Journal of Multidisciplinary Healthcare

Dovepress

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ORIGINAL RESEARCH

Dyslipidemia and Its Associated Factors Among *Helicobacter pylori*-Infected Patients Attending at University of Gondar Comprehensive Specialized Hospital, Gondar, North-West Ethiopia: A Comparative Cross-Sectional Study

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Background: Dyslipidemia refers to a lipid profile disturbance due to decreased high-density lipoprotein cholesterol and elevated low-density lipoprotein cholesterol, triglycerides, and total cholesterol. *Helicobacter pylori* infection can lead to some appetite-related disorders that may cause deregulated absorption of nutrients in the digestive system, contributing to changes in serum lipids. The purpose of this study is to assess dyslipidemia and its associated factors among *H. pylori*-infected patients attending at University of Gondar Comprehensive Specialized Hospital.

Methods: A comparative cross-sectional study was conducted on 231 *H. pylori*-positive and control groups, which were included by the convenience sampling technique from March to May 2021 at University of Gondar Specialized Hospital. Sociodemographic and behavioral characteristic data were collected using a pretested questionnaire, and 5mL of venous blood were used to determine the lipid profiles using DxC 700 AU chemistry analyzer. The data were analyzed using SPSS version 25. Mann–Whitney *U*-test and multivariable logistic regression were applied, and P-value <0.05 is considered statistically significant.

Results: The magnitude of dyslipidemia among *H. pylori*-infected patients was 71.8% (95% CI: 62.7–79.7). There was a statistically significant difference in lipid profiles between *H. pylori*-infected patients and control groups. The median (IQR) of lipid profiles in *H. pylori*-infected patients and control groups were for low-density lipoprotein: 108 (89.8, 145.5) vs 95 (79.45, 115.8, P<0.001), for triglycerides: 93 (65,117) vs 83 (58.5, 102, P=0.031), and cholesterol: 143 (119.5, 169,) vs 125 (110,143, P<0.001) mg/dl, respectively. *Helicobacter pylori* infection, alcohol drinking, unable to read and write, primary school, and secondary school were a significant associated variables with dyslipidemia (P<0.05).

Conclusion: There was a median lipid profile statistically significant difference between *H. pylori*-positive and control groups. *Helicobacter pylori* infection, educational status, and alcohol drinking habit had statistically significant association with dyslipidemia.

Keywords: associated factors, dyslipidemia, *H. pylori*

Background

Dyslipidemia refers to a lipid profile disturbance, including both hyperlipidemia and hypolipidemia.[1] It is categorized by a decreased high-density lipoprotein cholesterol (HDL-C) and an elevated low-density lipoprotein cholesterol (LDL-C), triglycerides (TGs), and total cholesterol (TC).[2]

The invading of the stomach by *H. pylori* causes reliable disturbance of the stomach, which can affect some biochemical parameters (lipid profiles) in the patient. The underlying possible mechanisms for these conditions 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.[3] Scientific facts indicate

Received: 31 March 2022
Accepted: 28 June 2022
Published: 15 July 2022

that *H. pylori* infection can lead to some appetite-related disorders and significant changes in body weight. Dysregulated absorption of nutrients and the effects of the inflammatory response system caused by *H. pylori* infection contribute to changes in serum lipids. The change of lipid profiles may also be due to the effects of the inflammatory response system caused by *H. pylori* infection. Several lines of evidence indicate that the secretion of inflammatory cytokines by cells induced by chronic infection of gram-negative bacteria is related to the change of lipid profiles.[5]

An experimental investigation indicated that interleukin-8, which is overexpressed in *H. pylori*-infection, increases the recruitment of T lymphocytes and smooth muscle cells, contributing to atherosclerosis.[6] In addition to this, lipopolysaccharide (LPS) affects circulating macrophages and increases free radical production. It is known that free radicals oxidize LDL, the result of which (oxidized LDL) transforms macrophages into foam cells are known to be essential in atherosclerosis pathogenesis.[7] With the presence of LPS in the cell walls of *H. pylori*, there is the stimulation of large quantities of cytokines (TNF-α, and IL-6) which inhibit lipoprotein lipase activity.[8,9]

The commitment of dyslipidemia to cardiovascular diseases (CVDs) is apparent from several longitudinal considerations that have outlined the affiliation of high levels of LDL-C, TC, TG, and low levels of HDL-C with CVD.[11–13] Dyslipidemia is a class of TC and TG metabolism disorders that have consequences for the cardiovascular system, causing pathologies such as vascular coronary disease and atherosclerosis. An increase in 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.[14]

Different studies have reported different findings regarding the relationship of *H. pylori* disease and its relation to changes in serum lipid profile. Several studies yield various and sometimes contradictory results: increased, normal, and decreased levels of lipids.[15–18] In *H. pylori* patients, observational studies have found a strong correlation between rising levels of LDL-C or decreasing levels of HDL-C and increased risk of coronary artery disease (CAD) events.[19] Other studies showed that *H. pylori* could play a role in the development of ischemic heart disease through various means, such as endothelial cell colonization, lipid profile changes, hypercoagulation, platelet aggregation, molecular mimicry mechanism induction, and low-grade systemic inflammation progression.[20]

Dyslipidemia is the most asymptomatic metabolic disorders, which is analyzed incidentally or through screening. However, in serious cases, the patient can show one of the indications of the complications (either coronary or peripheral artery illness) such as leg pain, chest pain, dizziness, palpitations, swelling of lower limb or veins (eg, in neck or stomach), and blacking out.

