

Immunosuppressive treatment for immune thrombocytopenia which masked Graves' disease

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ABSTRACT

A 71-year-old female patient followed primary immune thrombocytopenia (ITP) was admitted to endocrinology unit with excessive sweating. We started methimazole for Graves' disease. Without any additional immunosuppressive treatment, at week 12 of methimazole therapy, thyroid stimulating hormone (TSH) levels returned to normal, and platelet counts rose to tolerable levels. When her hospital records were analyzed, they revealed that a year ago, when she had been diagnosed with ITP, her TSH values had been suppressed. After immunosuppressive therapy, her platelet values were maintained at normal levels, and during her control visits, her TSH levels were measured twice and were within normal limits. We think that immunosuppressive therapy for ITP without considering thyroid function tests may result in a transient euthyroid state, which potentially masks Graves' disease accompanying immunosuppressive therapy and associated recurrent ITP attacks.

Keywords: Graves' disease, immune thrombocytopenia, methimazole

Introduction

Coexistence of hyperthyroidism and primary immune thrombocytopenia (ITP) is a rather rare condition. Therefore, little is known about the outcomes of these two diseases when associated. Underlying untreated Graves' disease leads to worsening of the ITP and rendering it refractory to standard therapy.^[1] We report a case of Graves' disease masked by immunosuppressive therapy for ITP.

Case Report

A 71-year-old female patient presented with a complaint of excessive sweating for nearly a month to a hematology unit where she had been followed-up with after a diagnosis of ITP. Her laboratory tests revealed manifest hyperthyroidism in addition to thrombocytopenia, and hence she was referred to our endocrinology unit. On her physical examination, her pulse rate was 112 bpm, and her skin was warm and damp. In addition, a diffusely hypertrophic thyroid gland was palpated. Palpable lymphadenopathy and splenomegaly were not detected. On peripheral smear, only thrombocytopenia was found.

Increased levels of anti-thyroid peroxidase, anti-thyroglobulin, and thyroid-stimulating antibody and enhanced uptake of radioactive iodine were observed, and the diagnosis of Graves' disease was confirmed. Methimazole therapy at daily doses of 30 mg was initiated. Without any additional immunosuppressive treatment, at week 12 of methimazole therapy, thyroid stimulating hormone (TSH) levels returned to normal, and platelet counts rose to tolerable levels.

When her hospital records were analyzed, they revealed that a year ago, when she had been diagnosed with ITP, her TSH values had been suppressed. At that time, the patient had not described any complaint suggesting hyperthyroidism. She had been given intravenous immune globulin (0.5 g/kg/day for 4 days) and oral steroid (1 mg/kg/day) for the treatment of ITP. The patient, whose platelet counts returned to being within normal limits, was followed-up with for 6 months. During this period, her platelet values were maintained at normal levels, and during her control visits, her TSH levels were measured twice and were within normal limits. The patient's laboratory test results are shown in chronological order [Table 1].

Discussion

The association between Graves' disease and thrombocytopenia was first defined in 1931.^[2] Afterwards, concomitancy of

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Table 1: Laboratory test results of the patient in chronological order

Parameter (normal range)	October 2013 (at the time of the diagnosis of ITP was made)	January 2014 (the first control)	April 2014 (the second control)	May 2014 (at the time of diagnosis of Graves' disease was made)	July 2014 (at 3 months of the anti-thyroid therapy)
Free T ₃ (2-4.4 pg/mL)	-	-	-	5.1	2.09
Free T ₄ (0.93-1.7 ng/dL)	-	-	-	2.32	0.92
TSH (0.27-4.2 µIU/mL)	<0.01	1.01	1.24	<0.01	4.02
Platelets (150-400 K/uL)	12	201	176	32	109

TSH: Thyroid stimulating hormone, ITP: Immune thrombocytopenia

autoimmune thyroid diseases such as Hashimoto thyroiditis and Graves' disease with ITP has been reported many times.^[1]

In Graves' disease, the lifespan of platelets is shortened significantly, and moderate degrees of thrombocytopenia are frequently seen.^[3] The potential mechanism involved in this process is increased phagocytic activity of the reticuloendothelial system.^[4] The association between ITP and Graves' disease is explained by immune dysregulation, which is the main mechanism underlying both diseases and involves an increase in the levels of antiplatelet and anti-thyroid antibodies in these patients.^[1]

Because of the strong fundamental autoimmune mechanism underlying both diseases, it is known that the treatment of associated autoimmune thyroid disorder contributes to the remission of ITP. It has been reported that the treatment of underlying Graves' disease in patients refractory to standard immunosuppressive therapy ensured remission of ITP.^[5] Besides, only anti-thyroid treatment without additional immunosuppressive treatment enabled remission of ITP.^[6]

In our case, Graves' disease had been overlooked during the diagnostic work-up for ITP. Although spontaneous remissions can be seen in Graves' disease, we think that immunosuppressive therapy given for the treatment of ITP-induced a transient euthyroidism in Graves' disease. During the 6 months following ITP therapy of our patient, whose TSH values remained at normal levels, Graves' disease and concomitant ITP recurrences were observed, and only anti-thyroid therapy increased platelet counts to tolerable levels.

Conclusion

In the presence of thrombocytopenia, the patients should be carefully evaluated for hyperthyroidism. It should be kept in mind that immunosuppressive therapy without considering thyroid function tests may result in a transient euthyroid state, which potentially masks Graves' disease accompanying immunosuppressive therapy and associated recurrent ITP attacks.

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