

Evaluation of Lipid Profile and Inflammatory Marker in Patients with Gastric *Helicobacter pylori* Infection, Ethiopia

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Introduction: *H. pylori* are gram-negative, microaerophilic helical-shaped bacteria with multiple flagella and commonly exist in the stomach. This infection may cause significant mucosal inflammation and damage, leading to ulcers in the stomach. It can also affect organ systems external to the gastrointestinal tract. To assess cardiovascular risk factors and to predict cardiovascular disorders, we are evaluating and comparing lipid profile and inflammatory marker between *H. pylori*-positive and negative patients.

Objective: To evaluate and compare lipid profile (TC; TG; LDL; HDL) and inflammatory marker (hs-CRP) in dyspeptic patients with and without *H. pylori* infection.

Methods: Comparative cross-sectional study was conducted from September 2020 to January 2021 at Debre Markos Referral Hospital, Debre Markos Health Center, and Hidassie Health Center, Ethiopia. Each of 50 *H. pylori*-positive and negative dyspeptic patients were studied. The data were checked for completeness and analyzed by SPSS version 25.0 Software. A p-value < 0.05 was considered statistically significant.

Results: Serum mean high-density lipoprotein (HDL) values were 37.54 ± 7.98 mg/dL and 43.12 ± 7.86 mg/dL ($p < 0.05$) for *H. pylori*-positive and negative dyspeptic patients, respectively, and median serum high sensitive C reactive protein (hs-CRP) levels were 6.29 mg/L (1.66–41.34) and 3.35 mg/L (0.39–10.01) ($p < 0.05$) for *H. pylori*-positive and negative dyspeptic patients, respectively.

Conclusion: *H. pylori* infection significantly alters serum high-density lipoprotein (HDL) and high sensitive C reactive protein (hs-CRP) levels in dyspeptic patients, as a result, increase the potential risk of cardiovascular diseases.

Keywords: *H. pylori*, dyspepsia, lipid profile, hs-CRP, inflammation, cardiovascular diseases

Introduction

The Genus *Helicobacter* has different species but only *H. pylori* can survive in human beings. Other species of *helicobacter* can survive in different animals.¹

H. pylori are a spiral-shaped, gram-negative, microaerophilic rod with 4–7 flagella. The flagella help in the settlement of the bacterium to the gastric mucosa layer.²

H. pylori produce outer membrane vesicles that contain different virulence factors encompassing cell-bound components, secreted molecules, and antigens that could be injected into a host cell by the type IV secretion system (T4SS) or direct secretion of soluble compounds to the gastric area. Outer membrane vesicles are described as 20–300 nm blebs, which are secreted during the logarithmic phase

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of Gram-negative bacteria growth. Outer membrane vesicles can be crossing the cellular barrier which is the effective way of *H. pylori* antigens and virulence factors translocation via the gastric mucosa towards the immune cells. *H. pylori* outer membrane vesicles contains lipopolysaccharide, Vacuolating cytotoxin gene A (VacA), Cytotoxin associated gene A (CagA), blood group antigen-binding adhesin, sialic acid-binding adhesin, outer inflammatory protein A, *H. pylori* neutrophil-activating protein, adherence associated lipoprotein, and urease. Those proteins and lipids are involved in the pathogenesis of *H. pylori* infection.³

The virulence factors of *H. pylori* can cause gastric inflammation, disruption of the gastric mucosal barrier, and extra gastric effect.⁴ Chronic inflammations are the critical component of the disease process of *H. pylori*, which is the initial step starting from superficial gastritis to chronic gastritis, intestinal metaplasia to dysplasia, and until invasive adenocarcinoma.⁵

The virulence factors of *H. Pylori* have a greater contribution to the disease process by inducing gastric inflammation and disruption of the gastric mucosal barrier. *H. pylori* promote gastric inflammation by stimulating interleukin (IL-8) secretion from the gastric cell, inducing neutrophil-endothelial cell interactions, activating platelet-activating factor, injecting the lipopolysaccharide of *H. pylori* to the gastric mucus coat, and secreting urease enzyme.⁶

H. pylori also disrupt the gastric mucosal barrier because by expressing phospholipases (A2 and C), mucinase like gene, vacuolating cytotoxin, reactive oxygen species, and induce programmed cell death.^{4,7}

