V. Internal Medicine Section

1. A CASE OF GRAVE'S DISEASE AND THROMBOCYTOPENIA

Author: Pascari Otilia

Scientific adviser: Stela Vudu, MD, Department of Endocrinology, *Nicolae Testemitanu* State University of Medicine and Pharmacy of the Republic of Moldova

Introduction. Thrombocytopenia may coexist with Grave's disease. However, the mechanisms of decrease in platelet count alongside with autoimmune thyroid disease have not been comprehensively investigated.

Case presentation. A case of Grave's disease is reported in a 55-year-old man who has been receiving continuous treatment with synthetic antithyroid drugs for 7 years. Laboratory analysis showed suppressed thyroid stimulating hormone (TSH) 0.01 mlU/ml, normal free T4 level 21.60 pmol/l, increased free T3 6.99 pmol/l and thyroid stimulating immunoglobulin (TSI) 23.60 IU/L. Complete blood test revealed severely decreased platelets 3 x 10^9 / l, decreased hemoglobin 110.00 g/l, leucocytes 9.58 x 10^9/l, hematocrit 21.90%, Prothrombin Index 11%, International normalized ratio (INR) 7.00. The patient has recovered from viral pneumonia caused by SARS-COV-2, 1 month prior to the medical consultation.

Discussion. Thrombocytopenia observed in hyperthyroidism may be mediated by both metabolic and immunological phenomena. In our case, the following causes may be considered: (1) an overlapping autoimmune process; (2) antithyroid drugs creating destructive antibodies; (3) COVID-19 disease. An autoimmune process is capable of triggering both conditions by activation of the reticuloendothelial system by thyroid hormones along with a cross-reaction between thyroid antibodies and platelet epitopes [1,3]. Carbimazole generates a drug-dependent immune response against platelets, involving platelet endothelial cell adhesion molecules and thus provoking thrombocytopenia [4]. SARS-CoV-2 can induce thrombocytopenia by mass production of cytokines causing progenitor destruction in the bone marrow with decreased primary platelet production. Infection increases the number of autoantibodies and immune complexes that destroy platelets. Lung damage by decreasing pulmonary capillary bed and evoking the fragmentation of megakaryocytes, leads to a reduced number of circulatory platelets [2].

Conclusion. A severe thrombocytopenia was detected in a patient with Grave's disease, in whom the number of platelets was 50 times below the lower limit of the reference range. Coexistence of thrombocytopenia may be explained by (1) an overlapping autoimmune process; (2) the effect of carbimazole; (3) its occurrence or aggravation secondary to COVID-19.