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Original Research Article

Profile of Lipid Markers during *Helicobacter pylori* Infection in Type 2 Mellitus Diabetics and non-Diabetics Subjects at Brazzaville University Hospital

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Abstract

***Background*:** H. pylori is a micro-aerophilic, a gram-negative, spiral shaped pathogenic bacterium that specifically colonizes on the gastric epithelium and it is one of the most common human bacterial pathogens in wide world. Type 2 Diabetes Mellitus is known as non-insulin-dependent diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia associated with disturbances of the various metabolisms, in particular lipid, carbohydrate and protein. ***Materials and Methods*:** In this study, we aimed to determine the profile of lipid markers in T2DM and non-diabetics patients during Helicobacter pylori infection. We carried out a descriptive cross-sectional study over a period from June to November, 2021. Ninety patients were selected divided into two groups, each subdivided into two subgroups. 44 type two diabetes of mellitus patients and 46 non-diabetic patients were included. ***Result*:** In ninety patients, the average age of diabetic patients was 51 ± 11 years old. The average age of non-diabetic patients was 40±15 years old. Out of 90 patients in our study population, we had a predominance of women compared to men and infected women represented 76.47% and men 23.52% of the overall population. H. pylori infection was more common in diabetics compared to non-diabetics. In this current study we have observed the disturbance of the lipid profile to variable degrees in diabetics and non-diabetic patients whose H.pylori infection was positive with p-values less than 0.005. The univariate correlation between H.pylori infection and lipid markers showed that H. pylori were associated with abnormalities including cholesterol LDL, cholesterol HDL and triglycerides in our study population. ***Conclusion*:** According to Our study H.pylori infection was linked with disturbances of lipid markers and the univariate showed that the H. pylori was susceptible to increase and to fall down lipid profile.

Keywords: Helicobacter pylori, Type 2 diabetes of mellitus and non-diabetics, lipid markers.
T2DM: Type 2 Diabetes Mellitus and NDT: Non-*Diabetics.*

1. INTRODUCTION

Discovered in 1982 by Barry Marshall and Robin Warren *Helicobacter pylori (H. pylori)* ubiquitous bacterium is a gram-negative bacterium and a very common pathogen, which has infected more than half of the of the worldwide population [1]. Its prevalence is higher in developing countries exceeding 95% in some African countries [18]. The prevalence of infection *H. pylori* in Congo-Brazzaville is estimated at 89% among patients received for upper digestive endoscopy at the Gastroenterology and Internal Medicine department in 2015 at CHU-Brazzaville [2]. This infection has formidable consequences; the diagnosis is not always made in time because the infection can remain silent for several months and the search for the infection at *H. pylori* cannot be considered in the absence of symptoms. The infection with *H. pylori* is associated with many gastrointestinal diseases including gastritis, gastric and duodenal ulcers, gastro-esophageal reflux, MALT lymphoma and gastric cancer, it is also involved in other extra gastrointestinal pathologies such as diabetes

mellitus, thyroiditis, ischemic heart disease, high blood pressure, dermatological diseases, rheumatologic diseases and cerebro-vascular diseases [3]. Type 2 diabetes mellitus (T2DM) a metabolic disorder characterized by chronic hyperglycemia, is considered a major public health problem; its pathophysiology is complex and multiple factors are involved in the occurrence of T2DM. Its prevalence is now experiencing exponential growth in the world in general, and particularly in Africa. The interrelation between *H. pylori* infection and T2DM was first explored by Simon and colleagues in 1982. However certain biological mechanisms support this relationship:

First, colonization of the gastric mucosa by *H.pylori* leads to an acute infiltration of polymorphonuclear cells which is gradually replaced by an immunologically mediated infiltration with mononuclear cells, triggering a major local inflammatory response, which can cause simple gastritis. This in the absence of treatment can develop into chronic gastric and gastric ulcer.

Second Infection of *H. pylori* induced gastritis can potentially affect gastric hormone secretion.

Among the gastrointestinal hormones involved in the pathogenesis of diabetes are leptin, ghrelin, somatostatin and gastrin, which are involved in insulin regulation.

