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ANTIPHOSPHOLIPIID SYNDROME AS A CAUSE OF STROKE

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Abstract: Hypercoagulation syndrome is an actual multidisciplinary problem of the last decade. Violations of hemostasis, leading to hypercoagulation syndrome, are manifested by various diseases in neurology, obstetrics, rheumatology, and surgery. Antiphospholipid syndrome is the most common form of hypercoagulation syndrome and most often develops at a young age, in children and even newborns, and in females 5 times more often than in males. Antiphospholipid syndrome requires special attention for timely and early diagnosis to prevent severe complications.

Keywords: antiphospholipid syndrome, antiphospholipid bodies, neurological manifestations, ischemic stroke, prevention

One of the urgent problems of modern medicine is the prevention of non-communicable diseases, taking into account the pathogenetic mechanisms underlying their development [1,2]. Antiphospholipid syndrome (antiphospholipid antibody syndrome, lupus antibody syndrome, Hughes syndrome) is a systemic autoimmune disease associated with hypercoagulation and caused by the synthesis of antiphospholipid antibodies (APLA): anticardiolipin antibodies (aCL), lupus anticoagulant (LA), antibodies to b2-glycoprotein I (anti-b2-GP I). Most often, APLS is considered in the context of gynecological pathologies, as one of the causes of abortion, although this disease occurs in any population and has an erased clinical picture. The alertness of a doctor of any specialization regarding this condition is especially important, since timely prescribed therapy improves the quality of life of patients and long-term prognosis, preventing the development of life-threatening complications.

The exact prevalence of APLS in the population remains unknown due to lack of statistics. The disease occurs mainly among young and middle-aged people, compared with the elderly. The exact percentage ratio between the sexes has not been identified, however, secondary APLS more often affects women.

The most common manifestations of APLS are deep vein thrombosis (38.9%), thrombocytopenia (29.6%), stroke (19.8%), pulmonary embolism (14.1%), superficial thrombophlebitis (11.7%), transient ischemic attack (11.1%), hemolytic anemia (9.7%), epilepsy (7%) and obstetric morbidity [3].

In the pathogenesis of APLS, the leading role belongs to organ-specific autoantibodies that react with antigenic determinants of

phospholipids - antiphospholipid antibodies (aPL). It should be noted that autoantibodies are traditionally associated with autoimmune diseases, although they are present in minimal amounts in all healthy individuals.

According to the ICD 10 classification, the diagnosis of APLS can be made if the patient has at least one clinical and one laboratory criterion. Clinical criteria include arterial or venous thrombosis of various localization (peripheral venous thrombosis, myocardial infarction, ischemic stroke, infarction of any organ, etc.), objectified by imaging (ultrasound, angiography) or histological methods [4,5].

Laboratory criteria include the presence of a positive lupus anticoagulant (LA), positive anticardiolipin antibodies in a titer of more than 40 GPL, MPL, positive antibodies to β2-glycoprotein I (aβ2GP-1) isotypes G or M (> 99th percentile).

In this case, a prerequisite is to obtain positive results in at least two studies conducted at least 12 weeks apart. Knowledge and observance of these diagnostic criteria is of fundamental importance, since, according to the results of the experience and research of foreign colleagues, there is currently an unjustified overdiagnosis of APLS in the field of neurology [6,7].

The neurological manifestation of APLS is represented by stroke (19.8%), myelopathy (less than 1%), Sneddon syndrome, convulsive syndrome (7%), chorea (1.3%), headache and migraine (20.2%), dementia (2 .5%), eye syndromes (15-88%), multiple sclerosis, Guillain-Barré syndrome and peripheral neuropathy [8]. The most severe neurological complication of APLS is stroke.

Cerebral circulation disorders, combined with the production of aPL, in most cases debut at a young age (up to 45 years), much less often - in childhood or older [9]. Women get sick more often (81%), which is associated with the specifics of their hormonal background, which favors the development of an immunopathological process and a procoagulant state. Pregnancy, the postpartum period, dysmenorrhea, and premenopause can be provoking factors for NMC in APLS in women, which clinically confirm the importance of hormonal changes in the implementation of the procoagulant state present in APLS [10].

The main mechanism for the development of ischemic CVD in APLS is cerebral artery thrombosis in situ due to a hypercoagulable state induced by aPL production. The clinical spectrum of ischemic brain lesions in APS is extensive and includes manifestations from transient ischemic attacks to focal lesions such as amaurosis, extensive cerebral infarction, ataxia, and dementia. Most often, a vascular lesion in APLS affects the territory of the middle cerebral artery [11].

It should be noted that in APLS there is a tendency to recurrence of stroke, which is often preceded by transient ischemic attacks and very often

cerebrovascular accidents occur without symptoms and turn out to be an accidental finding in the study of magnetic resonance imaging.

Diagnosis of cerebrovascular accident associated with the production of aPL is based on their clinical features: young age of patients, intact main arteries of the head, small or medium sizes of cerebral infarcts. Of great diagnostic importance is the presence in patients of the main and additional systemic manifestations of APLS. Differential diagnosis of APLS is carried out with a wide range of diseases occurring with vascular disorders. It is believed that APLS should be suspected in the development of thrombotic disorders (especially multiple, recurrent, with unusual localization), thrombocytopenia, obstetric pathology in young and middle-aged individuals in the absence of risk factors for these pathological conditions [10].

The basis for the treatment and prevention of recurrent cerebral ischemia is the use of indirect anticoagulants, aspirin and heparin. In the acute period of a stroke, in most cases it is recommended to use direct-acting anticoagulants - heparin, fraxiparin, clexane. Anticoagulants of indirect action, small doses of aspirin, or a combination of both are also used to prevent recurrent cerebrovascular accident. The use of anticoagulants should be under constant clinical and laboratory monitoring of patients.

Thus, APLS is one of the causes of ischemic CVD, recurrent cerebrovascular accident, multi-infarct dementia, and thrombosis of the veins and sinuses of the brain in young patients. Diagnosis is based on a comprehensive assessment of neurological and systemic manifestations of the disease in combination with the detection (at least twice) of at least one of the diagnostically significant aPL. Early diagnosis and timely administration of anticoagulants can prevent the development of recurrent strokes and dementia. Therefore, of no small importance in this belongs to doctors and narrow specialists of primary care, who are entrusted with the main function in carrying out both primary and secondary prevention.

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