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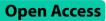
Association between infection with *Helicobacter pylori* and metabolic syndrome among diabetic patients attending Jimma medical center in Jimma city, Ethiopia: a cross-sectional study

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Abstract

Background Previous studies have implicated the role of H. pylori infection in developing the metabolic syndrome. However, findings remain contradictory, and data from developing countries are scarce.

Methods We employed a cross-sectional study design to assess the relationship between H. pylori infection and metabolic syndrome among diabetic patients attending Jimma Hospital, Ethiopia. An interviewer-led questionnaire administered to study participants provided information on sociodemographic factors, and medical records were used to obtain medical history information. Metabolic parameters, including plasma glucose, triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), body-mass index (BMI), waist circumference (WC), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were collected. H. pylori infection status was assessed using IgG Enzyme-linked Immunosorbent Assays (ELISA). The effect of H. pylori infection on metabolic syndrome and metabolic parameters was determined using multivariate linear and logistic regressions.

Results We found H. pylori infection status was positively but not significantly associated with metabolic syndrome (AOR = 1.507, 95% CI: 0.570–3.981, $p = 0.408$). When the analysis was restricted to individual metabolic parameters, *H. pylori* positivity was significantly associated with lower HDL-c and higher SB, respectively.

Conclusions Our result confirms that individual metabolic parameters, not an overall metabolic syndrome, are significantly associated with *H. pylori* infection. Future studies should examine the relationship between *H. pylori* and metabolic syndrome, considering gastrointestinal conditions such as GERD, GU, and DU.

Keywords Ethiopia, Helicobacter pylori, Metabolic syndrome, Metabolic parameters

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Introduction

Helicobacter pylori is a gram-negative bacterium affecting about 50% of the world's population and is more prevalent in developing countries [1, 2]. A recent review of the global epidemiology of *Helicobacter pylori* in adults from 1970 to 2016 and children and adolescents from 2000 to 2021 indicated a consistently higher prevalence in Africa, the Eastern Mediterranean regions, and Middle and South America [3]. Moreover, a pooled analysis of *H. pylori* data involving 410,879 participants from 73 countries revealed variations in the prevalence of *H. pylori* by region, age, and sex, which are linked with different economic and social conditions [2].

H. pylori persistently inhabits the gastric mucosa in the absence of treatment [4] although the majority of those infected are asymptomatic. There is good evidence supporting the role of *H. pylori* in the development gastric and duodenal ulcer, distal gastric adenocarcinoma, peptic ulcer disease, and primary gastric mucosa associated lymphoid tissue (MALT) lymphoma [5, 6]. Given the ability of *H. pylori* virulence factors to modulate host immune response [7], there is growing interest in investigating the effects of *H. pylori* in extragastroduodenal diseases. A growing body of evidence has supported an association between *H. pylori* and extragastroduodenal diseases including cardiopulmonary, hematologic, neurologic, dermatologic, and metabolic diseases [8–11]. Our group in Ethiopia has found higher anemia prevalence, decreased growth trajectory, and reduced platelet indices among *H. pylori*-infected children compared with non-infected children [12–14], and provided supportive evidence for the potential role of *H. pylori* in extragastroduodenal diseases from a resource limited setting. However, these investigations didn't explore the involvement of *H. pylori* in metabolic syndrome.

The potential association between *H. pylori* and metabolic syndrome is a topic of great interest as metabolic syndrome affects an estimated one-quarter of the world population, and it is becoming increasingly prevalent [15, 16]. Metabolic syndrome is defined by a variety of factors that predispose patients to greater risk of cardiovascular disease, type II diabetes, as well as significantly higher total mortality [17]. Metabolic parameters that predispose patients to developing metabolic syndrome include central obesity in addition to abnormal glucose levels, triglyceride levels, systolic blood pressure, diastolic blood pressure, as well as high-density lipoprotein and low-density lipoprotein cholesterol levels. Pervious clinical and epidemiological studies investigating the association between *H. pylori* infection status and prevalence of metabolic syndrome reported a positive association [18–28]. While two studies to date have found no such association [29, 30]. More recently, a meta-analysis conducted by Azami et al. suggests that collective findings overwhelmingly support a significant association between H. pylori infection and metabolic syndrome [8]. H. pylori infection has been linked to impaired insulin signaling in hepatocytes and increased expression of various inflammatory factors involved in developing insulin resistance [9]. Insulin resistance is a key factor associated with metabolic syndromes [31]. While metabolic syndrome is influenced by multiple genetic and environmental risk factors [32], proving the association between H. pylori and metabolic syndromes could make H. pylori one of the treatable causes of metabolic syndromes.