Third, colonization of the gastric epithelium by H. pylori causes chronic active inflammation by infiltrating gastric sub mucosal neutrophils and monocytes, which can lead to damage to the gastric mucosa. The pro-inflammatory cytokines released will have different metabolic effects; many tissues are affected by pro-inflammatory cytokines, which would cause recognizable features of T2DM [4-7]. Dyslipidemia is a complicated condition that significantly contributes to unfavorable cardiovascular outcomes and occurrence of T2DM. Gastrointestinal inflammation caused by *H. pylori* can influence the absorption of glucose and lipids, which are abnormal in DM patients [8]. In Congo Brazzaville there is not yet a published study describing the association between infection *H. pylori* and type 2 diabetics. The current study was carried out to determine the profile of lipid markers in T2DM and non-diabetics patients during the infection of *Helicobacter pylori*.

2. MATERIALS AND METHODS

The Gastroenterology and External Medicine Department of Brazzaville University Hospital was chosen for the recruitment and sampling of our patients and the laboratory of the Faculty of Health Sciences for the analysis of the samples. We carried out a descriptive cross-sectional study over a period from June to November 2021, during a period of six months. Our study population consisted of male and female subjects with type 2 diabetes and non-diabetics.

2.1. Sampling method and size

We carried out a systematic non-probabilistic sampling among T2DM and non-diabetic patients seen in the Gastroenterology and Internal Medicine department of the University Hospital Center of Brazzaville consulting for gastro-duodenal symptoms, in whom an infection of H. pylori was suspected.

94 patients were selected. Among these patients, four were excluded due to failure to deposit stools. We had a total of 90 patients divided into two groups, each subdivided into two subgroups according to the results of the search for the antigen of *H. pylori* in stools (Fig 1):

Table 1: Characteristics of patients grouping

Groups	Characteristics
Group 1	44 patients with type 2 DM
Group 2	46 patient non-diabetics

2.2. Inclusion criteria

Included in this study were:

1) The non-diabetic patients of 18 to more years old.
2) Type 2 diabetic patients of 40 to more years old followed by the Endocrinology and Metabolic diseases service of CHU-Brazzaville and living with diabetes for less than ten years old due to that patients living with diabetes for more ten years old had more diabetics Complications than those who had less ten years old according to data from the literature.
3) All consultants in the Gastroenterology and External Medicine consultant service CHU-Brazzaville for gastro-duodenal warning signs making suspect *H. pylori* infection.

2.3. Exclusion criteria

Have not been included in our study patients with:

1) Diabetes mellitus type1;
2) Patients with hemorrhage digestive;
3) Patients on antibiotic treatment following clarithromycin and amoxicillin as well as a proton pump inhibitor for fewer months.
4) Others treatments for eradicating *H. pylori* infection.

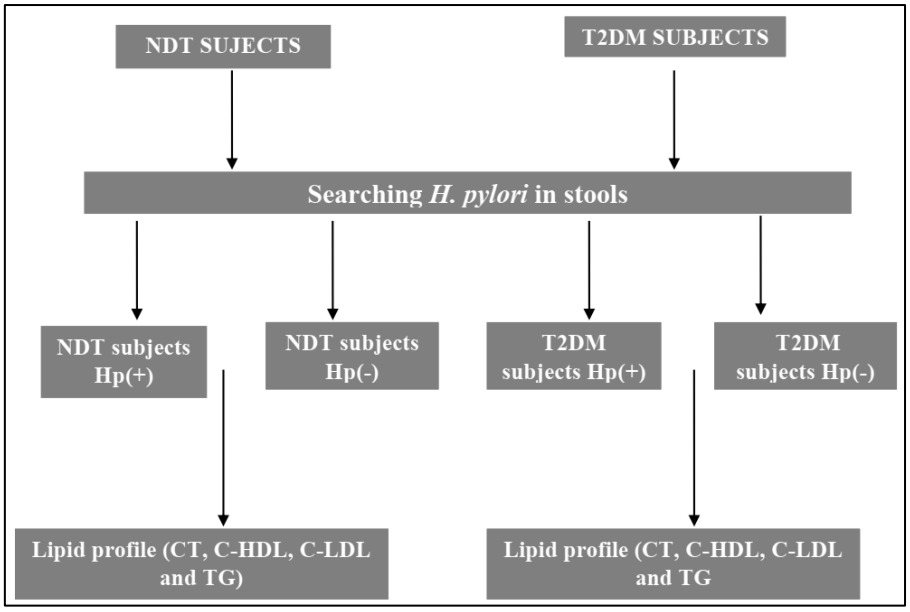

Figure 1: Operational diagram of the study population

2.4. Blood collection procedure

The blood collection was carried out in a dry tube and for each patient these three tubes were collected. It was done at the elbow crease in the morning after a 12-hour fast using a vacuite needle by collecting 5 ml of blood in a dry tube away from any medication and alcohol intake. The blood collected in the dry tube was used for the lipid profile measurement.

