from invading microorganisms, and their high levels are an indication of inflammation. The number of white blood cells is associated with an increased risk of cardiovascular disease [26].

The presence of *H. pylori* in the gastric mucosa leads to the activation of innate defense mechanisms by the host's body, and this leads to stimulation of the expression of several anti-inflammatory and antibacterial factors by epithelial cells in the host's stomach [27]. The results of the current study showed in Table 3 that there was a significant difference at ($P < 0.05$) in the number of white blood cells between the three study groups. The difference in the first group was due to the bacterial infection by *H. pylori,* even if the infection was old. But the presence of latent bacteria in the body towards pathological agents stimulates the immune response and an increase in the number of white blood cells, *H. pylori* is an active stimulator of both innate and adaptive immune responses [28]**.** There was also a statistically significant difference in the second group compared to the control group, although the second group were individuals who did not have *H. pylori*, due to several factors that may be due to smoking, which increases pathological factors and thus increases white blood cells [29].

Also, neutrophils, lymphocytes, monocytes, eosinophils, and basophils showed significant and statistically significant differences between the three study groups. Neutrophil count by itself could predict both acute and chronic CVD [30-31]. Interestingly, an elevated neutrophil ratio has not only been associated with CVD but also with short term adverse CVD outcomes, including mortality, coronary artery disease (CAD), stroke and heart failure [18].

Inflammation plays an active role in the initiation of heart disease and the process of atherosclerosis and disease progression. The presence of white blood cells in general is a sign of inflammation, and the presence of subtypes of white blood cells predicts the presence of atherosclerosis and other heart diseases. Lymphocytes play an important role in modulating the inflammatory response. In heart disease patients, in addition to its involvement in the immune system's regulatory pathway [32],

Monocytes are produced as a result of an inflammatory condition where inflammatory cytokines are increased in patients with coronary arteries or heart infarction [33].A result of the catalytic activation of monocytes, various cytokines are released and low-density lipoprotein can be oxidized, then endothelial cells with monocytes produce large amounts of MCP 1- which inhibits the anticoagulant properties of the vascular wall [34].

The increased coronary risk was associated with elevated neutrophils, eosinophils, lymphocytes, monocytes, or basophils[35].

Eosinophils are white blood cells with multiple functions, implicated in the pathogenesis of many inflammatory processes including allergic diseases. infection of tumor immune tissue, bacterial and viral infections, and helminths. Eosinophils are recruited from the circulation to inflammatory sites where they modulate immune responses through a range of mechanisms [36]. Eosinophils are considered as a biomarker in staging patients with coronary artery disease. In the inflammatory state, eosinophils secrete a protein called ribonuclease-binding proteinase, which is associated with hyperbaric heparin, which is secreted exclusively by eosinophils and serves as a tool to monitor hypersensitivity inflammation. Acid-base positive protein levels are associated with adverse cardiac events. Including myocardial infarction, recurrent coronary events and sudden death [35].

Basophils are effective cells in the allergic response and may be responsible for anaphylaxis in, there is evidence that anaphylaxis is frequently associated with the effect of the heart muscle due to coronary artery spasm and thrombosis that appears in the coronary artery, there is a study showing that there are patients suffering from cardiogenic shock and

myocardial infarction, so they are treated with anti-allergic drugs, and this indicates that the heart, especially the coronary arteries, is affected by hypersensitivity, and therefore the basophils increase at that time [37].There are reports of sudden death due to coronary artery spasm affecting the coronary arteries. In a patient with recurrent episodes of coronary artery spasm, who died suddenly after stopping his treatment, an increase in the number of basophils and mast cells was found [20-38].

The results of the current study, Table (4), indicated that there was a decrease in the level of high-density lipoprotein in the first and second groups compared with the third group, but it was not statistically significant. These results are consistent with the study [39] and the study of [40], The low level of high-density lipoprotein may be due to several reasons, either due to diabetes [41] or cigarette smoking [42] or due to inflammatory factors [43]. Also, women who use oral contraceptives that contain progestin[44] have low HDL cholesterol.

