Helicobacter pylori and Iron Deficiency Anemia

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Since Helicobacter pylori was first isolated by Warren and Marshall in 1983 (1), there has been general agreement that the bacterium is closely associated with gastroduodenal disorders including gastritis, peptic ulceration, and gastric cancer (2). Furthermore, it has recently been shown that H. pylori infection is associated with several extragastric disorders. These include chronic urticaria (3), idiopathic thrombocytopenia (4), iron deficiency anemia (5, 6), ischemic heart disease (7), and Guillain-Barre syndrome (8). The association between H. pylori and these extragastric disorders is supported by the finding that they resolve on eradication of H. pylori. Although the basic mechanisms by which H. pylori infection in the stomach mediates the above-mentioned disorders is unclear, it has been suggested that they result from the inflammatory process elicited by H. pylori in the stomach and the subsequent systemic consequences.

The most common cause of iron deficiency anemia (IDA) is insufficient iron intake, blood loss from the intestinal tract, poor absorption, and malabsorption or diversion of iron in the reticulo-endothelial system. Moreover, H. pylori infection as a cause of refractory IDA has been reported by Dufour et al (5): Based on findings that recovery from IDA can occur after H. pylori eradication therapy and without supplemental iron, it has been suggested that this bacterium actually blocks iron metabolism. Since this initial report, the several supplementary trials by clinical investigation have been performed and the relation between H. pylori infection and IDA has been confirmed (9, 10). In a double-blind controlled trial, H. pylori eradication in 10 to 17-year-olds with IDA resulted in a significant increase in serum hemoglobin level at eight weeks post-therapy, compared with a control group (individuals with IDA and not receiving H. pylori eradication therapy) (11). In the control group, there was no change in the hematologic profile and iron stores, and anemia was refractory to the iron treatment. The result of the study clearly demonstrated that there exists a subset of patients in which H. pylori infection plays a role in the disease pathogenesis and where eradication therapy can be an effective treatment.

There are several hypotheses as to how H. pylori may block iron absorption. In those individuals with H. pylori-related gastritis, both a low level of acid secretion, as well as a low ascorbic acid level in gastric juice has been observed (12). Moreover, H. pylori eradication reverses gastric acid secretion and the ascorbic acid level. This probably explains at least partly the reduction in iron absorption associated with H. pylori infection (13).

Like many other microorganisms, H. pylori requires iron for growth. H. pylori infection of the stomach raises the local level of lactoferrin (LF), an iron-binding glycoprotein that captures iron from transferrin. This thus permits H. pylori to increase iron uptake. Since the turnover of H. pylori is very rapid, iron stores within the bacterium tend to be lost rapidly from the host (14–16).

Currently, the diversion of iron to H. pylori residing in the gastric mucosa seems the most biologically plausible explanation for H. pylori’s role in IDA (16). However, it does not explain why IDA develops in only some H. pylori-infected individuals. It is feasible that the variation in response of individuals to H. pylori may be the result in variation between infecting H. pylori strains and/or variation in the host’s requirement for iron. For example, in childhood where the iron-balance is potentially negative, H. pylori may be more likely to play a role in the onset of IDA.

See also p 971.

Results of early studies and case reports indicating an association between H. pylori infection and IDA were based mainly on data from children and young adults, and they suggested the existence of an underlying specific ferrokinetic state during this period of life. In contrast, case reports and studies focusing on H. pylori infection and IDA in adults are now being increasingly seen (9, 17–19). For example, in a recent clinical seroepidemiological study, it was reported that serum ferritin levels were lower in anti-H. pylori antibody-positive adults (19). In conclusion, it is highly probable that the association between H. pylori infection and iron deficiency is a more common condition than previously thought (16). Further studies are required to determine the mechanism by which H. pylori-induced gastritis is associated with IDA.

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References