2.5. Blood sample processing

After sampling, the samples obtained were transported from the CHU-B sampling location to the laboratory in a cooler containing the cold accumulators. The tubes were then placed on the bench for pretreatment, which consisted of leaving the tubes at room temperature. The tubes were centrifuged for 10 minutes at 3,000 rpm. The serums obtained were used for the analysis of lipid markers.

2.6. Biochemical analyzes

- Fasting blood glucose, total cholesterol, HDL cholesterol and triglycerides were measured using the cobasc311 analyzer.
- Determination of serum LDL cholesterol level

The concentration of low-density lipoprotein (LDL-C) was determined using the formula of Friedewald and colleagues from 1972 provided that the triglycerides were less than 3.5g/l [9].

LDL-c = CT- HDL-c – TG/5.6

2.7. Stool collection procedure

We gave the patient a sterile pot containing a spatula and a paper towel while explaining to him the procedure for collecting stools: Collect fresh stools in the morning, defecate on clean mud avoiding contact between urine and stools, take a dab of saddle using the spatula, put a dab of saddle in the sterile jar, close the jar hermetically and put it in the plastic packaging intended for this purpose.

2.8. Searching of *H. pylori* antigen in stool

The search for the monoclonal antigen of *H. pylori* was performed through rapid stool tests using the JusChek kit. We followed the following steps to search for *H. pylori* in the stool: Unscrew the cap of the sample collection tube then randomly into the fecal sample in three different sites to collect 50mg of feces, Put the applicator in the tube containing the sample collection swab. Extraction, Close the tube and shake vigorously to mix the sample and the extraction buffer well, distribute 3 drops or 90µl of the mixture in the sampling well of the cassette and set the stopwatch to 10 minutes according to the instructions of the manufacturer of the reagent. Read the results. Positivity or negativity of the test to prick the sample collection applicator appearance of two bands (one in the control zone and the other in the test zone). The appearance of a single band at the level of the control zone marked the negativity of the test and the *H. pylori*: the test was considered positive by the appearance of a single band at the level of the test zone translated an invalid test.

2.9. Ethical approval

Type two diabetic and non-diabetic patients consulting in the gastroenterology department and of the Internal Medicine of the hospital center University of Brazzaville and spreading to our criteria written informed consent was obtained for each of them. The Brazzaville Health Sciences Research Ethics Committee (CERSA) approved our study.

2.10. Statistical analysis

Excel 2013 software was used for the design and development of the database and RStudio software for data processing. Quantitative variables were

expressed as means ± standard deviation, and qualitative variables were expressed as numbers and percentages. The comparison of the qualitative variables was made by the Pearson chi2 test and the comparison of the quantitative variables was made by the mann-whitney test.

2.11. Limits of study

The main limitations of our study were the small size of the sample and the period of realization of this work, which was relatively short. The size of sample was small compared to the large cohorts carried out in internationals studies because all the biological examinations carried out for the population of our study were at our expense.

3. RESULTS

Table 2 shows the distribution of the prevalence of the study population according to gender in both groups. The overall distribution of the study population was 90 patients; men were 22 in number with prevalence of 28.2% of diabetic patients and 37.4% of non-diabetics patients, women were 36 with a prevalence of 71.8% of diabetic patients and 62.6% of non-diabetics patients, the sex ratio was 0.32.

Table 2: Distribution of the prevalence of the study population according to gender

Gender	Diabetics Subjects n (%)	Non-diabetics Subjects n (%)
Men	8 (28.2)	18 (37.4)
Women	36 (71.8)	28 (62.6)
Total	**44 (100)**	**46 (100)**

Table 2 shows the distribution of the prevalence of the study population according to age in both groups. The average age of our study population was 45 ± 14 years with extremes ranging from 18 years and 70 years. The average age of diabetics was 51 ± 11 years and non-diabetics were 40 ± 15 years. The most represented age group was that of 30 to 50 years with a prevalence of 52% in diabetic patients and 34% in non-diabetic patients. Statistical analysis of the results showed no statistically significant difference.

