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STUDY OF BLOOD COAGULATION PARAMETERS AND ACTIVITY INDICES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS

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Annotation

Objective. To evaluate the blood coagulation (hemostasis) indicators and disease activity indices in patients with systemic lupus erythematosus (SLE).

Materials and methods. A total of 75 patients (73 women and 2 men) with SLE, who were under observation at the Rheumatology Center of the 1st Clinic of Tashkent Medical Academy, were involved in the study. The patients were diagnosed with SLE based on the criteria of the American College of Rheumatology (ACR), and the APS diagnosis was made according to the Sapporo criteria. SLE activity was evaluated based on SLAM, SLEDAI, and ECLAM indices.

Results. In patients with SLE: Spontaneous platelet aggregation increased 5 times ($p<0.01$); the concentration of von Willebrand factor antigen (FV:Ag) increased more than 3 times; platelet activation was observed along with a 27% decrease in aggregation with collagen ($p<0.01$); a direct correlation was found between activity indices and the following indicators: RFMK (ECLAM, $r=0.51$), Fibrinogen (SLAM, $r=0.34$), D-dimer (ECLAM, $r=0.5$), Spontaneous platelet aggregation (ECLAM, $r=0.5$). AT III activity showed a negative correlation with SLEDAI: $r = -0.73$.

Conclusion. Hemostasis system changes in SLE may indicate the development of thrombotic disorders in advance. At the same time, these indicators indicate the need for correction with medications. There is a direct relationship between the activity of the coagulation system and SLE activity indices.

Keywords: SLE, antithrombin III, D-dimer, activity indices, soluble fibrin-monomer complexes.

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease with a wide range of clinical manifestations. The clinical picture of systemic lupus erythematosus has a number of specific features and is characterized by a polysyndromal pattern, rapidly progressing, and often fatal as a result of the rapid development of functional insufficiency of one or another internal organ. The disease often begins with general weakness, rapid weight loss and a long-term persistent

increase in temperature, joint syndrome, and in some cases Raynaud's phenomenon, and then develops into damage to internal organs and systems. syndromes characteristic of inflammation occur.In patients with SJS, increased apoptosis of endothelial cells is associated with increased tissue factor levels, which leads to accelerated atherothrombotic processes [8]. Molecular links between inflammation and coagulation are undeniable: inflammation activates hemostasis through intravascular tissue factor expression, activation of adhesion molecules, and inhibition of fibrinolytic and anticoagulant systems; thrombin, in turn, promotes the development of inflammation [9]. Inflammatory mediators (e.g., endotoxin and TNF-α) can cause the release of tissue factor from monocytes and possibly from the endothelium, thereby initiating the coagulation cascade [10,11]. The interest in studying the activation of procoagulant mechanisms in SJS is reflected in several scientific studies. In particular: a correlation has been found between some coagulation parameters and disease activity indices (SLEDAI and SLAM) [12,13]; a correlation has been noted between coagulation markers and plasma α-DNA levels in patients with active SQY; a pro-thrombotic state and compensatory fibrinolytic processes are secondary to subclinical intravascular coagulation [14]; coagulation changes are especially pronounced in SQY patients with secondary AFS [15,16]. However, some studies have not found a correlation between hemostasis disorders and SQY activity [17,18]. Therefore, further studies are warranted to determine the relationship between inflammation and blood coagulation processes in patients with SQY.

Materials and Methods

A total of 75 patients under observation at the IADK Rheumatology Center of the 1st Clinic of the Tashkent Medical Academy participated in the study.SQY Patients (73 women and 2 men) with rheumatoid arthritis were enrolled. Patients were diagnosed with SQY based on the American College of Rheumatology (ACR) criteria, and AFS was diagnosed based on the Sapporo criteria. Disease activity was assessed using the following indices: ECLAM [21], SLEDAI [22], SLAM [23].

