# Helicobacter pylori Seropositivity in Nigerians with Type 2 Diabetes Mellitus

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## Citation

## Abstract

Background: Data on the relationship between H. pylori infection and diabetes mellitus are scarce and discordant.
Objective: A case-control study has been conducted to investigate the relationship between infection with Helicobacter pylori and type 2 diabetes mellitus in Abakaliki, south eastern Nigeria; a region with high H. pylori endemicity.
Materials & Methods: The study population comprised 60 type 2 diabetics (32 males and 28 females) and 60 non-diabetics (27 males and 33 females) aged 29 to 72 years. Hematological and biochemical parameters were determined using standard techniques while Helicobacter pylori detection in serum was done by an enzyme-linked immunosorbent assay for the identification of immunoglobulin G.
Results: There was no statistically significant difference (p > 0.05) in the prevalence of H. pylori infection and dyspeptic symptoms between diabetics and non-diabetics. Although H. pylori-infected diabetics were significantly older (63.71 vs. 51.95 years; p = 0.00) and had significantly lower fasting plasma glucose (7.96 vs. 11.58 mmol/l; p = 0.01) than the non-infected diabetics, the two groups had comparable hematological and biochemical parameters. Conclusion: H. pylori seropositivity and dyspeptic symptoms were similar in type 2 diabetics and non-diabetics, although H. pylori infection when present in diabetics appears to influence glycaemic status, the mechanism of which remains largely unknown.

## INTRODUCTION

Infection with Helicobacter pylori has been recognized as a public health problem worldwide [1] affecting approximately 50% of the world population and more prevalent in developing than the developed countries [2]. Helicobacter pylori infection has been associated with both gastrointestinal and non-gastroenterological conditions such as peptic ulcer (gastric and duodenal), gastric cancer and cardiovascular disease [3,4,5]. As one of the most common chronic bacteria infections in the world [6], coupled with the susceptibility of diabetic patients to a wide range of infections as a result of chronic elevation of blood glucose level and impairment of immune functions [7,8], researchers have hypothesized an association between infection with Helicobacter pylori and diabetes mellitus [1,9]. However, studies to date have failed to confirm this hypothesis as results have been discordant [10,11,12]. In Nigeria, there is paucity of information on the association between H. pylori infection and diabetes mellitus. The aim of this study is to investigate the relationship between infection with Helicobacter pylori and type 2 diabetes mellitus in Abakaliki, South Eastern Nigeria; a region with high H. pylori endemicity [13].

## MATERIALS AND METHODS

This study was conducted in Abakaliki at the Ebonyi State University Teaching Hospital (EBSUTH) from May to November 2007. The study area has been previously defined [13]. The Research and Ethics Committee of Ebonyi State University Teaching Hospital, Abakaliki approved the proposal for this study. Participants were known type 2 diabetes mellitus patients on regular management at the Medical Out-patient Clinic of the Department of Internal Medicine, Ebonyi State University Teaching Hospital (EBSUTH), Abakaliki. Before enrolment, patients were adequately educated on the need for this study after which willing participant signed written informed consents. At entry into the study each participant was administered with a structured questionnaire to obtain sociodemographic data such as age, sex, level of education, occupation, smoking habits, alcohol intake, duration of diabetes, drug treatment, and symptoms of dyspepsia. Dyspeptic symptoms were regarded as present if the patient complained of any of the following: epigastric pain, bloating, nausea, and vomiting, early satiety, weight loss, GI bleeding or combinations of

these symptoms and absent if none was present. Height and weight were measured with the subject in light clothes without shoes, and BMI (Kg/m2) was calculated. In all, a total of sixty (60) patients (32 males and 28 females) aged 29 to 72 years were enrolled. Sixty (60) non-diabetic patients; with fasting plasma glucose ? 6.1mmol/l (27 males and 33 females) matched for age and sex and without family history of diabetes mellitus, not pregnant, or on oral contraceptive pills who were attending the Medical Out-patient Clinic for treatment other than D/M were recruited as controls. Seven milliliters (7ml) venous blood were obtained between 08:00 and 10.00 a.m. after a 12 hour fasting period of which 3ml were dispensed into EDTA bottles for hematological and lipid profile determinations, 2ml into fluoride oxalate bottles for glucose estimation while the remaining 2ml of blood was dispensed into dry glass test tubes for clotting and retraction to take place after which serum were used for total cholesterol determination. Both plasma and serum were obtained after samples were centrifuged at 2000g for five minutes.