Table 3: Distribution of the prevalence of the study population according to Age

Gender	Diabetics Subjects (years)	Non diabetics Subjects (years)	p-value
Men	50.1 ± 10.1	34.2 ± 8.8	0.023
Women	48.6 ± 8.4	42.3 ± 12.6	0.08
Total	**51 ± 11**	**40 ± 15**	**0.07**

Table 3 shows the distribution of Sex, Age and BMI according to *H. pylori* infection. In this study, infected women represented 76.47% and men 23.52% of the overall population. Infected women represented 86.20% and men 13.79% of diabetic patients and concerning non-diabetics patients infected women represented 63.63% and men 36.36%. The most infected age group in the diabetic population was 51 to 61 years old in diabetics and 18 to 28 years old in the non-diabetic population. The average of BMI was 28.03±3.12 in infected diabetics and 27.51±4.25 in non-infected diabetic patients and Statistical analysis did not show significant difference and in non-diabetics infected patients the average was 26.78±5.02 and 24.98±5.18 in non-diabetics patients non-infected. Statistical analysis of the results showed no statistically significant difference.

3.1. Lipid marker profile

3.1.1. Profile of Total cholesterol distribution in both groups

Figure 2 shows Total Cholesterol distribution in both groups of our study population. In non-diabetic patients with *H. pylori* negative a distribution of Total cholesterol was almost normal with an average of 1.58±0.27 (table 4) and in non-diabetics with *H. pylori* positive the distribution was almost asymmetric with an average of 1.66±0.52 (Table 4). This difference was not significant. For the case of diabetics and diabetic patients with *H.pylori* negative had an asymmetric distribution in more than 25% of patients with an average of 1.8±0.32.

In diabetics with *H. pylori* positive distribution was significant in more than 75% of patients with an average 2.12±0.58. Statistical analysis showed a statistically significant difference with p-value < 0.005.

3.1.2 Profile of cholesterol HDL and LDL distribution in both groups

Figure 3 and 4 shows HDL and LDL cholesterol distribution in both groups. In non-diabetic patients with *H. pylori* negative a distribution of HDL cholesterol was almost normal with an average of 3.6±0.9 (table 4) and in non-diabetics with *H. pylori* positive the distribution was symmetric with an average of 0.40±0.35 (Table 4).

This difference was not significant. For the case of diabetics and diabetic patients with *H.pylori* negative had, an asymmetric distribution with an average of 0.54±0.20 and in diabetics with *H. pylori* positive distribution was less significant with an average 0.33±0.12.

3.1.3. Profile of Triglycerides distribution in both groups

In non-diabetic patients with *H. pylori* negative a distribution of HDL cholesterol was almost normal with an average of 0.84±0.58 (Table 4).

In non-diabetics with H. pylori positive, the distribution was asymmetric with an average of 1.72±0.33 (Table 2). This difference was statistically significant.

In diabetic subjects and diabetic patients with *H.pylori* negative had an asymmetric distribution with an average of 1.52±1.97 and in diabetics with *H. pylori* positive distribution was significant with an average 2.69±1.44. Statistical analysis showed a statistically significant difference with p-value < 0.005.

Concerned LDL cholesterol profile, the non-diabetic patients with *H. pylori* negative a distribution of LDL cholesterol was almost normal with an average of 0.96±0.33 (Table 2) and in non-diabetics with *H. pylori* positive the distribution was asymmetric with an average of 1.35±0.40 (Table 2).

This difference was not significant. For the case of diabetics and diabetic patients with *H.pylori* negative had, an asymmetric distribution with an average of 0.59±0.33 and in diabetics with *H. pylori* positive distribution was more significant with an average 1.85±0.59. Statistical analysis showed a statistically significant difference with p-value < 0.005.