H. pylori infection not only causes a gastric problem but also induces extra gastric diseases. Common extra gastric diseases related to *H. Pylori* include iron deficiency anemia, Henoch Schönlein Purpura, immune thrombocytopenic purpura, chronic urticaria, hepatocellular carcinoma, laryngeal cancer, insulin resistance, metabolic syndrome, cardiovascular diseases, and asthma.^{8,9}

The potential mechanism of *H. pylori* infection to induce cardiovascular disorders is related to inflammation and immunity.¹⁰ The common mechanisms that *H. Pylori* infection promoting cardiovascular diseases (CVDs) are:

Endothelial Dysfunction

Infected with *H. pylori* leads to an increase in inflammatory adhesion molecules such as intracellular adhesion molecule-1 and inflammatory mediator cytokines such as

hs-CRP, IL-1, IL-6, and tumor necrosis factor- α (TNF- α). These molecules affect the microvascular vasomotor functions and resulting in vasoconstriction and endothelial dysfunction.¹¹

H. pylori infection causes malabsorption of nutrition such as vitamin B12 and folic acid which leads to an increase in serum homocysteine levels and results in endothelial dysfunction.¹²

H. pylori infection cause alteration of asymmetric dimethylarginine (ADMA) which plays a crucial role at the beginning of *H. pylori*-induced endothelial dysfunction. ADMA is a competitive inhibitor of endothelial nitric oxide synthase (NOS) which reduces nitric oxide production from L-arginine by nitric oxide synthase in the endothelium to carry out its fundamental role in the regulation of vascular tone.⁵

Oxidative Stress

H. pylori infection resulting not only in inflammation but also causes the accumulation of reactive oxygen species (ROS) and oxidative DNA damage in the gastric mucosal layer. Therefore, the accumulation of ROS has been resulting in the initiation of multiple disease processes which have a contribution to the pathogenesis of cardiovascular disorders through the expression of adhesion molecules, stimulation of vascular smooth muscle proliferation and migration, apoptosis in the endothelium, and oxidation of lipids.⁵

Autoimmunity

The autoimmune responses triggered by cross-reactions between *H. pylori* antigens, such as cytotoxin-associated gene A (CagA), heat shock proteins (HSPs), and self-antigens, also help the role of *H. pylori* in the pathogenesis of atherosclerosis.¹³

Alter Lipid Metabolism

H. pylori infection is also linked with an atherogenic modified lipid profile. These alterations are may be mediated by cytokines, particularly TNF- α , which inhibits lipoprotein lipase and thus results in the mobilization of lipids from tissues as well as increased serum TG and reductions in HDL. An altered lipid profile, including an increased LDL level and decreased HDL level, is one of the most important risk factors for atherosclerotic disease.¹⁴

However, there are different kinds of controversial pieces of evidence that show the interrelationship of *H.*

pylori infection and predictive risk factors of cardiovascular disorders such as lipid profile and hs-CRP levels. This research was conducted to assess cardiovascular risk factors and to predict cardiovascular disorders, we are evaluating and comparing lipid profile and inflammatory marker between *H. pylori*-positive and negative patients that will help for better management of *H. pylori*-infected patients to reduce further complications and to perform a different kind of preventive and therapeutic measurement by the health professionals and insight to pay attention to laboratory investigation of lipid profile and hs-CRP in *H. pylori*-infected dyspeptic patients. The term dyspepsia comprises a wide and common clinical entity that presents in one of the three ways: Epigastric pain/burning (epigastric pain syndrome), Postprandial fullness, or Early satiety.¹²

Materials and Methods

A comparative hospital-based cross-sectional study was conducted at Debre Markos Referral Hospital, Debre Markos Health Center, and Hidassie Health Center from September 2020 to January 2021, Debre Markos, Ethiopia. Each of 50 *H. pylori*-positive and negative dyspeptic patients were selected during a health check-up in the outpatient department (OPD) health by purposive sampling technique those fulfill inclusion criteria. Sample sizes of 100 participants were used by using a sample size calculation formula for comparing the mean between two independent populations. All dyspeptic patients attending at Debre Markos Referral Hospital, Debre Markos Health Center, and Hidassie Health Center during the data collection period were included in the study but subjects with liver disease, renal dysfunction, obesity, diabetes, cardiovascular disorders, malignancy, rheumatoid arthritis, gouty arthritis, who receive the antihyperlipidemic drug, and patients with a history of *H. pylori* eradication therapy were excluded from the study. After we obtain informed consent from the study participants, 5 mL of blood was drawn and the blood was allowed to stand for 30 minutes for complete clotting, centrifuged at 3000 rpm for 15 minutes to extract serum, and stored at -70°C before the day of analysis.

