



Co-Occurance of Graves Disease and Immune Thrombocytopenia: A Case Report

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Introduction

Cytopenias can be observed in patients with Graves Disease (GD). However, there are very few number of case reports about isolated thrombocytopenia in GD.

We present a case of immune thrombocytopenia incidentally found in the evaluation of GD.

Case Presentation

A 48-year old female was admitted to our hospital with palpitation and anxiousness. The thyroid function tests were concordant with thyrotoxicosis. She had two subcentimetre nodules in the right thyroid lobe. Thyroid gland had increased iodine uptake and thyroid scan showed a diffuse hyperactive thyroid gland. Graves Disease (GD) was diagnosed. Complete blood count and liver function tests were performed before antithyroid treatment. She had isolated thrombocytopenia (37000/ μ L). She had no history of bleeding, chronic illness, medication or herbal drugs. Peripheral smear was concordant with platelet count. Anti-nuclear antibody was negative. Immune thrombocytopenia was diagnosed. As she had platelet levels above 20000/mL we decided to initially start methimazole therapy without steroid therapy. In the first month of methimazole therapy, platelet counts were between 25000-45000/ μ L. Six weeks after methimazole therapy, when her free T4 and free T3 levels were in the normal range, her platelet count began to decrease. Prednisolone treatment was initiated for immune thrombocytopenia. Platelet count rapidly increased to normal levels. Steroid therapy was gradually tapered and stopped after two months. Four months after discontinuing steroid treatment, she is now euthyroid with methimazole therapy, and her platelet count is between 100000-120000/ μ L.

Discussion

There are only a few case reports describing co-occurrence of GD and immune thrombocytopenia in the literature.

In our case, methimazole was initiated without corticosteroid therapy. The rationale of this treatment was not only for normalisation of thyroid function tests but also for the potential of improving immune thrombocytopenia. But treatment failed for improving platelet count despite successful normalisation of free thyroid hormone levels.

A hypothesis of cross reaction between antithyroid antibodies and platelet epitopes has been suggested which has not been cleared yet. One would think that euthyroidism might improve immune thrombocytopenia. However, in our case, platelet levels were still low although euthyroidism was achieved. Therefore, etiologies other than cross reactions between antithyroid antibodies and platelet epitopes may explain this issue which should be investigated.

References

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