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ARAŞTIRMA / RESEARCH

Transient pseudothrombocytopenia associated with Graves' disease

Graves hastalığına eşlik eden geçici yalancı trombositopeni

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To the Editor,

Graves' disease, an autoimmune disease characterized by antibodies against the thyroid stimulating hormone (TSH) receptor, is the most common cause of hyperthyroidism. Association of pseudothrombocytopenia (PTP) with Graves' disease was reported in only one case and the course of PTP was not defined in this paper[1]. A case with PTP associated with Graves' disease was presented in this report. After clinical healing and normalization of thyroid stimulating immunoglobulin (TSI), PTP was resolved completely.

A 27-year-old woman admitted with palpitation, sweating and weight loss complaints. These complaints have started since six months. She had weight loss of 3 kg in spite of good appetite in this period. She had no fever or diarrhea. Body temperature was 37.2 °C, arterial blood pressure was 120/60 mmHg and pulse was 104/min and rhythmic. The skin was moist. She had the nervous appearance. The thyroid gland was diffusely enlarged and painless. White blood cell count was 6.8 x109/L, red blood cell count 4.55 x1012/L, hemoglobin 11.9 g/dL, hematocrit 35.2%, mean corpuscular volume 77.4 fL, mean corpuscular hemoglobin 26.3 pg and platelet count 33 x109/L in complete blood count with ethylenediaminetetraacetic acid (EDTA). Biochemical analysis, erythrocyte sedimentation rate and urine analyses were normal. Tests for thyroid disease were free T3 16.63 pg/mL (2.2-4.2), free T4 5.23 ng/dL (0.65-1.7), TSH 0.004 µIU/mL (0.4-4.2), TSI 69.42 U/L (0-14), anti-thyroglobulin antibody (anti-Tg ab) 5 IU/mL (5-100) and anti-thyroid peroxidase antibody (anti-TPO ab) 1.49 IU/mL (1-16). Thyroid ultrasonography revealed diffuse enlargement in the thyroid gland. Anti-nuclear antibody and anti-double strand DNA antibody were negative. Platelet clumps were seen sufficiently in peripheral smear examination. Platelets were counted mean 16.2 numbers in microscopic evaluation with x1000 augmentation. Complete blood count was repeated with heparin and platelet count was found to be 180 x109/L. PTP associated with Graves' disease was diagnosed and propylthiouracil 600 mg/day and propranolol 40 mg/day were started. Drugs' doses were adjusted with regular controls. White blood cell count was 5.8 x109/L, red blood cell count 4.61 x1012/L, hemoglobin 11.5 g/dL, hematocrit 35.8%, mean corpuscular volume 77.6 fL, mean corpuscular hemoglobin 25.0 pg and platelet count 167 x109/L in complete blood count with EDTA after three months from diagnosis. Platelet count was found as 168 x109/L with heparin. Free T3 3.21 pg/mL (2.2-4.2), free T4 0.825 ng/dL (0.65-1.7), TSH 1.37 µIU/mL (0.4-4.2), TSI 15.37 U/L (0-14), anti-Tg ab 5 IU/mL (5-100) and anti-TPO ab 3.98 IU/mL (1-16) were found. Platelet clumps were observed in peripheral smear.

PTP is a condition caused by anti-platelet antibodies in which agglutination of platelets in the complete

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blood count tube leads to incorrectly low platelet counts. It should be considered first in patients with low platelet counts without a bleeding diathesis. This phenomenon could be observed in healthy subjects, but it could also accompany certain diseases. The incidence is 0.09–0.21%[2]. Misdiagnosis of PTP as true thrombocytopenia leads to unnecessary laboratory tests and unwarranted exposure to transfusion and related complications.

Table 1. Values of platelet count and thyroid hormones at diagnosis and after three months

	At diagnosis	After three months
Platelet count with EDTA (x109/L)	33	167
Platelet count with heparin (x109/L)	180	168
Free T3 (2.2-4.2 pg/mL)	16.63	3.21
Free T4 (0.65-1.7 ng/dL)	5.23	0.825
TSH (0.4-4.2 μIU/mL)	0.004	1.37
TSI (0-14 U/L)	69.42	15.37
Anti-Tg ab (5-100 IU/mL)	5	5
Anti-TPO ab (1-16 IU/mL)	1.49	3.98

TSH= Thyroid Stimulating Hormone, TSI= Thyroid Stimulating Immunoglobulin, Anti-Tg Ab= Anti-Thyroglobulin Antibody, Anti-TPO= Anti-Thyroid Peroxidase Antibody

The association between hyperthyroidism and thrombocytopenia was described firstly in 1931[3]. The shortened platelet life span, increased reticuloendothelial phagocytic activity, immune thrombocytopenia related to thyroid autoantibodies, genetic predisposition to the concurrence of immune thrombocytopenia and hyperthyroidism are most commonly reported mechanisms[4-6]. Graves' disease may be associated with autoimmune hematological disorders such as immune thrombocytopenic purpura[7]. Szczepinski et al reviewed 217 cases with EDTA-dependent PTP. In this review, association of PTP with Graves' disease was reported in only one case. This case had also anti-glycoprotein Ib autoantibody and the course of PTP was not defined in this paper[1]. As a conclusion, PTP is frequently misdiagnosed which lead to inappropriate treatments. Therefore, this situation should be kept in mind primarily in the evaluation of thrombocytopenic patients.

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