The effects of *H. pylori* infection on lipid profiles are still unknown. Several studies have shown varying and occasionally contradictory results, including raised, normal, and decreased lipid levels, all of which have been reported. This study will be used as a source of information about lipid profiles of *H. pylori*-infected patients for the physicians for early detection, treatment, and prevention of lipid abnormalities. This study will also provide supportive evidence for policymakers and evidence-based information to the scientific community about the lipid profiles among *H. pylori*-infected patients. Additionally, this study will serve as baseline information for other researchers in the study area. Therefore, the aim of this study is assessing dyslipidemia and its associated factors among *H. pylori*-infected patients attending at University of Gondar Comprehensive Specialized Hospital from March 10/2021 to May 10/2021, Gondar, North-West Ethiopia.

Materials and Methods

Study Population, Area, Design and Period

A hospital-based comparative cross-sectional study design was conducted among *H. pylori*-infected patients attending the outpatient department of University of Gondar Comprehensive Specialized Hospital (UGCSH), which is located in Gondar town, Amhara Region, North-West Ethiopia. Gondar is found in the North-West of Ethiopia at about 727 km and 180 km away from the capital city Addis Ababa and Bahirdar respectively. It is at 12°3′ N latitude and 37°28 ′E.[29] The current population of Gondar city is 378,000 and has a total area of 192.3 km^2 with undulating mountainous topography.[30] Currently, the hospital has a catchment population of over 7 million and serving as a referral hospital for all populations in the Central Gondar zone and neighboring district areas. All *H. pylori*-infected patients attending at

UGCSH during the study period and who can participate in the study were included in the study population. Age-matched healthy *H. pylori*-negative adults who visited Gondar Blood Bank during the study period were included in the control groups.

Eligibility Criteria

Inclusion Criteria

All *H. pylori*-infected adult patients aged greater than or equal to 18 years and who were voluntarily to participate were included in the study. All H. *pylori*-negative individuals aged greater than or equal to 18 years and who were willing to participate in the study were included in the control groups.

Exclusion Criteria

Study participants withTB, drug users, antiretroviral treatment users, participants with hypertension and diabetes mellitus (DM) were excluded from the study by screening and reviewing their medical records. Patients who were severely ill were also excluded from the study.

Sample Size Determination and Sampling Techniques

The sample size was determined by using open Epi, version-3 software by considering the following assumptions; mean difference of TG on a study done at the United States of America (USA),[28] sample 1 mean=177.2, SD1=87.5, sample 2 mean=148, SD2=68.2, 95% confidence level, and 80% power (0.84) (power approach two mean difference formula). This gives a total of 228 (114 confirmed *H. pylori* patients and 114 healthy *H. pylori*-negative control groups) study units. A convenience sampling technique was employed to select study participants at UGCSH and adult healthy controls from the Gondar Blood Bank. When the study participants visited the medical OPD with complaint of gastritis, they were tested for *H. pylori*. If the patients were tested positive for *H. pylori*, they were asked to fill written consent for their participation in our study.

Operational Definition

Dyslipidemia was considered when total cholesterol >200 mg/dl and/or triglycerides >150 mg/dl and/or LDL-C> 130 mg/dl and/or, HDL-C <40 mg/dl; male and/or, HDL-C<50 mg/dl; female.[31] Alcohol intake was defined as people who never drink any alcohol and people who do not drink alcohol now but did in the past (non-drinkers), and people who drink alcohol one or more days per week (regular drinkers), and (past drinkers) (ex-drinker).[32] Cigarette smoking was also defined as non-smoker (individuals who never smoke, and those who smoke before but not current) and smoker (individuals who are currently smoking).[32] 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).[33] Physical exercise was considered when a study subject has experience of doing the physical exercise once per day for 20–30 minutes as a continuous activity.[34]

Data Collection and Laboratory Methods

The socio-demographic characteristics of study participants were collected by using a questionnaire prepared for this study. The questionnaire consisted of the study participant's age, sex, ethnicity, religion, marital status, residence, occupation, educational status, income, and behavioral characteristics such as the habit of physical exercise, smoking habit, and a habit of alcohol drinking, and cigarette smoking. Data for these characteristics were collected by trained nurses through a face-to-face interview. The study participants were interviewed after written informed consent was taken.

