Diabetes & Metabolic Syndrome: Clinical Research & Reviews xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Diabetes & Metabolic Syndrome: Clinical Research & Reviews

journal homepage: www.elsevier.com/locate/dsx

Original Article

The effects of *helicobacter pylori* eradication on modification of metabolic syndrome parameters in patients with functional dyspepsia

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ARTICLE INFO

Article history:
Available online xxx

Keywords:
Helicobacter pylori
Dyspepsia
Treatment
Metabolic syndrome

ABSTRACT

Background: Helicobactor pylori (*H. pylori*) have the potential role in the pathogenesis of various extra-gastric disorders such as metabolic disorders. But, it is now questioned about whether *H. pylori* eradication reduces or induces the risk for metabolic disorders especially in patients with dyspepsia. Hence, the present study aimed to assess the effects of *H. pylori* eradication on criteria of metabolic syndrome.

Methods: H. pylori infected patients with dyspepsia were included. The patients were treated with omeprazole (20 mg, q12 h), amoxicillin (1 g, q12 h), and clarithromycin (500 mg, q12 h) for two weeks, then *H. pylori* eradication was evaluated by C^{14} Breathing test (UBT) 6 weeks after the end of the treatment. Demographic data, clinical manifestation and metabolic parameters were recorded before and three months after completing treatment regimen. The data was analyzed by SPSS version 16.0.

Results: Of 110 patients were initially enrolled, 91 patients completed the study. Overall eradication rate was 61.5%. Significant differences in the serum level of total cholesterol(180.7 ± 34 vs. 172.1 ± 28, $p = 0.001$), LDL(107.0 ± 25 vs. 100.8 ± 20, $p < 0.001$), HDL(46.2 ± 8.7 vs. 48.9 ± 8.6, $p < 0.001$), fasting blood sugar (93.7 ± 12 vs. 90.9 ± 10, $p = 0.001$), hemoglobin A1c(5.37 ± 0.52 vs 5.25 ± 0.53, $p = 0.006$), and as well as for waist circumference(92.2 ± 14 vs. 91.4 ± 13.9, $p = 0.03$) was found after treatment. Data for body weight, systolic and diastolic blood pressure and triglyceride level remained without any significant changes.

Conclusion: H. pylori eradication could relatively reduce the risk of metabolic syndrome criteria such as fasting blood sugar, hemoglobin A1c, lipid profile and waist circumference.

1. Introduction

The metabolic syndrome (MS) is a common metabolic phenomenon with high prevalence and adverse consequences such as coronary artery disease in almost all countries. MS as a potential risk factor for increasing atherothrombotic risk is originally resulted from interplay between genetic and environmental factors [1]. In other words, the main pathophysiological basis of MS is sourced from the combination of some metabolic disturbances including obesity, insulin resistance, endothelial dysfunction, elevated blood pressure, atherogenic dyslipidemia, hyper-coagulation, chronic stress, and genetic susceptibility [2,3]. Along with these metabolic factors, the close link between infectious conditions and MS risk has been recently suggested. The causality between MS and infections can be explained by inducing production and secretion of some pro-inflammatory cytokines leading metabolic manifestations [4,5]. One of the main organisms that may be related to increased risk for MS is *H. pylori* infection [6]. *H. pylori* is gram-negative bacterium that is colonized in the gastric epithelium and is identified as a main cause for different gastric problems such as peptic ulcer disease, chronic gastritis, functional dyspepsia, and even gastric carcinoma. The progression of this infection leads to some immunological reactions such as mononuclear cellular infiltration and cytokines secretion leading extra-gastric manifestations [7,8].

