# Detection of Anti-*Helicopacter pylori* IgG Antibody, Anti-GAD antibody, Anti-Beta islets cell in a sample of serum with type-2 Diabetes mellitus patients

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#### Abstract:

We have investigated twenty five patients with type-2 diabetes mellitus aged (35-60) years and fifteen healthy persons as control group to detect Anti-*Helicobacter pylori* IgG antibody. All studied groups were carried out to measure fasting blood sugar, anti- Glutamic acid decarboxylase (GAD), anti- $\beta$  islets cells antibody by IFAT, Anti-*H. pylori* IgG antibody by ELISA technique. There was significant elevation in the concentration of fasting blood sugar than in control group (P < 0.05), the patients had negative results for anti-GAD antibody and anti- $\beta$  islets cells antibody, there were significant differences (P < 0.05) of anti-*H. pylori* IgG antibody in 28 % of patients had type-2 diabetes than control group. This lead to suggestion that type -2 diabetes mellitus patients are more susceptible to *H. pylori*.

#### Key words: type-2 diabetes mellitus, Helicobacter pylori, metabolic disorder.

#### **Introduction:**

Type-2 diabetes mellitus patients are more prone to infection because of the impairment of their immune status. *H. pylori* is one of the common chronic infection worldwide [1]. The elements of the innate immune system, such as C-reactive protein may predict risk of developing type-2 diabetes mellitus [2].

In diabetic patients the frequency of non-ulcer, non-gastric dyspepsia is two time higher than in non-diabetics persons and strictly association with autonomic neuropathy, acting as a favoring factor occurrence and recurrence of gastrointestinal disease [3].

Many patients with diabetes have upper and lower gastrointestinal (GI) symptoms [4]. Infection with *H. pylori* had been linked with chronic atrophic gastritis [5]. The rate of *H. pylori* infection was similar between the type-2 diabetic group with or without

gastrointestinal symptoms [6]. H. pylori is common infection in diabetics who do not have metabolically controlled hyperglycemic and these are individuals who are colonized by H. pylori infection in the gastric antrum [7]. There was a significant association between *H. pylori* infection and type-2 diabetes mellitus [8]. Both the natural history of gastrointestinal symptoms and factor influencing symptoms in diabetes mellitus were unknown. Hence the aim of this study is to investigate the association of H. pylori infection in type-2 diabetes mellitus.

### Materials and methods:

The study included twenty five patients with type-2 diabetes mellitus aged (35-60) years without gastric intestinal infections and fifteen healthy blood donor taken as control group. The work were done in Private

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studied group measured All fasting blood sugar by Human company, Germany. Anti-H. pylori IgG were measured in present study by ELISA test from Euroimmune. Germany, anti-Glutamic acid decarboxylease (GAD) and anti-B islets by IFAT from Euroimmune, Germany.

#### **Results and discussion:**

The results of present study showed that there was a significant elevation in the concentration of fasting blood sugar ( $250\pm5.3$ ) mg/dl than in control group ( $92.8\pm1.5$ ) mg/dl as shown in figure (1).



Fig. (1): Mean level of fasting blood sugar concentration mg/dl in serum of women had diabetic mellitus and control group.

Where the results of anti-GAD antibody and anti- $\beta$  islets cell antibody by IFAT test of diabetes mellitus type-2 patients serum were negative as in figure (2 and 3).



Fig. (2): Negative result of anti-GAD antibody in women sera had diabetic mellitus type-2



Fig. (3): Negative result of anti- $\beta$  islets cell antibody in women sera had diabetic mellitus typ-2

The demographic study showed that 7(28%) patients with diabetic mellitus type-2 had sero positive results of *H. pylori* compared to control group 0%, as in figure (4).



Fig. (4): Sero positive of *H. pylori* in sera of studied groups

The result of the present study showed no significant differences (p> 0.05) between sero positive samples of *H. pylori* and patient's age, while there were significant differences (P<0.05) in compare with sex, the male had showed high percentage.