Laboratory Tests

H. Pylori was detected by linear *H. Pylori* Ag cassette, which is a chromatographic immunoassay for the qualitative detection of *H. Pylori* antigen in the human fecal specimen (Joaquim Costa 18 2^a planta. 08390 Montgat (Barcelona) SPAIN). Serum HDL, Triglyceride, total cholesterol, and hs-CRP levels were analyzed by Cobas C 501

chemistry analyzer (Roche Diagnostic, USA) but serum levels of LDL-cholesterol were calculated by Friedewald formula. All biochemical analysis was done in the Ethiopian public health institute, Addis Ababa, Ethiopia. The reference range of HDL (40–60 mg/dL), TG (<130 mg/dL), TC (<200 mg/dL), LDL (<130 mg/dL) and hs-CRP (<5 mg/L) in adults.

Data Processing and Analysis

Data analysis was done by SPSS software version 25.0 package. Simple descriptive statistics are used to present the socio-demographic data. While Pearson Chi-square (χ^2) tests and Pearson correlation are used to describing the association between variables. Continuous variables with normal distribution are presented as mean \pm standard deviation and skewed variables are presented as median (range). Student's *t*-tests, ANOVA test, and Mann–Whitney-test were used to compare variables. A *p*-value of <0.05 at a 95% confidence level is considered to be statistically significant.

Result

Socio-Demographic Data of Dyspeptic Patients

This study was conducted on 100 dyspeptic patients with 50 *H. pylori*-positive and 50 *H. pylori*-negative dyspeptic patients. The mean age of *H. pylori*-positive and negative dyspeptic patients was 43.63 ± 13.31 and 35.54 ± 12.59 years, respectively. In *H. pylori*-positive dyspeptic patients, 26 participants were males and 24 participants were females. The age category of this study indicated that in *H. pylori*-infected patients 4 participants were ages ≤ 20 years, 16 participants were ages from 21 to 40 years, and 30 participants were ages from 40 to 60 years. *H. pylori* status is significantly associated with the age category of dyspeptic patients at *p*-value < 0.05.

Serum Lipid Profile and hs-CRP Levels in Dyspeptic Patients

Serum HDL and hs-CRP levels were significantly different between *H. pylori*-positive and negative dyspeptic patients (Table 1).

Serum Lipid Profile and hs-CRP Levels in Dyspeptic Patients Based on Gender

There is no significant difference between males and females in serum levels of lipid profile and hs-CRP

Table 1 The Comparison of Serum Lipid Profile and hs-CRP Levels Between *H. pylori*-Positive and Negative Dyspeptic Patient at DMRH, DMHC, and HHC, Debre Markos, Ethiopia (Each of 50 HP+ and HP- Participants) (N=100)

Variables	<i>H. pylori</i> Status		P-value
	HP+	HP-	
Serum HDL in mg/dL	37.54 ± 7.976	43.12 ± 7.86	0.001
Serum TC in mg/dL	172.36 ± 28.22	164.56 ± 30.04	0.183
Serum LDL in mg/dL	109.73 ± 32.78	102.05 ± 28.13	0.212
Serum TG in mg/dL	125.28 ± 61.67	117.82 ± 93.16	0.638
Serum hs-CRP in mg/L	6.29 (1.66–41.34)	3.35 (0.39–10.01)	0.001

Notes: *H. pylori*, *helicobacter pylori*; HP+, *H. pylori* positive; HP-, *H. pylori* negative; DMRH, Debre Markos Referral Hospital; DMHC, Debre Markos Health Center; HHC, Hidassie Health Center. Serum lipid profiles are presented as mean ± SD and serum hs-CRP levels are presented as median (range).

Abbreviations: HDL, high-density lipoprotein; TC, total cholesterol; LDL, low-density lipoprotein; TG, triglyceride; hs-CRP, high sensitive c-reactive protein.

(Table 2). In females, both serum HDL and hs-CRP levels were significantly different between *H. pylori*-positive and negative dyspeptic patients but, in males, only hs-CRP levels were significantly different between *H. pylori*-positive and negative dyspeptic patients (Table 3).

Serum Lipid Profile and hs-CRP in Dyspeptic Patients Based on Age Groups

Serum LDL and TC levels were significantly different among different age categories in *H. pylori*-positive patients (Table 4).

