Prevalence of *Helicobacter pylori* infection among Diabetes mellitus type two patients in Misurata-Libya

Salem Ramadan Sariti and Shireen Mohamed Eldanfaria
Life science department-Libyan Academy, Misurata-Libya

Abstract

Helicobacter pylori (H.pylori) is a spiral-shaped gram-negative rod. It is associated with antral gastritis, duodenal (peptic) ulcer disease, and gastric carcinoma. Other *Helicobacter* species that infect the gastric mucosa exist but rare.

H.pylori is present on gastric mucosa of less than 20% of persons under age 30 but increases in prevalence to 40-60% in persons of age 60, including individuals who are asymptomatic, In developing countries, the prevalence of infection may be 80% or higher in adult.

Methods: This study had been conducted in diabetic and endocrine centre in Misurata- Libya. 212 subjects were involved, 195 subjects are type 2 diabetic patients and 17 non-diabetic subjects (control group). From each subject, a blood sample and a stool sample had been collected for *H. pylori* test by using the Rapid Serological Test (IgG detection) and the Antigen Stool Test (*H.pylori* Antigen detection).

Results: This study involved 195 T2DM patients and 17 control subjects. It showed that participants with T2DM have a higher prevalence of *H.pylori* infection 71.28% when compared to the control participants 58.82% with a significant statistical difference (p-value= 0.029).

Conclusion: This study demonstrated high *H.pylori* prevalence among the T2DM patients, which gave a general impression leads to belief that the *H.pylori* infection could be an important risk factor for T2DM disorder.

Key words: *Helicobacter pylori* infection, peptic ulcerd, *H.pylori* prevalence, diabetic and endocrine centre in Misurata-Libya

Introduction:

Helicobacter pylori (H.pylori) is a spiral-shaped gram-negative rod. It is associated with antral gastritis, duodenal (peptic) ulcer disease, and gastric carcinoma. Other *Helicobacter* species that infect the gastric mucosa exist but rare.

H.pylori is present on gastric mucosa of less than 20% of persons under age 30 but increases in prevalence to 40-60% in persons of age 60, including individuals who are asymptomatic, In developing countries, the prevalence of infection may be 80% or higher in adult. Person-to-person transmission of *H.pylori* is likely because intrafamilial clustering of infection occur. Acute

epidemics of gastritis suggest a common source for *H.pylori* (Brooks et al, 2004).

A growing body of evidence has linked *H.pylori* infection and insulin resistance IR in Diabetes mellitus (DM) patients (Eshraghian, 2009), which is defined by a state where insulin can no longer effectively induce glucose disposal in skeletal muscle or suppress endogenous glucose in the liver (Dinneen et al, 1992). Insulin resistance and abnormal insulin secretion are central to developing type two of diabetes mellitus (T2DM) disorder, and most studies support the view that IR precedes defects in insulin secretion (Moller, 1991). The first direct evidence for an association between chronic *H.pylori* infection and IR came from a study had been done by Aydemir et al, 2005 where the study showed high homeostatic model assessment insulin (HOMA-IR) scores in *H. pylori*–positive individuals. Furthermore, a Japanese study in 2009 that included a large population of 1107 asymptomatic subject also showed that *H.pylori* significantly and independently to IR (Gunji et al, 2009).

A study from Saudi Arabia showed that the association of Metabolic sclerosis (MS) and *H.pylori* is still controversial with emphasis on the possible linkage between them. However, the high prevalence of both MS and *H.pylori* infection might explain the coincidence (AL baker, 2011). Moreover, in the Medical Services Clinics in Gaza Strip, a study was done

randomly by selecting 129 T2DM patients, It showed that 70% of the selected samples were positive to the *H.pylori* test and that gave a strong relationship between *H.pylori* infection and insulin resistance risk factors (El-Sakka et al, 2013).

This work was aimed to determine the *H. pylori* prevalence among T2DM patients in Misurata-Libya.

Materials and Methods:

This study had been conducted in diabetic and endocrine centre in Misurata- Libya. 212 subjects were involved, 195 subjects are type 2 diabetic patients and 17 non-diabetic subjects (control group). From each subject, a blood sample and a stool sample had been collected for *H. pylori* test by using the Rapid Serological Test (IgG detection) and the Antigen Stool Test (*H.pylori* Antigen detection).

Statistical analysis: All the obtained date have been subjected and statistically analysed by using Minitab 16 program. The results for positive samples were expressed as percentages, and statistical analysis was carried out by using t test, two proportion test and the correlation test to determine the degree of correlation which measure the degree of linear correlation between two variables and the direction of this link. A probability p-value of ≤ 0.05 was considered as significant whenever appropriate.

