Helicobacter pylori infection and idiopathic thrombocytopenic purpura: description of 21 newly diagnosed cases

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From the first observation of Gasbarrini et colleagues in 1998,1 many other studies have documented the association between Helicobacter pylori infection and idiopathic thrombocytopenic purpura (ITP) and a significant increase of platelet count after bacterial eradication,2-6 but other authors disagreed with these findings.7-9 In a recent review on this topic,10 we found that bacterium eradication was associated with a complete or partial platelet response in 55% of the cases so far reported in the literature. However, most of these studies have a short follow-up period and regard patients previously treated or under immunosuppressive treatment when anti-*H. pylori* therapy was started. Although an adequate follow-up is crucial in order to evaluate the efficacy of any treatment for ITP, given the high percentage of relapses of this disease, the absence of concomitant therapies which may confound the results of bacterial eradication is, in our opinion, equally important. In fact, in our previous study with 16 adult ITP patients with documented H. pylori infection, we observed the best results, in terms of platelet increase, in the group of patients who had never been treated with immunosuppressive therapy.[6]

To confirm our previous observation we have evaluated the efficacy of eradication therapy in 21 consecutive, newly diagnosed, *H. pylori* positive ITP patients (11 males and 10 females, median age 55.1 years [range 25-73 years]). We followed these patients between January 2000 and July 2003 (median follow up 18.1 months, range 10-42 months) and we never administered immunosuppressive drugs. H. pylori infection was assessed by urea breath test and HIV, HCV positivity or other underlying autoimmune diseases were excluded. The patients received eradication therapy as previously described.6 As shown in Figure 1, *H. pylori* was eradicated in 19 patients (90.5%) and a complete (CR) or partial (PR) platelet response was observed in 9 of them (47.4%). The mean platelet count before and after eradication treatment was 57.3±28.1×10^9/L and 104.6±37.4×10^9/L, respectively (p< .01, t test). None of the two patients who were still H. pylori positive after the treatment had a significant platelet count increase during the follow-up. Interestingly, we observed a further platelet increase, although statistically not significant, in eradicated patients during the follow-up period. After the follow-up, the mean platelet count raised up to 119.8±40.3×10^9/L and the rate of response up to 73.7% (14 CR/PR). This finding is in contrast with that observed after ITP steroid treatment and may be due to the different mechanisms of action of these two treatments.

Idiopathic thrombocytopenic purpura is a heterogeneous condition with a great variability of laboratory and clinical presentations. In our opinion, these differences may reflect different pathogenetic mechanisms which could finally explain the discrepancy in response rates reported in the studies so far published.

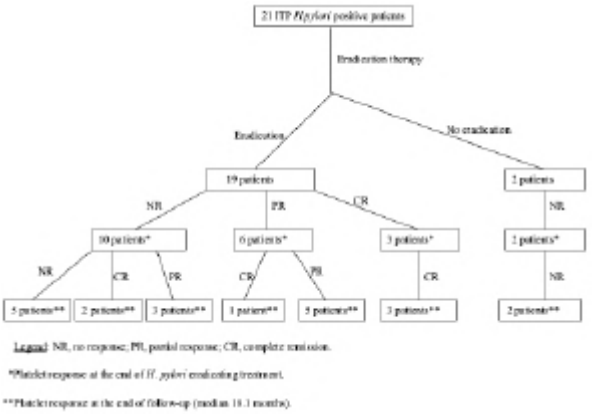

Figure 1. Platelet response to eradication therapy in 21 ITP H.pylori positive patients.

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