Height was measured using a height measure scale. Participants stood erect on the stadiometer's floorboard with their backs to the stadiometer's vertical backboard. Both heels of the feet were placed together on the vertical board, with both heels touching the base. The feet were at a 60-degree angle, slightly outward. The participant's shoes and hats were removed during the height assessment. The height measurement was recorded to the nearest 0.1 cm.[35] Before weighing the study subjects, the weight scale was set at zero. Then, the participants were asked to remove extra layers of clothing,

shoes, jewelry, and any items in their pockets. After the participants were asked to step on the scale backward (for confidentiality), their body weight was recorded to the nearest 0.1 kg (100 g).[35] The waist circumference was measured using a tape measure at the level of the iliac processes and the umbilicus to assess abdominal (central) obesity.[36] The spatial distance between each corresponding hip bone in proportion to the buttocks was determined by measuring the hips with a tape measure.[37]

After receiving informed consent from the study participants, 8–12 hours fasting 5 mL of venous blood was collected preferably at the antecubital area by applying a tourniquet. Before collecting the sample, the puncture area of the vein was disinfected by using 70% alcohol. The serum was collected using a serum separator tube at the medical ward department of UGCSH. After collection, specimens were transported to the clinical chemistry unit of the UGCSH laboratory for analysis. The collected blood sample was left for 30 minutes at room temperature. Then, the blood samples were centrifuged for 5 minutes at 3500 revolutions per minute (rpm) to separate serum from formed elements. All these procedures were done by the laboratory technologist and principal investigator by applying standard operating procedures (SOPs). The HDL-C, LDL-C, TG, and TC were analyzed by DxC 700 AU auto analyzer (Beckman Coulter, USA). The stool antigen *H. pylori* RapiCard™ Insta tests were performed for the recruitment of study participants.

Data Quality Assurance and Management

The questionnaire was prepared in both languages English and Amharic (which is the local language). Five percent[4] of the sample size was pre-tested at Poly health center. Data collectors were trained in data collection to eliminate technical and observer bias. After completion of each questionnaire, cross-checking was done between the data collector and principal investigator to assure the completeness of the data collected. The label on the test tube and the study participants' unique identification number on the questionnaire were checked. Before patient sample processing, quality controls (normal and pathological) were performed, and the study participants' result was taken after confirmation of the controls was okay.

Data Analysis and Interpretation

The results were organized and summarized using frequency, and percentage for categorical variables, median (interquartile range) for continuous variables, and tables. The model of fit was checked by Hosmer and Lemeshow's goodness fit statistic. The Kolmogorov–Smirnov and Shapiro Wilk normality test were conducted to check the normality of continuous variables. Since the continuous variables were not normally distributed, Mann–Whitney *U*-tests were used to compare the median and interquartile range of these variables. Multivariable logistic regression analysis was done to control possible confounders and to determine factors statistically associated with dyslipidemia. The variables were selected by the backward selection method. P-values less than 0.05 were considered statistically significant.

Results

Socio-Demographic Characteristics of Study Participants

This study included 117 *H. pylori*-infected individuals and 114 *H. pylori*-negative healthy control groups with response rate of 100%. Among the study groups, 117 (50.65%) were females, ranging in age from 18 to 63 years, with a median (IQR) age of 31 (22, 40) years. The median (IQR) age of *H. pylori*-positive and control groups was 32 (22, 42.5), and 31 (20, 38) years, respectively. The urban residence of study participants was 155 (67.10%), and 121 (52.38%) were married. Based on educational status, 67 (29.00%) study participants were from secondary schools, 77 (33.33%) of occupation status were students (Table 1).

Behavioral and Anthropometric Characteristics of Participants

Based on the lifestyle condition of study participants, almost all of the study participants 230 (99.6%) were non-smokers, 214 (92.6%) did not drink alcohol, 229 (99.13%) did not chew khat, and 196 (84.8%) did not have regular physical exercise. The BMI value of the study participants, 184 (79.7%) had normal (18.5–25kg/m^2), 17 (7.4%) had overweight

Table 1 Sociodemographic, Behavioral and Anthropometric Characteristics of Study Participants Characteristics of Study Subjects (N=231, UGCSH, Gondar, 2021) (Pearson's Chi-Square Test)

Variables	Category	*H. pylori* Positive, N=117 N (%)	Control, N=114 N (%)	Total, N=231 N (%)	p-value
Age group	<40 years	68(58.1)	96(84.2)	164(71)	0.060
	≥40 years	49(41.9)	18(15.8)	67(29)	
Sex	Male	53(45.3)	61(53.5)	114(49.4)	0.486
	Female	64(54.7)	53(46.5)	117(50.6)	
Residence	Rural	62(52.99)	14(12.3)	76(32.9)	0.214
	Urban	55(47.01)	100(87.7)	155(67.1)	
Education status	Illiterate	41(35.0)	13(11.4)	54(23.4)	<0.001
	Primary	30(25.6)	6(5.3))	36(15.5)	
	Secondary	12(10.3)	55(48.2)	67(29.0)	
	≥College	34(29.1)	40(35.1)	74(32.0)	
Occupation	Student	26(22.2)	51(44.7)	77(33.3)	0.221
	Farmer	23(19.7)	6(5.3)	29(12.6)	
	Merchant	5(4.3)	10(8.8)	15(6.5)	
	House wife	37(31.6)	15(13.2)	52(22.5)	
	Gov't employee	18(15.4)	24(21.0)	42(18.2)	
	Private and others[a]	8(6.8)	8(7.0)	16(6.9)	
Marital status	Single	32(27.4)	59(51.8)	91(39.4)	0.119
	Married	74(63.2)	47(41.2)	121(52.4)	
	Separated[b]	11(9.4)	8(7.0)	19(8.2)	
Physical exercise	Yes	16(13.7)	19(16.7)	35(15.2)	0.25
	No	101(86.3)	95(83.3)	196(84.8)	
Body mass index	18.5–24.99 kg/m^2	91(77.8)	93(81.6)	184(79.7)	0.309
	≥25 kg/m^2	6(5.1)	11(9.6)	17(7.4)	
	<18.5 kg/m^2	20(17.1)	10(8.8)	30(13.0)	
Alcohol drinking	Yes	10(8.5)	7(6.10	17(7.4)	<0.001
	No	107(91.5)	107(93.9)	214(92.6)	
Waist circumference	<94 cm	106(90.6)	109(95.6)	215(93.7)	0.113
	≥94 cm	11(9.4)	5(4.4)	16(6.3)	
Hip circumference	<102 cm	97(82.9)	109(95.6)	206(89.2)	0.020
	≥102 cm	20(17.1)	5(4.4)	25(10.8)	

