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Research Article

Pattern of *Helicobacter pylori* infection in normal, overweight and obese adults in Nigeria

F.L. Ciroma[1*], M.B. Akor-Dewu[2], M.A. Kana[3], J.O. Ayo[4] and A. Mohammed[2]

Departments of Human Physiology, [1]College of Medicine, Faculty of Basic Medical Sciences, Kaduna State University, Nigeria, [2]College of Health Sciences, Faculty of Basic Medical Sciences, Ahmadu Bello University, Zaria, Nigeria, [3]Department of Community Medicine, College of Medicine, Faculty of Clinical Sciences, Kaduna State University, Nigeria and [4]Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria

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ABSTRACT

Background: Obesity is a state of chronic energy imbalance which has a multifactorial origin, but mainly believed to be due to overeating and underactivity. Prevalence of obesity is rising to an epidemic level world-wide. Obesity is associated with metabolic and cardiovascular complications, and recently, *Helicobacter pylori* infection (*H. pylori*). *H. pylori* is a gram-negative bacteria that colonizes the gastric mucosa, and is highly prevalent. It has been implicated in the pathogenesis of chronic gastritis, gastric cancer and inflammation. Previous studies observed *H. pylori* infection as a risk factor to development of obesity, but other reports suggest a protective role of *H. pylori* against obesity. There is inconsistent data across various population studies. **Aim:** To investigate the pattern of *H. pylori* in overweight and obese subjects in Nigeria. **Methods:** A total of 277 subjects (185 males and 92 females), within the age of 18- 72 years were recruited from a university in northern part of Nigeria. Anthropometric and blood pressure were measured, and participants were grouped into normal, overweight and obese. *H. pylori* was serologically assayed by ELISA. **Results:** A total of 149 (53.8%) subjects were within normal body mass index (BMI), 55 (19.9%) were overweight and 73 (26.4%) were obese. From a total of 125 respondents who were positive to *H. pylori*, 52% were within normal group, 19.2% were overweight, and 28.8% were obese. *H. pylori* infection was present in 64/185 (34.6%) of males and 61/92 (66.3%) of females in the studied population. Out of obese respondents, only 16% of obese males were positive to *H. pylori*, but up to 43% of obese females were *H. pylori* positive. **Conclusion:** In the present study, *H. pylori* infection was less in obese males and more in the normal group. In the females, *H. pylori* was more in the obese and less in normal and overweight groups.

INTRODUCTION

Obesity is a global epidemic (Smith and Smith, 2016), which is currently increasing in Nigeria (Akarolo-Anthony *et al*., 2014) and fast becoming a major health problem (Ogunbode *et al*., 2011; Adediran *et al*., 2012). Obesity is associated with co-morbid conditions such as hypertension and other cardiovascular disorders, metabolic diseases, cancers, peptic ulcers and *Helicobacter pylori* (*H. pylori*) infection (Renshaw *et al*., 2001).

H. pylori is a gram-negative bacterium which hasnncolonized the human stomach for thousands of years. *H. pylori* bacteria is a major cause for gastritis, peptic ulcer disease and gastric cancer (Laszewicz *et al*., 2014). Occurrence of this infection has been linked with poor sanitation and hygiene, low socio-economic status and high population density, common in underdeveloped and developing countries (Laszewicz *et al*., 2014). Studies have shown that occurrence of *H. p*ylori infection early in life causes reduced weight and growth retardation, suggesting that it alters host metabolism early in life (Khosravi *et al*., 2015). *H. pylori* bacteria, has in fact, been classified as a human carcinogen because of its strong association with gastric cancer (O'Connor *et al*., 2017).

