Original Article

Association between helicobacter pylori infection and serum iron profile

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Received: 14 June 2011
Revised: 18 July 2011
Accepted: 2 Aug 2011

Abstract

Background: It is clear that Helicobacter pylori (H. pylori) is a spiral bacterium that infects human and causes gastritis and peptic ulcer. The aim of this study was to determine the relationship between H .pylori infection and the iron status of the body.

Methods: This study was conducted in Shahid Beheshti Hospital of Babol University of Medical Sciences, Babol, Iran from August 2007 to July 2008. The study group consisted of 35 patients with H. pylori and 35 matched healthy subjects as the control group. The members of both groups were enrolled in the study voluntarily. Serum iron and total iron-binding capacity were measured by Darman-kav Standard kit. Ferritin was measured by Padtan-e-Elm Standard kit. The Collected data were analyzed by SPSS version 16. p-value of <0.05 was considered significant.

Results: The serum iron and total iron-binding capacity in H. pylori positive group were lower than in the control group (108.67±31.26 vs. 110.92±28.45 µg/dL, p=0.578) and (327.88±81.39 vs. 342.51±79.45 µg/dL, p=0.153) respectively. The mean of ferritin was significantly lower in H.pylori positive group (210.51±132.01 ng/ml, p=0.047) than H.pylori negative group (265.03±170.79 ng/mL, p=0.047).

Conclusion: The results of the present study show that serum iron and total iron binding capacity in H.pylori infected group was lower than the control group. H.pylori may impair iron metabolism. It is possible that H.pylori may both directly compete with the host for available iron by impairing its uptake.

Key words: Helicobacter pylori, Serum iron, Total iron-binding capacity, Ferritin, Iron deficiency.

Caspian J Intern Med 2011; 2(3): 266-269

There are reports denoting presence of an association between Helicobacter pylori (H.pylori) infection and iron-deficiency (1). Iron deficiency and H. pylori infection is a common medical problem. A number of correlational studies have confirmed the association between iron deficiency and H. pylori infection. Some microorganisms need the host's iron to grow (2). It is now clear that human lactoferrin and heme supports full growth of H. pylori even in a medium with no other iron resources (3). According to several studies, the absorption of iron takes place in the duodenum and the proximal jejunum (4). It may be hypothesized that there is convincing evidence that iron deficiency has many negative effects on health (5). Recent studies also showed that H. pylori infection can contribute to iron deficiency anemia (6). According to several studies H.pylori requires iron to survive and may play an important role in unexplained iron deficiency anemia (7). Earlier studies have suggested that H. pylori has a negative effect on the absorption of iron (8). The gastric hypoacidity in H. pylori infected stomach also contributes to iron deficiency anemia by impairing iron uptake (9).

Literature review confirmed the hypothesis that H. pylori infection is known to be an etiological agent of chronic gastritis and peptic ulcer disease (10-12). On the basis of studies published to date, H.pylori gastritis can lead to a malabsorption of iron and should be considered after blood loss has been ruled out (13-16).

However, despite the importance of this medical problem, relatively little research has been done on the iron status of H. pylori infection. The present study was carried out to assess the iron status in patients with H. pylori infection, using laboratory measurements of iron indicators including serum iron, total iron binding capacity and serum ferritin.

Methods

This study was conducted in Shahid Beheshti Hospital of Babol University of Medical Sciences, Babol, Iran From August 2007 to July 2008, The approval of the Ethics Committee of Babol University of Medical Sciences was also obtained.

A total of 70 subjects, including 35 patients with H.pylori infection (20 males and 15 females), and 35 matched healthy controls subjects (19 males and 16 females) who voluntarily participated in the study were enrolled. No blood samples were drawn for the purpose of the study unless an informed consent form was signed.

Helicobacter pylori infection confirmation

Infection with H.pylori was confirmed by endoscopy and by histology of biopsy specimens obtained through endoscopy. Three specimens were taken from the gastric antrum. The specimens were fixed in 10% formalin and embedded in paraffin.

H.pylori infection was determined by Giemsa staining of sections and identified as rods on the surface of the epithelium, microscopically. Sources of the blood loss such as erosive esophagitis and gastritis, gastric and duodenal ulcers were not found on endoscopy.

Exclusion and inclusion criteria

The criteria for inclusion in this study were positive results of pathology and endoscopic results. All cases had diagnostic inclusion criteria including both endoscopy and pathological indices for H. pylori infection. The included subjects in the study were never smokers, had no history of systemic diseases, had never been under gastrointestinal treatment protocols and had taken no antibiotics, anti-inflammatory or any other drugs over the past 6 months. The exclusion criteria were treatment within 10 days prior to the study with drugs known effect on iron metabolism. Subjects with any history of GI surgery, peptic ulcer and diabetes were eliminated in the study.

