Ulcerative Colitis Accompanied by Idiopathic Thrombocytopenic Purpura and *Helicobacter pylori* Infection

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Abstract

The coexistence of idiopathic thrombocytopenic purpura (ITP) and active ulcerative colitis (UC) has been reported. We herein report a rare case of UC accompanied by ITP and *Helicobacter pylori* (*H. pylori*) infection. A female UC patient was diagnosed with ITP. At that time, the UC was almost in remission and we suspected that the ITP was caused by some factor other than UC. Accordingly, we found *H. pylori* infection and administered *H. pylori* eradication therapy. Consequently, the patient’s serum platelet count recovered dramatically. Our report demonstrates the importance of conducting examinations for *H. pylori* infection in ITP patients, even those with UC.

Key words: ulcerative colitis, idiopathic thrombocytopenic purpura, inflammatory bowel disease, *Helicobacter pylori*

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Introduction

Idiopathic thrombocytopenic purpura (ITP), also known as primary immune thrombocytopenia, is a destructive thrombocytopenia caused by autoantibodies against platelets membrane antigens (1, 2). ITP often occurs secondary to other diseases, including autoimmune conditions (3). Ulcerative colitis (UC) is an intractable chronic inflammatory bowel disease of unknown etiology that can affect the entire colon. The coexistence of ITP and UC has also been reported, and ITP may therefore represent an immunologically-mediated extraintestinal manifestation of UC (3-9). On the other hand, Gasbarrini et al. (10) first showed that a high prevalence of *Helicobacter pylori* (*H. pylori*) infection is observed in ITP patients and that platelet recovery occurs following the administration of *H. pylori* eradication therapy in most cases. A recent meta-analysis comprising 788 ITP patients showed a strong association between *H. pylori* eradication and increased platelet counts (11). We herein report a rare case of UC accompanied by ITP and *H. pylori* infection.

Case Report

A 41-year-old woman had been diagnosed with proctitis type UC in 1999. Since that time, she had been treated with mesalamine and sometimes complained of mild mucous hematochezia. An endoscopic examination conducted in September 2009 showed mild proctitis (Fig. 1A). The results of hematological tests performed in August 2009 were normal, namely, the blood red cells count was 514×10⁴/μL, the white blood cells count was 7,500/μL and the platelet count was 24.5×10⁴/μL. However, a hematological examination conducted in February 2010 showed a significant decrease in the platelet count (6.4×10⁴/μL) (Fig. 2) with normal blood red cells count (479×10⁴/μL) and white blood cell (7,500/μL) counts. The patient’s coagulation parameters, including the prothrombin time (PT), activated partial thromboplastin time (APTT), antithrombin, the fibrinogen level and D-dimer, were all normal. The laboratory data for the patient’s liver

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and renal functions were also normal. However, the serum platelet-associated IgG (PA-IgG) level was elevated (107 pg/mL (normal range: 0-46)). A physical examination of the patient revealed no splenomegaly. We first suspected a diagnosis of drug-related thrombocytopenia and stopped the administration of mesalamine. However, discontinuing the mesalamine did not improve the patient’s platelet count. After consulting with a hematologist, the patient was diagnosed with ITP on the basis of the criteria constructed by the International Working Group for ITP (1), namely, no history of any autoimmune/immunodeficiency disorders, liver disease, drugs for thrombocytopenia, recent transfusions or inherited thrombocytopenia, normal physical examination findings without splenomegaly, a platelet count <10×10⁴/μL, normal counts of blood red cells and white blood cells and no abnormalities on a peripheral blood smear suggestive of other etiologies for thrombocytopenia. Since the International criteria for ITP (1) demonstrate that conducting a bone marrow aspiration examination is appropriate for establishing the diagnosis in patients over 60 years of age and in patients considering splenectomy, we did not perform a bone marrow examination in the current patient. Other etiologies for secondary thrombocytopenia, including viral infections, disseminated intravascular coagulation, hemolytic uremic syndrome and lymphoproliferative disorders, were ruled out in the present case. As a result, we considered that the ITP was most likely caused by the UC. However, no exacerbations of UC were observed at that time, and the patient’s serum C-reactive protein (CRP) level (0.02 mg/dL) was not elevated (Fig. 2). As previous reports have shown that ITP tends to occur in UC patients with extensive and significant colonic inflammation (5), we suspected that this patient’s ITP was caused by some factor other than UC. Accordingly, we performed a urea breath test, which revealed H. pylori infection, and gastroduodenal endoscopy, which demonstrated atrophic gastritis (Fig. 1B). Based on these findings, we determined that the ITP was most likely caused by H. pylori infection and initiated H. pylori eradication therapy with amoxicillin, clarithromycin and lansoprazole. Consequently, the patient’s H. pylori infection disappeared, and her serum platelet count recovered dramatically (Fig. 2). To date, the patient’s platelet count has remained within the normal range.

**Discussion**

Since Edwards and Truelove originally reported three patients with both ITP and UC among 624 patients with UC in 1964 (4), the coexistence of ITP and UC has continued to be reported. Almost always, UC coincides with or precedes the onset of ITP by days or years (5). ITP has been suggested to be causally associated with UC, most likely as a
result of immunostimulation by luminal antigens and altered immunoregulation (6). As described above, ITP tends to occur in UC patients with extensive and significant colonic inflammation (5), and thrombocytopenia has been frequently observed during flare-ups of colitis (3). Both diseases respond well to steroid treatment in approximately half of affected patients; however, other cases require splenectomy or colectomy (5, 7). Concerning the pathogenesis of H. pylori-induced thrombocytopenia, molecular mimicry between platelets and H. pylori antigens has been discussed (12, 13). In two studies, eradication was achieved in 87-100% of the H. pylori-positive patients, 54-58% of whom exhibited increased platelet counts (12, 13). Therefore, both UC and H. pylori infection can cause ITP; however, the treatment for ITP caused by each of these diseases is quite different.

In our patient, we believe that the ITP was caused not by UC but by H. pylori infection, as the ITP manifested when the UC was almost in remission and improved dramatically after the eradication of H. pylori. However, if ITP occurred in a UC patient during an episode of exacerbation or in a patient with severe colitis, it would be unusual to suspect an association with H. pylori early in the work-up. We reviewed cases of UC with ITP reported in the literature between 2003 and 2012 (3, 5, 7-9) and found that H. pylori tests were actually performed in only one case among five reports. In that report, the H. pylori test results of the patient were positive (9). These findings suggest that H. pylori tests might be performed in UC cases accompanied by ITP less frequently than expected. A low platelet count can lead to severe colonic bleeding in patients with UC, and their ITP should therefore be treated promptly. In this context, although the prevalence of H. pylori infection among patients with inflammatory bowel disease has been shown to be low (14), our case report demonstrates the importance of conducting examinations for H. pylori infection in patients with ITP, even when the ITP occurs in UC patients with or without severe colonic inflammation.

The authors state that they have no Conflict of Interest (COI).

References


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