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Dyslipidemia and Associated Factors Among Patients Suspected to Have *Helicobacter pylori* Infection at Jimma University Medical Center, Jimma, Ethiopia

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Background: Dyslipidemia is a group of plasma lipid and lipoprotein abnormality that is metabolically associated, and it is categorized by low HDL-C and increased LDL-C, TGs, and total cholesterol (TC) levels. Colonization of the stomach by *Helicobacter pylori* (*H. pylori*) causes chronic inflammation of the stomach wall which can change some biochemical factors in the patient. On the association of *H. pylori* infection and its contributions to change in serum lipid profile, different studies reported varying outcomes.

Objective: To assess the prevalence of dyslipidemia and associated factors among patients suspected for *H. pylori* infection in the outpatient department of Jimma University Medical Center, Jimma, Ethiopia.

Materials and Methods: A hospital-based cross-sectional study was conducted from January 03 to April 05, 2019, at Jimma University Medical Center on 369 *H. pylori* suspected patients. The study subjects were selected by convenient sampling technique. About 5mL of blood was collected from an overnight fasting individual. Data were edited, coded, and entered into Epidata version 3.1 and exported to (SPSS) version 25 for analysis. Bivariate analysis was used to screen those variables which were candidates for multivariate analysis.

Results: From the total study subjects 77.5% had at least one abnormality in lipid profile and 87.2% of *H. pylori* positive patients had at least one abnormality in lipid profile. Our study demonstrated that there was significant increase of mean ± SD of TC, TG, and LDL-C in *H. pylori* positive patients than *H. pylori* negative patients (P-value < 0.05). After adjusting for traditional dyslipidemia risk factors, *H. pylori* infection was an independent predictor of dyslipidemia (AOR 2.628, 95% CI 1.477–4.678, P=0.001).

Conclusion: An increase in prevalence of dyslipidemia among *H. pylori* positive patients indicates *H. pylori* infected patients have a possibility of altered lipid profile, therefore assessment of lipid profile in *H. pylori* infected patients is recommended.

Keywords: *Helicobacter pylori* infection, lipid profile, Jimma, Ethiopia

Introduction

Dyslipidemia is a group of plasma lipid and lipoprotein abnormality that is metabolically associated, and it is categorized by low HDL-C and increased LDL-C, TGs, and total cholesterol (TC) levels.[1] Lipids and lipoproteins are causes for coronary heart disease (CHD). It has been demonstrated that high levels of TC, TG, LDL-C, low HDL-C, and increased body mass index (BMI) are significantly associated with CHD.[2]

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Helicobacter pylori(*H. pylori*) is a spiral shaped, microaerophilic, gram-negative bacteria that was first isolated in 1982 from stomach biopsy specimens of patients with chronic gastritis. Approximately, half of the world's population is estimated to be infected with *H. pylori*, but the prevalence varies greatly among countries and the overall prevalence of *H. pylori* infection is strongly correlated with socioeconomic conditions.[3] *H. pylori* infection is commonly acquired in early life via oral-oral or fecal-oral pathways and chronic infection is strongly linked to the development of gastric cancer and peptic ulcer disease.[4]

Although *H. pylori* is the most predominant infection in the world, the epidemiologic link between the *H. pylori* infection and metabolic changes is a topic of debate and controversial.[5] The colonization of the stomach by *H. pylori* cause chronic inflammation of the stomach wall which can change some biochemical parameters in the patient.[6] *H. pylori* infection has linked to a variety of extra-gastric disorders, like coronary heart disease (CHD). The underlying possible mechanisms are chronic low-grade activation of the coagulation cascade, accelerating atherosclerosis, and antigenic mimicry between *H. pylori* and host epitopes leading to autoimmune disorders and lipid metabolism abnormality.[7] Due to gastrointestinal inflammation caused by *H. pylori*, absorption of glucose and lipids can be decreased.[8] An increase of TC, LDL-C and decrease in HDL-C levels in *H. pylori* infected people creates an atherogenic lipid profile which could promote atherosclerosis with its complications, myocardial infarction, stroke and peripheral vascular disease.[9] This was indicated in an experimental investigation that interleukin-8 (IL-8), which is over expressed in *H. pylori* infected mucosa production is stimulated by oxidized LDL-C by monocytes and thus, this potent chemoattractant cytokine increases the recruitment of T lymphocytes and smooth muscle cells, contributing to atherosclerosis.[10]

Now a day, infectious agents are being considered more frequently as causes of diseases that have been thought previously to be of non-infectious etiology like coronary heart disease. Additionally, lipopolysaccharide (LPS) affects circulating macrophages, and increase production of free radicals. Free radicals are known to oxidize LDL, the product of which (oxidized LDL), transform macrophages into foam cells, which are known to be important in the pathogenesis of atherosclerosis.[11] Moreover, products of Gram negative bacteria LPS, is recognized by toll like receptors (TLRs) on macrophages and other cells and these initiate marked changes in lipid and lipoprotein metabolism.[12]