Discussion

The Role of *H. pylori*-Infection in Lipid Metabolism

Earlier studies had indicated that *H. pylori* might be implicated in the alteration of serum lipid profile concentration and increase the potential risk of cardiovascular disorders.^{15–22}

But, some studies have reported that *H. pylori* infection did not significantly alter lipid profile.²³ We have found that the mean serum TG (125.28 ± 61.67 mg/dL), TC (172.36 ± 28.22 mg/dL), and LDL (109.73 ± 32.78 mg/dL) levels were higher among *H. pylori*-positive dyspeptic patients

Table 2 The Comparison of Serum Lipid Profile and hs-CRP Between *H. pylori*-Positive and *H. pylori*-Negative Dyspeptic Patients for the Same Gender at DMRH, DMHC, and HHC, Debre Markos, Ethiopia (N=100)

Variables	Sex	<i>H. pylori</i> Status		P-value
		HP+	HP-	
Serum TG in mg/dL	Males	120.673 ± 64.468	148.859 ± 83.918	0.378
	Females	130.275 ± 59.45	101.836 ± 50.83	0.057
Serum HDL in mg/dL	Males	39.242 ± 7.763	42.654 ± 8.854	0.19
	Females	35.7±7.952	43.355±7.428	0.001
Serum TC in mg/dL	Males	173.476 ± 28.589	162.6 ± 30.077	0.239
	Females	171.159±28.367	165.564 ± 30.43	0.484
Serum LDL in mg/dL	Males	110.226 ± 31.633	96.065 ± 31.304	0.157
	Females	109.196 ± 34.65	105.133 ± 26.32	0.617
Serum hs-CRP in mg/L	Males	6.74 (1.66–41.34)	3.42 (0.4–10.01)	0.04
	Females	5.78 (1.75–40.39)	3.33 (0.39–7.91)	0.001

Notes: *H. pylori*, *helicobacter pylori*; HP+, *H. pylori* positive; HP-, *H. pylori* negative; DMRH, Debre Markos Referral Hospital; DMHC, Debre Markos Health Center; HHC, Hidassie Health Center. Serum lipid profiles are presented as mean ± SD and serum hs-CRP levels are presented as median (range). The bold figures indicate statistically significant values ($p < 0.05$).

Abbreviations: HDL, high-density lipoprotein; TC, total cholesterol; LDL, low-density lipoprotein; TG, triglyceride; hs-CRP, high sensitive c-reactive protein.

Table 3 The Comparison of Serum Lipid Profile and hs-CRP Levels Between Male and Female Dyspeptic Patients at DMRH, DMHC, and HHC, Debre Markos, Ethiopia (N=100)

Variables	<i>H. pylori</i> Status	Sex of Participants		P-value
		Males	Females	
Serum TG in mg/dL	HP+	120.673 ± 64.468	130.275 ± 59.454	0.0588
	HP-	148.859 ± 83.918	101.836 ± 50.832	0.091
Serum HDL in mg/dL	HP+	39.242 ± 7.763	35.7 ± 7.952	0.118
	HP-	42.654 ± 8.854	43.355 ± 7.428	0.768
Serum LDL in mg/dL	HP+	110.226 ± 31.633	109.196 ± 34.652	0.913
	HP-	96.065 ± 31.304	105.133 ± 26.317	0.285
Serum TC in mg/dL	HP+	173.476 ± 28.589	171.159 ± 28.367	0.775
	HP-	162.6 ± 30.077	165.564 ± 30.433	0.745
Serum hs-CRP in mg/L	HP+	6.74 (1.66–41.34)	5.78 (1.75–40.39)	0.69
	HP-	3.42 (0.40–10.01)	3.33 (0.39–7.91)	0.68

Notes: *H. pylori*, *helicobacter pylori*; HP+, *H. pylori* positive; HP-, *H. pylori* negative; DMRH, Debre Markos Referral Hospital; DMHC, Debre Markos Health Center; HHC, Hidassie Health Center. Serum lipid profiles are presented as mean ± SD and serum hs-CRP levels are presented as median (range).