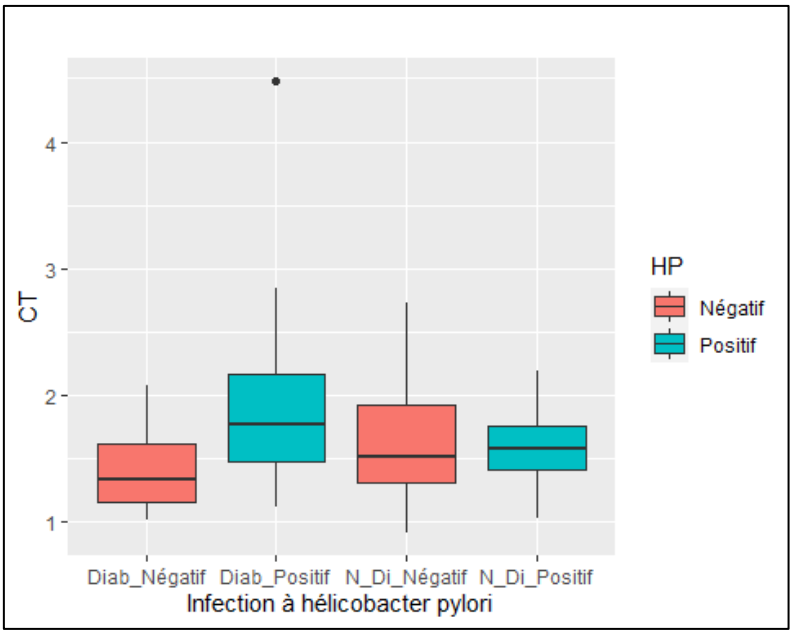

Fig 2: Distribution of Total cholesterol in both groups

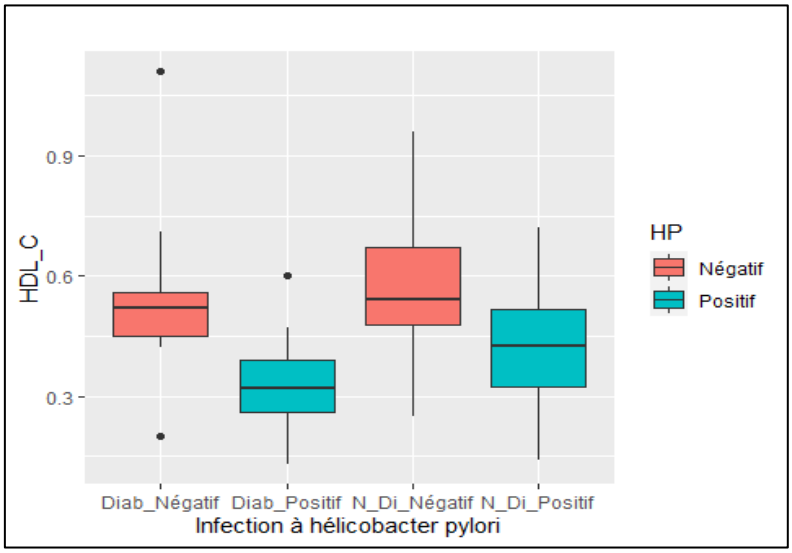

Fig 3: Distribution of HDL cholesterol in both groups

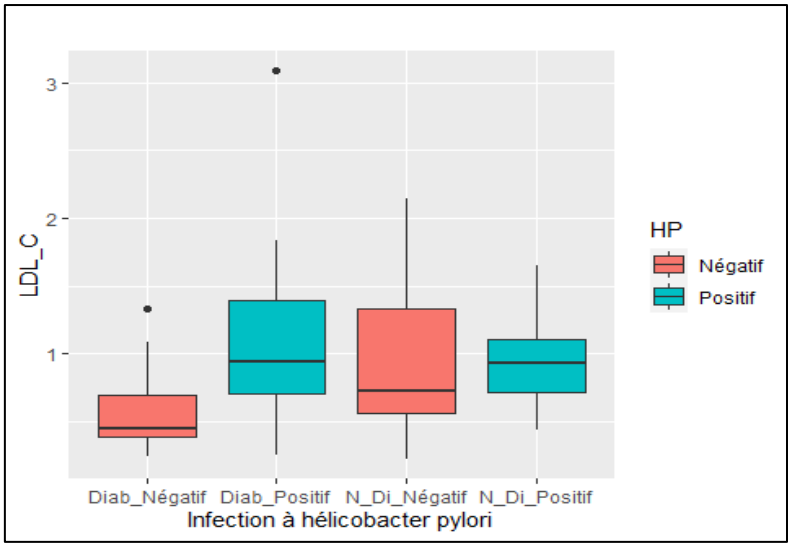

Fig 4: Distribution of LDL cholesterol in both groups

Table 4: Distribution of Sex, Age and BMI according to *H. pylori* infection

	Diabetics (n=44)		***p-value***	***Non Diabetics (n=46)***		***p-value***
	***H. pylori* positive n (%)**	***H.pylori* negative n (%)**		***H. pylori* negative n (%)**	***H. pylori* positive n (%)**	
Sex			**0.08**			0.06
Women	25(86.20)	12(80)		18(75)	14(63.63)	
Men	4(13.79)	3(20)		6(25)	8(36.36)	
Age (years)			**0.312**			0.221
18 – 28	-	-		7(29.17)	8(36.36)	
29 – 39	-	-		6(25.00)	5(22.72)	
40 -50	10(34.48)	6(40)		4(16.17)	3(13.63)	
51 – 61	11(37.93)	7(46.66)		5(20.84)	4(18.18)	
62 – 72	8(27.58)	2(13.34)		2(8.34)	2(9.11)	
BMI (kg/m^2)	28.03±3.12	27.51±4.25	**0.420**	26.78±5.02	24.98±5.18	0.348

Table 6 show univariate logistic regression of lipid markers in diabetics subjects according to *Helicobacter pylori* infection.

Uninfected diabetic patients have not a risk to develop dyslipidemia but diabetic patients with positive of *H. pylori* infection had a low risk of 1.7 of developing hypercholesterolemia with a percentage of 9.65% and 1.08 with a percentage of 7.51 for HDL. However, the risk was 2.02 with a percentage of 15.96 and 3.4 with a percentage 20.32 respectively for LDL and triglycerides in patients whose *H. pylori* infection positive and statistical analysis showed a statistically significant difference with p-value < 0.005.

However, the risk was 2.02 with a percentage of 15.96 and 3.4 with a percentage 20.32 respectively for LDL and triglycerides in patients whose *H. pylori* infection positive and statistical analysis showed a statistically significant difference with p-value < 0.005. Univariate logistic regression in non-diabetic patients, the risk of developing lipid disturbances was relatively low for the entire lipid profile and statistical analysis has no statistically significant difference (Table 7).

Table 5: Distribution of the averages of lipid markers according to the nature of H. pylori infection

Variables	***Diabetics (n=44)***		***p-value***	***Non diabetics (n=46)***		***p*-value**