Infection with *H. pylori* triggers a chronic inflammatory state which along with other mechanisms such as dyslipidemia, hyper-homocysteinemia, hypercoagulability, impaired glucose metabolism and endothelial dysfunction, contribute in pathogenesis of atherosclerosis. Studies have shown a positive relation between cytotoxic associated gene-A (Cag A) positive strain of *H. pylori* and vascular diseases such as coronary artery disease (CAD) and stroke.[13]

The other mechanisms postulated to be the link between *H. pylori* infection and atherosclerosis are the activation of endothelial dysfunction by endotoxins released from virulent strains of *H. pylori*, the autoimmune response by secreting heat-shock proteins.[14] Endothelial dysfunction leads to increased tension, vascular wall remodeling, vascular inflammation, increased platelet adhesion and aggregation. These processes play its role in the development of atherosclerosis.[15] For example, chronic *Streptococcus pneumoniae* infection seemed to be associated with an increase serum lipid profile considered to increase the risk of atherosclerosis, supporting the hypothesis that infections can play an indirect role in the pathogenesis of atherosclerosis.[16]

Study has indicated that the presence of *H. pylori* in gastrointestinal ulcers results in change in lipid profile of serum including: cholesterol, TG, and LDL-C, HDL-C lipoproteins.[17] But conflicting results also exist.[18] Other study indicate *H. pylori* could play a role in the development of ischemic heart disease through different ways such as colonization of endothelial cells, changes in lipid profile, hyper coagulation, platelet aggregation, induction of molecular mimicry mechanisms, and progression of low-grade systemic inflammation.[19]

Activation of human neutrophils, monocytes, and dendritic cells with *H. pylori* neutrophil-activating protein (*H. pylori*-NAP) strongly up regulates both interleukin 12 (IL-12) and interleukin 2 (IL-2) production, via TLRs and in the gastric mucosa of *H. pylori* infected patients, a considerable proportion of T helper cells (Th cells) that are specific for different *H. pylori* antigens, including *H. pylori* -NAP, CagA, urease, vacuolating cytotoxin gene A (VacA) and heat shock proteins.[10]

Currently a growing evidence suggests that there are extra-intestinal manifestations of *H. pylori* infection.[20] This research will help to assess the association of *H. pylori* infection and dyslipidemia.

Dyslipidemia comprises a group of disorders in the metabolism of TC and TG, which has implications in the

cardiovascular system, producing pathologies as vascular coronary disease and atherosclerosis.[21] CAD are a global health problem. Its etiology is associated with various risk factors like hypertension, diabetes mellitus (DM) and dyslipidemia. Infectious microorganism like *C. pneumoniae, H. pylori*, may represent an additive risk factor.[22] The epidemiological distribution of *H. pylori* infection is worldwide, based on regional prevalence estimates, there were approximately 4.4 billion individuals with *H. pylori* infection worldwide in 2015, Africa had the highest prevalence (70.1%; 95% CI, 62.6–77.7), and Oceania had the lowest prevalence (24.4%; 95% CI,18.5–30.4).[23]

The burden of chronic diseases is increasing in low- and middle-income countries, where as it remains stable in high-income countries, developing countries are encountering a growing burden of chronic diseases, which constitute a double burden combined with infectious diseases and nutritional problems[24] Epidemiologic and clinical report suggest that *H. pylori* infection is a contributing factor in the progression of atherosclerosis. But the specific cardiovascular disease risk factors associated with *H. pylori* remain unclear, and the infection remains a destructive, transmissible, with serious health consequences.[25]

Some studies suggest a significant relationship between *H. pylori* infection and atherogenesis.[26] *H. pylori* infection might play an important role in the pathogenesis of atherosclerosis.[16] Due to accumulation of fatty substances, cholesterol, cellular waste products, calcium, and fibrin in the inner lining of the arterial wall cause the formation of atherosclerotic plaques.[2]

The possible role of the *H. pylori* infection as a determinant of extra-gastric manifestations such as atherosclerotic processes and peripheral vascular disorders is matter of debate.[27]

Currently, there is no convincing evidence supporting the presence of *H. pylori* within atherosclerotic plaques, and sero-epidemiological evidence is contradictory. Studies indicated that *H. pylori* infection bring to changes in serum lipid profile and may increase the risk of atherosclerosis.[28] But there are studies, which do not support the idea of *H. pylori*-dependent dyslipidemia.[29] This study was conducted to investigate the association between *H. pylori* infection and changes in serum lipid profile in gastritis suspected patients. To the best of our knowledge, there is no study conducted in this area in particular and in Ethiopia in general, about the association between *H. pylori* infection, and dyslipidemia among adult gastritis suspected patients.

Materials and Methods

Study Area, Design and Period

A hospital-based cross-sectional study design was used to conduct the study. The study was conducted at OPD of JUMC, which is located in Jimma city 352 km southwest of the capital Addis-Ababa. The study was conducted from January 03 to April 05, 2019. Patients who were coming to adult cold OPD of JUMC with gastritis symptoms during the study periods was the study population and individual patients suspected for *H. pylori* infection was taken as the study subjects.