Decreased activity of the enzyme Lipoprotein Lipase (LPL), which leads to inhibition of the decomposition of triglycerides into fatty acids and glycerol, as well as an increase in the effectiveness of hepatic lipase (HL), as the high-density lipoprotein is rich in glycerol, and thus it is one of the basic materials that the liver lipase enzyme works on. (HL) This causes rapid removal of high-density lipoprotein from the circulatory system and, as a result, a decrease in its level in the serum [45].

The results of the current study, Table 4, showed that there was an increase in the level of low-density lipoprotein (LDL) in the first and second groups compared to the third group, but it was not statistically significant.

This is consistent with the study [46]. The reason for the increase in the concentration of (LDL) is the decrease in the activity of the enzyme Lipo Protein Lipase (LPL), which leads to the non-dissolution of triglycerides and the conversion of most of the (VLDL) to medium-density lipoprotein, and then the formation of low-density lipoprotein (LDL) and its increase [47]. In addition, an increase in cholesterol in the tissues and blood vessels reduces the activity of LDL receptors, and this results in the accumulation of LDL particles in high concentrations in the blood.

An increase in the level of LDL in the blood is a risk factor for the development of coronary heart disease, so this increase is undesirable, and the inefficiency of the hepatic receptors (Apo-B-100), which is important because it reduces the transfer of LDL to the liver tissues and then increases its level, or there may be a defect in the binding of LDL lipoprotein to the scavenging receptors in the liver through the protein part (Apo-B-100), which leads to an increase in its level [46]. When the process of removing LDL, which depends on the ability of the receptors, fails, the enzymes work to oxidize it across the surface of the vascular endothelium, turning it into oxidized LDL, especially when combined with high concentrations of triglycerides, since most of its LDL molecules are small and dense, and therefore they are more ready for the oxidation process [1].

In patients with coronary artery disease, progressive accumulation of cholesterol in the artery wall, many studies have found an association between high cholesterol levels and cardiovascular events, particularly coronary artery disease [48].

The current results show a significant increase in cholesterol and triglyceride levels for the first group compared to the second group, while both groups show significant differences compared to the third group. These results are consistent with several studies [6-49]. It is possible that elevated cholesterol and triglyceride lipids in patients are due to *H. pylori*, although the mechanism of how *H. pylori* infection affects lipids is still unknown, but a plausible explanation is that the systemic inflammatory response to the bacteria leads to changes in lipid and lipoprotein metabolism [13]. This means that chronic *H. pylori* infection has been postulated to shift the lipid profile towards an atherosclerotic direction through the action of pro-inflammatory cytokines, such as interleukins 1 and 6, interferon-alpha, and tumor necrosis factor-alpha (TNF-α). These cytokines are able to affect lipid metabolism in various ways, including activating adipose tissue lipoprotein lipase, stimulating hepatic fatty acid synthesis, influencing lipolysis and increasing hepatic Hydroxyl methyglutary-CoA (HMG-CoA) reductase activity [50]. Thus, *H. pylori* infection could play a role in the process of coronary atherosclerosis and may be a reliable indicator for cardiovascular disease risk assessment [2-51].

The results of the current study, Table 5, showed that there was a significant increase in fasting glucose concentration between the first and second groups compared to the third group. and statistically significant.

Our results showed that *H. pylori* infection is related to high blood sugar, as the results were consistent with a study [52]. The reason for the increased FBS level may be attributed to the fact that the bacteria induce insulin resistance due to the stimulation of systemic inflammation in patients with *H. pylori* infection [53]. The reason for the high level of glucose in diabetic patients is due to a lack of insulin secretion, a defect in the resistance of insulin receptors in the body, or an imbalance in the pancreas gland and metabolism, the presence of *H. pylori* infection is associated with the risk of metabolic syndrome and insulin resistance [54-55].

V. CONCLUSIONS

1) People who have coronary artery disease may be more susceptible to *H. pylori* infection due to a weak immune system or due to a defect in the white blood cells due to the stress caused by the disease.
2) High levels of triglycerides and high cholesterol lead to stress on the cells lining the inner layer of the coronary arteries, and thus lead to their hardening, which prevents their flexibility, which causes damage to the coronary arteries, in addition to a weak immune system due to stressed cells and the production of large quantities of white blood cells, which may accumulate in the bloodstream. This causes blockage of the coronary arteries.