While previous epidemiological observations and mechanistic insights provide support for a role of *H. pylori* infection in metabolic syndrome development, most studies have been conducted in high-income populations, neglecting data from developing countries where the prevalence of *H. pylori* is greater [33]. Therefore, the aim of this study was to assess the relationship between *H. pylori* infection and metabolic syndrome, as well as the relationship between *H. pylori* infection and metabolic syndrome indicators including glucose, triglycerides, HDL-C, waist circumference, systolic blood pressure, and diastolic blood pressure. We therefore used clinical setting data from a developing country to assess the relationship between *H. pylori* infection and metabolic syndrome among diabetic patients attending Jimma Medical Center in Jimma, Ethiopia.

Methods

Study setting

This study was conducted at Jimma Medical Center (JMC) in Jimma Town, Southwest Ethiopia. JMC is located in Jimma city, 352 km Southwest of Addis Ababa. Jimma, part of the Oromia region, sits at an elevation of 1780 m above sea level, and average daily temperatures range from 20 °C in July to 27 °C in January. The population of Jimma is estimated to be 208 000. Jimma Medical Center, comprised of 1600 staff members, provides service to a catchment population 15 million people, and is currently the only teaching and referral hospital in the southwestern part of Ethiopia. The chronic care clinic section of the center provides services for noncommunicable diseases including hypertension and diabetes. Over three-thousand diabetic patients attend the diabetic follow up clinic, which runs twice weekly and provides integrated care for diabetic patients. Physicians, nurses, and final year medical and B Sc nursing students all provide diabetic services at the clinic. Data collection took place from August 15, 2020, to October 30, 2020. A convenience sampling technique was used to select 321 diabetic patients. We recruited patients attending the JMC chronic clinic that were over 18 years old. Patients who were critically ill, pregnant, unconscious, or had communication difficulties were excluded from the

study. Patients who received antibiotics, proton pump inhibitors, H2 blockers, or bismuth treatment within one month prior to the study were also excluded.

Measurement and data collection
Questionnaires
Upon enrolling patients in this study, we then conducted structured questionnaires via face-to-face interviews, carried out by trained data collectors in the local language of patients, such as Afan Oromo or Amharic. These questionnaires were derived from the WHO stepwise approach to surveillance instruments, containing socio-demographic information, medical information, and modifiable risk factors such as smoking, alcohol consumption, khat chewing, fruit and vegetable consumption, type of oil used, and engagement in physical activity. Physical activity questionnaires were used to obtain data on the engagement of participants in physical activity in their daily lives. A participant engaging in greater than 150 min of moderate-intensity physical activity or greater than 75 min of high-intensity physical activity throughout the week, or greater than an equivalent combination of moderate- and high-intensity physical activity, was defined as physically active and labeled as "Yes". Participants who did not meet this physically active definition criteria were defined as physically inactive and labeled as "No". (Online supplementary information)

Anthropomorphic data
Anthropomorphic measurements including waist circumference (cm), weight (kg), and height (m) were measured, and body mass indices were computed as (weight (kg)) / [height (m)]^2. All anthropomorphic measurements were performed by a trained data collector. Participants were instructed to remove their shoes, and digital scales were used to measure each participant's weight. Standing height of shoeless participants was measured using a stadiometer. Waist circumference (WC) was measured around the midway portion between the lower border of the ribs and the bone crest at the widest portion over lightweight clothing using a soft tape measure, applying no pressure to the body.

Blood pressure data
A mercury-based sphygmomanometer was used to measure systolic blood pressure (SBP) and diastolic blood pressure (DBP). For blood pressure measurement, participants were instructed to lay and rest in a supine position for more than ten minutes, and then blood pressure measurements were obtained. Clinical data including but not limited to family history of diabetes, duration of diabetes, hypertension status, and medication for hypertension were obtained from patient self-reporting and analysis of personal health record files.