Data Collection

After a brief explanation, the study participants were asked for their consent to be interviewed by clinical nurses to give fasting blood sample. Then 5mL of blood was withdrawn by laboratory professionals from the study participants, who had fasted overnight (10–12 hours), for laboratory analysis. In addition, the questionnaire was filled by face to face interview and blood pressure were measured by clinical nurses.

Blood Pressure Measurement

An automatic digital sphygmomanometer (OMRON HEALTH CARE Co., Ltd. Kyoto, Japan) was used to measure patients' blood pressure. The patient was seated upright with their upper arm positioned on the bench and excess clothing was removed that might interfere with the BP cuff or constrict blood flow in the arm.

Anthropometric Measurement

Waist Circumference Measurement

Participants waist circumference was measured at the level of the iliac processes and the umbilicus with a tape measure to evaluate abdominal obesity.[30]

Height Measurement

Height was measured using height measure scale (Infiniti Med Lab Pvt. Ltd., India). Participants stand erect on the floor board of the stadiometer with their back to the vertical backboard of the stadiometer. The heels of the feet are placed together with both heels touching the base of the vertical board. Feet were pointed slightly outward at a 60-degree angle. During the height measurement, the participant's

shoes and hats were removed. The height measurement was recorded to the nearest 0.1 cm.[31]

Weight Measurement

First the weight scale (Infiniti Med Lab Pvt. Ltd., India) was turn to zero then participants were asked to remove extra layers of clothing, shoes, jewellery, and any items in their pockets, next the participant were asked to step on the scale backwards (for confidentiality) then body weight was evenly distributed between both feet, arms hang freely by the sides of the body, palms toward thighs and head is up and facing straight ahead then weight is recorded to nearest 0.1 kg (100 gm).[31]

Body Mass Index (BMI)

After measuring participant's height and weight, the BMI was calculated by dividing weight in (kg) by height squared (m^2). The WHO definition of obesity is based on various categorical cut-points based on the body mass index (BMI) of weight-for-height: underweight (<18.5 kg/m^2), normal weight (18.5–24.9 kg/m^2), overweight (25.0–29.9 kg/m^2), and obesity (≥30 kg/m^2).[32]

Blood Sample Collection and Laboratory Analysis

After obtaining consent from the study subjects by giving detail information about the study objective, venous blood was selected preferably at the antecubital area by applying a tourniquet, puncture area of the vein was disinfected by using 70% alcohol. About 5 mL of blood was withdrawn aseptically from the antecubital vein from fasting individuals using serum separator tube. Following collection, specimens was transported to clinical chemistry unit of JUMC laboratory for analysis. The collected blood sample was left for 30 minutes at room temperature for clotting. Then the clotted blood samples were centrifuged for 10 minutes at 3000 revolutions per minutes (rpm) to separate serum from formed elements. All these procedures were done by the principal investigator. The extracted serum was kept in Nunc tube under −20°c deep freeze until laboratory analysis. TG, HDL-C, and TC were measured in serum by using ABX Pentra 400 clinical chemistry analyzer (Horiba ABX SAS, Montpellier, France) fully automated auto analyzer by the direct end point enzymatic method.

Lipid Profile Tests

Total cholesterol was measured by CHOD-PAP, enzymatic photometric method. Absorbance is measured at 500 nm. Triglycerides was measured by (GPO-PAP METHOD), a series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol. Absorbance is measured at 500 nm. HDL-C is measured by direct assay in a homogeneous method for directly HDL-C levels in serum without the need for any off-line pretreatment or centrifugation steps. LDL-C was determined using Friedwald formula[34]. LDL-cholesterol was calculated from measured values of TC, TG and HDL-C according to the relationship: [LDL-C] = [TC] - [HDL-C] - [TG/5].

Determination of *H. pylori*

Study participants *H. pylori* status were determined by rapid antibody test strip Wondfo (one step *H. pylori* serum/plasma test). The performance of Wondfo (Guangzhou Wondfo Biotech Co., Ltd) one step *H. pylori* serum/plasma test had sensitivity of 99.0% and specificity of 99.2% when compared with *H. pylori* ELISA kit. ELISA kit has 99.5% sensitivity and 99.6% specificity to detect *H. pylori*.

Data Quality Assurance and Management

After completion of each questionnaire, cross checking was done between data collector and principal investigator to assure the completeness of the information gathered. The label on the test tube and subject's unique identification number on questionnaire was checked for similarity. After checking the expiry date of the reagents and control, ABX Pentra 400 clinical chemistry analyzer (Horiba ABX SAS, Montpellier, France), was checked for delivering correct result by using normal and pathological controls. Before any patient sample processed, dual quality controls (normal and pathological) was performed and the patient result was taken after the controls passed. Collected results were checked for completeness on a daily basis by the principal investigator.