Results:

The prevalence of *H.pylori* infection among T2DM patients and control subjects in this study outcome showed a positive correlation between T2DM and *H.pylori* infection. This study involved 195 T2DM patients and 17 control subjects. It showed that participants with T2DM have a higher prevalence of *H.pylori* infection 71.28% (139 sample out of 195 sample) with a confidence interval of (95% Cl 64.38% to 77.52%) when compared to it's prevalence in the control participants 58.82% (10 out of 17) with a confidence interval of (95% Cl 32.92 to 81.56), with a significant statistical difference (p-value= 0.029). As showed in tables 1, 2 and 3.

Table No. 1: A contingency table relating the true positive, false positive, true negative and false negative results of the IgG method and Ag method for T2DM group:

	IgG method	Ag method	P-value

Positive samples	118 (60.51%)	96(49.23%)	0.032
Negative samples	77 (39.48%)	99(50.77%)	0.032
Total no. of samples	195 (100%)	195 (100%)	--
No. true positive	75	75	--
No. false positive	21	43	0.004
No. true negative	43	21	0.004
No. false negative	56	56	--
Sensitivity	78.12%	63.56%	0.029
Specificity	56.57%	72.73%	0.027
Positive predictive value	63.56%	78.12%	0.029
Negative predictive value	72.73%	56.57%	0.027
Accuracy	67.17%	67.17%	NS**

* **Total positive**: **139** (**71.28%**).

Non-significant.

Table No. 2: A contingency table relating the true positive, false positive, true negative and false negative results of the IgG method and Ag method for the control group:

	IgG method	Ag method	P-value
Positive samples	7 (41.18%)*	8 (47.06%)*	0.046

Negative samples	10 (58.82%)	9 (52.94%)	0.015
Total no. of samples	17 (100%)	17 (100%)	--
No. true positive	5	5	--
No. false positive	2	3	NS**
No. true negative	7	7	--
No. false negative	3	2	NS**
Sensitivity	62.50%	71.43%	0.005
Specificity	77.78%	70%	0.004
Positive predictive value	71.43%	62.50%	0.005
Negative predictive value	70%	77.78%	0.004
Accuracy	70.59%	70.59%	NS**

* **Total positive in the control group**: **10** (**58.82%**).

NS=Non-significant.

Table No. 3: The *H. pylori* prevalence among T2DM patients and Control group:

	T2DM	Control group	p-value
No. of total Samples	195	17	
No. of positive Samples	139	10	0.029
H.pylori	71.28%	58.82%	

prevalence			

Discussion:

In this study, according to the obtained results, the prevalence of *H.pylori* in DMT2 patients was 71.28% (95% Cl 64.38% to 77.52%) which is higher than in the control group 58.82% (95% cl 32.92% to 81. 56%) with a significant statistical differences (p-value=0.029).

In fact, there are several lines of evidences to implicate increase susceptibility to the *H.pylori* infection in diabetic patients:

1- Diabetes mellitus induced impairment of cellular and humoral immunity may enhance individual's sensitivity to *H.pylori* infection.
2- Diabetes induced reduction gastrointestinal motility and acidic secretion may promote pathogen colonization and infection rate in guts.
3- Altered glucose metabolism may produce chemical changes in the gastric mucosa that promote *H.pylori* colonization.
4- Individuals with diabetes are more frequently exposed to pathogens than their healthy counterparts as they regularly attend hospital settings (He et al, 2014).

The above lines could explain the high prevalence rate of *H.pylori* infection among the T2DM group comparing to the control group. Similar results were also detected in a study conducted at Gaza strip, where more than 70.5% of the tested

samples were found to have positive *H.p*ylori test in diabetic patients (Mazen et al, 2013). In another study in Benghazi city-Libya, where 200 T2DM patients had been screened, the prevalence rate was 50.5% (Lemziany, 2012). However, other study did not find any significant difference between the DM group and control group with regard to *H.pylori* infection (Demir et al, 2008).

A study by So et al found that *H. pylori* titer could independently predicts abnormal pancreatic β-cell function in Chinese men (So et al, 2009). Additionally, Rahman et al also described a positive association between *H. pylori* infection and impaired insulin secretion (Rahman et al, 2009). Moreover, the insulin-producing pancreatic β-cells are especially susceptible to damage by inflammation and oxidative stress (Fosslien, 2001). Therefore, it is possible that inflammation caused by *H. pylori* infection results in deficits in insulin secretion. Furthermore, it was reported in a study by Hsieh et al that patients with *H. pylori* infection were more likely to have had impaired insulin secretion at a young age, which may increase the risk for T2DM (Hsieh et al, 2013). Many additional factors are likely involved in the relationship between *H.pylori* infection and T2DM. The T2DM affected both the cellular and humoral component of immune system, because of the risk of T2DM patients with the complication of the *H.pylori* infection is more comment. However, it could be that *H.pylori* infection plays a role in type

2 diabetes mellitus chronic inflammation, the secretion of gastric related hormones, and insulin secretion deficiency implicate *H.pylori* in a predisposition to diabetes.

