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Title: Dermatological manifestation of antiphospholipid antibody syndrome

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Introduction. There is a group of coagulation inhibitors, which are antiphospholipid antibodies (APLA). In patients with different phospholipid antibodies, recurrent thrombolytic therapy complications, brain lesions and pregnancy pathology were observed, which allowed identifying APLA syndrome

Materials and methods. During the study of changes in the coagulation, anticoagulation and fibrinolysis systems in dermatology patients, we examined three patients whose clinical and morphological manifestations of the disease allowed diagnosing them with thrombotic microangiopathy, characteristic of APLA syndrome (patient S. with multiple ulcerative and necrotic skin changes within 12 hours after taking Analgin, patient Ch. with multiple painful nodules on the upper and lower limbs, which had been observed for 10 years, dense-elastic, soldered to the skin, patient T. with multiple ulcers on the anterior surface of the lower legs that were unresponsive to treatment.

Results. The testing of hemostasis in all patients indicated the presence of DIC syndrome. The auto coagulation test (ACT) for the hypercoagulable type, the coagulation time at the 2nd minute was (%): control group - 23.48 ± 2.26; patient S. - 44.00; patient Ch. - 34.00, patient T. - 59.0. An elevated total fibrinogen level (g/l) was observed: control group 3.51±0.17, patient S. - 6.22, patient Ch. - 6.73, patient T. - 7.12; blood clot retraction (%): control group 67.52±1.37; patient S. - 100.01, patient Ch. - 105.72, patient T. - 95.31. A decreased spontaneous fibrinolysis (%) was observed: control group 16.31 ± 0.72; patient S. - 9.25, patient Ch. - 10.83, patient T. - 7.33; activated recalcification time (s): control group 64.05 ± 1.02, patient S. - 34.00, patient Ch. - 25.00, patient T. - 60.00; thrombin time (s): control group 16.30 ± 0.20; patient S. - 14.00, patient Ch. - 11.00, patient T. - 9.00. Prolonged euglobulin lysis time (min): control group - 316.1 ± 11.7; patient S. - 580.0, patient Ch. - 520.0, patient T. - 520.0; blood coagulation time according to Lee-White: control group - 559.1 ± 11.6; patient S. - 600, patient Ch. - 600, patient T. - 720; antithrombin III (%): control group - 100.0 ± 0.10, patient S. - 46.68, patient Ch. - 57.52, patient T. - 61.04. Increase in fibrinogen degradation products (μg/ml): control group - 2.10±0.09, patient S. - 128.0, patient Ch. - 256.0, patient T. - 64.

The results of a skin biopsy of the patient S.: under the unaltered epidermis in the venules and small veins of the reticular layer of the dermis and subcutaneous fatty tissue, there were multiple fibrin thrombi obstructing the lumen; unaltered vessel walls; mild perivascular, lymphohistiocytic infiltration. Patient Ch.: the integrity of the epidermis was intact; within the dermis and subcutaneous fat, conglomerates of dilated vessels of capillary type were visible, obturated with fibrin thrombi; thin vessel walls, swollen endothelial cells; endothelial proliferation in some areas. Patient T.: granulation tissue with slight lymphocytic infiltration in the area of the ulcer defect; some blood vessels of the dermis with signs of fibrinoid swelling in the underlying areas; fibrin and fibrin-platelet thrombi in the lumen of the vessels.

Discussion. Thus, the skin is an important indicator of the intensity and severity of DIC. The combination of morphological changes with clinical and laboratory tests will reveal its pathogenesis and will be the basis for diagnosing the internal pathology.