Abbreviations: HDL, high-density lipoprotein; TC, total cholesterol; LDL, low-density lipoprotein; TG, triglyceride; hs-CRP, high sensitive c-reactive protein.

than the mean serum TG (117.82 ± 93.16 mg/dL), TC (164.56 ± 30.04 mg/dL), and LDL (102.05 ± 28.13 mg/dL) levels of *H. pylori*-negative patients. But, the difference was not statistically significant. This was in agreement with studies done in Iran and Peru.^{23,24} But, in contrast with studies done in Jimma and Iraq.^{14,18} We have found that serum HDL

levels of *H. pylori*-positive dyspeptic patients (37.54 ± 7.98 mg/dL) were significantly lower than negative dyspeptic patients (43.117 ± 7.858 mg/dL). A similar result was reported in studies conducted in Iraq and a meta-analysis was done in Japan.^{18,25} This significant difference of HDL between *H. pylori*-infected and non-infected might be due to

Table 4 The Comparison of Serum Lipid Profile and hs-CRP Levels Among Different Age Group in Dyspeptic Patients at DMRH, DMHC, and HHC, Debre Markos, Ethiopia (N=100)

Variables	<i>H. pylori</i> Status	Age Categories			P-value
		≤ 20 Years	21–40 Years	41–60 Years	
TG in mg/dL	HP+	97.0 ± 12.405	121.963 ± 69.137	130.823 ± 61.559	0.578
	HP-	115.267 ± 77.0	109.732 ± 71.813	132.944 ± 129.307	0.735
HDL in mg/dL	HP+	32.25 ± 4.009	38.7 ± 9.385	37.63 ± 7.461	0.357
	HP-	45.667 ± 11.676	43.801 ± 8.127	40.963 ± 5.379	0.367
LDL in mg/dL	HP+	85.85 ± 37.236	97.49 ± 35.76	119.445 ± 27.416	0.027
	HP-	94.913 ± 24.379	102.039 ± 28.956	104.744 ± 29.135	0.773
TC in mg/dL	HP+	137.5 ± 32.734	160.931 ± 30.583	183.11 ± 19.779	0.001
	HP-	163.633 ± 38.344	160.33 ± 29.245	172.3 ± 28.637	0.453
hs-CRP in mg/L	HP+	20.37 (3.42–40.39)	4.32 (1.75–40.65)	6.32 (1.66–40.34)	0.44
	HP-	1.27 (0.45–4.19)	3.49 (0.39–7.91)	3.35 (0.40–10.01)	0.053

Notes: Serum lipid profiles are presented as mean ± SD and hs-CRP levels are presented as median. The p-value represents the significant level of lipid profile and hs-CRP in different age categories for *H. pylori*-positive as well as negative dyspeptic patients.

metabolic and inflammatory effects of *H. pylori* infection. Inflammation caused by *H. pylori* infection is bringing significant changes in the protein (apoA-I), and lipid content of HDL, as well as reducing the concentration of HDL.²⁶ Inflammation causes an increase in reactive oxygen species such as Myeloperoxidase and superoxide anion radical, that resulting in the generation of oxidized and modified lipoproteins and reduce the concentration of HDL.²⁷ The metabolic effects of *H. pylori* infection are mediated by cytokines, predominantly TNF- α , which inhibit lipoprotein lipase and that results in the mobilization of lipids from tissues along with an increase in serum TG and decrease in HDL levels.⁵

Serum HDL has an atheroprotective function through different mechanisms. The first one is, HDL has an anti-atherogenic component called paraoxonase-1, which is an enzyme produced in the liver and has various biochemical functions in the body including protection of oxidation/lipid peroxidation, inducing natural immunity, and detoxification.²⁸ It has also the ability to limit the expression of proinflammatory cytokines and to decrease the expression of leukocyte adhesion molecules when stimulated by inflammatory cytokines. Besides, HDL can induce the production of IL-10 which is an anti-inflammatory and atheroprotective cytokine.²⁶ However, in our study, *H. pylori*-positive dyspeptic patients' serum HDL levels were significantly reduced due to the metabolic and inflammatory effect of *H. pylori* infection as a result, they are potentially at higher risk for atherosclerosis and other cardiovascular diseases.

Except for the levels of HDL, the serum TG, TC, and LDL were found higher in males when compared with females. This was mainly due to the positive effect of estrogen on lipid profile in females and the negative effect of androgen on lipid profile in males.²⁹ In females', estrogen increases hepatic cell surface LDL receptors and consequently rapid clearance of LDL particles.³⁰ Estrogen suppresses lipase activity in the liver as a result HDL levels increase. It also increases lipoprotein lipase activity, and lipoprotein lipase is responsible for hydrolyzing TG.³¹ In males', testosterone increases the degradation of HDL molecule by stimulating the expression of genes encoding for hepatic lipase and scavenger receptor B. On the other hand, testosterone reduced the expression of LDL receptors and the activity of lipoprotein lipase. Therefore, HDL decrease, and LDL, TG and TC levels increased in males.²⁹ But, in our finding, there was no significant difference in lipid levels between males' and females' dyspeptic patients, and this might be due to the effect of *H. pylori* infection,

inflammation, and other confounding factors such as alcohol consumption, smoking, and khat chewing on lipid profile.