	***H. pylori* Negative (n=15)**	***H. pylori* Positive (n=29)**		***H. pylori* Negative (n=24)**	***H. pylori* Positive (n=22)**	
Cholesterol total (g/l)	1.8±0.32	2.12±0.58	**<0.001**	1.58±0.27	1.66±0.52	0.9
HDL-Cholesterol (g/l)	0.54±0.20	0.33±0.12	**<0.001**	3.6±0.9	0.40±0.35	0.06
LDL-Cholesterol (g/l)	0.59±0.33	1.85±0.59	**0.002**	0.96±0.33	1.35±0.40	0.03
Triglycerides (g/l)	1.52±1.97	2.69±1.44	**<0.001**	0.84±0.58	1.72±0.33	0.004

Table 6: Univariate logistic regression analysis of dyslipidemia in diabetic subjects according *H.pylori* infection

Variables	***Diabetics Subjects (n=44)***				***p*-value**
	H. pylori negative (n=15)	***H. pylori* positive (n=29)**			
		Percentage (%)	*Odd ratio (OR)*	*Confidence interval(95%CI)*	
Cholesterol total	-	9.65	1.7	3.80-4,45	**0.035**
HDL-cholesterol	-	7.51	1.08	3.87-1.02	**0.018**
LDL-cholesterol	-	15.96	2.02	0.00-0.16	**0.001**
Triglycerides	-	20.32	3.4	0.35-1.54	**0.007**

Table 7: Univariate logistic regression analysis of dyslipidemia in non-diabetic subjects according *H.pylori* infection

Variables	***Non diabetics Subjects (n=46)***				***p*-value**
	H. pylori negative (n=15)	***H. pylori* positive (n=29)**			
		Percentage (%)	*Odd ratio (OR)*	*Confidence interval(95%CI)*	
Cholesterol total	-	2.65	0.14	2.01-0.94	**0.084**
HDL-cholesterol	-	11.51	0.35	1.08-1.34	**0.4**
LDL-cholesterol	-	10.96	1.54	0.12-2.12	**0.05**
Triglycerides	-	8.56	1.48	0.35-1.54	**0.04**

4. DISCUSSION

Since its discovery still now, *H. pylori* continue to be spread around the world and she is become a common bacterial infection, which colonize stomach epithelium and cause non-gastrointestinal disorders. Ninety patients of the population of our study, we obtained a predominance of women 68 compared to men 22. The infected women represented 76.47% and the men 23.52%. Although the prevalence of *H. pylori* infection of women in this study was predominant, statistical analysis did not show any statistically significant difference. This result agrees with that of Mohammed *et al*., on the other hand it differs from that obtained by Jamshid *et al*., who had noted a male predominance [10, 11]. This difference could be explained by the fact that in our study there was a greater participation of women compared to men.

