The prevalence of overweight and obesity is increasing worldwide. Obesity and overweight are caused by several factors, including genetic, metabolic, behavioral and environmental. The increasing prevalence suggests that behavioral and environmental influences play a predominant role, rather than biological changes.

There are also associations between obesity and several diseases, including diabetes mellitus, hypertension, dyslipidemia, and ischemic heart disease. In addition, higher degrees of obesity (BMI $\geq 35$ kg/m$^2$) are linked to higher mortality rates.

Although medical programs favor non-surgical methods for weight loss, bariatric surgery is widely accepted as the only known effective treatment for morbid obesity.

H. pylori are microaerophilic, gram-negative organisms that leads to many gastroduodenal diseases such as chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue lymphoma (MALT) and gastric cancer. H. pylori are found to be involved with other system affections, such as cardiovascular, immunological and systemic disorders. Recently, H. pylori were associated with insulin resistance and obesity.

The prevalence of H. pylori infection in obese patients is still controversial. Many studies demonstrated a low prevalence of H. pylori in obese patients, others reported high prevalence and some found an increase in body mass index (BMI) after H. pylori eradication.

The aim of this study is to evaluate the prevalence of H. pylori in obese and non-obese patients.

METHOD

One hundred obese (endoscopy evaluation before bariatric surgery) and 100 non-obese subjects (endoscopy screening) were included in the study. Informed consent was obtained from the participants.

Inclusion criteria were as follows: 100 obese subjects with body mass index (BMI) $\geq 30$ kg/m$^2$ and 100 non-obese subjects with 18.5 $< $ BMI $< 25$ kg/m$^2$ according to WHO criteria.
Exclusion criteria were as follows: chronic liver or renal failure, malignancy, connective tissue disorders, history of eradication therapy and those with history of significant upper GI symptoms.

Documented data included clinical examination, patients’ weight and height, liver function tests, renal function tests, electrolytes, complete blood picture, fasting plasma glucose level and lipid profile. Ultrasound abdomen was performed. Upper gastroduodenoscopy using EPK-1 Pentax was performed to evaluate gastroduodenal pathology and random biopsies (one from the antrum and one from the body) for rapid urease test using Kimberly-Clark CLO test.

RESULT

The mean age for H. pylori positive patients was 38.6±11.06 years and 35.3±11.1 years for H. pylori negative patients, P=0.41. The mean BMI was 45.7±7.5 in obese patients and 22.22.8± in non-obese patients.

In obese patients, 18 (29%) males and 44 (71%) females were H. pylori positive, while in non-obese patients, 13 (36%) males and 23 (64%) females were H. pylori positive, P=0.041.

H. pylori in obese patients were seen in 62 (62%) patients and in non-obese patients were seen in 36 (36%) patients, P=0.052.

Fatty liver was seen in 32 (53.2%) H. pylori positive obese patients and in 11 (28.9%) of H. pylori negative, P=0.05. However, fatty liver in H. pylori positive non-obese patients was seen in 16 (44.4%) and in H. pylori negative non-obese patients was seen in 18 (28.1%), P=0.06.

We found that fatty liver is more common in H. pylori positive patients. H. pylori infection was considered a risk factor for the development of non-alcoholic fatty liver disease (NAFLD) because of its link with the pathogenesis of insulin resistance (IR)6.

H. pylori were more prevalent in obese patients than non-obese, it is similar to the result by Arslan et al who found that prevalence of H. pylori in obese patients was 66.2%, and in non-obese 35.2%10. The high prevalence of H. pylori in obese patients is attributed to the alteration of the innate and adaptive immunity which is usually found in obesity because morbidly obese patients have less mature monocytes, low macrophages and reduced polymorphonuclear (PMN) bactericidal capacity46. In morbidly obese, there is a significant reduction in natural killer cells activity when compared to normal individuals matched for age and gender11.

Another study suggested a suppression of peripheral blood mononuclear cells chemokine and cytokine production, with subsequent reduction of the immune response to infectious agents12. Moreover, animal studies have also suggested immune dysfunction in obese animals leading to an altered response to both bacterial and viral infection13.

On the other hand, some studies found that H. pylori infection reduce the ghrelin secretion and consequently decreases appetite, and they noticed increased weight after H. pylori eradication with demonstrated increased plasma ghrelin levels14.

The difference in the results of previous research may be attributed to using different testing modalities for H. pylori because serologic testing is less sensitive and specific (85% and 79%) compared to histology (>95% for both) and is considered inferior to rapid urease test for the diagnosis of H. pylori, because antibodies remain positive whether the infection is active or resolved15.

CONCLUSION

There is a high prevalence of H. pylori infection in obese patients tested before bariatric surgery, and our results raise the issue of whether preoperative esophagogastroduodenoscopy should routinely include biopsies to evaluate H. pylori infection in morbidly obese patients.

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