Is Hashimoto’s Thyroiditis Associated with Chronic Immune Thrombocytopenic Purpura?

Dear Editor,

Autoimmune disorders result from common process of disrupted self-tolerance. This describes why one autoimmune disease likely causes other autoimmune disorders.1 However, the literature about the occurrence of Hashimoto’s thyroiditis (HT) in previously diagnosed immune thrombocytopenic purpura patients is rarely reported. As there is few data about this theme in children, we described 4-year-old boy who developed autoimmune thyroid disease in a year after the diagnosis of ITP. Three years old patient with an unremarkable medical and family history was admitted to our hospital with acute onset of bruising and petechiae on his cheeks, legs, arms and back. Physical examination revealed no other abnormalities. Laboratory studies showed an isolated thrombocytopenia of 13000/L. After performing multiple diagnostic studies to exclude other hematologic and infectious disorders, the patient was diagnosed with immune thrombocytopenic purpura (ITP). Serum tests were negative for anti-EBV and anti-CMV IgM antibodies. Methylprednisone (30 mg/kg/day), intravenous immunoglobulin (2 gr/kg/dosage IVIG) and WinRho (60 mcg/kg/day Anti-D) were administered. After these treatments, his platelet count increased. The patient received methylprednisone (30 mg/kg/day), IVIG (1 gr/kg/dosage) or WinRho (60 mcg/kg/day) for persisting thrombocytopenia (8000-20000/L) with an interval of one month in first six months. Serologies for HIV and HTLV, ANA and anti dsDNA autoantibodies were negative. Subsequently IVIG (1 gr/kg/dosage), dexamethasone (28 mg/m²/day) and vincristine (1.5 mg/m²/dosage) were administered for persisting thrombocytopenia (8000-20000/L) in following four months. After ten months, cyclosporine was started, but thrombocytopenia persisted (8000-20000/L). The patient was diagnosed with chronic immune thrombocytopenic purpura (ITP). While we were performing multiple diagnostic studies, the patient showed elevated levels of antithyroid antibodies [antiperoxidase antibody (TPO): 361.67 IU/mL (<35) and antithyroglobulin antibody: 282.5 IU/mL (<40)], low free thyroxine (FT4): 0.63 ng/dL (0.75-1.80) and high thyrotropin (TSH): 14.2 mIU/mL (0.3-5.0)). Hashimoto’s thyroiditis was considered and treatment with levothyroxine was started (3 µg/kg/day). When diabetes antibodies were performed for investigating other autoimmune diseases, the patient had antiglutaric acid decarboxylase (anti-GAD) antibodies (1.46 (0-1)), and anti-insulin antibody (4.01 U (0-7U)) and islet cell antibody were negative. Because serum blood glucose, serum C-peptide level and insulin were normal, the insulin therapy was not administered.

To the best of our knowledge, we presented the first case of very young boy with chronic ITP associated with Hashimoto’s thyroiditis. Pratt et al investigated 31 children with immune thrombocytopenic purpura for positive antithyroid antibodies of which five patients tested positive.2 Lio et al reported on 20 children with idiopathic thrombocytopenic purpura who had no antithyroid antibodies.3 Our patient had Hashimoto’s thyroiditis and positive anti-GAD antibodies. These evidences indicate that chronic ITP might be related with a specific gene, which may cause the autoimmune response in the thyroid and pancreas glands. Defects in the autoimmune regulator gene might cause these disorders. Chronic ITP with autoimmune thyroid disease might share common pathogenic pathway and this defect might be classified as an autoimmune polyendocrine syndrome type. Further investigations into similar cases are needed to clarify this association. Patients with chronic ITP should be evaluated by thyroid function tests, including those for antithyroid antibodies, for prevent the development of overt hypo- and hyper-thyroidism.

References


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