(*Continued*)

Table 1 (Continued).

Variables	Category	*H. pylori* Positive, N=117 N (%)	Control, N=114 N (%)	Total, N=231 N (%)	p-value
Cigarette smoking	Yes	1(0.9)	0	1(0.4)	0.440
	No	116(99.1)	114(100)	230(99.6)	
Khat chewing	Yes	0	2(1.8)	2(0.9)	0.065
	No	117(100)	112(98.2)	229(99.1)	

Note: [a]others= priest, [b]separated (divorced and widowed).

(≥25kg/m^2), and 30 (13.0%) had underweight (<18.5kg/m^2). From the study participants, 215 (93.7%) had less than 94cm of waist circumference, and 206 (89.2%) had less than 102 cm of the hip circumference (Table 1).

The Magnitude of Dyslipidemia Among Study Participants

The overall prevalence of dyslipidemia in either of the four lipid profile parameters was 145 (62.8%) (95% CI: 56.2–69%). From *H. pylori*-infected patients, the prevalence of dyslipidemia in at least one of the parameters among the four lipid profiles were 71.8% (84/117) (95% CI: 62.7–79.7%) and 53.5% (61/114) (95% CI: 43.9–62.9%) were from control groups (Table 2).

The serum level of abnormal lipid profile among *H. pylori*-positive individuals were 53 (45.3%) HDL-C, 41 (35%) LDL-C, 12 (10.3%) TC, and 12 (10.3%) TG, whereas the prevalence of dyslipidemia among control groups were 54 (47.37%), 11 (9.6%), 9 (7.9%), and 1 (0.9%) of serum HDL-C, LDL-C, TC, and TG, respectively (Table 2).

Associated Factors of Dyslipidemia Among Study Individuals

Those variables that had a statistically significant associations with dyslipidemia (P<0.05) in bivariate analysis such as age, education status, *H. pylori* infection, alcohol drinking habits, and hip circumference were entered to multivariate analysis. In multivariable analysis, there was a significant association between dyslipidemia with educational status (p=0.001), alcohol drinking habits (p=0.001), and *H. pylori* infection (p=0.001) (Table 3).

Table 2 Dyslipidemia Among *H. pylori*-Positive Patients and Control Groups (N=231 at UGCSH, Gondar, 2021)

Lipid Profiles	*H. pylori* Status				Total	
	Positive		Control			
	N (%)	95% CI	N (%)	95% CI	N (%)	95% CI
Decreased HDL-C	53(45.3%)	36.1–54.8	54(47.4)	37.9–56.9	107(46.3)	39.8–53
Increased LDL-C	41(35)	26.5–44.4	11(9.6)	4.9–16.6	52(22.5)	17.3–29.4
Hypercholesterolemia	12(10.3)	3.4–11.2	9(7.9)	0–4.8	21(9.1)	3.0–9.4
Hypertriglyceridemia	12(10.3)	3.4–11.2	1(0.9)	3.1–13.4	13(5.6)	5.4–13.1
Total dyslipidemia	84(71.8)	62.7–79.7	61(53.5)	43.9–62.9	145(62.8)	56.2–69

Table 3 Multivariate and Bivariate Logistic Regression Analysis to Identify Associated Factors of Dyslipidemia Among Study Participants (N=231, Gondar, 2021)