Since the discovery of *H. pylori*, a variety of studies have assessed the potential involvement of it in the pathogenesis of various extra-gastric disorders. The association between *H. pylori*

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http://dx.doi.org/10.1016/j.dsx.2017.07.035
1871-4021/

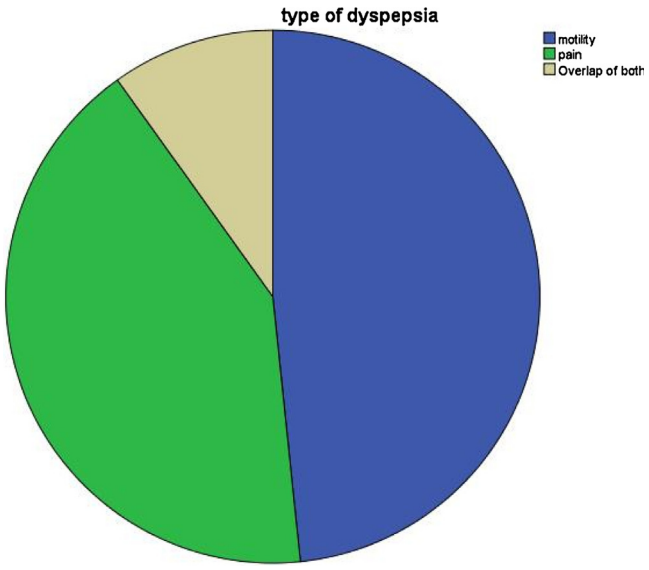

Fig. 1. Distribution Chart Frequency Type of Dyspepsia.

infection and risk for coronary artery disease have been demonstrated and thus eradication of *H. pylori* can attenuate the reduction in coronary artery lumen in CAD patients and also can reduce the risk for restenosis in coronary stenting [9,10]. The association between *H. pylori* infection and risk for coronary atherosclerosis can referred to the direct effects of *H. pylori* on progressing inflammatory-mediated coronary atheroma or to its impacts on increasing the risk for hyperlipidemia [11]. Some studies could indicate the elevation of serum lipid profiles such as total cholesterol, low-density lipoprotein cholesterol (LDL-c), lipoprotein Lp(a), apolipoprotein apo-B, and triglyceride concentrations in positive *H. pylori* patients [12]. In fact, infection by *H. pylori* can medicate secretion of active pro-inflammatory cytokines that can affect lipid metabolism via activating adipose tissue lipoprotein lipase and lipolysis as well as stimulating hepatic fatty acid synthesis [13]. Moreover, high prevalence of *H. pylori* in diabetic patients has been also revealed. However, some others could show lower prevalence of this infection in diabetic ones explaining contradictory effects of *H. pylori* infection on inflammatory biomarkers [14]. In total, it is now questioned whether *H. pylori* eradication reduces risk for metabolic disturbances or induce metabolic abnormalities. Hence, the present study aimed to assess the effects of *H. pylori* eradication on criteria of metabolic syndrome in patients with functional dyspepsia.

2. Materials and methods

This interventional before-after study was performed on patients aged higher than 18 years with the manifestations of functional dyspepsia (according to the ROME III criteria) that referred to gastroenterology clinic or endoscopy unit in Rasoul-a-Akram hospital in Tehran between April and September 2015. The exclusion criteria were pregnancy and breastfeeding, history of receiving HP eradicating regimens, pump proton inhibitors, H2 antagonists and/or antibiotics in recent 3 months, history of acute infectious disease during the last 3 months history of gastric surgery, cardiac or pulmonary diseases, endocrine abnormalities, chronic renal failure, liver diseases, history of cancer, typical symptom of gastro-esophageal reflux disease, irritable bowel syndrome, and major depression or anxiety disorders. According to the diagnostic criteria of functional dyspepsia, the patients were assessed in two categories of epigastric pain syndrome(ulcer-like dyspepsia), postprandial distress syndrome(dysmotility-type dyspepsia) and overlap of both of them. In those with warning signs or age >50 years, upper endoscopy was scheduled and the presence of HP infection was tested by rapid urease test (RUT), otherwise, the diagnosis of HP infection was based on noninvasive methods such as stool antigen test, serology, or urea breath test (UBT). Our study was approved in the ethic committee of Iran University of Medical Sciences (IUMS number: 1394–25804), Initially, the research plan was described for all participants and if they desired to participate in the study, demographic characteristics and systolic/diastolic blood pressure were recorded. Then, the patients' body weight and height were measured and the body mass index was calculated by dividing your weight in kilograms by your height squared in meters. After obtaining informed consent from the patients, venous blood samples were taken from all to assess the serum fasting levels of glucose, lipid profiles, C-Reactve Protein, and hemoglobin A1c. Then, the patients with positive *H.pylori* were treated with standard triple regimen (OAC) including omeprazole (20 mg, q12 h), amoxicillin (1 g, q12 h), and clarithromycin (500 mg, q12 h) for two weeks. Six weeks after treatment, a ^{14}C-urea breath test was performed for the re-evaluation of *H. pylori* eradication. Three months after completing treatment regimen, anthropometric and metabolic parameters were assessed in both groups of patients with successful and failed eradication treatment of *H. pylori*. The mood status with respect to depression and anxiety was also assessed using the beck inventory scaling method.

