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Short Communication

Plasma lipids in *Helicobacter pylori* infected pregnant women

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Dyslipidaemia is a common feature of pregnancy and *Helicobacter pylori* infection respectively. We hypothesised that pregnant women infected with *H. pylori* may have increased disorder in lipid metabolism than non-infected pregnant women and thus estimated the plasma lipids in pregnant women with *H. pylori* infection. Forty (40) pregnant women (GA ≥ 11 weeks) attending ANC of the Department of Obstetrics and Gynaecology of the Federal Medical Centre, Abakaliki, Nigeria and found to be *H. pylori* positive by a serological test and 40 age- and socioeconomically matched *H. pylori* seronegative pregnant women who served as controls participated in this study. Fasting plasma lipids were determined by standard colorimetric methods. *H. pylori* infected pregnant women had significantly ($p < 0.05$) higher BMI than their non-infected counterparts. Although the total cholesterol, triglyceride and LDL-cholesterol were higher in *H. pylori* infected than non-infected pregnant women, only LDL-cholesterol was statistically significant (3.38 ± 1.52 vs. 2.34 ± 1.19, $p = 0.001$). On the other hand, HDL-cholesterol was lower in the *H. pylori* infected than non-infected pregnant women (0.68 ± 0.23 vs. 0.81 ± 0.49, $p > 0.05$). *H. pylori* infected than non-infected pregnant women exhibit greater disordered lipid metabolism that may be predisposing to increased risk for coronary heart disease (CHD). Further studies using larger sample sizes are needed to confirm these preliminary findings.

Key words: *Helicobacter pylori*, dyslipidaemia, pregnant women, coronary heart disease.

INTRODUCTION

Infection with *Helicobacter pylori* has been recognized as a public health problem worldwide (Bener et al., 2002) affecting approximately 50% of the world population and more prevalent in developing than the developed countries (Pounder, 1995). *H. pylori* seroprevalence of 20% has been reported in pregnant women (Eslick et al., 2002) and is associated with nausea and vomiting (Quinlan and Hill, 2003). *H. pylori* infection has been associated with both gastrointestinal and non-gastroenterological conditions such as chronic active gastritis, peptic ulcer, gastric adenocarcinoma, type B low-grade mucosa-associated lymphoid tissue lymphoma, and cardiovascular disease (Kanbay et al., 2005; Aslan et al., 2006; Naito et al., 2002). Moreover, infection with *H. pylori* is associated with conditions such as atherosclerosis, insulin resistance, diabetes mellitus and some autoimmune disease (Naito et al., 2002). Studies have shown association between *H. pylori* infection and elevated plasma lipids (Hoffmeister et al., 2001; De-Luis et al., 1998). Pregnancy is a physiological condition associated with both anatomical and biochemical alterations all geared towards the sustenance of the growing foetus (Ajose et al., 2002). Hyperlipidaemia, as a feature of pregnancy, has been well documented in women populations of different races (Ajose et al., 2002). Because hyperlipidaemia which is a common feature of both pregnancy and *H. pylori* infection is a known risk factor for coronary heart disease (Shankar et al., 2008; Kinjo et al., 2002), we hypothesise that pregnant women with *H. pylori* infection may have increased disorder in lipid metabolism than non-infected pregnant women. This study is therefore aimed to assess the lipid profile in pregnant women with *H. pylori* infection.

MATERIALS AND METHODS

This study which is a part of a larger study (Micronutrient Study) was conducted among pregnant women attending antenatal clinic at Federal Medical Centre, Abakaliki, one of the tertiary health facilities in the South-eastern Nigeria. Of the three hundred (300)