CONFLICT OF INTEREST

Authors declare that they have no conflict of interest.

REFERENCES

[1] A. Onat *et al.*, "Predictors of abdominal obesity and high susceptibility of cardiometabolic risk to its increments among Turkish women: a prospective population-based study," *Metabolism.*, vol. 56, no. 3, pp. 348–356, 2007, doi: 10.1016/j.metabol.2006.10.016.

[2] J. Vafaeimanesh, S. F. Hejazi, V. Damanpak, M. Vahedian, M. Sattari, and M. Seyyedmajidi, "Association of helicobacter pylori infection with coronary artery disease: Is helicobacter pylori a risk factor?," *Sci. World J.*, vol. 2014, 2014, doi: 10.1155/2014/516354.

[3] G. H. Dahlén, J. Boman, L. S. Birgander, and B. Lindblom, "Lp(a) lipoprotein, IgG, IgA and IgM antibodies to Chlamydia pneumoniae and HLA class II genotype in early coronary artery disease," *Atherosclerosis*, vol. 114, no. 2, pp. 165–174, 1995, doi: 10.1016/0021-9150(94)05480-7.

[4] J. I. Elizalde *et al.*, "Platelet activation in mice and human Helicobacter pylori infection," *J. Clin. Invest.*, vol. 100, no. 5, pp. 996–1005, 1997, doi: 10.1172/JCI119650.

[5] B. Factors, "REVIEWS IN BASIC AND CLINICAL," pp. 306–323, 2008, doi: 10.1053/j.gastro.2007.11.009.

[6] J. Sasaki *et al.*, "Gender difference in coronary events in relation to risk factors in Japanese hypercholesterolemic patients treated with low-dose simvastatin," *Circ. J.*, vol. 70, no. 7, pp. 810–814, 2006, doi: 10.1253/circj.70.810.

[7] P. Cullen and G. Assmann, "High risk strategies for atherosclerosis," *Clin. Chim. Acta*, vol. 286, no. 1–2, pp. 31–45, 1999, doi: 10.1016/S0009-8981(99)00092-3.

[8] B. M. Kadhim, M. B. Salih, and H. S. Abdulhussein, "Molecular Detection of Helicobacter pylori by Real Time- PCR in Dyspeptic Patients Abstract :," vol. 6, no. 4, 2018.

[9] B. L. Smoak, P. W. Kelley, and D. N. Taylor, "Seroprevalence of Helicobacter pylori infections in a cohort of US army recruits," *Am. J. Epidemiol.*, vol. 139, no. 5, pp. 513–519, 1994, doi: 10.1093/oxfordjournals.aje.a117034.

[10] M. Ferrara, L. Capozzi, and R. Russo, "Effect of helicobacter pylori eradication on platelet count in children with chronic idiopathic thrombocytopenic purpura," *Hematology*, vol. 14, no. 5, pp. 282–285, 2009, doi: 10.1179/102453309X12473408860181.

[11] A. Prasad, J. Zhu, J. P. J. Halcox, M. A. Waclawiw, S. E. Epstein, and A. A. Quyyumi, "Predisposition to atherosclerosis by infections: Role of endothelial dysfunction," *Circulation*, vol. 106, no. 2, pp. 184–190, 2002, doi: 10.1161/01.CIR.0000021125.83697.21.

[12] H. Scharnagl, M. Kist, A. B. Grawitz, W. Koenig, H. Wieland, and W. März, "Effect of Helicobacter pylori eradication on high-density lipoprotein cholesterol," *Am. J. Cardiol.*, vol. 93, no. 2, pp. 219–220, 2004, doi: 10.1016/j.amjcard.2003.09.045.

[13] M. Yucel and S. Boyacioglu, "Does Eradication of Helicobacter pylori Infection Help Normalize Serum Lipid and CRP Levels ?," no. May 2018, 2005, doi: 10.1007/s10620-005-2764-9.

