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DIAGNOSIS AND TREATMENT ANTIPHOSPHOLIPID SYNDROME

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Annotation: Antiphospholipid syndrome is an autoimmune pathology based on the formation of antibodies to phospholipids, which are the main lipid components of cell membranes. Antiphospholipid syndrome can be manifested by venous and arterial thrombosis, arterial hypertension, valvular heart defects, obstetric pathology (habitual miscarriage, intrauterine fetal death, gestosis), skin damage, thrombocytopenia, hemolytic anemia. The main diagnostic markers of antiphospholipid syndrome are At to cardiolipin and lupus anticoagulant. Treatment of antiphospholipid syndrome is reduced to the prevention of thrombosis, the appointment of anticoagulants and antiplatelet agents.

Key words: Antiphospholipid syndrome, autoimmune pathology.

Antiphospholipid syndrome (APS) is a complex of disorders caused by an autoimmune reaction to phospholipid structures present on cell membranes. The disease was described in detail by the English rheumatologist Hughes in 1986. There are no data on the true prevalence of antiphospholipid syndrome; it is known that insignificant serum levels of antiphospholipid antibodies are found in 2-4% of practically healthy individuals, while high titers are found in 0.2%. Antiphospholipid syndrome is 5 times more often diagnosed among young women (20-40 years), although men and children (including newborns) can suffer from the disease. As a multidisciplinary problem, antiphospholipid syndrome (APS) attracts the attention of specialists in the field of clinical rheumatology, obstetrics and gynecology, and cardiology.

Reasons

The underlying causes of antiphospholipid syndrome are unknown. Meanwhile, factors predisposing to an increase in the level of antibodies to phospholipids were studied and determined. Thus, a high titer of antiphospholipid antibodies is observed against the background of:

- viral and bacterial infections (hepatitis C, HIV, infectious mononucleosis, malaria, infectious endocarditis, etc.)
- autoimmune diseases (systemic lupus erythematosus, rheumatoid arthritis, Sjogren's disease, periarteritis nodosa, autoimmune thrombocytopenic purpura);
- malignant neoplasms;
- taking medications (psychotropic drugs, hormonal contraceptives, etc.), discontinuing anticoagulants.

There is evidence of a genetic predisposition to increased synthesis of antibodies to phospholipids in individuals carrying HLA DR4, DR7, DRw53 antigens and in relatives of patients with antiphospholipid syndrome.

Pathogenesis

Depending on the structure and immunogenicity, "neutral" (phosphatidylcholine, phosphatidylethanolamine) and "negatively charged" (cardiolipin, phosphatidylserine, phosphatidylinositol) phospholipids are distinguished. The class of antiphospholipid antibodies

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that react with phospholipids includes lupus anticoagulant, anti-cardiolipin antibodies, beta2-glycoprotein-1-cofactor-dependent antiphospholipids, etc. Interacting with phospholipids of vascular endothelial cell membranes, platelets, and neutrophils, antibodies cause hemostasis disorders, which are expressed in a tendency to hypercoagulability. In general, the immunobiological mechanisms of the development of antiphospholipid syndrome require further study and clarification.

Classification

Taking into account the etiopathogenesis and course, the following clinical and laboratory variants of antiphospholipid syndrome are distinguished::

- primary – there is no association with any underlying disease that can induce the formation of antiphospholipid antibodies;
- secondary-antiphospholipid syndrome develops against the background of another autoimmune pathology;
- catastrophic – acute coagulopathy that occurs with multiple thrombosis of internal organs;
- AFL is a negative variant of antiphospholipid syndrome, in which serological markers of the disease (At to cardiolipin and lupus anticoagulant) are not detected.

Symptoms

According to modern views, antiphospholipid syndrome is an autoimmune thrombotic vasculopathy. In APS, the lesion can affect vessels of various caliber and localization (capillaries, large venous and arterial trunks), which causes an extremely diverse range of clinical manifestations, including venous and arterial thrombosis, obstetric pathology, neurological, cardiovascular, skin disorders, and thrombocytopenia.

The most common and typical sign of antiphospholipid syndrome is recurrent venous thrombosis: thrombosis of the superficial and deep veins of the lower extremities, hepatic veins, portal vein of the liver, and retinal veins. Patients with antiphospholipid syndrome may experience recurrent episodes of PE, pulmonary hypertension, superior vena cava syndrome, Budd-Chiari syndrome, and adrenal insufficiency.

Venous thrombosis in antiphospholipid syndrome develops 2 times more often than arterial thrombosis. Among the latter, cerebral artery thrombosis prevails, leading to transient ischemic attacks and ischemic stroke. Other neurological disorders may include migraines, hyperkinesis, convulsive syndrome, sensorineural hearing loss, ischemic optic neuropathy, transverse myelitis, dementia, and psychiatric disorders.