Variables		Dyslipidemia		COR (95% CI)	AOR (95% CI)	p-value
		Yes	No			
Age	<40 years	93	71	1	1	
	≥40 years	52	15	2.647 (1.379–5.081)**	1.6779(0.692–4.063)	0.081
Sex	Male	69	45	1	1	1
	Female	76	41	1.209(0.709–2.602)	0.860(0.364–2.033)	0.409
Marital status	Single	50	41	1	1	1
	Married	81	40	1.660(0.948–2.908)	1.328(0.611–2.887)	0.097
	Separated	14	5	2.296(0.763–6.908)	1.329(0.360–4.906)	0.099
Residence	Rural	52	24	1	1	1
	Urban	93	62	0.692(0.387–1.237)	2.308(0.869–6.128)	0.050
Education status	Unable to read and write	41	13	4.139(1.907–8.985)***	4.413(1.384–14.077)**	0.012*
	Primary	26	10	3.412(1.441–8.082)**	3.607(1.026–9.170)	0.045*
	Secondary	46	21	2.875(1.440–5.740)**	4.787(2.167–10.571)	<0.001***
	College and above	32	42	1	1	1
Occupation status	Student	48	29	1	1	1
	Farmer	20	9	1.343(0.539–3.341)	0.269(0.039–1.874)	0.331
	Merchant	10	5	1.208(0.376–3.887)	0.233(0.034–1.582)	0.124
	House wife	38	14	1.640(0.762–3.530)	0.229(0.038–1.373)	0.141
	Gov't employee	21	21	0.604(0.282–1.293)	0.360(0.070–1.841)	0.339
	Private	8	8	0.604(0.205–1.784)	0.082(0.011–0.621)	0.019
Alcohol drinking	Yes	3	14	9.204(2.562–33.062)**	9.767(2.616–36.467)**	0.001**
	No	142	72	1	1	1
BMI	18.5–24.99 kg/m^2	111	73	1	1	1
	>24.99 kg/m^2	12	5	1.578 (0.534–4.668)	2.286(0.614–8.505)	0.119
	<18.5	22	8	1.809(0.764–4.280)	1.869(0.679–5.139)	0.410
Exercise	Yes	25	10	1	1	
	No	120	76	1.583(0.720–3.480)	1.734(0.652–4.614)	0.221
H. pylori status	Negative	61	53	1	1	1
	Positive	84	33	2.212(1.282–3.816)**	3.377(1.637–6.966)**	0.001**
WC	<94 cm	132	83	1	1	1
	≥94 cm	13	3	2.725(0.754–9.850)	1.354(0.126–14.533)	0.858
HC	<102 cm	124	82	1	1	1
	≥102 cm	21	4	3.472(1.150–10.483)*	2.123(0.517–8.7140	0.128

Notes: *p<0.05, **p<0.01, ***p<0.001.

Abbreviations: AOR, adjusted odds ratio; BMI, body mass index; COR, crude odds ratio; CI, confidence interval; HC, hip circumference; WC, waist circumference.

Table 4 Comparison of Lipid Profiles Among Cases and Controls (Cases, N=117, Control-N=114, Gondar, Ethiopia, 2021) (Mann–Whitney *U*-Test

Variables	*H. pylori* Status		P-value
	Positive, Median (IQR)	Control, Median (IQR)	
Age	32(22, 42.5)	31(20, 38)	0.060
HDL-C	41(35, 47)	40(33.75, 44)	0.376
LDL-C	108(89.8, 145.5)	95(79.45, 115.8)	<0.001
TG	93(65, 117)	83(58.5, 102)	0.031
TC	143(119.5, 169)	125(110, 143)	<0.001
HC	89(82.5, 92)	88 (81.75, 93)	0.159
WC	75(69, 86)	77(70.75, 77)	0.837

Note: P<0.05=statistically significant value.
Abbreviations: HDL, high-density lipoprotein; HC, hip circumference; LDL, low-density lipoprotein; TC, total cholesterol; TG, triglycerides; WC, waist circumference.

Comparison of Lipid Profiles Among Study Participants

The median level of serum HDL-C was not statistically significant difference with *H. pylori*-positive and control groups (p=0.376). However, the median serum level of LDL-C, TG, and TC was a statistically significant difference between *H. pylori*-positive and control groups (p<0.05) (Table 4).

Discussion

The prevalence of dyslipidemia among *H. pylori* patients in either of the four lipid profiles was 71.8% (95% CI: 62.7–79.7), which was higher than a study conducted in Iran (60.4%).[22] Our findings, however, were lower than a study done in Ethiopia, which found that 87.2% of *H. pylori* patients had dyslipidemia.[21] The reason for the variation is most likely related to differences in lifestyle, study design, and sample size.

In our finding, the odds of dyslipidemia among peoples unable to read and write were 4.413 times higher (AOR: 4.413; 95% CI: 1.384–14.077, P=0.012), the odds of dyslipidemia being primary school education status were 3.067 times higher (AOR: 3.067; 95% CI: 1.026–9.170; P=0.045), and the odds of dyslipidemia among secondary school education status were 4.787 times higher (AOR: 4.787, 95% CI; 2.167–10.571, P<0.001) compared to higher educational status. Most cardiovascular risk factors were more common at lower educational levels in the most recent National Health Survey, indicating a trend toward better health outcomes as education levels increase.[38] People with a lower level of education are more likely to develop unhealthy eating habits and lifestyles, which can be worsened if their income rises without their level of education.[39]

Dyslipidemia was 9.767 times more common in alcoholics than non-alcoholics (AOR: 9.767, 95% CI: 2.616–36.467, P=0.001) which was consistent with the study done in Korea.[26] This is due to the impact of chronic ethanol drinks on cardiovascular health is through the effect on lipid metabolism.[40] The odds of dyslipidemia with *H. pylori* infection were 3.377 times higher than control groups (AOR: 3.377; 95% CI; 1.637–6.966, P=0.001). This finding was agreed with studies done in Ethiopia and Iran.[21,22]

In our finding, the lipid profiles showed that *H. pylori*-infected individuals have a statistically significant difference in LDL-C, TG, and TC compared with control groups. The HDL-C levels of *H. pylori*-positive and control study participants, however, did not differ significantly. This finding was consistent with a study done in Ethiopia and Iran.[21,22] This may be due to the impact of *H. pylori* infection on lipid metabolism.[41] The current study was consistent with studies conducted in other areas, indicating that *H. pylori* infection may alter serum lipid concentrations, potentially increasing the risk of CHD. *H. pylori*-positive individuals had a statistically significant higher concentration of LDL-C than those control groups (p<0.05).[15,21,24,27,28]

