The long-term efficacy of Helicobacter pylori eradication therapy in patients with idiopathic thrombocytic purpura

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*Helicobacter pylori* (H. pylori) is a gram negative, spiral bacillus that colonizes the human gastric mucosa. *H. pylori* has been recognized as the pathogenic agent of gastropathies such as chronic gastritis, gastro duodenal ulcers, gastric adenocarcinoma, and mucosa-associated lymphoid tissue lymphoma. The eradication therapy for *H. pylori* successfully restrains the recurrence of a peptic ulcer; furthermore, the possibility of preventing the development of gastric carcinoma has been suggested. Several studies have proposed that *H. pylori* infection may be associated with extra gastric diseases such as iron deficiency anemia, chronic idiopathic urticaria, and idiopathic thrombocytic purpura (ITP). Several investigators have shown that the eradication of *H. pylori* is often accompanied by a significant increase in platelet count in patients with *H. pylori*-positive ITP. Gasbarrini *et al.* speculated that cross-mimicry between platelets and *H. pylori* antigens, or chronic immunological stimulus caused by *H. pylori* infection, might be involved in the destruction of platelets in patients with *H. pylori*-positive ITP. Since most of these reports involved a small number of cases and had a short follow-up period, it was not clear whether the recovered platelet count after *H. pylori* eradication was maintained for a long period.

In this study, they followed ITP patients, in whom *H. pylori* was successfully eradicated, for more than 2 years in Japan and investigated the long-term effect of *H. pylori* eradication on platelet recovery in patients with *H. pylori*-positive ITP. *H. pylori* infection was assessed by the 13C urea breath test (UBT) or rapid urease test. The *H. pylori*-positive patients received standard antibiotic therapy for *H. pylori* eradication (750 mg of amoxicillin, 400 mg of clarithromycin, and 30 mg of lansoprazole, twice daily) for 1 week. Eradication was assessed by UBT more than 4 weeks after the eradication therapy. Standard therapies, including corticosteroids, splenectomy, intravenous gamma globulin, and immunosuppressive chemotherapy have been administered to patients with severe thrombocytopenia (platelet count less than $20 \times 10^9/L$) and bleeding symptoms. It was found that 20%-30% of ITP patients were refractory to current therapies. The platelet counts of *H. pylori*-positive ITP patients were examined 1 and 6 months after *H. pylori* eradication and at least once a year thereafter. They evaluated the effect of *H. pylori* eradication on platelet recovery within 6 months after the therapy.
complete response (CR) was defined as platelet counts greater than 120 × 10^9/L. A partial response (PR) was defined as platelet counts between 50 × 10^9/L and 120 × 10^9/L, or an incremental increase of 30 × 10^9/L from baseline platelet counts. No response (NR) was defined as no increase in the platelet count or an increase of less than 50 × 10^9/L. In this study, the patients showing CR and PR were defined as responders to *H. pylori* eradication. Veneri *et al.* analyzed the host factor that contributed to the different responses to eradication therapy in ITP patients. They observed that HLA-DQB1*03* was more frequently detected in responders than in non-responders. They suggested the HLA class II allele pattern as a possible prognostic factor for predicting the effect of *H. pylori* eradication. By studying the factors of both the bacteria and host, it may be possible to select an adequate treatment for ITP patients. The long-term use of corticosteroids has several side-effects, and splenectomy is an invasive therapy. On the other hand, no serious side effects were observed in patients that received the eradication therapy. It might be possible to select *H. pylori* eradication as the first-line therapy for treating patients with *H. pylori*-positive ITP.

In conclusion, eradication therapy for *H. pylori*-positive patients with ITP was effective in more than half of patients, with a favorable effect maintained for long periods. ITP could be classified into at least three subgroups: *H. pylori*-dependent ITP, *H. pylori*-positive but independent ITP, and *H. pylori*-negative ITP. Further studies are required to clarify other etiologic factors involved in autoimmune response to platelets to establish a new therapy for *H. pylori*-independent ITP.

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