Blood sample collection and processing
Blood samples were taken from the antecubital vein of patients with minimal tourniquet time. After that, the punctured area was disinfected with 70% ethanol, and the samples were collected in a serum separator tube. Five milliliters (mL) of venous blood were collected after an overnight fast (8–12 h), and then transported to the clinical chemistry unit of the JMC laboratory for analysis. The blood specimens were held at room temperature for 20–30 min to facilitate clot formation, then centrifuged at 3000 rpm for five minutes to separate the serum from the blood. After separation, the serum samples were stored in Nunc tubes at -20 ºC until analysis was performed.

Laboratory testing
All biochemical analyses were performed in the clinical chemistry unit of Jimma Medical Center Laboratory by the spectrophotometric method of the Cobas 6000 chemistry analyzer. Plasma glucose was determined by enzymatic hexokinase principle. Total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein (LDL-c), and triglycerides (TG) were determined by enzymatic colorimetric methods. From the same sample, *H. pylori* antibody levels were determined by the IgG Enzyme-linked Immunosorbent Assays (ELISA) method according to the manufacturer's instructions. This was performed by the ELISA reader in the screening room at the Jimma blood bank laboratory. A patient was considered to be positive for.

H. pylori infection if the IgG anti *H pylori* antibody concentration was >20 U/mL.

Data quality assurance
Questionnaires were prepared in English and translated to local language (Amharic and Afan Oromo). Data were collected by licensed laboratory technologist and nurses under the supervision of a principal investigator. To avoid mislabeling of samples, test tube labels were cross-checked with unique patient identification numbers. To ensure that specimens were free from hemolysis and lipemia, collected samples were visually checked. Blood samples were immediately processed and separated. All laboratory activities were completed according to standard operating procedures and reagents and instrumental equipment were thoroughly checked prior to analyzing patient samples.

Outcome and exposure variables
The primary study outcome was metabolic syndrome status. We defined metabolic syndrome according to modified International Diabetes Federation (IDF) criteria, present in Rafaeli et al. (2018) [26], which defined metabolic syndrome as two or more of the following conditions in addition to central obesity, defined as

BMI≥30 kg/m^2: triglycerides≥150 mg/dL, HDL<40 mg/dL, plasma glucose≥100 mg/dL. 'Exposure to *H. pylori* infection' was defined as a positive result provided by the ELISA reader.

Statistical analysis

Survey data and laboratory data from the JMC patient population was cleaned and coded for statistical analysis using IBM SPSS Statistics V.27. Before investigating the association between *H. pylori* infection and metabolic syndrome, univariate analyses were used to identify potential confounders. Variables associated with both the exposure (*H. pylori* infection) and outcome (metabolic syndrome status) variables in the crude analysis using a statistical significance cutoff of $p < 0.3$ were considered to be possible confounders. For our modified IDF definition of metabolic syndrome, these included family history of diabetes and coffee drinking status. The primary outcome of this analysis was metabolic syndrome status. Our hypothesis that *H. pylori* infection would be associated with metabolic syndrome status was assessed using univariate logistic regression to calculate a crude odds ratio and obtain a p value. Then multivariate logistic regression was performed, adding potential confounders to the model and then removing them according to the backward elimination technique. Additionally, we examined the relationship between *H. pylori* infection status and continuous variables (metabolic parameters) using univariate and multivariate generalized linear models. These variables included plasma glucose, triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), bodymass index (BMI), waist circumference (WC), systolic blood pressure (SBP), and diastolic blood pressure (DBP). First, we analyzed the crude mean difference between *H. pylori*-positive and *H. pylori*-negative individuals, and then we repeated the analysis while adjusting for possible confounders, using the backwards elimination technique.