Our finding shows that the median (IQR) of lipid profiles in *H. pylori*-positive individuals was significantly greater than in control groups for LDL-C: 108 (89.8, 145.5) vs 95 (79.45, 115.8), for TG: 93 (65, 117) vs 83 (58.5, 102), and TC:143 (119.5, 169) vs 125 (110, 143) mg/dl respectively with P value less than 0.05. This finding was comparable with a study conducted in Iraq[42] and Japan[18] that *H. pylori* infection was associated with an increase in serum cholesterol, TG, and LDL-C. Our findings were also in line with those of a Turkish study on *H. pylori*-infected patients' serum cholesterol, which was considerably higher (189.32 ± 45.15 vs 179.41 ± 36.37) mg/dl ($p<0.05$) when compared to the control study participants. The patient's group also had significantly higher serum TG and TC/HDL-c values compared to control groups (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.[23]

Our results were comparable with a study done in Ethiopia, the serum LDL-C concentration was higher in *H. pylori*-positive patients than *H. pylori*-negative groups 122 ± 37 vs 104.27 ± 34.71 mg/dl ($p<0.001$), serum TG (185.61 ± 74.82 vs 138.18 ± 60.17 mg/dl ($p<0.001$), and, serum TC (200.8 ± 43.48 vs 173.67 ± 42.41 mg/dl ($p<0.001$), respectively.[21] Our findings also supported by those of a study conducted in Sudan,[24] the United States,[28] China,[15] and Korea,[27] which found a statistical difference between patients and controls with TC, TG, and LDL-C values with a p-value of <0.05.

Our findings showed that *H. pylori* infection increased TC, LDL-C, and TG levels in infected participants when compared to control groups. This finding was in line with a Korean study that found *H. pylori* is associated with higher levels of TC and LDL-C.[14] This is due to a disturbance in food absorption in the digestive system, which causes alterations in serum lipids. The effects of the inflammatory response system caused by *H. pylori* infection may potentially be responsible for the alteration in lipid profiles.[5] Due to the presence of LPS in the cell walls of gram-negative bacteria like *H. pylori*, high amounts of cytokines (TNF-α, and IL-6) are released, inhibiting lipoprotein lipase action. The consequence being the mobilization of fat from tissues to blood is responsible for an increase in serum lipid levels.[8,43]

In contrast, our result was not comparable in a study done in Finland indicate that *H. pylori* infection was no significant difference in *H. pylori*-positive and control groups with serum TC, LDL-C, and TG. The serum HDL-C concentration in *H. pylori*-positive patients had a significant difference than in control groups ($p<0.001$).[16] Whereas, in our finding LDL-C, TG, and TC concentration was a statistical difference between cases and controls but not in HDL-C. Our findings also not supported by a study done in Iran shows that the value of TG, LDL-C were high and HDL-C was low in *H. pylori*-positive than control groups. But these findings were not a statistical difference between *H. pylori*-positive and control groups.[17] A study was done in China also shows that the value of TG and LDL-C was not statistically different between *H. pylori*-positive and control groups.[25] This is due to differences in the study design, source population, study participants they use, and the way they define dyslipidemia.

Conclusion

Based on our findings, we conclude that *H. pylori* patients have more likely to develop dyslipidemia than control (healthy) groups. There is a statistically significant association between *H. pylori* infection with cardiac and coronary risk factors like a high concentration of LDL-C, TG, and TC. Alcohol drinking habits, and education status were statistically significantly associated with dyslipidemia. For health professionals, monitoring and assessment of serum lipid profiles are important for the management of dyslipidemia. So, patients who had *H. pylori* infection should be assessed for their serum lipid profiles.

Abbreviations

AOR, adjusted odds ratio; BP, blood pressure; BMI, body mass index; CVDs, cardio vascular diseases; CAD, coronary artery disease; COR, crude odds ratio; DBP, diastolic blood pressure; *H. pylori, Helicobacter pylori*; HDL-C, high-density lipoprotein cholesterol; IL, interleukin; IQR, interquartile range; LPS, lipopolysaccharide; LDL-C, low-density lipoprotein cholesterol; NCDs, non-communicable diseases; SST, serum separator tube; SOPs, standard operating procedures; SPSS, Statistical Package for Social Science; TGs, triglycerides; TC, total cholesterol; WHO, World Health Organization.

Data Sharing Statement

All the primary data are available and if anyone has a reasonable interest to find the data, they can contact the corresponding author.

Ethics Approval and Consent to Participate

This study was done based on the declaration of Helsinki. Ethical approval was obtained from the Research and Ethical Committee of the School of Biomedical and Laboratory Sciences, the University of Gondar with the reference number of SBLS 2762 on March 08, 2021. Data collectors explained the purpose, confidentiality, and discomfort related to the study to each participant and obtained a fully informed written consent. The privacy of information was kept up during and after an interview in which coding was utilized for all the information collected and those who had lipid abnormalities were advised to visit the UGCSH Medical OPD for further diagnosis and treatment.

