Thirty-seven patients with idiopathic thrombocytopenic purpura (ITP) were treated with a standard Helicobacter pylori eradication regimen irrespective of H. pylori infection. Our results indicate that platelet recovery results from the disappearance of H. pylori itself, and is mediated, in part, through suppression of anti-platelet autoantibody production.

Effects of a Helicobacter pylori eradication regimen on anti-platelet autoantibody response in infected and uninfected patients with idiopathic thrombocytopenic purpura

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It has been proposed that Helicobacter pylori (H. pylori) infection is associated with idiopathic thrombocytopenic purpura (ITP), based on increased platelet counts after successful eradication of H. pylori. However, the prevalence of H. pylori infection is similar in ITP patients and the general population, suggesting that ITP is not necessarily linked to H. pylori infection. A critical question is how the H. pylori eradication regimen increases platelet counts. The most plausible explanation is that the disappearance of H. pylori has a therapeutic effect. However, a 1-week regimen of a combination of antibiotics should eradicate bacteria other than H. pylori, which may include a bacterium truly associated with the pathogenesis of ITP, or induce prominent changes in the normal flora. Moreover, immunomodulatory effects are reported for drugs used for H. pylori eradication. To evaluate these possibilities, we conducted an open-label, prospective study involving 37 consecutive patients with ITP (aged 24 to 73, 14 male) who satisfied the following criteria: thrombocytopenia persisting >6 months, normal or increased bone marrow megakaryocytes without morphologic evidence of dysplasia, no secondary immune or non-immune diseases that could account for thrombocytopenia, and a platelet count <50×10^9/L at ≥3 measurements during the preceding 3 months. None of the patients satisfied the classification criteria for systemic lupus erythematosus, but 13 (35%) had a low titer (≤80) of anti-nuclear antibodies. All patients were assessed for H. pylori infection and given amoxicillin (1.5g daily), clarithromycin (800 mg daily), and lansoprazole (60 mg daily) for 7 days, irrespective whether they did or did not have H. pylori infection.

All patients visited our hospital at 0, 1, 4, 8, 12, and 24 weeks, and all responders were followed for ≥56 weeks. The anti-GP IIb/IIIa autoantibody response was evaluated by detecting circulating B cells producing anti-GP IIb/IIIa antibodies. The patients were allowed to continue other therapy (danazol, n=4), provided their dosages were maintained at a constant level until 24 weeks, except for prednisolone (≥10mg daily), which was allowed to be decreased or discontinued after platelet counts reached >100×10^9/L.

The study protocol conformed to the ethical principles of the World Medical Association Declaration of Helsinki as reflected in a priori approval from the Institutional Review Board. Twenty-six patients (70%) who had positive results in a 13°C urea breath test plus serum anti-H. pylori antibodies or stool H. pylori antigen were regarded as H. pylori-positive, whereas 11 patients negative for all three tests were H. pylori-negative. Eradication was successful in all H. pylori-positive patients according to a negative urea breath test at 12 weeks. When a therapeutic response was defined as a platelet count >100×10^9/L at 24 weeks, 16 H. pylori-positive patients (62%) were responders, while none of the H. pylori-
Our study clearly showed it is likely that platelet recovery results from the eradication itself, rather than from other mechanisms, potentially similar to the actions of IVIG. These different actions of *H. pylori* eradication suggest that multiple processes are responsible for platelet recovery in ITP patients.

**References**