Results

Selected demographic characteristics and H. pylori infection status

A total of 321 patients were enrolled in this study. Of these participants, 62.6% (201/321) were male and a slight majority of 55.1% (177/321) lived in a rural area. The age range of the study population was 18–69 years and 29.3% (94/321) of participants were within the 60–69 years old range. The income range of the study participants spanned from no monthly income to monthly income≥4000 Birr and 38% (122/321) reported having no monthly income. Analyzing education, 31.5% (101/321) of study participants had no formal education while 43.6% (140/321) of participants had received primary education. Most participants 85.4% (274) were married and 81.9% (263) reported regularly drinking

coffee. The prevalence of *H. pylori* infection was 69.5% (223/321) (Table 1).

Metabolic syndrome components in study population

Of the entire study population, 8.4% (27/321) of participants were classified as having metabolic syndrome according to modified IDF criteria. All participants had glucose levels≥100 mg/dL, and 59.5% (191/321) participants had triglycerides≥150 mg/dL. Only 9% (29/321) of the population had elevated high-density lipoprotein (HDL-c) levels. Examining blood pressure, 42.7% of participants had elevated systolic blood pressure (SBP)≥130 mmHg, and 22.7% (73/321) had elevated diastolic blood pressure (DBP) (Table 2).

Univariate analysis for relationships between potential confounders and H. pylori infection

The crude association between demographic variables and *H. pylori* infection was examined using univariate logistic regression. Univariate logistic regression was performed between demographic variables and metabolic syndrome status and then performed between demographic variables and *H. pylori* status to identify potential confounders. Family history of diabetes and coffee drinking status were found to be potential confounders in relation to the modified IDF definition of metabolic syndrome which is defined as two or more of the following conditions in addition to central obesity, defined as BMI≥30 kg/m^2: triglycerides≥150 mg/dL, HDL<40 mg/dL, plasma glucose≥100 mg/dL. (Supplementary Tables 1 and 2). Interestingly, maternal education status, urban or rural residence, and age were not identified as significant confounders.

Association between H. Pylori infection and metabolic syndrome

In a multivariate logistic model between *H.* pylori status and metabolic syndrome status adjusted for potential confounders, patients infected with *H. pylori* showed higher odds of metabolic syndrome according to modified IDF criteria, yet results were not statistically significant (Adjusted odds Ratio (AOR)=1.507, 95% CI: 0.570–3.981, $p = 0.408$) (Table 3).

Association between H. Pylori infection and metabolic syndrome-related outcomes

Linear regression models related plasma glucose, triglycerides, high-density lipoprotein cholesterol, bodymass index, waist circumference, systolic blood pressure, and diastolic blood pressure as continuous outcomes to the individual estimates of *H. pylori* status. These models showed significant increase in waist circumference among patients infected with *H. pylori* (mean difference: 4.337, 95% CI: 0.151–8.523, $p = 0.042$). These models also

Table 1 Demographic characteristics of study subjects

Variable	N	Percent (%)
Sex		
Male	201	62.6
Female	120	37.4
Participant Occupation		
Employee	71	22.1
Farmer	110	34.3
Housewife	69	21.5
Other	71	22.1
Education		
Illiterate	101	31.5
Primary School	140	43.6
High School	35	10.9
Diploma and Beyond	45	14
Residence		
Rural	177	55.1
Urban	144	44.9
Diabetes Mellitus Type		
Type 1 DM	88	27.4
Type 2 DM	233	72.6
Hypertension Status		
Yes	135	42.1
No	186	57.9
Medication for Hypertension		
Yes	125	38.9
No	196	61.1
Family History of Diabetes		
Yes	27	8.4
No	294	91.6
Smoking Status		
Yes	5	1.6
No	316	98.4
Alcohol Consumption Status		
Yes	12	3.7
No	309	96.3
Fruit Consumption Status		
Yes	297	92.5
No	24	7.5
Vegetable Consumption Status		
Yes	299	93.1
No	22	6.9
Khat Chewing Status		
Yes	90	28
No	231	72
Type of Oil or Fat Used		
Liquid Oil	169	52.6
Solid Oil	152	47.4
Coffee Drinking Status		
Yes	263	81.9
No	58	18.1
Physical Activity		
Yes	115	35.8
No	206	64.2
Level of Physical Activity		

Table 1 (continued)