Acknowledgments

We would like to thank study participants, University of Gondar, and data collectors for their cooperation and financial, material, and reagent support.

Funding

The data collection and laboratory diagnosis were funded by University of Gondar.

Disclosure

The authors declare that they have no conflicts of interest in relation to this work.

References

1. Welty FK. Hypobetalipoproteinemia and abetalipoproteinemia. *Curr Opin Lipidol*. 2014;25(3):161. doi:10.1097/MOL.0000000000000072
2. Bays HE, Toth PP, Kris-Etherton PM, et al. Obesity, adiposity, and dyslipidemia: a consensus statement from the National Lipid Association. *J Clin Lipidol*. 2013;7(4):304–383. doi:10.1016/j.jacl.2013.04.001
3. Kusters JG, Van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. *Clin Microbiol Rev*. 2006;19(3):449–490. doi:10.1128/CMR.00054-05
4. Osawa H, Nakazato M, Date Y, et al. Impaired production of gastric ghrelin in chronic gastritis associated with Helicobacter pylori. *J Clin Endocrinol Metab*. 2005;90(1):10–16. doi:10.1210/jc.2004-1330
5. Khovidhunkit W, Kim M-S, Memon RA, et al. Effects of infection and inflammation on lipid and lipoprotein metabolism: mechanisms and consequences to the host. *J Lipid Res*. 2004;45(7):1169–1196. doi:10.1194/jlr.R300019-JLR200
6. Buzás GM. Metabolic consequences of Helicobacter pylori infection and eradication. *World J Gastroenterol*. 2014;20(18):5226. doi:10.3748/wjg.v20.i18.5226
7. Ellis R. Infection and coronary heart disease. *J Med Microbiol*. 1997;46(7):535–539. doi:10.1099/00222615-46-7-535
8. Sung KC, Rhee EJ, Ryu SH, Beck S-H. Prevalence of Helicobacter pylori infection and its association with cardiovascular risk factors in Korean adults. *Int J Cardiol*. 2005;102(3):411–417. doi:10.1016/j.ijcard.2004.05.040
9. Feingold KR, Grunfeld C. Lipids: a key player in the battle between the host and microorganisms. *J Lipid Res*. 2012;53(12):2487–2489. doi:10.1194/jlr.E033407
10. World Health Organization. Noncommunicable diseases. Fact sheet. Available from: http://www.who.int/mediacentre/factsheets/fs355/en/. Accessed March 23, 2017.
11. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. *JAMA*. 2007;298(3):309–316. doi:10.1001/jama.298.3.309
12. D'Agostino Sr RB, Pencina MJ, Massaro JM, Coady S. Cardiovascular disease risk assessment: insights from Framingham. *Glob Heart*. 2013;8(1):11–23. doi:10.1016/j.gheart.2013.01.001
13. Reinikainen J, Laatikainen T, Karvanen J, Tolonen H. Lifetime cumulative risk factors predict cardiovascular disease mortality in a 50-year follow-up study in Finland. *Int J Epidemiol*. 2015;44(1):108–116. doi:10.1093/ije/dyu235
14. Kim H-L, Jeon HH, Park IY, Choi JM, Kang JS, Min K-W. Helicobacter pylori infection is associated with elevated low density lipoprotein cholesterol levels in elderly Koreans. *J Korean Med Sci*. 2011;26(5):654. doi:10.3346/jkms.2011.26.5.654
15. Gong Y, Wei W, Jingwei L, Nannan D, Yuan Y. Helicobacter pylori infection status correlates with serum parameter levels responding to multi-organ functions. *Dig Dis Sci*. 2015;60(6):1748–1754. doi:10.1007/s10620-015-3522-2
16. Pohjanen V-M, Koivurova O-P, Niemelä SE, Karttunen RA, Karttunen TJ. Role of Helicobacter pylori and interleukin 6-174 gene polymorphism in dyslipidemia: a case–control study. *BMJ open*. 2016;6:1. doi:10.1136/bmjopen-2015-009987
17. Haeri M, Parham M, Habibi N, Vafaeimanesh J. Effect of Helicobacter pylori infection on serum lipid profile. *J Lipids*. 2018;2018. doi:10.1155/2018/6734809