## LABORATORY ASSAYS

Packed cell volume (PCV) and hemoglobin concentration were determined as described in a standard hematology textbook [14]. Fasting plasma glucose was determined by glucose oxidase method as described by Barham and Trinder [15] using reagent kit from Biosystem Laboratories, Spain. Serum total cholesterol and triglyceride concentrations were determined by enzymatic colorimetric assay as described previously [16] and modified by Richmond [17] and HDL-cholesterol and LDL-cholesterol were determined enzymatically in EDTA anticoagulated plasma after precipitation of other lipoprotein as described by Burstein et. al.[18] and Assmann et. al. [19] respectively, using kits from Biosystem Laboratories (Spain). Helicobacter pylori detection in serum was done by an enzyme-linked immunosorbent assay for the identification of immunoglobulin G [20]. All samples were analyzed fresh.

## STATISTICAL ANALYSIS

All statistical analyses were performed with Statistical Package for Social Science (SPSS) 7.5 Data were analyzed for mean and standard deviation. Proportions were expressed as percentage while significant tests were done with the X 2 test. The result was considered significant at p < 0.05.

## RESULTS

Table 1 shows the sociodemographic characteristics of diabetic and non-diabetic patients. The two groups were comparable in sex, age, indicators of socioeconomic status except for living accommodation, which was significantly different. The

**Figure 1**

Table 1: Sociodemographic characteristics of diabetic and non-diabetic patients

| Characteristics | Diabetics n = 60 | Non-diabetics n = 60 | p-value |
|---|---|---|---|
| Duration of illness (yrs) | 4.12 ± 4.08 | | |
| Age (yrs) | 56.90 ± 12.24 | 52.13 ± 12.06 | 0.136 |
| BMI (Kg/m²) | 25.12 ± 4.15 | 23. 64 ± 3.37 | 0.034* |
| Sex | | | |
| Male (n/%) | 32 (53) | 27 (45) | |
| Female (n/%) | 28 (47) | 33 (55) | 0.361 |
| Marital status | | | |
| Married (n/%) | 54 (90) | 39 (65) | |
| Separated/Divorced/Widowed (n/%) | 6 (10) | 21 (35) | 0.001* |
| Living accommodation | | | |
| Single room (n/%) | 12 (20) | 21 (35) | |
| Flat (n/%) | 27 (45) | 13 (22) | |
| Bungalow (n/%) | 21 (35) | 26 (43) | 0.019* |
| Occupation | | | |
| Civil servant (n/%) | 17 (28) | 14 (23) | |
| Artisan (n/%) | 8 (13) | 13 (22) | |
| Farming (n/%) | 19 (32) | 15 (25) | |
| Unemployed (n/%) | 16 (27) | 18 (30) | 0.558 |
| Alcohol consumption | | | |
| No | 51(85) | 40 (67) | |
| Yes | 9 (15) | 20 (33) | 0.019* |
| Tobacco intake | | | |
| No (n/%) | 51 (85) | 41 (68) | |
| Yes (n/%) | 9 (15) | 19 (32) | 0.031* |
| Level of education | | | |
| None (n/%) | 15 (25) | 13 (22) | |
| Primary (n/%) | 20 (33) | 13 (22) | |
| Secondary (n/%) | 12 (20) | 16 (26) | |
| Tertiary (n/%) | 13 (22) | 18 (30) | 0.391 |

*p < 0.05

diabetics have significantly higher BMI than non-diabetics and both groups exhibited significant behavioral difference (alcohol and tobacco consumption).

**Figure 2**

Table 2: Comparison of biochemical parameters of diabetic and non-diabetic patients (mean + SD)

|  | Diabetics n = 60 | Non-diabetics n = 60 | p-values |
|---|---|---|---|
| FBG (mmol/l) | 10.33 ± 5.07 | 5.76 ± 0.64 | 0.002* |
| Haemoglobin (g/dl) | 12.84 ± 1.33 | 13.47 ± 1.13 | 0.049* |
| Packed cell volume (%) | 38.74 ± 4.72 | 40.87 ± 3.57 | 0.043* |
| Total cholesterol (mmol/l) | 5.11 ± 1.26 | 4.62 ± 0.87 | 0.013* |
| HDL-cholesterol (mmol/l) | 1.09 ± 0.22 | 1.13 ± 0.23 | 0.472 |
| LDL-cholesterol (mmol/l) | 3.05 ± 0.87 | 2.65 ± 0.57 | 0.043* |
| Triglyceride (mmol/l) | 1.57 ± 0.20 | 1.48 ± 0.27 | 0.191 |