Variable	N	Percent (%)
inactive	206	64.2
Light	57	17.8
Moderate	15	4.7
Vigorous	43	13.4
Income (Birr)		
No Monthly Income (0)	122	38
1 to 1999	109	34
2000–3999	56	17.4
≥4000	34	10.6
Amount of Sleep		
7–8 h.	196	61.1
less than 6 h.	29	9
more than 9 h.	96	29.9
Participant Marital Status		
Single	35	10.9
Married	274	85.4
Divorced	12	3.7
Diabetes Duration		
1–5 yrs.	181	56.4
6–10 yrs.	86	26.8
≥11yrs.	54	16.8
Age		
18–29	44	13.7
30–39	49	15.3
40–49	57	17.8
50–59	77	24
60–69	94	29.2
H. pylori status		
Negative	98	30.5
Positive	223	69.5

Table 2 Prevalence of metabolic syndrome components in study population

Variable	N	Percent (%)
Glucose		
≥100 mg/dL	321	100
Tryglycerides (TG)		
<150 mg/dL	130	40.5
≥150 mg/dL	191	59.5
HDL-C		
>40 mg/dL(M) or >50 mg/dL (F)	29	9
≤40 mg/dL(M) or ≤50 mg/dL (F)	292	91
Systolic Blood Pressure (SBP)		
<130 mmHg	184	57.3
≥130 mmHg	137	42.7
Diastolic Blood Pressure (DBP)		
<85 mmHg	248	77.3
≥85 mmHg	73	22.7
Metabolic syndrome *		
Yes	27	8.4
No	294	91.6

*Metabolic syndrome defined according to IDF criteria: two or more of the following conditions in addition to central obesity, defined as BMI≥30 kg/m²: triglycerides≥150 mg/dL, HDL<40 mg/dL, plasma glucose≥100 mg/dL

showed significant increase in systolic blood pressure among patients who were infected with *H. pylori* (mean difference: 4.867, 95% CI: 0.350–9.384, p=0.035). When the analysis was adjusted for potential confounders found for JIS criteria, findings no longer remained statistically significant. When the analysis was adjusted for potential confounders using modified IDF criteria, there was a significant decrease in HDL-c observed among patients infected with *H. pylori* (adjusted mean difference: 6.651, 95% CI: 0.902–12.401, p=0.024). Additionally, the increase in systolic blood pressure among *H. pylori*-infected patients remained significant when adjusting for confounders that were found using the IDF definition of metabolic syndrome (mean difference: 5.650, 95% CI: -11.278 - -0.22, p=0.049) (Table 4).

Discussion

This study contributes to our understanding of the influence of *H. pylori* on metabolic syndrome status in diabetic patients in Ethiopia. We found *H. pylori* infection status was positively but not significantly associated with metabolic syndrome. We also found *H. pylori* positivity

Table 3 Univariate and multivariate logistic regression analysis of metabolic syndrome risk associated with *H. pylori* infection status

Variable	Metabolic Syndrome *N*(%)*		COR (95%CI)	*P*-value	AOR (95%CI) **	*P*-value
H. pylori Status	Yes	No				
Positive	21 (9.4)	202 (90.6)	1.594 (0.623–4.082)	0.331	1.507 (0.570–3.981)	0.408
Negative	6 (6.1)	92 (93.9)	1	-	1	-

*Metabolic Syndrome defined according to IDF criteria: two or more of the following conditions in addition to central obesity, defined as BMI≥30 kg/m2: triglycerides≥150 mg/dL, HDL<40 mg/dL, plasma glucose≥100 mg/dL

**Adjusted for family history of diabetes and coffee drinking status

Table 4 T-test analysis analyzing unadjusted and adjusted mean difference between *H. Pylori* infection status and metabolic parameters including blood glucose, triglycerides, HDL-C, WC, SBP, and DBP