Regarding body mass index, no statistically significant difference was observed between infected diabetic and non-diabetic patients in our study. Our results are consistent with amongst results found in the literature.

Lipid markers

The relationship between lipid markers and infection *H. pylori* has been the subject of our numerous studies. Analysis of our results showed that diabetic and non-diabetic patients infected with *H. pylori* had a disturbed lipid profile compared to those who were not infected. The correlation between lipid markers and infection with *H. pylori* showed that diabetics whose infection *H. pylori* was positive had an increase in low-density lipoprotein (LDL) with odd ratio 2.02 (95%IC0.00-0.16). triglycerides with odd ratio 3.4 (95%IC0.35-1.54) and a decrease in high-density lipoprotein (HDL) and also disturbances in total cholesterol levels. According Mukhtar and *al* study, H. pylori infection is associated with dyslipidemia and higher levels of oxidised LDL in T2DM patients [12]. Our results agree with those of Mysara *et al*., in Egypt and those of Yusuf and *al* in Türkiye who had noticed lipid disturbances in patients infected with *H. pylori* [13, 14].

Several hypotheses have been raised as to a plausible explanation for the disruption of these markers during an infection with *H. pylori* in diabetic and non-diabetic patients.

Chimienti *et al*., as well as Mysara *et al*., had noticed that disturbances in lipid metabolism were due to chronic inflammation induced by *H. pylori* which led to the production of pro-inflammatory cytokines such as interleukins 1 and 6, interferon- and tumor necrosis factor (TNF) which inhibited the activation of adipose tissue lipoprotein lipase and increased the stimulation of hepatic fatty acid synthesis and lipolysis [15, 13]. Pieniazek *et al*., had observed the same disturbances in lipid metabolism; this could be due to the pathogenicity island and the Cag protein that certain strains of *H. pylori* possess [16].

Our study provides evidences that *H. pylori* infection was associated with atherogenic lipid profile among patients with T2DM and non-diabetics infected patients. Furthermore, univariate logistic regression of lipid markers in diabetic's subjects according to *H. pylori* infection showed that patients with *H.pylori* infection had the higher risk of lipids disturbances. Sung *et al*., found that *H. Pylori* infection in healthy Korean adults was associated with atherogenic lipid profile (increase in total cholesterol, triglyceride, LDL cholesterol, and decrease in HDL cholesterol) [17]. According to Kanbay *et al*., *H. pylori* infection affected lipid metabolism in a way that increased the risk of atherosclerosis and has

been regarded as an independent risk factor for coronary artery disease [18]. However, Moghimi *et al.*, found that *H. pylori* eradication has been reported to modify some parameters of lipids and homeostasis [19]. In contrary other stated that there is no lipid profile disturbance in T2DM [3].

Nevertheless uninfected diabetic patients have not a risk to develop dyslipidemia but non diabetic patients with positive of *H. pylori* infection had a low risk of 1.7 of developing hypercholesterolemia with a percentage of 9.65% and 1.08 with a percentage of 7.51 for HDL.

5. CONCLUSION

The present study showed that infection with *H. pylori* infection was present and associated with disturbance lipid profile and increased levels of lipid markers in diabetic and non-diabetic patients. Moreover, eradication of *H. pylori* may play an important role for monitoring dyslipidemia in infected diabetic and non-diabetic is patients. However, the small size of our sample could not allow us to speak of a greater susceptibility to this infection within the diabetic population.

Conflicts of interest: There were no conflicts of interest.

Financial support and sponsorship: We declare that we have not received any financial support or sponsorship.

