RELEVANCE BETWEEN HELICOBACTER PYLORI INFECTION AND THROMBOCYTOPENIA

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ABSTRACT
Idiopathic thrombocytopenic purpura (ITP), also known as primary immune thrombocytopenic purpura and autoimmune thrombocytopenic purpura, is defined as isolated thrombocytopenia with normal bone marrow and the absence of other causes of thrombocytopenia. ITP is a bleeding condition in which the blood doesn't clot as it should. This is due to a low number of blood cell fragments called platelets. It is important to maintain an adequate platelet count in patients with ITP in order to avoid hemorrhagic during surgery. The association between helicobacter pylori infection and the effect of it were observed in an ITP patient over 6 months. Presence of Helicobacter pylori indicated to the increased platelet count and provides a new insight for a non-immunosuppressive treatment in selective ITP patients.

Key words: Helicobacter pylori, Platelet, Idiopathic thrombocytopenic purpura, primary immune thrombocytopenic purpura.

1. INTRODUCTION
The idiopathic thrombocytopenic purpura (ITP), first described by P.G. Werlhof [1], with characterized by premature destruction of autoantibody-coated platelets causing purpura, epistaxis, gingival bleeding, menorrhagia, and easy bruising. GI bleeding, hematuria, retinal hemorrhage, and even intracranial hemorrhage. Several studies showed some idiopathic thrombocytopenic purpura (ITP) patients with complete platelet recovery after Helicobacter pylori extermiate and cited the cause of persistent thrombocytopenia as inability to exterminate Helicobacter pylori. In this paper, we report on our efforts to take control of the helicobacter pylori infection in a case of 33 years old male with ITP.

2. CASE REPORT
A 33 year old male was referred to the Department of Surgery, with untreated chronic ITP, he suffered from bleeding gums and Gingival enlargement with the appearance of generalized purple spots all over the body. It was not associated with fever but had one episode per day of severe abdominal pain with vomiting. Retrospection of his past history revealed similar kind of attacks in the past 4 years and this was his second episode. An upper gastrointestinal endoscopy showed fundal and corpus hemorrhagic gastritis and biopsy test for the presence of H. pylori infection confirmed active infection by mucosal polymerase chain reaction (PCR). Other parameters of the patient upon investigation on the first day showed blood pressure of 80/60mm Hg with a platelet count of 30 x 10³/mm³ and hemoglobin was found to be 2g%. The patient was conservatively managed by giving 2 units of packed cells followed by 2 units of platelet with plasma for 3 days, due to the positivity to H. pylori infection, the patient was kept on pantazol (40mg, once daily), ampicilin (500mg) & sanmox (1.2g, 8 hrly) with baclecin (500mg, twice daily) for minimum 7 days. The patient was discharged on the eleventh day with a platelet count of 80 x 10³/mm³, hemoglobin 5.8 g%, blood pressure was 120/900mm Hg. The patient continued to take pantazol (40mg, once daily) for 5 weeks. The patient continued to take steroids for a period of 35 days in tapered doses (once the alternate day) and discontinued after 35 days. The platelet count of the patient was monitored every 12 days and was found to be normal after completion of six weeks of PPI intake. Endoscopy was performed again at the end of the treatment, which showed reduction in the fundal gastritis and absence of Helicobacter pylori infection by PCR. A control endoscopy performed along with platelet count after 12 weeks of the eradication therapy showed normal fundus with no evidence of ulceration in any part of the stomach, and platelet count was in the normal range.
3. DISCUSSION & CONCLUSION
Helicobacter pylori is a microaerophilic, Gram-negative, spiral-shaped, flagellated bacterium that colonises the mucous layer of the human stomach [2]. Several lines of direct and indirect evidence suggest that infectious agents may influence the occurrence or the course of some autoimmune disease [3], the relationship between H. pylori infection and idiopathic thrombocytopenic purpura (ITP) has been investigated since 1998, when an Italian group reported a significant increase of the platelet count in eight of the 11 ITP patients in whom the bacterium was eradicated [4]. Emilia, et al [5] and Gasbarrini, et al [6] both in Italy. Reported that 43 % and 61 % respectively of a total of 48 AITP cases were infected with Helicobacter pylori whereas the prevalence of Helicobacter pylori in the general population in Italy is 45% [7]. others have reported neither an increased incidence of H. pylori in patients with ITP nor improvement in platelet counts after eradication [8]. In addition, both H. pylori infection and ITP are associated with a T helper 1 type immune response characterized by increased levels of interferon-γ and interleukin-2; hence, H. pylori associated increase of the cytokine profiles may contribute to the development of immune thrombocytopenia. In addition to this, few studies have also demonstrated a temporal association between the disappearance of anti-CagA antibodies in the serum and improvement of ITP [9]. The reason for these inharmonious results is uncertain but may reflect the results of studying diverse patient populations, failing to control for administration of concomitant therapies, or variable effects of genetically diverse H. pylori strains in remote geographic regions. Additionally, our study demonstrated H. pylori infection using PCR in contrast to other studies which had used serological assays or histopathological tests to confirm this infection. Initial treatment of the patient with steroids was mandatory so as to rescue the patient from the critical condition and should not be a limitation of the report, as later it is seen that patient platelets did not decrease at tapered doses and even after stopping it completely. The data so far reported indicate that the prevalence of H. pylori infection in ITP mirrors the prevalence of H. pylori infection in the general population, although eradication of H. pylori infection led to a good platelet response in our chronic ITP patient. It is also important to initiate studies in broad number of subjects from different geographical areas with probably longer follow-up simultaneously, so as to evaluate the molecular pathway that links H. pylori with ITP.

4. REFERENCES