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Case Report

Success of *Helicobacter pylori* Eradication Therapy and Long Lasting Platelet Recovery in an Iranian Patient with Severe Immune Thrombocytopenia

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ABSTRACT

Background: *Helicobacter pylori* is a gram-negative spirochete that usually affects the gastric mucosa and has a prevalence of more than 50% of people worldwide. It has been revealed that *Helicobacter pylori* infection is not only associated with chronic gastritis and peptic ulcers, but also with some hematologic disorders such as immune thrombocytopenia, iron deficiency anemia and gastric mucosa-associated lymphoid tissue lymphoma. Immune thrombocytopenia is an acquired autoimmune disorder with low platelet count due to devastation of autoantibody-coated platelets.

Case report: In the present study, it was reported an Iranian patient with severe immune thrombocytopenia (platelet count 1×10^9/L) and *Helicobacter pylori* infection who achieved a long lasting platelet recovery after *Helicobacter pylori* eradication therapy. The patient was followed up for two years and no failure in platelet response was observed.

Conclusion: Accordingly, *Helicobacter pylori* treatment is not only useful in immune thrombocytopenia patients with mild and moderate thrombocytopenia but also may be beneficial in severe cases of immune thrombocytopenia.

Keywords: *Helicobacter pylori*, Purpura, Thrombocytopenic, Idiopathic, Blood Platelets

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Introduction

Helicobacter pylori (*H. pylori*) is a gram-negative spirochete that usually affects the gastric mucosa. The prevalence of *H. pylori* infection is more than 50% of people worldwide; however, it is more prevalent in undeveloped countries (1). The role of *H. pylori* in development of several diseases including chronic gastritis, peptic ulcers, and gastric adenocarcinoma is well-established. According to the studies, *H. pylori* infection is also associated with some hematologic disorders such as immune thrombocytopenia (ITP), iron deficiency anemia, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma (2,3). The association of ITP and *H. pylori* infection has been proposed by a large number of studies. Immune thrombocytopenia is an acquired autoimmune disorder which is characterized by a low platelet count (less than 100×10^9/L). The disorder stems from devastation of autoantibody-coated platelets and is usually accompanied by mucocutaneous bleeding diathesis (4). It has been reported that eradication therapy of *H. pylori* infection in ITP patients may be accompanied by platelet recovery, however, studies showed contradicting findings in this regard.

Previous studies are mainly from Japan and Italy and data on other countries are limited. Based on the two studies conducted by Payandeh et al. (2012 & 2013) on Iranian population, it was revealed that platelet count response following *H. pylori* treatment is mainly observed in ITP patients with mild thrombocytopenia and if the thrombocytopenia is severe, the platelet response is less probable (5,6). Conversely, here, an Iranian patient with severe ITP (platelet count 1×10^9/L) and *H. pylori* infection who showed a long lasting increment in platelet count after *Helicobacter pylori* eradication therapy was reported. The patient was followed up for two years after eradication therapy and no failure in platelet recovery was observed.

Case report

A 37-year-old woman was referred to Imam Khomeini Hospital of Tehran. She was represented with severe thrombocytopenia with platelet count of 1×10^9/L. She was manifested by mucocutaneous bleeding events and petechiae on the lower limbs. The patient had no history of taking medications that cause thrombocytopenia. Complete blood count (CBC) showed normal RBC and WBC count with normal morphology in peripheral blood smear. Laboratory investigations revealed that autoimmune tests including antinuclear antibody (ANA) and anti-double stranded DNA (anti-dsDNA) were negative. Lactate dehydrogenase (LDH) was in normal range and indirect antiglobulin test (IAT; indirect coombs) was negative. Liver function tests, thyroid function test, and hepatitis B, C and HIV were all normal.

Eventually the patient was diagnosed with ITP and received methylprednisolone (500 mg for 3 days) and intravenous immunoglobulin (IvIg) with a dose of 0.5 g/kg body weight/day (29 vials, in 6 days). Three days after the treatment, a CBC was performed and showed that the platelet count reached 60×10^9/L, and with further IVIG vials administration, platelet count reached 82×10^9/L. The patient was also assessed for *H. pylori* infection. It was demonstrated that anti *H. pylori* antibodies including both IgG and IgM were positive. *H. pylori* antigen in stool was positive by enzyme-linked immunosorbent assay (ELISA). Cytotoxin-associated gene A (CagA) antigen was also positive. The patient was treated for *H. pylori* (Clarithromycin 500 mg bd, pantoprazole 40 mg daily and bismuth bd) and simultaneously corticosteroids were administered. Two months after anti *H. pylori* therapy, stool ELISA was negative for *H. pylori* antigen. During *H. pylori* eradication, the platelet count was assessed each month and the results showed an increase in platelet number, which eventually reached 200×10^9/L after six months. The patient was followed up for two years and no failure in platelet recovery was observed.

Discussion

The mechanism by which *H. pylori* treatment results in platelet recovery in ITP patients is not clearly known. However, there are some theories that can explain this association. The first one refers to the production of antibodies against some antigens of *H. pylori* bacterium which also cross react against platelet antigens, a phenomenon called molecular mimicry. Some potential targets of these antibodies are CagA protein and Lewis antigens which are present on specific strains of *H. pylori*. There is also another theory which proposes the causative role of molecular mimicry of such antigens and platelet specific antigens in inducing the onset of ITP. Increased destruction of platelets in an antibody-dependent manner has been also reported in the

concomitant presence of bacterial agents and antiplatelet antibodies (7).

Generally, about 50% of adult ITP patients with *H. pylori* infection show platelet responses (8). Although there are several factors which may predict the patients' response to eradication therapy, but none is a distinct predictor. There are variable data regarding platelet responsiveness from different countries. According to the literature, platelet responsiveness is mainly observed in patients from Japan and Italy where the prevalence of infection in adult ITP patients is almost 70% and 50%, respectively (7). The severity of ITP and its duration are other effective factors. Patients with a platelet count below 30×10^9/L are less likely to reach a platelet response and patients with recent onset of ITP have a higher chance for platelet recovery compared with those with a chronic severe disease (9). In contrast, here, a case of severe ITP with a platelet count of 1×10^9/L at baseline who achieved a platelet response after eradication therapy was reported.

The variety of patients' outcome may be also explained by type of the causative strain and genetic background of the patient. It has been proposed that patients with HLA-DQB1*03 have a higher chance of response following treatment for *H. pylori* (8).

Despite all the variation in the patients' response, *H. pylori* eradication therapy is considered as a beneficial treatment in ITP patients with positive H. Pylori infection. In responders, the use of immunosuppressive drugs will be diminished significantly, and in some patients, splenectomy is no longer required. Therefore, routine screening of *H. pylori* for ITP patients has been suggested by several studies.

Conflict of interests

None.

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