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consenting participants (GA ≥ 11 weeks) recruited between August 2007 and September 2008, seventy-seven (77) were found to be positive for *Helicobacter pylori* infection by a serological test (Pronovost, 1994). Forty (40) of these *H. pylori* seropositive pregnant women eventually qualified for the study while participants that were hypertensive (n = 10), diabetic (n = 15), HIV-seropositive (n = 2), alcoholics (n = 3), on lipid lowering drugs (n = 5) and with history of liver disease (n = 2) were excluded. Forty (40) age- and socio-economically matched apparently healthy *H. pylori* seronegative pregnant women served as controls. The Ethics and Research Committee of the Federal Medical Centre Abakaliki approved the protocol for this study. Socio-demographic data like age, parity, living accommodation educational level, and occupation were collected by a structured questionnaire. Height and weight were measured with the subject in light clothes without shoes, and BMI (Kg/m2) was calculated. Five millilitres (5.0 ml) of venous blood samples were collected between 08.00-10.00 h after 8-12 h overnight fasting into EDTA bottle and centrifuged at 2000 g for 5 min to prepare plasma. Total cholesterol and triglyceride concentrations were determined by enzymatic colorimetric assay as described previously (Siedel et al., 1983; Nagele et al., 1984) and HDL-cholesterol was determined enzymatically after precipitation of other lipoprotein as described by Warnic et al. (1982) while LDL-cholesterol was calculated using Friedewald equation (1972). All samples were analysed within 24 h after collection.

Statistical analysis

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 comparison of mean plasma lipids were done with one-way analysis of variance (ANOVA) with significant level set at $p < 0.05$.

RESULTS

From Table 1, both the *H. pylori* infected and non-infected pregnant women have comparable educational level ($p = 0.272$), living accommodation ($p = 0.445$) and occupation ($p = 0.563$).

The *H. pylori* infected pregnant women were older (Age = 28.95 ± 4.89 vs. 27.38 ± 4.84 yrs and have had more deliveries (Parity = 2.70 ± 1.56 vs. 2.53 ± 1.55) than the non-infected subjects, although the differences were not statistically significant (Table 2). However, the *H. pylori* infected pregnant women have significantly ($p < 0.05$) higher BMI than their non-infected counterparts.

Table 3 shows that although the total cholesterol, triglyceride and LDL-cholesterol were higher in the *H. pylori* infected than non-infected pregnant women only LDL-cholesterol reached statistically significant level (3.38 ± 1.52 vs. 2.34 ± 1.19 mmol/l, $p = 0.001$). On the other hand, HDL-cholesterol was lower in the *H. pylori* infected than non-infected pregnant women, although this was not statistically significant (0.68 ± 0.23 vs. 0.81 ± 0.49 mmol/l, $p > 0.05$).

DISCUSSION

This study has shown that pregnant women infected with *H. pylori* have higher BMI and plasma lipid than the non-infected pregnant women of comparable age, parity and socioeconomic status. To the best of our knowledge, this is the first study that evaluates the plasma lipid in *H. pylori* infected pregnant women. Significantly higher BMI in hyperlipidaemic *H. pylori* infected than non-infected pregnant women found in the present study corroborates earlier reports of a positive and significant association between BMI and triglycerides (Donahue et al., 1985). Similarly, the Minneapolis Children's Blood Pressure Study also showed same results (Prineas et al., 1980). In addition to obesity (Pihl and Jurimae, 2001) and BMI (Bertolli et al., 2003) lipid profile has been reported to be associated with lifestyle (Serter et al., 2004), age (Maki et al., 1997), intra-abdominal adiposity (Mannabe et al., 1999; Mari et al., 1999), and waist to hip ratios (Okosun et al., 2000). It may therefore be speculated that increased BMI in combination with *H. pylori* infection in pregnancy may induce enhanced response to insulin leading to reduced plasma glucose levels (Ioannis et al., 2003) that may consequently lead to altered lipid metabolism as shown by the present study. Decreased HDL-cholesterol found in *H. pylori* infected than non-infected pregnant women in this study (although not statistically significant) may indicate decreased rate of reverse cholesterol transport and therefore accumulation of triglyceride-rich lipoproteins (Shankar et al., 2008). Hyperlipidaemia, a common finding in both pregnancy and *H. pylori* infection is an established risk factor for coronary artery disease (Shankar et al., 2008; Kinjo et al., 2002). The higher plasma lipid levels in *H. pylori* infected than non-infected pregnant women in the present study therefore suggests that *H. pylori* may predispose pregnant women with *H. pylori* infection to increased risk for coronary heart disease (CHD). More interesting is the finding of statistically significant elevation in the LDL-cholesterol in the infected than the non-infected women. It has been found that the blood level of HDL-C bears an inverse relationship with the risk of atherosclerosis and coronary heart disease (Khoo et al., 1997). Indeed, Kanbay et al. (2005) in a study concluded that *H. pylori* infection may affect lipid metabolism in a way that could increase the risk of atherosclerosis. They further posited that *H. pylori* infection may be regarded as an independent risk factor for CHD based on the findings that *H. pylori* eradication increased plasma HDL-C in *H. pylori* infected patients. Also eradication of *H. pylori* infection in type 1 diabetic patients has been found to modify some parameters of lipid. *H. pylori* infection by altering lipid profile and promoting the synthesis of acute phase reactants may be suggested as a possible pathophysiology of CHD in infected pregnant women (De-Luis et al., 1998). However, it is yet to be ascertained if *H. pylori* eradication would improve or not the hyper-lipidaemia observed in *H. pylori* infected pregnant women. In conclusion, *H. pylori* infected than non-infected pregnant women tend to experience greater disordered lipid metabolism and in a manner predisposing to increased risk of CHD. This preliminary findings need to be

