

Several mechanisms have been hypothesized to explain the possible effect of *H. pylori* infection on iron stores. A more likely mechanism is decreased iron absorption from hypo- or achlorhydria resulting from chronic gastritis⁽²⁹⁾. Gastric hydrochloric acid facilitates iron absorption by reducing non-heme iron from the ferric to ferrous form. Another important effect of *H. pylori* gastritis that may cause reduced iron absorption is a decrease in gastric juice ascorbic acid concentration. Ascorbic acid facilitates iron absorption by reducing iron to the ferrous form⁽³⁰⁾. Ascorbic acid is secreted into gastric juice, and it has been shown that gastric juice ascorbic acid levels are significantly lower in *H. pylori* -infected vs. uninfected persons^(31,32), another mechanism to explain decreased iron absorption associated with *H. pylori* infection is increased hepcidin production from hepatocytes in response to IL-6 production associated with *H. pylori* gastritis⁽³³⁾. Another possible mechanism by which *H. pylori* could result in decreased availability of iron is sequestration of iron in lactoferrin in the gastric mucosa. *H. pylori* takes up iron from human lactoferrin through a receptor-mediated method^(34, 35), and lactoferrin secretion in the gastric mucosa appears to be influenced by the *H. pylori* organism^(36, 37).

Another hypothesized mechanism to explain an association between *H. pylori* infection and iron deficiency is uptake of iron by the *H. pylori* organism. Like many bacteria, *H. pylori* require iron as a growth factor, and it possesses a 19-kDa iron-binding protein resembling ferritin (Pfr), that may play a role in storage of excessive iron by the bacteria⁽³⁸⁾. Acquisition and storage of iron in *H. pylori* are controlled by the ferric uptake

regulator gene product (Fur), which regulates transcription of iron uptake genes and Pfr iron storage⁽³⁹⁾.

Scientists have long known of *H. pylori*, but only in the last 10 years it has been recognized as a potential health threat. It causes stomach ulcers and gastrointestinal cancer and may play a role in the incidence of many other diseases.

Materials and Methods

Patients:

A total of 64 patients (41 females and 23 males), aged between 14 and 66 years, were screened for this study. Patients attended the Gastroenterology Unit at AL-Kadhimiya teaching hospital in Baghdad from 1st April to October 2007 because of recurrent abdominal pain and other gastrointestinal complaints, such as vomiting. All subjects filled out a questionnaire with regard to their general health and were excluded if they had been previously treated for *H. pylori* infection. The study was approved by the ethics committee of the Hospital. After an overnight fast, each patient underwent esophagogastroduodenoscopy, during which four antral biopsies were taken from within 2 cm of the pylorus using sterilized biopsy forceps (Olympus 16K; Olympus Corp., Tokyo, Japan). Biopsy specimens for the urease test were taken before those used for histological examination to avoid contamination with formalin.

Ultra rapid urease test:

Each specimen was subjected to Ultra Rapid Urease test as mentioned by Berry V, Sagar V⁽⁴⁰⁾ but with some modification. Briefly the medium used for the test was urea broth. It consists of urea, phenol red indicator and distilled water. 10 gm of urea was dissolved in