Data Analysis

Information from the questionnaires and laboratory analysis was entered into Epidata version 3.1, and exported to SPSS version 25 software (IBM Corporation, USA). Lipid profile result was expressed as mean ± SD. Descriptive statistics were used to analyze the data. For categorical variables, percentage and frequencies were used, whereas mean, standard deviation and range were used for continuous variables. Tables were used to assist data presentation and independent sample *t*-test was used to compare the different studied parameters between groups of *H. pylori*

positive and negative subjects. Bivariate logistic regression analysis was conducted to see the existence of crude association and to select candidate variables (with P-value below 0.25) to multivariable logistic regression. Logistic regression analysis was done to control possible confounders and to determine factors that may be significantly associated with dyslipidemia. P-value ≤ 0.05 was considered as a cut point for statistical significance in the final model.

Results

Socio-Demographic and Clinical Characteristics

A total of 369 patients suspected for *H. pylori* infection were included in the present study. Among the study subjects 194 (52.6%) were females and 175 (47.4%) were males aged from 20 years to 74 years with the mean ± SD age of 41.03 ± 13.55 years. Majority of the study participants 182 (49.3%), were Muslim by religion and 211 (57.2%), were Oromo by ethnicity. Regarding to participant's residence 213 (57.7%) were from urban and 156 (42.3%) live in rural area. Occupational status and smoking were significantly associated to *H. pylori (*p<0.05). From the total study participants 173 (46.9%) were positive and 196 (53.1%) were negative for rapid *H. pylori* serum antibody test.

Regarding life style condition of study participants, majority 344 (93.2%) were nonsmoker, 329 (89.2%) did not drink alcohol, 248 (67.2%) did not chew khat, and 313 (84.8%) did not have regular physical exercise. Majority of study participant routine diet was Shiro (Injera and wott) 221 (59.9%). Study participants who had known clinical disease were 29 (7.9%) had known diabetes mellitus, 40 (10.8%) had known renal disease, and 56 (15.2%) had known hypertension. Majority of study participant 265 (71.8%) had normal (18.5–25 kg/m^2), 55 (14.9%) had overweight (25–29.9 kg/m^2), and 49 (13.3%) underweight (<18.5kg/m^2) had BMI values respectively. There was no statistically significant association with *H. pylori* infection to known history of diabetes (P=0.876), hypertension (P=0.168), renal disease (P=0.078), khat chewing (P=0.10), and alcohol drinking (P=0.676) as indicated in Table 1.

Table 1 Socio-Demographic Characteristics, and *H. pylori* Antibody Status with Its Association among Adult Cold OPD Patients at JUMC, Jimma, Ethiopia (N=369)

Variables	Category	*H. pylori* Ab Positive No (%)	*H. pylori* Ab Negative No (%)	P-value
Age in years		42.68 ± 13.66	39.58 ± 13.32	0.028
Sex	Male Female	89 (24.1) 84 (22.8)	86 (23.3) 110 (29.8)	0.146
Marital status	Single Married Divorced Widowed	36 (9.8) 114 (30.9) 8 (2.2) 15 (4.1)	44 (11.9) 128 (34.7) 9 (2.4) 15 (4.1)	0.972
Educational status	Illiterate Read and write Elementary High school and above	54 (14.6) 24 (6.5) 38 (10.3) 57 (15.4)	53 (14.4) 27 (7.3) 54 (14.6) 62 (16.8)	0.625
Residence	Urban Rural	107 (29.0) 66 (17.9)	106 (28.7) 90 (24.4)	0.132
Occupational status	Civil servant House wife Merchant Private worker Farmer Other	37 (10) 27 (7.3) 10 (2.7) 23 (6.2) 45 (12.2) 31 (8.4)	30 (8.1) 60 (16.3) 5 (1.4) 29 (7.9) 42 (11.4) 30 (8.1)	0.014
Smoking	Yes No	19 (5.1) 154 (41.7)	6 (1.6) 190 (51.5)	0.003
Physical exercise	Yes No	28 (7.6) 145 (39.3)	28 (7.6) 168 (45.5)	0.612
History of DM	Yes No	14 (3.8) 159 (43.1)	15 (4.1) 181 (49.1)	0.876
History of hypertension	Yes No	31 (8.4) 142 (38.5)	25 (6.8) 171 (46.3)	0.168
History of renal disease	Yes No	24 (6.5) 149 (40.4)	16 (4.3) 180 (48.8)	0.078
Khat chewing	Yes No	64 (17.3) 109 (29.5)	57 (15.4) 139 (37.7)	0.10
Drinking alcohol	Yes No	20 (5.4) 153 (41.5)	20 (5.4) 176 (47.7)	0.676

Notes: P-value by Pearson chi-square for categorical variable; and by independent *t*-test for continuous variable; other: student.

Abbreviations: OPD, outpatient department; JUMC, Jimma University Medical Center; *H. pylori, Helicobacter pylori*; Ab, antibody; No, number; DM, Diabetes Mellitus

Prevalence of Dyslipidemia Among *H. pylori* Positive and Negative Subjects

In this study we found an overall prevalence of 286/369 (77.5%) for dyslipidemia in at least one of the four lipid profiles. 151 (87.2%) *H. pylori* positive had dyslipidemia in at least one of the four lipid profiles. Individual who had abnormality in all of the four-lipid profile were 25 (6.8%). From the total study subjects 154 (41.7%) had low HDL-C,

99 (26.8%) had high LDL-C, 130 (35.2%) had high TC and 196 (53.1%) had high TG. The distribution of abnormal lipid profile among *H. pylori* positive subjects were 89 (51.4%), 116 (67.05%), 66 (38.1%) and 68 (39.3%) by serum TC, TG, LDL-C and HDL-C respectively, whereas the prevalence of dyslipidemia among *H. pylori* negative individual were 41 (20.9%), 80 (40.8%), 33 (16.8%) and 86 (43.8%) by serum TC, TG, LDL-C and HDL- C respectively. Statistically significant mean value was observed in TC ($P < 0.001$), LDL-C ($P< 0.001$) and TG ($P< 0.001$) in *H. pylori* positive subjects.