REFERENCES

1. Parsonnet, J. (1993). Helicobacter pylori and gastric cancer. *Gastroenterology Clinics of North America*, *22*(1), 89-104.
2. Ontsira Ngoyi, E. N., Atipo Ibara, B. I., Moyen, R., Ahoui Apendi, P. C., Ibara, J. R., Obengui, O., ... & Megraud, F. (2015). Molecular detection of Helicobacter pylori and its antimicrobial resistance in Brazzaville, Congo. *Helicobacter*, *20*(4), 316-320.
3. Oluyemi, A., Anomneze, E, Smith, S., & Fasanmade, O. (2012). Prevalence of the activity of a biomarker of infection with *Helicobacter pylori* in patients with type 2 diabetes in Lagos. *BMC Res Notes*, 205, 1183-1185.
4. Popescu, D., Andronescu, D., & Babeș, P. A. (2017). Association between Helicobacter Pylori infection and insulin resistance: A systematic review. *Romanian Journal of Diabetes Nutrition and Metabolic Diseases*, *24*(2), 149-154.
5. Buzás, G. M. (2014). Metabolic consequences of Helicobacter pylori infection and eradication. *World journal of gastroenterology: WJG*, *20*(18), 5226-5234.
6. Buring, J., & Ridker, P. (2001). C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. *JAMA*, 286(3), 327-334.
7. Mégraud, F., & Lehours, P. (2007). *Helicobacter pylori* detection and antimicrobial susceptibility testing. *Clin Microbiol Rev*, 20, 280-322.
8. Wotherspoon, A. C., Ortiz-Hidalgo, C., Falzon, M. R., & Isaacson, P. G. (1991). Helicobacter pylori-associated gastritis and primary B-cell gastric lymphoma. *The Lancet*, *338*(8776), 1175-1176.
9. Mégraud, F., & Lehours, P. (2007). *Helicobacter pylori* detection and antimicrobial susceptibility testing. *Clin Microbiol Rev*, 20, 280-322.
10. Mohammed, N., Haitham, A., Azeem, A., Antably, Mohamed, T., & Sayed, A. (2015). Prevalence of infection with *Helicobacter pylori* in patients with type 2 diabetes mellitus. *Medical journal al-azhar assiut Rev*, *13*(4), 23-33.
11. Jamshid, V., Mahmoud, P., Mohammad Reza, S., & Mohammad, B. (2014). *Helicobacter pylori* infection and insulin resistance in the diabetic and non-diabetic population. *Log global scientist Hindawi Rev*, *10*(11), 391-250.
12. Mukhtar, M. H., Nasif, W. A., & Babakar, A. T. (2026) *Helicobacter pylori* infection is associated with dyslipidemia and increased levels of oxidized LDL in type 2 diabetes mellitus. *Journal of diabetes mellitus*, 6, 185-190.
13. Mysara, M., Mogahed, M., Amira, H., & Allam, M. (2017). *Helicobacter pylori* infection in type 2 diabetic patients and its relation to smoking. *Life Science Journal*, *14*(1), 53-59.
14. Yusuf, K., & Eroğlu, H. (2015). Relationship Helicobacter pylori infections in diabetics patients and inflammations, metatbolic syndrome and complitions. *Int J Chronic Dis*, 29, 128.
15. Chimienti, G., Russo, F., Lamanuzzi, B. L., Nardulli, M., Messa, C., Di Leo, A., ... & Pepe, G. (2003). Helicobacter pylori is associated with modified lipid profile: impact on Lipoprotein (a). *Clinical biochemistry*, *36*(5), 359-365.
16. Pareek, R., & Kannan, M. (2014). Prevalence of infection *H. Pylori* in type 2 diabetic patients in rural Rajasthan - a case control study. *Inter J Med Sci Clin Invent*, *1*(1), 1-14.
17. Sung, K. C., Rhee, E. J., Ryu, S. H., & Beck, S. H. (2005). Prevalence of Helicobacter pylori infection and its association with cardiovascular risk factors in Korean adults. *International journal of cardiology*, *102*(3), 411-417.
18. Kanbay, M., Gur, G., Akcay, S., & Yilmaz, U. (2005). Helicobacter pylori seroprevalence in patients with chronic bronchitis. *Respiratory medicine*, *99*(10), 1213-1216.
19. Moghimi, J., Toussy, J., Babaei, M., Mousavi, S., & Tamado, M. (2007). Effets of *Helicobacter pylori* eradication on short control of glicemia in patients with type 2 diabeyes mellitus. *Saudi Med J*, 28, 475-476.