Damage to the cardiovascular system in antiphospholipid syndrome is accompanied by the development of myocardial infarction, intracardiac thrombosis, ischemic cardiomyopathy, and arterial hypertension. Quite often, there is a lesion of the heart valves-from minor regurgitation detected by echocardiography, to mitral, aortic, tricuspid stenosis or insufficiency. As part of the diagnosis of antiphospholipid syndrome with cardiac manifestations, differential diagnosis with infectious endocarditis, myxoma of the heart is required.

Renal manifestations may include both mild proteinuria and acute renal failure. On the part of the gastrointestinal tract, antiphospholipid syndrome is associated with hepatomegaly, gastrointestinal bleeding, mesenteric vascular occlusion, portal hypertension, and spleen infarction. Typical lesions of the skin and soft tissues are represented by retinal livedo, palmar and plantar erythema, trophic ulcers, gangrene of the fingers; musculoskeletal system-aseptic

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necrosis of the bones (femoral head). Hematological signs of antiphospholipid syndrome include thrombocytopenia, hemolytic anemia, and hemorrhagic complications.

In women, APS is often detected in connection with obstetric pathology: repeated spontaneous termination of pregnancy at various times, delayed intrauterine development of the fetus, fetoplacental insufficiency, gestosis, chronic fetal hypoxia, and premature birth. When managing pregnancy in women with antiphospholipid syndrome, the obstetrician-gynecologist should take into account all possible risks.

Diagnostics

Given the multiple organ involvement, the diagnosis and treatment of antiphospholipid syndrome requires combining the efforts of doctors of various specialties: rheumatologists, cardiologists, neurologists, obstetricians and gynecologists, etc. Antiphospholipid syndrome is diagnosed on the basis of clinical (vascular thrombosis, burdened obstetric history) and laboratory data. The diagnosis is considered reliable if at least one basic clinical and laboratory criterion is combined:

- Laboratory markers. The main immunological criteria include detection in blood plasma of medium or high titers of At to cardiolipin IgG/IgM class and lupus anticoagulant twice within six weeks. Additional laboratory signs of antiphospholipid syndrome are false positive RW, positive Coombs reaction, increased titer of antinuclear factor, rheumatoid factor, cryoglobulins, and antibodies to DNA. The study of UAC, platelets, biochemical blood analysis, and coagulogram is also shown.
- Instrumental diagnostics. To confirm thrombosis of internal organs, ultrasound examination of the vessels of the head and neck, kidney vessels, arteries and veins of the extremities, eye vessels, etc. is performed. Changes in the valves of the heart valves are detected during echocardiography.
- Examination during pregnancy. Pregnant women with antiphospholipid syndrome need monitoring of blood coagulation parameters, dynamic ultrasound of the fetus and Dopplerography of uteroplacental blood flow, cardiotography.

Differential diagnostic measures should be aimed at the exclusion of:

- DIC-syndrome;
- hemolytic-uremic syndrome;
- thrombocytopenic purpura, etc.

The main goal of antiphospholipid syndrome therapy is to prevent thromboembolic complications. Regime moments provide for moderate physical activity, refusal to stay stationary for a long time, engage in traumatic sports and long air travel. Women with antiphospholipid syndrome should not be prescribed oral contraceptives, and before planning pregnancy, it is necessary to consult an obstetrician-gynecologist. During the entire gestation period, pregnant patients are shown to take small doses of glucocorticoids and antiplatelet agents, administer immunoglobulin, and inject anticoagulants under the control of hemostasiogram parameters.

Drug therapy for antiphospholipid syndrome may include the appointment of indirect anticoagulants, direct anticoagulants, and antiplatelet agents. Preventive anticoagulant or antiplatelet therapy for most patients with antiphospholipid syndrome is carried out for a long time, and sometimes for life. In the catastrophic form of antiphospholipid syndrome, high doses

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of glucocorticoids and anticoagulants are indicated, plasmapheresis sessions, transfusion of fresh frozen plasma, etc.

Forecast

Timely diagnosis and preventive therapy can avoid the development and recurrence of thrombosis, as well as hope for a favorable outcome of pregnancy and childbirth. In secondary antiphospholipid syndrome, it is important to monitor the course of the underlying pathology and prevent infections. Prognostically unfavorable factors include a combination of antiphospholipid syndrome with SLE, thrombocytopenia, rapid increase in the At titer to cardiolipin, and persistent arterial hypertension. All patients diagnosed with antiphospholipid syndrome should be monitored by a rheumatologist with periodic monitoring of serological markers of the disease and hemostasiogram indicators.

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