In a study they found the association between diabetic mellitus type-2 patients and peptic ulcer caused by H. pylori. Diabetic mellitus gastrointestinal related motor dysfunction is common and affect the esophagus, stomach and lower GI tract, several pathogenic there are mechanisms may involve in these GI symptoms, such as autonomic neuropathy, diabetic peripheral neuropathy [9].

Infection with *H. pylori* has been linked with chronic atrophic gastritis in Diabetic mellitus [5]. *H. pylori*-related disease are associated with male gender in adult, this predominance of *H. pylori* is not apparent in children [10]. Other study showed that the rate of *H. pylori* infection in Hong Kong Chinese subjects with diabetic mellitus type-2 around 50%, similar to control subjects [6], while the other showed higher frequency of *H. pylori* infection in dyspeptic patients affected with diabetic mellitus type-2 than in nondiabetes subject [3].

There was higher prevalence of *H. pylori* infection in obese diabetic patients 23.6% than the non-diabetic subject 11.8%, there were different symptoms such as gastrointestinal, chronic bronchitis, nausa, anemia, abdominal pain, diarrhea and vomiting were common in diabetic patients with *H. pylori* [1].

Other reported that the prevalence of *H. pylori* infection in diabetic mellitus type-2 range from 30-78% [11]. The variability in the prevalence rates may be related to the epidemiology distribution of *H. pylori*, it had been speculated that the

alteration in glucose metabolism may promote H. pylori colonization [12]. The present study showed that there was association between H. pylori infection in patients had diabetic mellitus type-2, these finding are generally explained by the impairment of cellular and humeral immunity in diabetic, by reduction of both gastrointestinal motility and acid secretion and by the effect of a higher secretion of proinfammatory cytokines attributable to *H*. pylori gastric infection [6, 7, 13].

In a study they found that patients with type – 2 diabetic mellitus had *H. pylori* 61% of the patients [14]. Other showed that diabetic mellitus are more prone and ar risk to a quire *H. pylori* infection, therefore proper monitoring of blood glucose level and screening for *H. pylori* infection are effective preventive measures for this life threatening infection [15]. There was association between *H. pylori* and insulin resistance [16].

*H. pylori* seropositivity and dyspeptic symptoms were similar in type -2 diabetic mellitus, although *H. pylori* infection when present in diabetics appears to influence glycemic status, the mechanism of which remains largely unknown [17].

The presence of *H. pylori* infection increased the risk of gastrointestinal cancer in diabetics and this is because hyperglycemia may be an effect modifying factor that promotes the carcinogenic effects of *H. pylori* in gastric mucosa [18].

## References

- Bener, A.; Uduman, S.A. and Ameen, |A. 2002. Prevalence of *Helicobcter pylori* infection. J. Communicable Dis., 34:179-184.
- 2. Crook, M.2004. Type-2 diabetes mellitus: a disease of the innate immune system. Diabetic Medicine, 21:203-207.

- 3. Genritle, S.; Turco, S.; Oliviero, B. and Torella, R.1998. The role of autonomic neuropathy as a risk factor of *Helicobacter pylori* infection in dyspeptic patients with type-2 diabetes mellitus. Diabetes Research and clinical practice,42(1):41-48.
- 4. Rothstein, R.D. 1990. Gastrointestinal motility disorders in diabetes mellitus. Am. J. Gastronterol, 85:782-783.
- Parsonnet, R.D.; Friedman, G.; Vandersteen, D.1991. *Helicobacter pylori* infection and risk of gastric carcinoma. New. Eng. J. Med., 325:1127-1131.
- Gary, T.C.; Francis, K.; Wing-Bun, C. and *etal.* 2001. *Helicobacter pylori* infection in Chinese subject with type-2 diabetes. Endocrain research, 27(1):171-177.
- Bytzer, P.; Talley, N.J.; Leemon, M. 2002. Gastrointestinal symptoms in diabetes mellitus are associated with poor glycemic control and diabetic complication. Am. J. Gastronterol., 97:604-611.
- Bener, A.; Micallef, R.; Affifi, M. 2007. Association between type-2 diabetes mellitus and *Helicobacter pylori* infection. Turkish J. Gastronetol., 18(4):225-229.
- Kim, J.; Park, H.; Young, S. 2010. Diabetic factor associated with gastrointestinal symptoms in patients with type-2 diabetes. World J. Gastroentrol., 16(14):1782-1787.
- 10. Martel, C. and Parsonnet, J. 2006. *Helicobacter pylori* infection and gender; a metaanalysis of population-based prevalence survey. Digestive Dis. Sci., 51(12):910-915.
- 11. Anastosis, R. and Goutases, C. 2002. *Helicobacter pylori*