Conclusion:

This study demonstrated high *H.pylori* prevalence among the T2DM patients, which gave a general impression leads to belief that the *H.pylori* infection could be an important risk factor for T2DM disorder.

References:

- Albaker, Waleed. I. (2011). *Helicobacter pylori* Infection and Its Relationship to Metabolic Syndrom: Is It a Myth or Fact? Saudi Journal of Gastroenterology, 17(3): 165-169.
- Aydemir, S., Bayraktaroglu, T., Sert, M., Sokmen, C., Atmace, H., Mungan, G., Gun, B. D., Borazan, A., Ustundag, Y. (2005). The effect of Helicobacter pylori on insulin resistance. Dig Dis Sci 50:2090-2093.
- Brooks, Geo. F., Carroll, Karen. C., Butel, Janet. S., Morse, Stephen. A. (2004). Medical Microbiology. The McGraw-Hill Companies,United State of America:The McGraw-Hill Companies Vol. 24, pp. 275-277.
- Demir, M,.Gokturk, H. S.,Ozturk, N. A.,kulaksizoglu, M., Serin, E,.Yilman, U. (2008). *Helicobacter pylori* prevalence in diabetes mellitus patients with dyspeptic symptoms and its

relationship to glycemic control and late complications. Digestive Diseases and Sciences, 53 (10), pp. 2646-2649.

- Dinneen S, Gerich J and Rizza R.(1992). Carbohydrate Metabolism in Non-Insulin-Dependent Diabetes Mellitus. N Engl J Med 327: 707-713
- El-Sakka M, Abu Jabal E, Nasser L. The role of Helicobacter pylori infection, and malnutrition among type 2 diabetic medical services patients in the Gaza Strip. Int J Clin Med 2013;4:556–560.
- Eshraghian, A., Hashemi, S. A., Hamidian, Jahromi. A., Eshraghian, H., Masoompour, S. M., Davarpanah, M. A., Eshraghian, K., Taghavi, S. A. (2009).
- Fosslien, E. (2001). Mitochondrial medicine--molecular pathology of
- Defective oxidative phosphorylation. Ann Clin Lab Sci. 31: 25-67.
- Gunji, T., Matsuhashi, N., Sata, H., Fujibayashi, K., Okumura, M., Sasabe, N., Urabe, A. (2009). Helicobacter pylori infection significantly increases insulin resistance in the asymptomatic Japanese population. *Helicobacter* 14: 144-150.
- He, Cong,. Yang, Zhen,. Lu, Hua. (2014). *Helicobacter pylori* infection and diabetes: Is it a myth or a fact?. Word Journal of Gastroenterology, 20(16): 4607-4617.

- Hsieh, M. C., Wang, S. S., Hsieh, Y. T., Kuo, F. C., Soon, M. S., Wu, D. C. (2013). *Helicobacter pylori* infection associated with high HbA1cand type 2 diabetes. Eur J Clin Invest.43: 949-956.
- Lemziany, Osama. Abubaker. (2012). Prevalence of *Helicobacter pylori* infection among Type 2 Diabetic patients in Benghazi city. pp. 46-47.
- Mazen, A., El-Sakka, Ektemal. M., AbuJabl, Luay. M. Nasser. (2013). The Role of *Helicobacter pylori* Infection, and Malnutrition among Type 2 Diabetic Medical Services Patients in the Gaza Strip. International Journal of Clinical Medicine, 4:556-560.
- Moller, D., E. Flier, J., S. (1991). Insulin resistance-mechanisms, syndromes, and implications. N Engl J Med 325: 938-948.
- Rahman, M. A., Cope, M. B., Sarker, S. A., Garvey, W. T., Chaudhur, H. S., Khaled, M. A. (2009). *Helicobacter pylori* Infection and Inflammation: Implication for the Pathophysiology of Diabetes and Coronary Heart Disease in Asian Indians. J. Life Sci. 1: 45-50.
- So, W.Y., Tong, P. C., Ko, G. T., Ma, R. C., Ozaki, R., Kong, A. P., Yang, X., Ho, C. S., Lam, C. C., Chan, J. C. (2009). Low plasma adiponectin level, white blood cell count and *Helicobacter pylori* titer independentlypredict abnormal

pancreatic beta-cell function.*Diabetes Res ClinPract*2009.**86**: 89-95.