[14] C. Y. Jeon *et al.*, "Helicobacter pylori infection is associated with an increased rate of diabetes," *Diabetes Care*, vol. 35, no. 3, pp. 520–525, 2012, doi: 10.2337/dc11-1043.

[15] M. Madjid and O. Fatemi, "Components of the complete blood count as risk predictors for coronary heart disease: In-depth review and update," *Texas Hear. Inst. J.*, vol. 40, no. 1, pp. 17–29, 2013.

[16] D. S. Mahdi, M. Thameraltemimi, A. Endocrinologist, S. D. Endocrine, and H. Directorate, "The Prediction of Macrovascular Complications in Individuals with Type 2 diabetes mellitus with Different Risk Factors in ThiQar," vol. 1, no. 1, 2023.

[17] C. Ciacci *et al.*, "Helicobacter pylori impairs iron absorption in infected individuals," vol. 36, pp. 455–460, 2004, doi: 10.1016/j.dld.2004.02.008.

[18] S. Kim, M. Eliot, D. C. Koestler, W. C. Wu, and K. T. Kelsey, "Association of neutrophil-to-lymphocyte ratio with mortality and cardiovascular disease in the jackson heart study and modification by the duffy antigen variant," *JAMA Cardiol.*, vol. 3, no. 6, pp. 455–462, 2018, doi: 10.1001/jamacardio.2018.1042.

[19] H. A. H. Abu-Zeid and J. M. Chapman, "Hemoglobin level and ischemic heart disease: Relationship to known risk factors," *Prev. Med. (Baltim).*, vol. 6, no. 1, pp. 120–129, 1977, doi: 10.1016/0091-7435(77)90009-3.

[20] R. C. Schlant, S. Forman, J. Stamler, and P. L. Canner, "The natural history of coronary heart disease: Prognostic factors after recovery from myocardial infarction in 2789 men. The 5-year findings of the coronary drug project," *Circulation*, vol. 66, no. 2 I, pp. 401–414, 1982, doi: 10.1161/01.CIR.66.2.401.

[21] D. W. Brown, W. H. Giles, and J. B. Croft, "Hematocrit and the risk of coronary heart disease mortality," *Am. Heart J.*, vol. 142, no. 4, pp. 657–663, 2001, doi: 10.1067/mhj.2001.118467.

[22] M. Franchini, M. Cruciani, C. Mengoli, G. Pizzolo, and D. Veneri, "Effect of Helicobacter pylori eradication on platelet count in idiopathic thrombocytopenic purpura: A systematic review and meta-analysis," *J. Antimicrob. Chemother.*, vol. 60, no. 2, pp. 237–246, 2007, doi: 10.1093/jac/dkm195.

[23] P. C. Elwood, A. D. Beswick, D. S. Sharp, J. W. G. Yarnell, S. Rogers, and S. Renaud, "Whole blood impedance platelet aggregometry and ischemic heart disease. The Caerphilly Collaborative Heart Disease Study," *Arteriosclerosis*, vol. 10, no. 6, pp. 1032–1036, 1990, doi: 10.1161/01.atv.10.6.1032.

[24] G. Y. H. Lip, "Target organ damage and the prothrombotic state in hypertension," *Hypertension*,

vol. 36, no. 6, pp. 975–977, 2000, doi: 10.1161/01.HYP.36.6.975.

[25] M. Yazici, A. Kaya, Y. Kaya, S. Albayrak, H. Cinemre, and H. Ozhan, "Lifestyle modification decreases the mean platelet volume in prehypertensive patients," *Platelets*, vol. 20, no. 1, pp. 58–63, 2009, doi: 10.1080/09537100802613449.

[26] L. E. Charles, D. Fekedulegn, T. McCall, C. M. Burchfiel, M. E. Andrew, and J. M. Violanti, "Obesity, white blood cell counts, and platelet counts among police officers," *Obesity*, vol. 15, no. 11, pp. 2846–2854, 2007, doi: 10.1038/oby.2007.338.