Measurement of Iron parameters

Venous blood was taken in the morning after an overnight fasting. Blood was drawn into tubes containing EDTA and chilled on ice. Plasma was separated by centrifugation at 2000 rpm for 15 min at 4 ºC. Samples were shipped to the laboratory for biochemical assessments after the sample collection was completed. Plasma samples were immediately used for assay.

The indicators of iron status measured were serum iron, total iron binding capacity, and serum ferritin. Serum iron and total iron-binding capacity were measured by Darman-kav standard kit. Ferritin was measured by Padtan-e-Elm standard kit.

Statistics

The results were summarized as Mean±SD. The analyses were performed using the SPSS version 16. Whether or not, there was a difference between the groups, with regard to the normally distributed variable was examined by independent t-test. P-values less than 0.05 were taken as statistically significant.

Results

We assessed 70 subjects, 35 with H. pylori positive and 35 controls. The H.pylori positive group (20 men and 15 women with mean age 45.54±15.62 yr) and the control group (19 men and 16 women with mean age 44.94±14.88 yr). No significant differences were found between mean ages (p=0.384).

The results of biochemical studies revealed that serum iron and total iron-binding capacity in H. pylori positive group were lower than in the control group (108.67±31.26 vs. 110.92±28.45 µg/dl, p=0.578) and (327.88±81.39 vs. 342.51±79.45 µg/dl, p=0.153), respectively.

In contrast, the mean of ferritin was significantly lower in H. pylori positive group (210.51±132.01 ng/ml) than H. pylori negative group (265.03±170.79 ng/ml, p=0.047), as shown in figure 1.

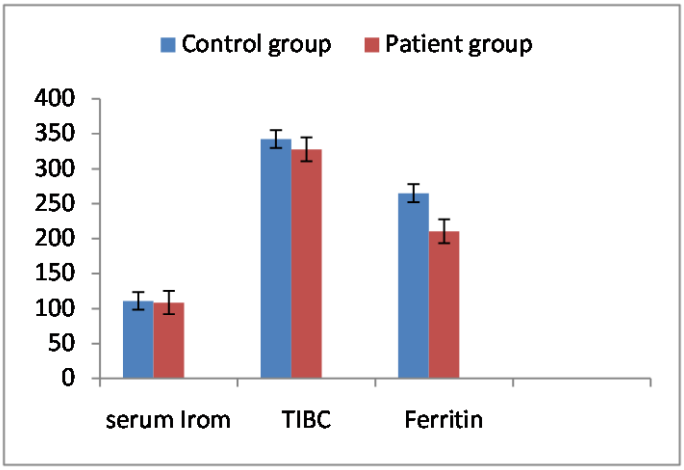

Figure 1. (Part I) Serum iron level in the control group and helicobacter pylori positive group. Each column represents Mean±SD of serum iron (µg/dl). (Part II) Total iron-binding capacity in the control group and helicobacter pylori positive group. Each column represents Mean±SD of total iron binding capacity (µg/dl). (Part III) The mean of serum ferritin in the control group and helicobacter pylori positive group. Each column represents Mean±SD of serum ferritin (ng/ml). (In each part column 1; control group, column 2; patient group).

Discussion

The mechanism by which infection with H. pylori causes iron metabolism disturbance is of considerable interest. However, despite the importance of this, relatively little research has been done yet. In the present study, we found that the mean of ferritin was significantly lower in H. pylori positive group than H. pylori negative group. These data confirm an earlier study (1, 6). Our data indicated that serum iron and total iron-binding capacity in H. pylori positive group were lower than in the control group. This observation was in agreement with the results obtained by other investigators (6, 7). The results suggested that H. pylori infection impaired iron uptake. This role is maybe related to directly competing with the host for available iron or by impairing iron uptake.

In general, several possible mechanisms for the association between anemia and H. pylori infection must be considered in understanding its details. Intermittent bleeding may be present. There is an attractive alternative hypothesis that it is possible that H. pylori may act as an iron-acquisition mechanism in vivo, forming a parasitic relationship to compete with the host for iron. It is now clear that H. pylori infection may progress into diffuse corpus gastritis. These conditions may play an important role in gastric hypoacidity.

On the other hand, as high gastric acidity facilitates the solubilization of non-heme iron, iron uptake may be impaired in subjects with H. pylori infection due to loss of iron. The mechanisms by which H. pylori infection may lead to iron metabolism disorder remains obscure and further work is necessary to clarify this issue.

Acknowledgments

This study was supported by the Research Council of Babol University of Medical Sciences (Gr 1045). We gratefully thank the assistance of our colleagues in our hospital.

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