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ISCHEMIC STROKE IN A PATIENT WITH SYSTEMIC LUPUS ERYTHEMATOSUS AND SECONDARY ANTIPHOSPHOLIPID SYNDROME

Abstract

The article presents a clinical case of an onset of antiphospholipid syndrome in a patient with systemic lupus erythematosus and ischemic stroke. Systemic lupus erythematosus is a non-modifiable risk factor for ischemic stroke.

Key words: *risk factors, ischemic stroke, systemic lupus erythematosus, antiphospholipid syndrome*

Conflict of interests

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BP — blood pressure, APS — antiphospholipid syndrome, IS — ischemic stroke, SLE — systemic lupus erythematosus

The main causes of ischemic strokes (IS) are considered hypertension, atherosclerosis, cardiac disorders, diabetes mellitus. However, sometimes a key role in the development of "vascular catastrophe" can be played by rarer causes, such as autoimmune diseases.

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease of unknown etiology characterized by hyperproduction of organ-specific autoantibodies to various components of the cell nucleus with the development of immune inflammatory damage to tissues and internal organs. Below is a clinical case, a feature of which is the development of IS due to secondary antiphospholipid syndrome (APS), outside the clinical signs of SLE activity.

A female patient, 48 years old, was admitted to hospital with complaints of pronounced weakness in the left extremities, impaired speech quality. "Classic" vascular risk factors (smoking, alcohol abuse, hypercholesterolemia and others) could not be identified. From medical history it is known that patient A. suffers from SLE for a long time, for treatment of which she takes prednisolone in a daily dose of 2.5 mg for the last 8 years. There were no previous thrombotic episodes. In addition, for the correction of blood

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pressure (BP) she takes Diroton at a dose of 2.5 mg once a day. BP variations began to raise concerns during therapy with corticosteroids. The highest BP was 140/90 mm Hg. According to the patient, in the middle of the day she felt sick; while trying to get up, she fell, unable to keep her balance. Witnesses called the ambulance team, and she was hospitalized in the Neurological Department for patients with acute cerebrovascular accident. On admission, the patient's condition was assessed as severe. Her somatic status was stable. BP was 160/90 mm Hg. Heart rate was 68 bpm. Respiratory rate was 18 breaths per minute. According to neurological examination, her consciousness is clear. Regarding cranial nerves function, the smoothness of the nasolabial fold on the left and the deviation of the tongue to the left were determined. No movement in the left extremities was revealed. Anisoreflexia, S > D. Positive pathological reflexes (carpal Rossolimo sign, Babinsky reflex) were revealed on the left. In the rest, state with no features. According to laboratory examination methods, the following changes were revealed: hemoglobin 111 g/l, hematocrit 32.7 %, platelets 128×10^9/l, fibrinogen 5.4 g/l, ESR 20 mm/h. Lupus anticoagulants: the result is doubtful, 1.2 (norm <1.2) in the confirming test; anti-DNA antibodies are found: COI 163.5 (norm up to 25), anti-cardiolipin Abs 50.38 (norm up to 10 U/ml). ECG: sinus rhythm, increased load on the left ventricle, diffuse changes in myocardium. Spiral brain computed tomography revealed a violation of cerebral circulation of ischemic nature in the right parietal, frontal and temporal lobes. According to the results of duplex scanning, brachiocephalic arteries are passable, additional formations in the arteries were not revealed. Due to closed ultrasonic temporal acoustic windows, the study of the intracranial arteries is impossible. By echocardiography, no abnormalities, the ejection fraction is 64 %. Consultation was performed, and the following diagnosis was made: "Systemic lupus erythematosus, chronic course, with activity III, with the skin, blood vessels, liver, gastrointestinal tract, kidneys, nervous system, joints, and heart affected. Secondary antiphospholipid syndrome with damage to the central nervous system. Ischemic stroke in the territory supplied by the right middle cerebral artery. Mild dysarthria. Left hemiplegia." Multisystem lesion occurred in previous exacerbations of SLE, and this information was obtained from the patient's medical records. Pulse therapy was performed: solution of prednisone 1,000 mg No. 3 with subsequent switch to the tablet form of 60 mg per day, solution of cyclophosphamide 1,000 mg No. 1; antiplatelet therapy was prescribed including Cardiomagnyl 75 mg 1 tablet per day. During the stay of the patient in the Department: in neurological status without positive changes with preservation of left-sided hemiplegia. After 6 weeks, laboratory re-examination of blood — antibodies against phospholipids of 53.48 U/ml, antibodies against cardiolipin of 65.16 U/ml.

According to the 2012 American Rheumatology Association SLICC SLE, among central nervous system lesions, seizures and psychosis are included in SLE. At the same time, SLE with the ongoing secondary APS is much more often (in 20–30 % of cases) complicated by IS or transient cerebrovascular disease [1]. APS is a clinical and laboratory syndrome characterized by the formation of antibodies against own phospholipids in combination with autoimmune damage to systems and organs, most often in the form of venous and arterial thrombosis of any localization, obstetric pathology (intrauterine fetal death, miscarriage, abortion) and thrombocytopenia. The basis of occlusion in APS is non-inflammatory thrombotic vasculopathy. In addition, SLE and APS are actually risk factors for an earlier IS onset. This is due to additional risk factors that are typical for this pathology — immunopathological damage to blood vessels, persistence of immune complexes in the blood, specific drug therapy (glucocorticosteroids, hydroxychloroquine, cyclophosphamide). A number of studies have shown an earlier onset of cerebrovascular diseases in patients with SLE than in the control healthy group [2, 3]. There is also a tendency to recurrent IS, which took place in our patient a year later, in the territory supplied by the left middle cerebral artery with the formation of a focus in the parietal temporal lobe with the development of subtotal sensorimotor aphasia, despite the intake of

antiplatelets, anticoagulants and a maintenance dose of prednisolone.

Thus, the development of stroke in patients with SLE may be due to secondary APS and requires the assay of IgG ABs against phospholipids, lupus anticoagulant and determining the activity of the disease with the assay of antinuclear antibodies and antibodies to double-stranded DNA. For the prevention of cerebrovascular disease in SLE it is necessary to carry out the correction of risk factors (level of SLE activity, dyslipidemia, hypertension) to prevent thrombotic complications of oral anticoagulants and antiplatelet agents.

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