[27] J. T. George, P. K. Boughan, H. Karageorgiou, and M. Bajaj-Elliott, "Host anti-microbial response to Helicobacter pylori infection," *Mol. Immunol.*, vol. 40, no. 7, pp. 451–456, 2003, doi: 10.1016/S0161-5890(03)00158-5.

[28] K. Robinson, R. H. Argent, and J. C. Atherton, "The inflammatory and immune response to Helicobacter pylori infection," *Best Pract. Res. Clin. Gastroenterol.*, vol. 21, no. 2, pp. 237–259, 2007, doi: 10.1016/j.bpg.2007.01.001.

[29] S. Lavi *et al.*, "Smoking is associated with epicardial coronary endothelial dysfunction and elevated white blood cell count in patients with chest pain and early coronary artery disease," *Circulation*, vol. 115, no. 20, pp. 2621–2627, 2007, doi: 10.1161/CIRCULATIONAHA.106.641654.

[30] T. Bhat, H. Bhat, M. Raza, G. Khoueiry, M. Meghani, and M. Akhtar, "Neutrophil to lymphocyte ratio and cardiovascular diseases : a review," *Expert Rev. Cardiovasc. Ther.*, vol. 11, no. 1, pp. 55–60, 2013.

[31] L. Guasti *et al.*, "Neutrophils and clinical outcomes in patients with acute coronary syndromes and/or cardiac revascularization: A systematic review on more than 34,000 subjects," *Thromb. Haemost.*, vol. 106, no. 4, pp. 591–599, 2011, doi: 10.1160/TH11-02-0096.

[32] C. Weber, A. Zernecke, and P. Libby, "The multifaceted contributions of leukocyte subsets to atherosclerosis: Lessons from mouse models," *Nat. Rev. Immunol.*, vol. 8, no. 10, pp. 802–815, 2008, doi: 10.1038/nri2415.

[33] L. Yao, K. Huang, D. Huang, J. Wang, H. Guo, and Y. Liao, "Acute myocardial infarction induced increases in plasma tumor necrosis factor-α and interleukin-10 are associated with the activation of poly(ADP-ribose) polymerase of circulating mononuclear cell," *Int. J. Cardiol.*, vol. 123, no. 3, pp. 366–368, 2008, doi: 10.1016/j.ijcard.2007.06.069.

[34] J. Mestas and K. Ley, "Monocyte-Endothelial Cell Interactions in the Development of Atherosclerosis," *Trends Cardiovasc. Med.*, vol. 18, no. 6, pp. 228–232, 2008, doi: 10.1016/j.tcm.2008.11.004.

[35] N. G. Kounis, G. D. Soufras, G. Tsigkas, and G. Hahalis, "White Blood Cell Counts , Leukocyte Ratios , and Eosinophils as Inflammatory Markers in Patients With Coronary Artery Disease," 2015, doi: 10.1177/1076029614531449.

[36] Y. M. Æ. F. Levi-schaffer, "Mast cells and eosinophils : the two key effector cells in allergic inflammation," 2009, doi: 10.1007/s00011-009-0042-6.

[37] N. George, K. Periklis, G. Hahalis, and A. Mazarakis, "The heart seems to be the primary site and the target of anaphylaxis resulting in the development of Kounis syndrome," vol. l, pp. 119–120, 2012, doi: 10.1007/s11739-012-0786-9.

[38] R. C. Schlant, "The Natural History of Coronary Heart Disease : Prognostic Factors After Recovery from Myocardial Infarction in 2789 Men," 1974.

[39] P. Keul, A. Polzin, K. Kaiser, M. Gr, and L. Dannenberg, "Potent anti-inflammatory properties of HDL in vascular smooth muscle cells mediated by HDL-S1P and their impairment in coronary artery disease due to lower HDL-S1P : a new aspect of HDL dysfunction and its therapy," pp. 1–14, 2018, doi: 10.1096/fj.201801245R.

[40] T. Duparc *et al.*, "Serum level of HDL particles are independently associated with long-term prognosis in patients with coronary artery disease: The GENES study," *Sci. Rep.*, vol. 10, no. 1, pp. 1–12, 2020, doi: 10.1038/s41598-020-65100-2.