Variables	H. pylori Positive Mean [SD]	H. pylori Negative Mean [SD]	Crude Mean Difference (95% CI)	*p*-value	Adjusted Mean Difference (95% CI)	*p*-value
Glucose	174.844 [4.582]	177.342 [6.912]	2.498 (-18.814–13.819)	0.763	10.318 (-43.660–23.024)*	0.543
TG	186.284 [6.753]	177.432 [10.187]	8.852 (-15.193–32.898)	0.469	17.813 (-47.587–11.961) †	0.24
HDL-C	30.820 [0.630]	32.887 [0.950]	2.067 (-4.309–0.176)	0.071	6.651 (0.902–12.401) §	0.024
WC	89.602 [1.176]	85.265 [1.773]	4.337 (0.151–8.523)	0.042	4.321 (-14.092–2.912) **	0.197
SBP	126.520 [1.269]	121.653 [1.914]	4.867 (0.350–9.384)	0.035	5.650 (-11.278–0.22) ††	0.049
DBP	78.287 [0.770]	76.092 [1.162]	2.195 (-0.548–4.938)	0.116	0.394 (-6.033–5.244) §§	0.891

† adjusted for age, coffee drinking status, amount of sleep, marital status

§ adjusted for age

** adjusted for age, DM type, coffee drinking status, amount of sleep, marital status

†† adjusted for age, DM type, marital status

§§ adjusted for age, DM type, coffee drinking status, marital status

was significantly associated with lower HDL-c and higher SB, respectively.

Several other studies have examined the effects of *H. pylori* infection on various parameters of metabolic syndrome, and have found positive association between *H. pylori* with lower HDL-c levels [22–24], and higher systolic blood pressure [22]. These individual findings are supported by a meta-analysis conducted by Upala et al. (2016), which also found that, overall, *H. pylori* positivity is significantly associated with higher systolic blood pressure ($p=0.01$) and lower HDL-c levels ($p<0.01$) [34]. However, more recent cross-sectional studies in the United States of America and Iran have found no significant difference in HDL-c levels between *H. pylori*-positive and negative individuals [35–37].

In this study, our finding of no significant association between *H. pylori* infection and metabolic syndrome status is supported by few studies. Naja et al. (2012) and Takeoka et al. (2016) which also found that odds of metabolic syndrome did not significantly differ between H. pylori- positive and negative individuals, respectively [29, 30]. Similarly, Chen et al. (2019) found that *H. pylori* was not significantly associated with metabolic syndrome in females only [21]. However, this explanation disagrees with previous studies that have found an association between *H. pylori* infection and metabolic syndrome in apparently healthy populations [18–28]. In a meta-analysis conducted by Azami et al. (2021) showed a significant association between *H. pylori* positivity and metabolic

syndrome with a pooled odds ratio of 1.19 (95% CI 1.05–1.35) [8]. The magnitude of the odds ratio from this result didn't not materially different from our observations, although ours failed to reach statistically significant. This inconsistency could be due to variations in age, outcome ascertainment, and differences in the method used to assess H. pylori status.

Several hypotheses have been proposed regarding the mechanism by which *H. pylori* induces the development of metabolic syndrome. As *H. pylori* is known to disrupt gastric barrier function through the dysregulation of epithelial tight junctions [38], this predisposes the gut to mucosal damage [39]. It has been shown that mucosal damage induces the production of pro-inflammatory cytokines [7], which may affect glucose and lipid metabolism [40, 41], as these cytokines promote systemic inflammatory response [38, 42]. Interestingly, Mokhtar et al. found that *H. pylori* eradication in H. pylori-positive patients with functional dyspepsia resulted in a significant reduction of LDL levels, plasma glucose counts, and waist circumference Field [39], suggesting that *H. pylori* affects metabolic parameters and increased metabolic syndrome risk through interaction with the gastric epithelium. These findings are further substantiated by others who found that gastroesophageal reflux disease (GERD) and gastric ulcer (GU) Field [19], as well as a duodenal ulcer (DU) [26], were predictive for metabolic syndrome. Our failure to conclude a significant association between *H. pylori* positivity and metabolic syndrome

may be explained by the lack of data on *H.* pylori-induced gastrointestinal abnormalities such as GU, DU, or GERD in the study population.