Association Between *H. pylori* Infection and Lipid Profile Among Study Participants

The mean level of serum TC, TG, LDL- C was significantly higher and serum HDL- C was not significant between *H. pylori* positive and *H. pylori* negative patients ($p < 0.001$) as shown in Table 2.

Correlation Between *H. pylori* Infection and Lipid Profile

There was statistically positive correlation between serum TC with weight (r=0.399, p<0.001), BMI (r=0.432, p<0.001), and WC (r=0.320, p<0.001) whereas there was no statistically significant correlation between serum TC with age (r=0.044, p=0.566), SBP (r=0.029, p=0.706), and DBP (r=0.102, p=0.183) as indicated in Table 3.

Table 2 Mean Serum Value of Lipid Profile Among *H. pylori* Positive and Negative Adult Patients at JUMC, Jimma, Ethiopia (N=369)

Variable	H. pylori Status		P-value
	Positive (Mean ± SD)	Negative (Mean ± SD)	
TC	200.80± 43.48	173.67± 42.41	<0.001
TG	185.61± 74.82	138.18± 60.17	<0.001
HDL-C	41.79± 8.70	41.91± 9.87	0.900
LDL-C	122.00± 37.00	104.27± 34.71	<0.001
Age	42.68 ± 13.66	39.58 ± 13.32	0.280
SBP	120.24 ± 15.82	116.89 ± 14.28	0.033
DBP	78.13 ± 9.21	76.00 ± 8.35	0.021
WC	79.38 ± 9.40	77.79 ± 10.51	0.129
HC	90.43 ± 10.38	87.84± 10.85	0.020

Note: P-value done by independent sample *t*-test.

Abbreviations: JUMC, Jimma University Medical Center; TC- total cholesterol; TG, triglycerides; HDL- C, high density lipoprotein cholesterol; LDL- C, low density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; WC, waist circumference; HC, hip circumference; *H. pylori, Helicobacter pylori*; SD, Standard Deviation.

Regarding to the magnitude of lipid profile among study subjects 89 (46.9%) *H. pylori* positive and 41 (11.1%) *H.* pylori negative had TC greater than 200mg/dl. The frequency and percentage of lipid profiles are indicated in Table 4.

Bivariate and Multivariate Analyses of Factors Associated with Total Cholesterol

On multivariate analysis, having BMI > 25kg/m^2 (AOR 0.243, 95% CI, 0.096–0.617, P= 0.003), and having weight >56kg (AOR 0.403,95% CI, 0.220–0.737, P=0.003) were independently associated with serum cholesterol concentration. However, being female sex (AOR 1.254, 95% CI, 0681–2.309, P=0.467), was not associated with serum cholesterol concentration as shown in Table 5.

Bivariate and Multivariate Analyses of Factors Associated with Dyslipidemia

On multivariate analysis of dyslipidemia (at least one abnormality in one of lipid profile), After adjusting for traditional dyslipidemia risk factors, *H. pylori i*nfection was the only independent predictor of dyslipidemia (AOR 2.628, 95% CI 1.477–4.678, P=0.001). However, other risk factors were not associated with dyslipidemia by multivariate analysis ($P > 0.05$) (Table 6).

Discussion

H. pylori infection is a causative agent for the development of peptic ulcer and gastric cancer.[21] There are evidence that indicate the role of *H. pylori* infection had pathogenesis of various extra gastric diseases.[34] In the present study we found that, the prevalence of dyslipidemia, at least in one of the four lipid profile, in *H. pylori* positive patients is 151/173 (87.2%). Our result is higher than the study done in Iran with a prevalence of dyslipidemia in *H. pylori* infected individual of 60.4%.[35] The probable reason of variation of the result might be due to geographical area, source population, sample size and the way they define dyslipidemia.