*Address for correspondence:
Email: fatimaumaralkali@gmail.com
Tel: +234 8037169285

The relationship between *H. p*ylori and obesity is controversial. Epidemiologic studies have demonstrated a high prevalence of *H. pylori* infection and obesity, especially in the developing countries. Previous studies have reported an increase in the prevalence of *H. pylori* infection among obese individuals (Arslan *et al*., 2009; Li *et al*., 2012; Zhang *et al*., 2015), thereby suggesting that *H. pylori* infection may predispose to obesity. Chen *et al*. (2018) observed higher BMI in subjects with *H. pylori* when compared with subjects who were *H. pylori* negative, and the infection increased the risk of being obese in age-group of less than 50 years. Studies from developed countries have, however, reported a decline in infection rate by *H. pylori*, coinciding with increasing prevalence of obesity (Lender *et al*., 2014). This may suggest a protective role of *H. pylori* infection against development of obesity. Currently, obesity is continuously being linked with *H. pylori* infection, although there is inconsistent and inconclusive evidence (Carabotti *et al*., 2014). Ciroma *et al*. (2015) opiened that *H. pylori* may be involved in the pathophysiology of obesity probably by eroding gastric mucosa cells that are involved in secreting ghrelin, an orexigenic hormone, or through dysregulated ghrelin secretion. Furthermore, *H. pylori* eradication was shown to increase gastric secretion of ghrelin, leading to increased appetite and weight gain (Nwokolo *et al*., 2003). Notwithstanding, a recent study has failed to provide evidence for association between *H. pylori* and BMI or obesity in adults (Hollander *et al*., 2017). In any case, studies on *H. pylori* and obesity are limited in Nigeria, therefore this study aims to determine how *H. pylori* is spread within normal, overweight and obese respondents in Nigeria.

MATERIALS AND METHOD

Study Design and Study Population

The study is a cross-sectional descriptive study which involved members of Kaduna State University community, males and females within the age of 18 - 72 years. Participants who had normal body mass index (BMI) between 18.5 to 24.9 kg/m^2 served as the control (normal). Overweight group were those within BMI of 25 to 29.9 kg/m^2, and obese group were BMI > 30 kg/m^2.

Ethical Approval

Ethical clearance was obtained from Health Research Ethics Committee, Kaduna State Ministry of Health, Kaduna (Reference No: MOH/ADM/744/Vol.1/61). All participants consented to take part in the study, which was conducted according to the Helsinki principles and American Physiological Society Guiding Principles for Research, involving animals and human beings (World Medical Association, 2009).

Exclusion criteria

Individuals who smoke, frequently consume alcohol, pregnant women, known diabetics, subjects with known endocrine disorders, peptic ulcers, hypertension and those taking medication for any of the mentioned ailments were excluded from the study.

Blood Serum and sample collection

Blood sample (10 ml) was collected; 3 ml was immediately stored in a sample tube containing dipotassium ethylenediaminetetraacetic acid (K_2EDTA) for haematological analyses, The remaining blood sample (7 ml) was emptied into labeled plain tubes to obtain the serum for other analyses. *H. pylori* was detected by serological testing using the appropriate ELISA kit. After laboratory analyses, participants who were found with some abnormal values were again referred to the university sick-bay for consultation with a physician.

RESULTS

Out of a total of 277 respondents in the study (185 males and 92 females), 149 were within normal BMI (18.5-24.9 kg/m^2), 55 were overweight (25-29.9 kg/m^2) and 73 were obese (>30 kg/m^2). Mean age of obese subjects was significantly higher when compared with normal subjects, but not significant between the overweight and obese. Both systolic and diastolic blood pressure was not significantly different between normal, overweight and obese (Table 1). *H. pylori* infection was serologically positive in 125 (45.13 %) and serologically negative in 152 (54.87%) of the participants. Out of total participants who tested positive, 65 (43.62%) were