Our findings should be interpreted considering the following limitations. First, the cross-sectional design of our study makes it difficult to establish causal findings since we did not have patient data prior to *H. pylori* infection. Longitudinal studies are needed to improve our understanding of the effects of *H. pylori* infection. Similarly, *H. pylori* may be a symptom of other conditions, such as other infections or socioeconomic status. We collected demographic and lifestyle information using self-reported questionnaires, which may be susceptible to misclassification and recall bias. However, the questionnaire had previously been effective in a similar population in Ethiopia, increasing the validity of our findings. Furthermore, enrolling patients with known type I or type II diabetes mellitus introduces the possibility that diabetes status may be associated with *H. pylori* infection or metabolic syndrome. To account for these possibilities, we adjusted our findings for markers of socioeconomic status that had been significantly associated with the outcome variable (metabolic syndrome). A further potential limitation is our method of *H. pylori* detection. As we used the IgG Enzyme-linked Immunosorbent Assays (ELISA) method to diagnose *H. pylori*-positive individuals, Shin et al. (2012) found that serological detection methods were less sensitive than histological methods, and thus less capable of establishing a correlation between *H. pylori* and metabolic syndrome. However, no other studies have examined the association between *H. pylori* infection and metabolic syndrome using ELISA methods to detect *H. pylori* and ELISA methods have been proven to be a useful tool with high diagnostic performance in African settings [43]. Finally, the potential of reverse causality may also explain found associations between H. pylori and metabolic syndrome. However, *H. pylori* infection in developing countries such as Ethiopia frequently occurs early in life [44], which makes it unlikely that patient metabolic parameters abnormality preceded to *H. pylori* infection.

The definition of metabolic syndrome used for this study was another limitation of this study. We opted to use the modified International Diabetes Federation (IDF) criteria used by Rafaeli et al. (2018), which used a BMI cutoff of ≥ 30 kg/m^2 as a proxy for determining central obesity as opposed to waist circumference values [26]. However, Body-mass index has been used as substitute for waist circumference because BMI and waist circumference have been found to be associated with BMI [45]. Although our study has limitations, this is the first study to investigate the potential link between H. pylori infection and metabolic syndrome in diabetic patients in Ethiopia. We gathered detailed lifestyle and demographic information to address potential confounding factors. We used comprehensive clinical laboratory data to define the study's exposure and outcome objectively.

In conclusion, we found that *H. pylori* infection was significantly associated with lower levels of HDL-c and higher systolic blood pressure. However, we didn't find a significant association between *H. pylori* infection status and overall metabolic syndrome. Future studies should seek to examine the relationship between *H. pylori* and metabolic syndrome in light of other gastrointestinal conditions.

Abbreviations

MALT	Mucosa Associated Lymphoid Tissue
HDL-c	High-Density Lipoprotein cholesterol
LDL-c	Low-Density Lipoprotein cholesterol
TG	Triglycerides
GERD	Gastroesophageal reflux disease
BMI	Body mass index
IDF	International Diabetes Federation
GU	Gastric Ulcer
DU	Duodenal Ulcer
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
WC	Waist Circumference
ELISA	Enzyme-linked Immunosorbent Assays

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-024-09840-w.

Supplementary Material 1

Supplementary Material 2

Author contributions
BT conceived and designed the study, and critically reviewed the manuscript. TI participated in the study design, data collection, and analysis, and drafted the initial manuscript. WC supervised the field data collection and critically reviewed the manuscript. WR, AG, and EZ assisted in data analysis and interpretation and critically reviewed the manuscript. All authors have read and approved the final manuscript.

Data availability
The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The Institutional Review Board (IRB) of Jimma University, Ethiopia approved the study. Informed consent was obtained from each patient before recruitment after explaining the study's objectives in the language they understood. The patients were informed that participation in the study would be voluntary and free. They were also informed that refusing or withdrawing from the study wouldn't jeopardize their right to receive any services. To ensure participant privacy, confidential numerical identifiers are assigned to each study subject, and all participant information remains password protected in electronic files. All methods were carried out in accordance with the relevant ethical guidelines and regulations of the Jimma University Institutional Review Board (IRB).

Consent for publication
All study subject enrolled in the study have provided their informed consent for the publication of the research results. In line with the IRB guide ethical guidelines and regulations all personal data or identifiable information were anonymized to protect individual privacy.

Competing interests
The authors declare no competing interests.

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