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LETTER TO THE EDITOR

# Primary immune thrombocytopenia responding to antithyroid treatment in a patient with Graves' disease

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Dear Editor,

A 22-year-old woman presented with generalized petechiae for 1 week, and complete blood count revealed an isolated thrombocytopenia of $6\times10^9$/L. Blood film examination was normal except thrombocytopenia. Physical examination showed no palpable lymphadenopathy or hepatosplenomegaly. Bone marrow biopsy showed megakaryocytic hyperplasia only, and a diagnosis of acute primary immune thrombocytopenia (ITP) was made. Intravenous immune globulin (IVIg; 0.5 $g\,kg^{-1}\,day^{-1}$ for 4 days) was started with no increase in platelet count. Oral prednisolone at 50 mg daily (1 mg/kg) was added subsequently, but the platelet count remained below $10\times10^9$/L for another 2 weeks. Although she had no clinical symptoms and signs of thyrotoxicosis, thyroid function test was requested in view of the presence of a small goiter. The blood test revealed a raised free T4 with suppressed TSH level, and the antithyrogobulin and anti-microsomal antibodies were also increased, compatible with a diagnosis of Graves' disease. Carbimazole was therefore initiated while prednisolone was continued concurrently for the ITP. Intriguingly, the platelet count showed a progressive improvement with a parallel reduction of the free T4. At 3 and 5 weeks after starting the carbimazole, the platelet counts were 83 and $182\times10^9$/L when the dose of prednisolone was also reduced to 30 and 15 mg, respectively.

The association between hyperthyroidism and thrombocytopenia was first described in 1931 [1]. Autoimmune thyroid disorders including Graves' disease and Hashimoto's thyroiditis have subsequently been reported in patients with ITP. In a recent standardization of terminology, ITP with concurrent autoimmune disease has been designated as secondary ITP which may require a different approach to management [2]. This distinction, however, is not well defined in cases of ITP associated with autoimmune thyroid diseases. Simultaneous presentation of ITP and autoimmune thyroid disease is often seen, but the timing between the two diagnoses can vary from months to years [3]. Several mechanisms connecting thyroid disorders and thrombocytopenia have been described. Mild thrombocytopenia is frequently observed in patients with Graves' disease [4]. Platelet life span has been shown to be significantly shortened in patients with hyperthyroidism [5]. Increased reticuloendothelial phagocytic activity by upregulation of Fc receptor expression or activity is a potential mechanism [6]. Significant immune dysregulation in patients with thyroid dysfunction and ITP is evidenced by the increased prevalence of antiplatelet and antithyroid antibodies in these patients [7]. Platelet-associated IgG or specific platelet antibodies occurred in 83% and 86% of patients with ITP with and without autoimmune thyroid disease, respectively [7]. Thyroid autoantibodies, on the other hand, were detected in 89% of patients with ITP and autoimmune thyroid disease [7]. Genetic predisposition to the concurrence of ITP and hyperthyroidism has also been suggested. HLA B8 is the commonest antigen reported in patients with isolated ITP and Graves' disease [8–10]. Given the well-established association between the two conditions, treatment of the coexisting thyroid disorder has been reported to induce remission of ITP or result in an improved response to standard ITP therapy [3]. As illustrated in our case, treatment of the Graves' disease did result in a significant improvement of thrombocytopenia that had been refractory to IVIg and steroid treatment.

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In cases where response to standard ITP treatment is suboptimal, the thyroid status of the patients should be evaluated and corrected accordingly.



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