infection in diabetic patients. Prevalence and endoscopic finding. Eur. J. Inter. Med., 13:376-379.

- Perdichizzi, G.; Bottari, M. and Pallio, S. 1996. Gastric infection by *H. pylori* and antral gastritis in hyperglycemic obese and diabetic subject. New Microbiol., 19:149-154.
- Murray, L.J.; Bamford, K.B.; Oreilly, D.P. 1995. *Helicobacter pylori* infection relation with cardiovascular risk factor and social factor. Br. Heart. J., 74:497-501.
- 14. Demir, M.; Gokturk, H. and Ozturk, N. A. 2008. *Helicobacter pylori* prevalence in diabetic mellitus patients with dyspeptic symptoms and its relationship to glycemic control and late complication. Dig. Dis. Sci, 53:2646 – 2649.
- 15. Devrajani, B.; Shah, S. and Soomro, A. 2010. Type - 2 diabetic mellitus: A risk factor for *Helicobacter pylori* infection: A hospital based case - control study. Int. J. Diabetes, 30(1): 22 -28.
- 16. Gen, R.; Demir, M. and Ataseven, H. 2010. Effect of *Helicobacter pylori* eradication on insulin resistance, serum lipid and low – grade inflammation. South Med. J., 103: 190 – 196.
- 17. Nicholas, C.; Emmanuei, C. and Brown, N. 2008. *Helicobacter pylori* seropositivity in Nigerians with type – 2 diabetic mellitus. Inter. J. Trop. Med., 4(2): 217 – 222.
- Marimthu, S.; Vijaragavan, P. and Moysich, K. 2011. Diabetic mellitus and gastric carcinoma. J. Carcinog., 10: 30 – 35.

# التحري عن أضداد بكتيريا Helicobacter pylori للنوع IgG ، وأضداد حامض الكلوتاميك ، وأضداد خلايا جزر بيتا في عينة من مصول مرضى مصابين بداء السكري النمط الثاني

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#### الخلاصة:

تم أخذ خمسة وعشرون عينة مصل دم لأشخاص مصابين بداء السكري النمط الثاني تتراوح أعمار هم ما بين H. عنه عشر عينة مصل دم لأشخاص أصحاء كمجموعة سيطرة لغرض التحري عن أضداد pylori في مصول مرضى السكري النمط الثاني. خصعت كل مجاميع الدراسة لأختبار تركيز سكر الدم، أضداد الكلوتاميك، أضداد خلايا جزر بيتا بأستخدام تقنيات التألق المناعي ، وأضداد IgG لبكتيريا IgG بطريقة الأمتزاز المناعي المرتبط بالأنزيم، كانت نتائج أضداد حامض الكلوتاميك وأضداد خلايا جزر بيتا سالبة لمجموعة مرضى داء السكري النط الثاني ، وكما لوحظ أرتفاع معنوي لمستوى أضداد خلايا جزر بيتا سالبة المجموعة مرضى داء السكري النط الثاني ، وكما لوحظ أرتفاع معنوي لمستوى أضداد وقداد وقد pylori وبنسبة 28 % في مصول مجموعة مرضى السكري النمط الثاني مقارنة بمجموعة السيطرة، وقد تشير الدراسة بأن مرضى داء السكر النط الثاني أكثر عرضة للإصابة ببكتيريا H. pylori .