Laboratory Diagnosis of Antiphospholipid Auto-antibody Syndrome in Materials of National Center of Hemostasis and Trombosis in Martin

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In the years of 1992–1996 (after establishing NCHT in Martin) we examined around 400 patients with suspicion for trombophilic status. Antiphospholipid antibody syndrome (AAS) is a clinical syndrome of venous and arterial trombosis, repeated loss of fetus and and trombocytopenia [1]. AAS were diagnosed in 33 patients, 11 males and 22 females. Primary AAS (without basic clinical signs) was diagnosed in 16 patients and secondary (with other symptoms) in 17 patients. 8 patients were positive for the presence of antibody against lupus anticoagulans (LA), 10 patients were positive for anticardiolipid antibodies (ACA) and 15 patients were LA and ACA positive. On the basis of individual caogulation screening tests as well as ELISA tests of titer evaluation and evaluation of isotype of ACA we estimated the relevance and prediction value of future trombosis.

Skin diseases (systemic lupus erythematosus) were the common causes of APS. The most frequent clinical signs of patients were venous and arterial trombosis, trombosis to CNS, repeated loss of fetus, reumatic difficulties, diabetes mellitus, asthma.

LA antibodies are linked with prolonged coagulation time of the whole blood and prothrombine time and therefore the estimation of lupus inhibitor is based on its ability to bound to phospholipids and to inhibit coagulation tests dependent on phospholipids [2]. ACA expresses no in vitro coagulation activity of LA and is dependent on cofactor beta 2 glycoprotein inhibitor (beta 2-GPI). Both tests are required to repeat after 3 moths.

Along with known findings of in vitro plasma tests we have also found a decreased levels of natural inhibitors of coagulation (ATIII, PC, PS, HC II) as well as levels of coagulation factors (FXII, FXI, FX, FIX, FVIII, FVII, FV, FII) and other markers of activated or damaged trombocytes (level of trombomodulin-TM, level of beta 2GPI). Further, we have shown a cross reactivity with tumor markers (CA-15-3) and the presence of antibodies bound on the surface of trombocytes has been proved by flow cytometry. In general, lupus inhibitor persists in patients with primary AAS, although it can spontaneously disappear. At secondary AAS, the therapy of basic autoimmune disorder leads to the reduction of disappearance of inhibitor activity. Patients with both ACA and trombosis require prolonged anticoagulation therapy and interruption in therapy may be started only if ACA are absent for 4–6 months.

REFERENCES

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