Table 1: Anthropometric and mean blood pressure of overall study participants

Variable	Normal (n = 149)	Overweight (n = 55)	Obese (n = 73)	p-value
Age (years):	27.6 ± 6.79	37.0 ± 12.00	38.4 ± 2.08[a]	0.000**
Blood pressure (mm Hg)				
SBP	116.5 ± 1.15	119.1 ± 2.64	115.1 ± 1.54	NS
DBP	80.5 ± 1.90	78.9 ± 1.24	79.0 ± 0.71	NS

a = significant when compared with normal, ** significant at 0.001 level. Values are Means ± SEM

Table 2: *Helicobacter pylori* infection in overall study population according to body mass index and sex

	Number tested	Number seropositive to *H. pylori* (%)	Number seronegative to *H. pylori* (%)
Normal	149	65 (43.62)	84 (56.38)
Overweight	55	24 (43.64)	31 (56.38)
snObese	73	36 (49.32)	37 (50.68)
Total	277	125 (45.13)	152 (54.87)
Males	185	64 (34.59)	121 (65.41)
Females	92	61 (66.30)	31 (33.69)

of normal BMI; 24 (43.64%) were overweight while 36 (49.32%) were obese (Table 2). In male respondents, 64/185 (34.59%) were positive and 121/185 (65.41%) were negative. But in the females 61/92 (66.30%) were positive and 31/92 (33.70%) were negative to the infection (Table 2).

Furthermore, out of the total participants who were obese, 28% of males and 72% of females were positive to *H. pylori* infection (Figure 1).

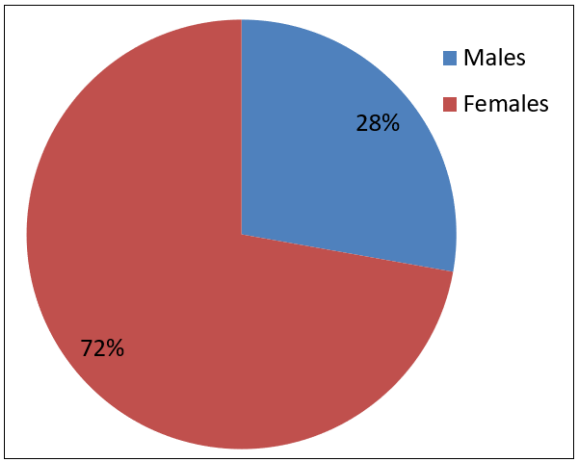

Fig. 1: *Helicobacter pylori* infection in obese study participants

Within the males, up to 62% of normal BMI tested positive to *H. pylori*, while 22% of overweight and only 16% of obese were *H. pylori* positive (figure 2). Greater percentage of males within normal BMI tested positive to *H. pylori*.

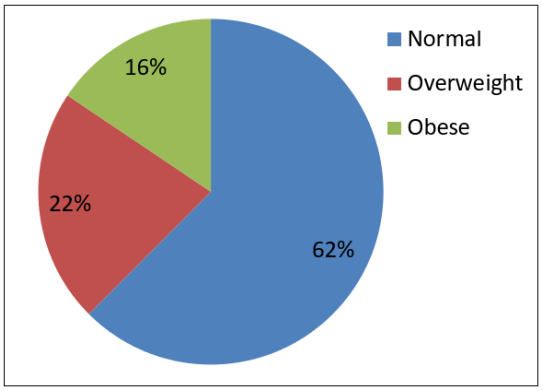

Fig. 2: Pattern of *Helicobacter pylori* Infection in Male participants According Body mass index

On the other hand, in the females, 41% of normal were positive to *H. pylori*, while only 16% of overweight and 43% of obese were *H. pylori* seropositive (figure 3). Greater percentage of obese women tested positive to *H. pylori*.