*$p < 0.05$

Legend

FPG: fasting plasma glucose; HDL: High density lipoprotein; LDL: Low density lipoprotein

The diabetics and non-diabetics were significantly different in hematological and biochemical parameters estimated ($p < 0.05$) except for HDL-cholesterol and triglyceride (table 2).

**Figure 3**

Table 3: Prevalence of infection and dyspeptic symptoms in diabetic and non-diabetic patients

|  | Diabetics n = 60 | Controls N = 60 | p-values |
|---|---|---|---|
| **H. pylori infection** |  |  |  |
| Positive (%) | 21(35) | 17 (28) |  |
| Negative (%) | 39 (65) | 43 (72) | 0.432 |
|  |  |  |  |
| **Dyspeptic symptoms** |  |  |  |
| Absent (%) | 29 (48) | 37 (62) |  |
| Present (%) | 31 (52) | 23 (38) | 0.142 |

*$p < 0.05$

There was no statistically significant difference in the prevalence of H. pylori infection and dyspeptic symptoms between diabetics and non-diabetics (table 3). Helicobacter pylori-infected diabetics showed no statistically significant difference when compared with non-infected patients with regards to BMI, diabetic duration, hemoglobin; packed cell volume, total cholesterol, HDL-cholesterol, LDL-cholesterol and triglyceride (table 4), although H. pylori-infected

diabetics have slightly higher HDL-cholesterol and triglyceride.

**Figure 4**

Table 4: Comparison of anthropometrics and biochemical parameters among -positive and negative diabetics

|  | H. pylori positive n = 21 | H. pylori negative n = 39 | p- values |
|---|---|---|---|
| Age (yrs) | 63.71 ± 10.04 | 51.95 ± 12.06 | 0.00* |
| BMI (Kg/m²) | 24.45 ± 4.70 | 25.29 ± 4.17 | 0.48 |
| Duration of illness(yrs) | 4.43 ± 3.56 | 4.62 ± 5.13 | 0.88 |
| FPG (mmol/l) | 7.96 ± 3.75 | 11.58 ±5.18 | 0.01* |
| Haemoglobin (g/dl) | 12.73 ± 1.12 | 12.94 ± 1.31 | 0.53 |
| Packed cell volume (%) | 38.71 ± 4.70 | 39.08 ± 4.50 | 0.77 |
| Total cholesterol (mmol/l) | 4.95 ± 1.25 | 5.27 ± 1.17 | 0.33 |
| HDL-cholesterol (mmol/l) | 1.11 ± 0.18 | 1.06 ± 0.25 | 0.50 |
| LDL-cholesterol (mmol/l) | 3.00 ± 0.78 | 3.11 ± 0.94 | 0.65 |
| Triglyceride (mm0l/l) | 1.51 ± 0.23 | 1.47 ± 0.20 | 0.33 |

*$p < 0.05$

Legend
BMI: Basal metabolic index; FPG: fasting plasma glucose; HDL: High density lipoprotein; LDL: Low density lipoprotein

The H. pylori-infected diabetics were significantly older (63.71 vs. 51.95 years; p = 0.00) and had significantly lower fasting plasma glucose (7.96 vs. 11.58 mmol/l; p = 0.01) than the non-infected diabetics.

## DISCUSSION

This study shows a comparable prevalence of H. pylori infection in diabetics and non-diabetics (35% vs. 28%; p = 0.432) and did not support an association between infection with H. pylori and diabetes mellitus. Although this result corroborates similar studies elsewhere, and the prevalence comparable to prevalence rates reported by other authors: 33% vs. 32% [21], 28.1% vs. 29.25% [8], 37.3% vs. 35.2% [11], it is lower than 50.8% vs. 56.4% reported by Ko et. al. [22] in Chinese subjects with type 2 diabetes mellitus and non-diabetics respectively. However, it is in contrast with higher prevalence of H. pylori infection earlier reported in diabetes mellitus than in non-diabetics by some authors [1,9,12,23]. Our findings also contradict the lower prevalence of H. pylori infection reported in diabetic patients than in healthy population [24,25]. The lack of statistically significant difference in dyspeptic symptoms between diabetics and non-diabetics also corroborates the findings of Anatesios et. al. [11]. We also found that the fasting plasma glucose was significantly lower in H. pylori infected diabetics than non-