Our result is comparable with study done in Iran on evaluation of serum lipid profiles among *H. pylori* infected and non-infected. The study of the lipid profile showed that infected groups have statistically significantly increased rate of TC, TG, LDL-C and HDL-C compared with the non-infected groups in all p<0.001.[17] The only difference from our study was with HDL-C which was not significant in *H. pylori* positive patients, the association

Table 3 Correlation Analysis of Lipid Profile with Predictors in *H. pylori* Positive Subjects (N =173 (46.9%)) Among Adult patients at JUMC, Jimma, Ethiopia

Predictors	TC		TG		HDL-C		LDL-C	
	r	p	r	p	r	p	r	P
Age	0.044	0.566	0.068	0.374	−0.155	0.131	0.049	0.520
Sex	−0.006	0.939	−0.064	0.404	0.108	0.158	−0.003	0.967
Physical exercise	−0.091	0.234	−0.131	0.085	−0.081	0.290	−0.033	0.664
Weight	0.399	0.000	0.396	0.000	0.024	0.756	0.304	0.000
BMI	0.432	0.000	0.382	0.000	0.012	0.875	0.351	0.000
WC	0.320	0.000	0.254	0.001	0.089	0.245	0.253	0.001
Smoking	−0.054	0.483	0.029	0.704	0.099	0.196	−0.097	0.204
Khat chewing	−0.016	0.837	0.024	0.752	0.084	0.274	−0.046	0.552
Alcohol drinking	−0.092	0.229	−0.024	0.752	−0.010	0.898	−0.095	0.214
SBP	0.029	0.706	−0.104	0.174	0.019	0.802	0.067	0.378
DBP	0.102	0.183	0.082	0.282	0.100	0.192	0.059	0.444
Duration of gastritis	−0.106	0.164	−0.073	0.338	−0.125	0.100	−0.063	0.411

Abbreviations: JUMC, Jimma University Medical Center; *r*, Pearson correlation coefficient; *p*,P-value for correlation; TC, Total Cholesterol; TG, Triglyceride; LDL-C, Low Density Lipoprotein-Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; BMI, body max index; SBP, systolic blood pressure; DBP, diastolic blood pressure; WC, waist circumference; *H. pylori, Helicobacter pylori.*

Table 4 Clinical Factors versus *H. pylori* Status Among Adult Patients at JUMC, Jimma, Ethiopia (N=369)

Lipid Profile		H. pylori (+) No (%)	H. pylori (-) No (%)	COR	95% CI	
					Lower	Upper
TC	<200 mg/dl ≥200 mg/dl	84 (22.8) 89 (46.9)	155 (42.0) 41 (11.1)	0.250	0.158	0.394
HDL-C	>40 mg/dl ≤40 mg/dl	105 (28.3) 68 (18.5)	110 (29.9) 86 (23.4)	0.279	0.181	0.430
LDL-C	<130 mg/dl ≥130 mg/dl	117 (31.7) 56 (15.2)	177 (48.0) 19 (5.1)	0.224	0.552	1.268
TG	<150 mg/dl ≥150 mg/dl	70 (19.0) 103 (27.9)	139 (37.7) 57 (15.4)	0.279	0.127	0.397

Abbreviations: JUMC, Jimma University Medical Center; TC, Total Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; LDL-C, Low Density Lipoprotein-Cholesterol; TG, Triglyceride; No, number, % percentage; COR, crude odd ratio; CI, confidence interval; *H. pylori, Helicobacter pylori.*

might be due to the effect of *H. pylori* infection on lipid metabolism.[36]

The current result is in agreement with a study done in Finland which states that *H. pylori* infection might modify the serum lipid concentrations in a way that could increase the risk of CHD. *H. pylori* positive had significantly (P = 0.03) higher concentrations of serum TG than those who were *H. pylori* negatives.[37] Our results were also in consistent with the study conducted in Korea to investigate whether *H. pylori* is associated with dyslipidemia revealed that *H. pylori* infection was significantly associated with higher TC level (r = 2.114, P < 0.001), higher LDL-C level (r = 3.339, P < 0.001), lower HDL-C level (r= −1.237, P < 0.001), and higher DBP (r = 0.539, P = 0.001).[38] The result of serum lipid profile such as LDL-C, TG, and TC obtained in the present study were higher in *H. pylori* positive individuals when compared to *H. pylori* negative individuals.

Our result is also similar to a study conducted in Iraq determine lipid profile in patients with gastritis found that *H. pylori* was associated with increase in serum cholesterol, TG and LDL-C and decrease in serum HDL-C and the positivity rate was 55.83%.[28]

In our study the mean ± SD of *H. pylori* positive patients were higher than *H. pylori* negative patients for TC, TG, and LDL-C (200.80 ± 43.48 mg/dl vs 173.67 ± 42.41), (185.61 ± 74.82mg/dl vs 138.18± 60.17), and (185.61 ± 74.82mg/dl vs104.27 ± 34.71) respectively

Table 5 Associated Factors for Serum Total Cholesterol Concentration Among Adult Cold OPD patients at JUMC, Jimma, Ethiopia (N=369)