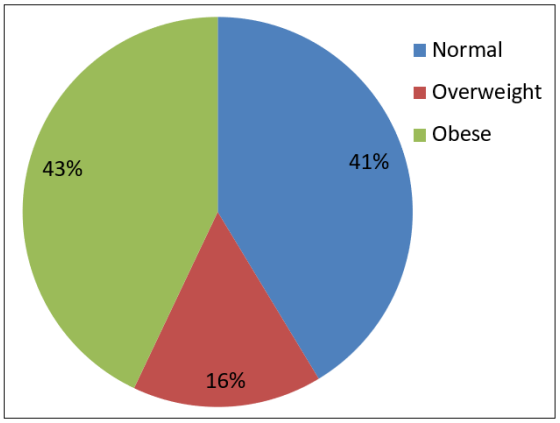

Fig. 3: Pattern of *Helicobacter pylori* Infection in Female participants According to body mass index

DISCUSSION

Pattern of Helicobacter pylori infection

The overall prevalence of *H. pylori* in the study was 45.13%, which was lower than 70% - 90% that was reported by Carabotti *et al*. (2014). Reason for the lower prevalence rate in the present study may be because the study was conducted within a university setting, which is assumed to have better sanitation and improved hygiene. Poor sanitary conditions and hygiene have been found to increase the risk of infection by *H. pylori* bacteria (Laszewicz *et al*., 2014). The prevalence of *H. pylori* in obese

respondents appears to be much higher in obese females than in obese males, suggesting that obesity may occur more in women with *H. pylori* infection. Or, since *H. pylori* bacteria is said to be acquired early in life during childhood, it may be that the bacteria predisposes females to develop obesity. In other words, *H. pylori* infection may be a risk factor to development of obesity in women. Although our study cannot propose that obesity in women may have been caused by *H. pylori* infection, the bacteria is known to colonize the gastric mucosa right from childhood (Taylor and Blaser, 1991), therefore the bacteria may most likely have appeared before onset of obesity.

Male participants within normal BMI were observed to have more occurrence of *H. pylori* infection, in this study. Again, since the bacterium is thought to colonize the body earlier during childhood, it may be that *H. pylori* somehow protects males against obesity. In other words, *H. pylori* infection occurred most in males with normal weight, and it occurred least in males with obesity. This finding could imply that presence of *H. pylori* in normal weight males may play a protective role against development of obesity. A possible mechanism could probably be explained in the sense that since *H. pylori* infection affects the gastric mucosa, it may have affected more gastric mucosal cells that produce ghrelin (Paoluzi *et al*., 2014). Ghrelin is an energy-regulating hormone, secreted by cells in the gastric mucosa, and it stimulates food intake (Kojima *et al*., 1999). Although a causative relationship between *H. pylori* and decline in ghrelin secretion is inconclusive, large majority of studies investigating levels of circulating ghrelin and ghrelin expression in the stomach in patients with *H. pylori* infection indicate that the bacterium has a negative impact on ghrelin production and/or secretion (Jeffery *et al*., 2011). Therefore, large percentage of males in the present study who are *H. pylori* positive and within normal weight status, may perhaps be due to ghrelin suppression together with its appetite-stimulating effects, probably brought about by gastric atrophy caused by *H. pylori* infection. This perception gives more weight in support of studies which propose that the absence of *H. pylori* could lead to obesity because they observed increased BMI and weight gain after eradication of *H. pylori* infection (Nwokolo *et al*., 2003).

Although our study cannot conclusively state that *H. pylori* in females predisposes the females to obesity, or increase the risk of the females to obesity; and *H. pylori* plays a protective role against obesity in the males, it generates a hypothesis on the selective role of *H. pylori* in males and females towards obesity in the studied population.

Nonetheless, the relationship between *H. pylori* and obesity remains controversial because of conflicting findings across various populations, and different methods of detecting *H. pylori* (Chen *et al*., 2018). Therefore, more studies involving larger samples and populations are needed to ascertain the actual stance.

CONCLUSION

H. pylori infection was observed to be progressively higher as BMI tends towards normal in male respondents. In other words, *H. pylori* was seen to affect more normal weight males, while it least occurs in obese males. But in the females, it poses a different pattern; highest level of infection by *H. pylori* was found within obese females.

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