confirmed in studies involving larger number of participants.

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REFERENCES

Ajose OA, Fasuba OB, Thomas KD, Bolodeoku JO (2002). Serum lipids and lipoprotein cholesterol profiles in pregnant Nigerian women. J. Clin. Sci. 2 (1-2): 9-13.

Aslan M, Horoz M, Nazligul Y Bolukbas C, Bolukbas FF, Selek S, Celik H, Erel O (2006). Insulin resistance in H. pylori infection and its association with oxidative stress. World J. Gastroenterol. 12 (42): 6865-6868.

Bener A, Uduman SA, Ameen A, Alwash R, Pasha MAH, Usmani MA, Al-Naili SR, Amiri KMA (2002). Prevalence of Helicobacter pylori infection among low socio-economic workers. J. Communicable Dis. 34: 179-84.

Bertolli A, Di-Daniele N, Ceccobelli M, Ficara A, Girasoli C, Lorenzoa D (2003). Lipid profile, BMI, body fat distribution and aerobic fitness in men with metabolic syndrome. Acta. Diabetol. 40 (Suppl): S130-S133.

De Luis DA, Lahera M, Canton R, Boixeda D, San Roman AL, Aller R, de la Calle H (1998). association of Helicobacter pylori infection with cardiovascular and cerebrovascular disease in diabetic patients. Diabetes Care 21 (7): 1129-1132.

Donahue RP, Orchard TJ, Kuller LH, Drash AL (1985). Lipids and lipoproteins in young adult population. Am. J. Epidemiol. 240:458-67.

Eslick GD, Yan P, Xia H H-X, Murray H, Spurrett NJ, Talley E (2002). Foetal intrauterine growth restrictions with *Helicobacter pylori* infection. Aliment. Pharmacol. Ther. 16 (9): 1677-1682.

Friedewald WT, Levy RI, Fredrickson DS (1972). Estimation of the concentration of low-density-lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin. Chem. 18: 499-502.

Hoffmeister A, Rothenbacher D, Bode G, Persson K, Marz W, Nauck MA, Brenner H, Hombach V, Koenig W (2001). Current infection with Helicobacter pylori, but not seropositivity to Chlamydia pneumoniae or cytomegalovirus, is associated with an atherogenic, modified lipid profile. Arterioscler Thromb. Vasc. Biol. 21 (3): 427-432.

Ioannis DK, Ioannis S, Panagiotis D, Vasilios G, Konstantinos D, Aggeliki D (2003). Impact of body mass index on fasting blood glucose concentration among Helicobacter pylori carriers. Dig. Dis. 21: 262-265.

Kanbay M, Gür G, Arslan H, Yilmaz U, Boyacioglu S (2005). Does eradication of Helicobacter pylori infection help normalize serum lipid and CRP levels? Dig Dis. Sci. 50(7): 1228-12231.