Variable	Category	Total Cholesterol		Bivariate Analysis		Multivariate Analysis	
		<200mg/dl No (%)	≥200mg/dl No (%)	COR 95% CI	P-value	AOR 95% CI	P-value
Age	< 40 year ≥40 year	146 (39.6) 93 (25.2)	51 (13.8) 79 (21.4)	[a] 0.411 (0.265–0.637)	<0.001	[m] 0.467 (0.276–0.790)	0.005
Sex	Female Male	133 (36.0) 106 (28.7)	61 (16.5) 69 (18.7)	0.705 (0.459 −1.082) [b]	0.109	1.254 (0.681–2.309) [n]	0.467
H. pylori	Neg. Pos.	155 (42.0) 84 (22.8)	41 (11.1) 89 (24.8)	[c] 4.006 (2.540–6.316)	<0.001	1 0.273 (0.158–0.470)	<0.001
BMI	< 25kg/m^2 ≥ 25kg/m^2	228 (61.8) 11 (3.0)	86 (23.3) 44 (11.9)	[d] 0.094 (0.047–0.191)	0.000	[u] 0.243 (0.096–0.617)	0.003
DBP	< 90mmHg ≥ 90mmHg	228 (61.8) 11 (3.0)	117 (31.7) 13 (3.5)	[e] 0.434 (0.189–0.999)	0.050	[o] 0.703 (0.266–1.855)	0.476
WC	< 94cm ≥ 94cm	229 (62.1) 10 (2.7)	106 (28.7) 24 (6.5)	[f] 5.185 (2.394–11.230)	<0.001	[v] 0.669 (0.198–2.264)	0.518
Weight	< 56kg ≥ 56kg	139 (37.7) 100 (27.1)	29 (7.9) 101 (27.4)	[g] 0.207 (0.127–0.336)	<0.001	[w] 0.403 (0.220–0.737)	0.003
Alcohol drinking	No Yes	220 (59.6) 19 (5.1)	109 (29.5) 21 (5.7)	[h] 2.231 (1.151–4.323)	0.017	[p] 1.192 (0.499–2.846)	0.692
Residence	Rural Urban	118 (32.0) 121 (32.8)	38 (10.3) 92 (24.9)	[i] 2.361 (1.498–3.722)	<0.001	[q] 0.357 (0.192–0.662)	0.001
HC	< 102cm ≥102cm	225 (61.0) 14 (3.8)	108 (29.3) 22 (6.0)	[j] 0.305 (0.150–0.620)	0.001	[r] 0.649 (0.248–1.695)	0.377
Diet	Vegetable Meat	181 (49.1) 58 (15.7)	81 (22.0) 49 (13.3)	[k] 0.530 (0.334–0.841)	0.007	[s] 0914 (0.503–1.660)	0.768
Khat chewing	No Yes	169 (45.8) 70 (19.0)	79 (21.4) 51 (13.8)	[l] 1.559 (0.995–2.442)	0.053	[t] 0.434 (0.227–0.828)	0.011

Note: [a, b, c … w] are identifier reference variables for comparison.

Abbreviations: OPD, outpatient department; JUMC, Jimma University Medical Center; CI, Confidence interval; COR, Crud Odds Ratio; AOR, Adjusted Odds Ratio; WC, waist circumference; HC, hip circumference; *H. pylori, Helicobacter pylori*; BMI, body mass index; DBP, diastolic blood pressure; kg, kilogram; cm, centimeter; mmHg, millimeter mercury.

with (P< 0.05). These findings are also supported with the results of a study conducted in Turkey on serum lipid profile in *H. pylori* infected patients, serum cholesterol concentrations were significantly higher in patients group when compared with healthy group (189.32±45.15 vs 179.41±36.37) mg/dl (p <0.05), serum TG and TC/HDL-C concentrations also were significantly higher in patients group (169.46±68.53 vs 135.67±94.35) mg/dl (p<0.05) and 3.93±1.23 vs 3.51±1.62, (p <0.05) respectively.[39]

The result of the present study also indicates that *H. pylori* infection increases TC, LDL-C and TG level of infected subjects compared to the negative subjects. It might be, due to LPS present in the cell walls of gram-negative bacteria *H. pylori*, there is stimulation of large quantities of cytokines (TNF-α and IL-6) which inhibit lipoprotein lipase activity. The consequence being mobilization of lipid tissue through an increase in serum TG level and in contrast, a decrease in serum HDL cholesterol level.[14] Abnormal high increase of TC (P < 0.001) and LDL-C (P < 0.001) and TG level (p<0.001) were observed in the *H. pylori* positive patients compared to *H. pylori* negative patients. Our results were in agreement with the study done by Kim et al who showed that *H. pylori* is independently associated to increased level of TC, and LDL-C.[9]

Our result was in contrast to a study conducted in Croatia, and Spain the result indicate that *H. pylori* infection was not an independent risk factor for acute myocardial infarction this is due to difference in the study design,

Table 6 Associated Factors for Dyslipidemia Among Adult Cold OPD Patients at JUMC, Jimma, Ethiopia (N=369)