We have found that serum LDL and TC levels in *H. pylori*-infected patients were significantly correlated with the age of dyspeptic patients and significantly higher among the age group > 40 years old. This was inconsistent with studies conducted in Iran.¹⁷ This significant association might be due to hormonal changes following increasing in age. In females, estrogen levels are decreasing after menopause which is resulting in the reduction of hepatic cell surface LDL receptors and consequently slowing the catabolism of LDL.³⁰ Besides, the reduction of estrogen causes dysregulation of lipoprotein lipase activity and results in to increase in LDL and TC levels.³¹

The Role of *H. pylori*-Infection in Inflammation

H. pylori-infection increases gastric and systemic inflammation through different mechanisms such as inducing inflammatory cytokines, inducing neutrophil-endothelial cell interactions, activate platelet-activating factor, by secreting lipopolysaccharide and urease enzyme which is a powerful stimulus of mononuclear phagocyte activation and inflammatory cytokine production.^{4,6}

H. pylori infection also induces an immune-inflammatory reaction through activation of cyclooxygenase enzyme-2 (COX-2), which leads to an increment of production of prostaglandin (PGE2).³²

A study conducted in Japan showed that serum hs-CRP levels were significantly higher in *H. pylori*-positive than those negative dyspeptic patients.¹³ This was in agreement with the present study with hs-CRP levels for *H. pylori*-positive (14.148 ± 20.59 mg/dL) and *H. pylori*-negative (3.51 ± 8.39 mg/dL; $p < 0.01$). This significant increment of hs-CRP levels among *H. pylori*-positive dyspeptic patients might be due to the inflammatory effect of *H. pylori* bacterium since the bacterium contain various virulent factors such as CagA and VacA. CagA, a highly virulent factor that is produced by *H. pylori* bacterium that has a greater role in *H. pylori*-induced inflammation. *H. pylori* cause gastric and extra gastric low-grade systemic inflammation. *H. pylori* infection promotes inflammation by activating nuclear factor Kappa-B (NF- κ B) which mediates inflammation through activation of pro-inflammatory cytokines such as IL-6, TNF- α , and IL-18. The activated IL-6 stimulates acute phase reactant protein production such as hs-CRP from the liver and is used as an inflammatory marker.⁹

In a study conducted in Japan, serum hs-CRP levels were significantly higher in females than males *H. pylori*-positive dyspeptic patients.¹⁵ This variation of serum hs-CRP levels between males and females might be probably due to the effects of androgen hormone on inflammatory mediators. Androgen hormone has an immunosuppressive effect and reduces the concentration of inflammatory markers including hs-CRP levels in males. The immunosuppressive effect of androgens could be either a direct effect on the expression of inflammatory genes or an indirect effect via inhibition of nuclear factor- κ B activation.³³ But, in our study, serum hs-CRP levels were insignificantly different between males and females *H. pylori*-positive dyspeptic patients. The variation might be due differences in statistical analysis.

In the present study, serum hs-CRP levels were significantly different between *H. pylori*-positive and negative dyspeptic patients in both sex and this significant difference might be due to the inflammatory effect of *H. pylori* bacteria both in males and females.⁹

Conclusion and Recommendation

H. pylori significantly alters serum HDL and hs-CRP levels. However, serum TC, TG, and LDL levels were higher in *H. pylori*-positive dyspeptic patients than negative dyspeptic patients, but statistically insignificant. The decreased HDL and the increased hs-CRP levels in *H. pylori*-infected dyspeptic patients are potentially increasing the risk for atherosclerosis, endothelial dysfunction, and other cardiovascular disorders. Lipid profile and inflammatory markers need to be tested in *H. pylori*-positive chronic dyspeptic patients to prevent and predict future complications.

Ethics Approval and Informed Consent

This study was conducted in accordance with the Declaration of Helsinki. The ethical clearance with reference number (SOM/BCHM/092/2012) was obtained from the Departmental Research and Ethics Review Committee, Department of Biochemistry, College of Health Sciences, Addis Ababa University. We collect data after we obtain written informed consent from each study subject.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest for this work.

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