LETTERS TO THE EDITOR

The Influence of Helicobacter pylori Eradication on Soluble CD40 Ligand and Oxidative Stress: A Step Forward to Prevent Atherosclerosis

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To the Editor We have recently read the paper by Kebapcilar et al published in Internal Medicine about the influence of Helicobacter pylori (H. pylori) eradication on leptin, soluble CD40 ligand, oxidative stress and body composition in patients with peptic ulcer (1). Some points should be noted in appraisal of this study.

The title tells us that the study population consisted of patients with peptic ulcer disease, however no specific descriptions are included. Then, the authors concluded that H. pylori eradication improves ulcer healing but there was no discussion about the peptic ulcer status in study participants and improvement after eradication therapy. We wonder why the authors performed endoscopy for all of these patients with dyspepsia. The generally accepted guidelines for the approach in patients with dyspepsia without any alarming symptoms and signs, especially in those under 55 years old, is a trial of acid suppressing regimen or H. pylori eradication according to the prevalence of H. pylori infection in the general population (2). As reflected in the results, the mean age of patients was 32.9 years. Thus, it seems that endoscopy was not the first step in evaluation of these patients. Furthermore, using both histology and urea breath test for confirmation of infection is questionable.

Their results showed that the serum leptin levels were not significantly altered after eradication for H. pylori but the decline in fat mass was significant. As leptin is associated with fat stores, it is anticipated that diminished fat mass accompanies the decrease in the serum leptin level. Comments from the authors seem to be necessary to explain this paradox as it was one of the aims of this study.

Another point is the condition of fasting plasma glucose. In the abstract the authors stated that they measured fasting glucose before and after the eradication regimen but the desirity of plasma glucose is not clear in the main text or in the table.

Despite the above-mentioned problems, this study provides further evidence toward the association of H. pylori infection, insulin resistance and type II diabetes. Since the CD40/CD40 ligand pathway is involved in insulin resistance and type II diabetes (3), this can be targeted as a new area of research to clarify the association of H. pylori infection, insulin resistance and type II diabetes.

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References