Results were presented as mean ± standard deviation (SD) for quantitative variables and were summarized by absolute frequencies and percentages for categorical variables. Normality of data was analyzed using the Kolmogorov-Smirnoff test. Categorical

Table 1
The change in anthropometric and metabolic parameters after HP eradication.

Parameters	Total Number = 91		Epigastric pain syndrome (Ulcer-like dyspepsia) (N = 38/91)		Postparendial distress syndrome (dismotility-like dyspepsia) (N = 44/91)		Overlap of both (N = 9/91)	
	3-month Difference	P-value	3-month Difference	P-value	3-month Difference	P-value	3-month Difference	P-value
Triglyceride	5.54 ± 37.59	0.163	13.49 ± 45.24	0.078	0.71 ± 31.13	0.879	−2 ± 29	0.0001*
Cholesterol	8.64 ± 25.02	<0.001*	7.67 ± 20.74	0.031*	0.71 ± 31.13	0.879	3 ± 25	0.0039*
HDL	−2.69 ± 6.37	<0.001*	−2.13 ± 7.29	0.840	10.40 ± 28.35	0.018*	0.1 ± 4.025	0.008*
LDL	6.23 ± 18.00	<0.001*	4.19 ± 15.95	0.118	−3.51 ± 5.89	<0.001*	0.1 ± 21	0.0001*
FBS	2.84 ± 7.81	<0.001*	2.54 ± 7.39	0.044*	9.34 ± 18.67	0.002*	−1 ± 9	0.0025*
HbA1C	0.12 ± 4.55	0.006*	0.16 ± 0.36	0.784	3.97 ± 7.58	<0.001*	−1 ± 0.3	0.0032*
Weight	0.15 ± 3.18	0.646	0.32 ± 2.12	0.359	0.21 ± 0.41	0.002*	0.0 ± 1	0.09
Systolic BP	0.40 ± 9.10	0.678	−0.69 ± 8.71	0.636	0.44 ± 4.07	0.942	−3 ± 7	0.003*
Waist cir.	0.76 ± 3.48	0.038*	1.54 ± 3.61	0.014*	0.53 ± 9.62	0.712	1 ± 4	0.004*
Diastolic BP	1.50 ± 8.46	0.96	1.25 ± 8.97	0.409	0.01 ± 2.97	0.999	2 ± 6	0.0001*
BMI	0.25 ± 1.22	0.841	0.11 ± 0.81	0.393	1.55 ± 8.58	0.231	0.023 ± 0.632	0.0032*

LDL; low-density lipoprotein, HDL; high-density lipoprotein, FBS; fasting blood sugar, HbA1C; hemoglobin a1 c,BP; blood pressure,Waist cir; waist circumference ,BMI; body mass index.
* P values of 0.05 or less were considered statistically significant.

Please cite this article in press as: M. Mokhtare, et al., The effects of *helicobacter pylori* eradication on modification of metabolic syndrome parameters in patients with functional dyspepsia, Diab Met Syndr: Clin Res Rev (2017), http://dx.doi.org/10.1016/j.dsx.2017.07.035