18. Shimamoto T, Yamamichi N, Gondo K, et al. The association of Helicobacter pylori infection with serum lipid profiles: an evaluation based on a combination of meta-analysis and a propensity score-based observational approach. *PLoS One*. 2020;15(6):e0234433. doi:10.1371/journal.pone.0234433
19. Kopin L, Lowenstein C. In the clinic. Dyslipidemia. *Ann Intern Med*. 2010;153(3):Itc21. doi:10.7326/0003-4819-153-3-201008030-01002
20. Azizian R, Khosravi A, Azizian M. The association of the human leukocyte antigen (HLA) with the pathogenesis of Helicobacter pylori. *J Pure Appl Microbiol*. 2013;7(3):2183–2189.
21. Abdu A, Cheneke W, Adem M, Belete R, Getachew A. Dyslipidemia and associated factors among patients suspected to have Helicobacter pylori Infection at Jimma University Medical Center, Jimma, Ethiopia. *Int J Gen Med*. 2020;13:311. doi:10.2147/IJGM.S243848
22. Karim I, Zardari AK, Shaikh MK, Baloch ZAQ, Shah SZA. DYSLIPIDEMIA. *Prof Med J*. 2014;21(05):956–959. doi:10.29309/TPMJ/2014.21.05.2529
23. Al-Fawaeir S, Zaid MA, Awad AA, Alabedallat B. Serum lipid profile in Helicobacter pylori infected patients. *Am J Physiol Biochem Pharmacol*. 2013;2(2):1–4.
24. Ali MAE. Assessment of serum high sensitivity C-reactive protein, lipid profile and magnesium among Sudanese Patients with H. pylori Infection. (In Khartoum State). Sudan University of Science & Technology; 2019.
25. Jia E-Z, Zhao F-J, Hao B, et al. Helicobacter pylori infection is associated with decreased serum levels of high density lipoprotein, but not with the severity of coronary atherosclerosis. *Lipids Health Dis*. 2009;8(1):1–7. doi:10.1186/1476-511X-8-59
26. Kim D-H, Son BK, Min K-W, et al. Chronic gastritis is associated with a decreased high-density lipid level: histological features of gastritis based on the updated Sydney system. *J Clin Med*. 2020;9(6):1856. doi:10.3390/jcm9061856
27. Kim TJ, Lee H, Kang M, et al. Helicobacter pylori is associated with dyslipidemia but not with other risk factors of cardiovascular disease. *Sci Rep*. 2016;6(1):1–8. doi:10.1038/s41598-016-0001-8
28. Tang DM, Chascsa DM, Chou JY, et al. Helicobacter pylori infection is strongly associated with metabolic syndrome, and weakly associated with non-alcoholic fatty liver disease, in a US Hispanic population. *GastroHep*. 2019;1(6):325–331. doi:10.1002/ygh2.375
29. Atsede D, Tegegne MAN, Meseret K, Desta KG, Nedessa HMB. CITY PROFILE GONDAR. SES social inclusion and energy management for informal urban settlements; 2015. Available from :https://docplayer.net/202405349-City-profil-gondar-ses-social-inclusion-and-energy-management-for-informal-urban-settlements html#show_full_text. Accessed July 7, 2022.
30. Macrotrends. Gondar, Ethiopia Metro Area Population 1950–2021. Available from: https://www.macrotrends.net/cities/20926/gondar/population. Accessed July 7, 2022.
31. Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the international diabetes federation task force on epidemiology and prevention; national heart, lung, and blood institute; American heart association; world heart federation; international atherosclerosis society; and international association for the study of obesity. *Circulation*. 2009;120(16):1640–1645. doi:10.1161/CIRCULATIONAHA.109.192644
32. Megerssa Y, Gebre M, Birru S, Goshu A, Tesfaye D. Prevalence of undiagnosed diabetes mellitus and its risk factors in selected institutions at Bishoftu Town, East Shoa. *Ethiopia J Diabetes Metab*. 2013;1:S12
33. Nhanes C. *Anthropometry Procedures Manual*. Atlanta (GA): CDC; 2007.
34. Corbridge SJ, Nyenhuis SM. Promoting physical activity and exercise in patients with asthma and chronic obstructive pulmonary disease. *J Nurse Pract*. 2017;13(1):41–46. doi:10.1016/j.nurpra.2016.08.022
35. Zaret B, Battler A, Berger H, et al. Report of the joint international society and federation of cardiology/world health organization task force on nuclear cardiology. *Eur Heart J*. 1984;5(10):850–863. doi:10.1093/oxfordjournals.eurheartj.a061576
36. Bayram F, Kocer D, Gundogan K, et al. Prevalence of dyslipidemia and associated risk factors in Turkish adults. *J Clin Lipidol*. 2014;8(2):206–216.
37. Cameron N, Schell L. *Human Growth and Development*. Academic Press; 2012.
38. Health. Mo. National Health Survey ENS Chile 2009–2010. Santiago; 2010. Available from: http://webminsalcl/portal/url/item/bcb03d7bc28b64dfe040010165012d23pdf. Accessed March 03, 2017.
39. Egerter S, Braveman P, Sadegh-Nobari T, Grossman-Kahn R, Dekker M. *Issue Brief 6: Education and Health*. Princeton, NJ: Robert Wood Johnson Foundation; 2009.
40. Brinton EA. Effects of ethanol intake on lipoproteins and atherosclerosis. *Curr Opin Lipidol*. 2010;21(4):346–351. doi:10.1097/MOL.0b013e32833c1f41
41. Murray LJ, Bamford KB, O'Reilly D, McCrum EE, Evans AE. Helicobacter pylori infection: relation with cardiovascular risk factors, ischaemic heart disease, and social class. *Heart*. 1995;74(5):497–501. doi:10.1136/hrt.74.5.497
42. Moutar F, Alsamarai A, Ibrahim F. Lipid profile in patients with gastritis. *World J Pharm Pharm Sci*. 2016;5(7):74–90.
43. Grunfeld C, Gulli R, Moser A, Gavin L, Feingold K. Effect of tumor necrosis factor administration in vivo on lipoprotein lipase activity in various tissues of the rat. *J Lipid Res*. 1989;30(4):579–585. doi:10.1016/S0022-2275(20)38349-8