Variable	Category	Dyslipidemia		COR (95% CI)	P-value	AOR (95% CI)	P-value
		Yes No (%)	No No (%)				
Age in year	<40 ≥40	144 (39.0%) 142 (38.5%)	53 (14.4%) 30 (8.1%)	[a] 0.574 (0.347–0.950)	0.031	[n] 0.696 (0.391–1.240)	0.219
Sex	Female Male	141 (38.2) 145 (39.3)	53 (14.4) 30 (8.1)	[b] 1.817 (1.097–3.008)	0.020	[o] 0.851 (0.436–1.659)	0.635
Residence	Rural Urban	110 (29.8) 185 (50.1)	46 (12.5) 37 (10.0)	[c] 0.503 (0.307–0.824)	0.006	[p] 1.803 (0.989–3.287)	0.055
Occupational status	Unemployed Employed	106 (28.7) 180 (48.8)	42 (11.4) 41 (11.4)	[d] 1.740 (1.063–2.847)	0.028	[q] 1.107 (0.606–2.024)	0.741
Physical exercise	Yes No	48 (13.0) 238 (64.5)	8 (2.2) 75 (20.3)	[e] 1.891 (0.856–4.175)	0.115	[r] 0.709 (0.286–1.758)	0.458
History of HTN	No Yes	239 (64.8) 47 (12.7)	74 (20.1) 9 (2.4)	[f] 1.617 (0.757–3.455)	0.215	[s] 0.998 (0.427–2.335)	0.997
Smoking	No Yes	264 (71.5) 22 (6.0)	80 (21.7) 3 (0.8)	[g] 2.222 (0.648–7.617)	0.204	[t] 1.183 (0.297–4.704)	0.812
Khat	No Yes	187 (50.7) 99 (26.8)	61 (16.5) 22 (6.0)	[h] 1.468 (0.851–2.531)	0.167	[u] 1.477 (0.747–2.921)	0.262
Regular diet	Vegetable Meat	198 (53.7) 88 (23.8)	64 (17.3) 19 (5.1)	[i] 1.497 (0.846–2.648)	0.166	[v] 1.061 (0.560–2.013)	0.855
Weight	<56kg ≥56kg	113 (30.6) 173 (46.9)	55 (14.9) 28 (7.6)	[j] 0.333 (0.199–0.555)	<0.001	[w] 0.612 (0.328–1.139)	0.121
WC	<94cm ≥94cm	253 (68.6) 33 (8.9)	82 (22.2) 1 (0.3)	[k] 0.093 (0.013–0.694)	0.210	[x] 0.148 (0.016–1.377)	0.093
HC	<102cm ≥102cm	255 (69.1) 31 (8.4)	78 (21.1) 5 (1.4)	[l] 0.527 (0198–1.402)	0.200	[y] 1.453 (0.454–4.655)	0.529
H. pylori	Negative Positive	151 (46.9) 135 (36.6)	22 (6.0) 61 (16.5)	[m] 0.322 (0.185–0.553)	<0.001	[z] 2.628 (1.477–4.678)	0.001
BMI	<24.99kg/m^2 ≥24.99kg/m^2	233 (63.1) 53 (14.4)	81 (22.0) 2 (0.5)	[n] 0.109 (0.026–0.456)	0.002	[a2] 0.345 (0.070–1.696)	0.190

Note: [a, b, c … a2] are identifier reference variables for comparison.

Abbreviations: OPD, outpatient department; JUMC, Jimma University Medical Center; COR, Crud Odds Ratio; AOR, Adjusted Odds Ratio; CI, confidence interval; WC, waist circumference; HC, hip circumference; *H. pylori*, *Helicobacter pylori*; HTN, Hypertension; BMI, body mass index; kg, kilogram; cm, centimeter; mmHg, millimeter mercury.

source population and study subjects they use prospective where as our study is simple cross sectional.[40]

Our study revealed that *H. pylori* was an independent risk factor with a significant positive association to TC, TG, LDL-C but *H. pylori* was inversely related with HDL-C. This findings is comparable with other study conducted in Korea.[38]

The mechanism of extra gastric manifestation of *H. pylori* infection to induces an acute polymorphonuclear infiltration in the gastric mucosa, which is gradually replaced by an immunologically mediated, chronic, predominantly mononuclear cellular infiltration. The mononuclear infiltration is characterized by the local production and systemic diffusion of pro-inflammatory cytokines that can affect remote tissues and organic systems.[41] These cytokines inhibit lipoprotein lipase and mobilization of fat from tissues to blood are responsible for these pathogenic alterations.[42]

Conclusion and Recommendations

H. pylori infected patients are at higher risk of developing dyslipidemia. The presence dyslipidemia among *H. pylori* positive patients shows the possible modification of serum lipid profile. So, it is possible to conclude that *H. pylori* positive patients are more likely to have modified lipid profile than *H. pylori* negative patients. Monitoring and evaluation of TC, TG, LDL-C, and HDL-C in *H. pylori* infected patient is important. Further studies should be conducted with larger sample size using prospective study design and advanced test for *H. pylori* determination to investigate the effect of *H. pylori* infection on serum lipid profiles. There for assessment of lipid profile in H. pylori infected patient is recommended.

Ethics and Consent Statement

Data collection were carried out after approval of the research proposal by the institutional review board (IRB) of Jimma University, institute of health with letter protocol number IHRPG/567/2018. All participants provided written informed consent, and that this study was conducted in accordance with the Declaration of Helsinki. Confidentiality was maintained by using unique identification numbers instead of individual names.

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Author Contributions

All authors contributed to data analysis, drafting and revising the article, gave final approval of the version to be published, and agree to be accountable for all aspects of the work.

Disclosure

All the authors declare that they have no conflicts of interest.

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