[41] M. Femlak, A. Gluba-Brzózka, A. Ciałkowska-Rysz, and J. Rysz, "The role and function of HDL in patients with diabetes mellitus and the related cardiovascular risk," *Lipids Health Dis.*, vol. 16, no. 1, pp. 1–9, 2017, doi: 10.1186/s12944-017-0594-3.

[42] B. M. He, S. P. Zhao, and Z. Y. Peng, "Effects of cigarette smoking on HDL quantity and function: Implications for atherosclerosis," *J. Cell. Biochem.*, vol. 114, no. 11, pp. 2431–2436, 2013, doi: 10.1002/jcb.24581.

[43] X. Guo and L. Ma, "Inflammation in coronary artery disease-clinical implications of novel HDL-cholesterol-related inflammatory parameters as predictors," *Coron. Artery Dis.*, vol. 34, no. 1, pp. 66–77, 2023, doi: 10.1097/MCA.0000000000001198.

[44] E. j. Cassel, "The New England Journal of Medicine Downloaded from www.nejm.org at PARTNERS HEALTHCARE on October 20, 2010. For personal use only. No other uses without permission. From the NEJM Archive Copyright © 2010 Massachusetts Medical Society.," 2010.

[45] A. V. A. N. D. E. Regulatie and V. A. N. L. Lipase, "ASPECTS OF THE REGULATION OF LIVER LIPASE," no. december, 1986.

[46] A. Huang, X. Qi, L. Wei, M. Zhang, and S. Zhou, "Non-HDL-c/TC: A Novel Lipid-Related Marker in the Assessment of Severity of Coronary Artery Lesions and Cardiovascular Outcomes," *Cardiol. Res. Pract.*, vol. 2019, 2019, doi: 10.1155/2019/5931975.

[47] R. Yang, G. Le, D. Ph, A. Li, J. Zheng, and Y. Shi, "Effect of antioxidant capacity on blood lipid metabolism and lipoprotein lipase activity of rats fed a high-fat diet," vol. 22, pp. 1185–1191, 2006, doi: 10.1016/j.nut.2006.08.018.

[48] B. J. G. Mb and C. Dphil, "Efficacy and safety of more intensive lowering of LDL cholesterol: a

meta-analysis of data from 170 000 participants in 26 randomised trials," *Medicine (Baltimore).*, vol. 2011, pp. 359–360, 2010, doi: 10.1016/S0084-3873(11)00278-1.

[49] M. M. Ibrahim, A. Ibrahim, K. Shaheen, and M. A. Nour, "Lipid profile in Egyptian patients with coronary artery disease," *Egypt. Hear. J.*, vol. 65, no. 2, pp. 79–85, 2013, doi: 10.1016/j.ehj.2012.08.005.

[50] S. A. Hamed *et al.*, "Vascular Risks and Complications in Diabetes Mellitus : The Role of Helicobacter Pylori Infection," vol. 17, no. 2, pp. 86–94, 2008, doi: 10.1016/j.jstrokecerebrovasdis.2007.10.006.

[51] M. Riad, "Association of Helicobacter pylori infection with coronary artery disease: is it an independent risk factor?," *Egypt. Hear. J.*, vol. 73, no. 1, 2021, doi: 10.1186/s43044-021-00185-2.

[52] M. Haeri, M. Parham, N. Habibi, and J. Vafaeimanesh, " Effect of Helicobacter pylori Infection on Serum Lipid Profile ," *J. Lipids*, vol. 2018, pp. 1–5, 2018, doi: 10.1155/2018/6734809.

[53] J. Vafaeimanesh, M. Parham, M. Seyyedmajidi, and M. Bagherzadeh, "Helicobacter pylori infection and insulin resistance in diabetic and nondiabetic population," *Sci. World J.*, vol. 2014, 2014, doi: 10.1155/2014/391250.

[54] K. Mansori *et al.*, "Helicobacter pylori infection as a risk factor for diabetes: A meta-analysis of case-control studies," *BMC Gastroenterol.*, vol. 20, no. 1, pp. 1–14, 2020, doi: 10.1186/s12876-020-01223-0.