Khoo KL, Tan H, Leiw YM (1997). Serum lipids and their relationship with other coronary risk factors in healthy subjects in a city clinic. Med. J. Malaysia 52: 38-52.

Kinjo K, Sato H, Sato H (2002). Prevalence of H. pylori infection and its link to coronary risk factors in Japanese patients with acute myocardial infarction. Cir. J. 66: 805-810.

Maki KC, Kritsch K, Foley S, Soneru I, Davidson MH (1997). Age dependence of the relationship between adiposity and serum LDL-C in men. J. Am. Clin. Nutr. 16: 578-583.

Mannabe E, Aoyagi K, Tachibana H, Takemoto T (1999). Relationship of intra-abdominal adiposity and peripheral fat distribution to lipid metabolism in an Island population in Western Japan: Gender differences and effect of menopause. Tohoku J. Exp. Med. 188: 189-202.

Mari O, Furuya R, Ohkawa S, Yoneyama T, Nisikino M, Hishida A, Kumagai H (1999). Altered abdominal fat distribution and its association with serum lipid profile in non diabetic haemo-dialysis patients. Nephrol. Dial. Transplant 14: 2427-2432.

Nagele U, Hagele EO, Sauer G, Wiedemann E, Lehmann P, Wahlefeld AW, Gruber W (1984). Reagent for the enzymatic determination of serum total triglycerides with improved lipolytic efficiency. J. Clin. Chem. Clin. Biochem. 22: 165-174.

Naito Y, Yoshikawa T (2002). Molecular and cellular mechanisms involved in Helicobacter pylori-induced inflammation and oxidative stress. Free Radic. Bio.l Med. 33: 323-336.

Okosun S, Richard ICS, Richard BO, Cooper S, Forrester T (2000). Association of waist circumference with risk of hypertension and type 2 diabetes in Nigerians Jamaicans and Africa-Americans. Diabetes Care 21: 1836-1842.

Pihl E, Jurimae T (2001).relationship between body weight change and cardiovascular risk factors in male former athletes. Int. J. Obes. Relat. Metab. Disord. 25: 1057-1062.

Prineas RJ, Gillum RF, Horibe H, Hannan PJ (1980). Stat. The Minneapolis Children's Blood Pressure Study: standards of measurement for children's blood pressure. Hypertention 2 (Suppl.): S18-24.

Pounder RE, Ng D (1995). The prevalence of Helicobacter pylori infection in different countrie. Aliment. Pharmacol. Ther. 9: 33-39.

Pronovost AD, Rose SL, Pawlak JW, Robin H, Schneider R (1994). Evaluation of a new immunodiagnostic assay for Helicobacter pylori antibody detection: Correlation with histopathological and microbiological results. J. Clin. Microbiol. 32: 46-50.

Quinlan JD, Hill DA (2003). Nausea and Vomiting of Pregnancy. American Family Physician 68: 121-128.

Shankar V, Kaur H, Dahiya K, Gupta MS (2008). Comparison of fasting and postprandial lipid profile in patients of coronary heart disease. Bombay Hosp. J. 50 (3): 445-449.

Serter R, Demirbas B, Korukluoslu B, Culha C, Cakal E, Aral Y (2004). the effect of L-thyroxine replacement therapy on lipid based cardiovascular risk in subclinical hypothyroidism. J. Endicrinol. Invest. 27: 897-903.

Siedel J, Hegele EO, Ziegenhorn J, Wahlefeld AW (1983). Reagent for the enzymatic determination of total serum cholesterol with improved lipolytic efficiency. Clin. Chem. 29: 1075-1080.

Warnick GR, Benderson JM, Alberts JJ (1982). Quantitation of high-density- lipoprotein subclass after separation by dextran sulphate and $Mg2^{+}$ precipitation. Clin. Chem. 28: 1574.

Witztum JL, Steinberg D. Role of oxidized low density lipoproteinin atherogenesis. J. Clin. Invest. 1991(88):1785-1792.