infected patients, indicating that H. pylori infection and H. pylori related gastrointestinal/gastroduodenal disorder may be related to glycaemic status. However, this is in contrast with the finding of Ko et. al. [22]. Peach and Barnnet [26] had previously shown that women infected with H. pylori had lower mean fasting plasma glucose concentration than did non-infected women. Although no histological study was done on our subjects, the lower fasting plasma glucose in H. pylori-infected than non-infected diabetics may partly be attributed to alteration in gastric mucosa as high prevalence of severe acute gastritic inflammation/ulcer disease has been reported in diabetic patients with little or no symptoms of dyspepsia [27]. H. pylori gastritis has been found to enhance glucose- and meal- stimulated insulin release, probably by increasing gastrin secretion [28]. However, no association has yet been documented between H. pylori infection and delayed gastric emptying or upper gastrointestinal symptoms in diabetics [29]. Again, Dursun et. al.[7] reported that H. pylori infection has no effect on insulin sensitivity/glucose metabolism in non-obese young adults. It has also been demonstrated that glycaemic controls deteriorate with age in healthy non-diabetic individuals, a phenomenon which has been attributed to small but steady decline in pancreatic beta cell function [30]. Whether these apply to diabetic/or H. pylori-infected diabetics is not known. Interestingly, in the present study, H. pylori-infected diabetics were significantly older than non-infected patients (63 vs. 51 years; p = 0.00). Thus it may be speculated that in diabetics infected with H. pylori, there is modification of glycaemic status, the mechanism of which remains largely unknown. In our subjects, one possibility is the early acquisition of H. pylori in area with high H. pylori endemicity. H. pylori may have been acquired earlier in life [25] independent of glycaemic status and prior to the development of type 2 diabetes mellitus and subsequently confers some degree of protection against excessive elevation of blood glucose. Wu et. al. [31] has hypothesized that lack of H. pylori infection, especially during childhood might enhance the risk of development of morbid obesity (a known risk factor for diabetes mellitus), based on their finding of inverse relationship between morbid obesity and H. pylori infection. Incidentally, H. pylori-infected diabetics in the present study have lower but insignificant BMI (24.45 vs. 25.29 Kg/m2; p = 0.48) and diabetic duration (4.43 vs. 4.62 years; p = 0.88) than non-infected subjects, thus supporting the protective roles of H. pylori-infection in the development of obesity (high BMI). However, obesity, in combination with H. pylori infection has been proposed to enhance response to insulin leading to

reduced fasting blood glucose levels among H. pylori positive obese persons in comparison with H. pylori positive lean persons [32]. It is known that obesity /high BMI causes decreased insulin sensitivity probably by cytotoxicity, TNF-? and resistin [33]. A recent study has however shown that H. pylori infection significantly worsen metabolic controls in children and adolescents with type 1 diabetes mellitus [34] and H. pylori eradication had been shown not to have significant effect on glycated hemoglobin levels [35]. Nevertheless, Yamagata et. al.[36] in The Hisayama Study demonstrated that hyperglycemia is a possible cofactor increasing the risk posed by H. pylori infection. The lack of significant difference in lipid profiles between H. pylori-infected and non-infected diabetics is in consonance with the finding of Dursen et. al. [7], thus affirming the metabolic neutrality of H. pylori infection in terms of serum lipids. However, H. pylori eradication has been reported to modify some parameters of lipids and homeostasis [37]. According to one study, H. pylori infection affect lipid metabolism in a way that could increase the risk of arteriosclerosis and has been regarded as an independent risk factor for coronary artery disease [4].

## CONCLUSION

Type 2 diabetes mellitus patients showed no significant difference in H. pylori seropositivity and dyspeptic symptoms from non-diabetics. H. pylori infection when present in diabetics appears to influence glycaemic status. Further studies are needed to clarify the mechanism underlying this effect and to elucidate the effect of H. pylori eradication on glycaemic controls in H. pylori –infected diabetic patients.

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