Treatment of Severe Thrombocytopenia with Intravenous Immunoglobulins and Corticosteroids in a Patient Receiving Continuous Ambulatory Peritoneal Dialysis

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Dear Sir,

While bleeding tendency and platelet dysfunction are known in uremic patients, severe thrombocytopenia is rarely encountered. We describe a uremic patient with acute severe thrombocytopenia who was undergoing continuous ambulatory peritoneal dialysis (CAPD). The patient was successfully treated with intravenous immunoglobulins and corticosteroids. Peritoneal dialysis was maintained during treatment for thrombocytopenia.

A 57-year-old Japanese man, who was receiving CAPD, was referred to our department because of acute severe thrombocytopenia. CAPD was initiated 5 years earlier after uremia due to chronic glomerulonephritis had been diagnosed. His platelet count ranged from $150 \times 10^9/l$ to $280 \times 10^9/l$ during this 5-year period, but fell to $100 \times 10^9/l$ six days before admission and continued to decrease (to $3 \times 10^9/l$ on admission) (fig. 1). He had hemorrhagic vesicles in the oral mucosa, gingival bleeding, and ecchymoses on both legs. He was also hoarse due to bleeding of the vocal cords. Megakaryocytes in bone marrow were plentiful and increased in size. The level of platelet-associated IgG (PAIgG) was increased (68.2 ng/107 cells). There were no symptoms or laboratory findings to suggest the presence of autoimmune disease. We made a presumptive diagnosis of thrombocytopenia caused by immunologic platelet destruction. High-dose steroid therapy was considered inappropriate because of the risk of significant side effects, especially since the patient had developed bacterial

of therapy, to $71 \times 10^9/l$ on hospital day 3, to $85 \times 10^9/l$ on hospital day 5, and to $141 \times 10^9/l$ on hospital day 11. Gingival bleeding stopped on day 2, and the hemorrhagic vesicles in his mouth disappeared by day 7. The hoarseness also gradually improved.

peritonitis 2 years earlier. He received high-dose intravenous immunoglobulin therapy (400 mg/kg/day) for 5 days and low-dose oral corticosteroid therapy for 7 weeks (initial dose 0.5 mg/kg/day). His platelet count increased to $27 \times 10^9/l$ one day after the start

Immunoglobulin

(400 mg/Kg/day)

September

July 1996

August Admission

Fig. 1. Clinical course. The platelet count gradually fell before admission and continued to decrease on admission. The patient received high-dose intravenous immunoglobulin therapy for 5 days (arrows) and low-dose oral corticosteroid therapy for 7 weeks (shaded square). His platelet count increased 1 day after the start of therapy and to above $100 \times 10^9/l$ on hospital day 7. It decreased slightly after 11 days but remained above $70 \times 10^9/l$ and then gradually increased to within the normal range as the corticosteroid dose was tapered.

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There was no worsening of uremia and peritonitis did not occur. His platelet count decreased slightly after 11 days but remained above $70 \times 10^9/l$ and then gradually increased to within the normal range as the corticosteroid dose was tapered (fig. 1).

We first thought that our patient had drug-induced thrombocytopenia because he was taking several medications [1,2]. Phen-acetin induced lymphocyte stimulation supporting this theory. He had been taking phenacetin once a week for 3 years. However, withdrawal of phenacetin did not result in a complete recovery. We then suspected acute idiopathic thrombocytopenic purpura (ITP), although he had no history of an antecedent infection. ITP usually occurs in children and young adults, but occasionally occurs in older patients [1, 3]. His bone marrow and elevated PAIgG level were characteristic of ITP.

The appropriate therapy for acute ITP is controversial. A brief course of corticosteroid therapy is recommended in the early

stages of severe acute thrombocytopenia. Corticosteroids impair reticuloendothelial function, thereby diminishing platelet destruction [4]. Intravenous administration of polyvalent immunoglobulin sometimes induces a remission in patients with refractory ITP. The mechanism of its therapeutic effect is not entirely clear, but evidence suggests that immunoglobulin blocks the Fc receptors of reticuloendothelial cells [5].

Severe thrombocytopenia caused by immunologic platelet destruction is uncommon but potentially fatal, in uremic patients [6]. We treated our patient with immunoglobulin and a corticosteroid in an effort to avoid life-threatening complications, such as intra-cranial hemorrhage. The present findings suggest that high-dose immunoglobulin and low-dose corticosteroid therapy may have an important role in the treatment of uremic patients with immunologic thrombocytopenia, especially in patients in whom high-dose corticosteroids are inappropriate.

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