Inspections:

The studies included a comprehensive assessment of hemostasis and immunological parameters, and all analyzes were performed before prescribing (or changing) medications. The following laboratory methods were used: Coagulometric analyses: Platelet count was calculated. Activated partial thromboplastin time (APTT) was determined. Thrombin time (TPT) was measured. Prothrombin activity was assessed by the Quick method. Fibrinogen concentration was determined by the RA Rutberg method. Soluble fibrin-monomer complexes (RFMK) were assessed using the orthophenanthroline test. Protein S activity was measured. All studies were carried out according to the instructions developed on the basis of reagents from the manufacturer "Technology-Standard" in Barnaul [24]. The amount of D-dimer was assessed using the latex test method from the company "Roche" (D-dimer-Test). Platelet aggregation: According to the GVR Born method, using the laser aggregation analyzer "Biola 230 LA": Spontaneous aggregation, Ristocetin, ADP and collagen-induced aggregation were assessed (inducers - "Sigma", USA) [25].

Immunological studies: Anti-cardiolipin antibodies (aKL) — IgG and IgM isotypes — were detected using solid-phase ELISA.

Antibodies against natural (native) DNA were measured using an equimolar ELISA using ready-made kits manufactured by Sigma Chemical (USA). Antithrombin III concentration was determined using an equimolar ELISA using reagents from DAKO (Denmark) and international standards from Sigma (USA). Von Willebrand factor antigen (FV:Ag) was determined using an equimolar immunoenzyme method using reagents from DAKO (Denmark) and international standards from NIBSC (England).

Results and discussion

Prevalence of clinical signs. The most common clinical signs among patients with SQY were: Arthralgia (joint pain) – 73.3% of cases, Photosensitivity – 65.1%, "Butterfly" or "winged" rash on the face – 59.3%, Central nervous system (CNS) involvement – 55.5%, Headache/migraine – 46.5%, Kidney damage – 43%, Reticular livaedo (network-like appearance of skin capillaries) – 21%. Among the clinical signs of hemostasis system disorders, the following prevailed: Phlebitis (inflammation of the vessel wall) – 22.1% of patients, History of acute cerebral circulation disorders (ACC) – 9%, Phlebothrombosis at the time of examination – in 5 patients (5.8%), Trophic ulcers on the soles of the feet – 8.1% of patients.

Hemostasiological and biochemical indicators

According to the results of the analysis (based on the table): The platelet count was similar in patients and controls, and 19% of patients with SQY had thrombocytopenia. Among patients with AFS, this was 50%. In patients with SQY: Spontaneous platelet aggregation increased 4 times ($p<0.01$), FV:Ag concentration increased more than 3 times, Plasma serotonin levels were significantly higher. This indicates that platelet activity occurs against the background of damage and activation of endothelial cells. This is confirmed by the following correlations: between the level of FV:Ag and spontaneous aggregation ($r=0.41$; $p<0.01$), between spontaneous aggregation and the Livaedo sign ($r=0.38$; $p<0.01$). Platelet aggregation with collagen decreased by 27% ($p<0.01$). This was correlated with: a decrease in platelet serotonin levels ($r=0.25$; $p<0.01$), an increase in plasma serotonin and adrenaline levels ($r=-0.24$; $p<0.01$). A decrease in platelet storage granules, although rare, was manifested in the form of petechiae (hemorrhagic spots). This was also confirmed by correlation analysis ($p<0.01$). In addition, spontaneous aggregation and increased aggregation with ADP were associated with the following thrombotic signs: Leg ulcers ($r=0.36$; $p<0.01$). Possible cause of impaired platelet aggregation with collagen: The presence of antibodies against glycoprotein receptors (GP IIb/IIIa, GPIa/IIa, GPIb/IX). This is confirmed by the following correlations: Between aggregation with collagen and the level of IgG anti-cardiolipin ($r=-0.33$; $p<0.01$), aggregation with collagen and IgM anti-cardiolipin ($r=-0.39$; $p<0.01$). Changes in coagulation hemostasis In patients with SQY, along with platelet-vascular hemostasis, plasma hemostasis was also significantly activated. Coagulation tests showed a hypercoagulable state in SQY patients. However, the results of the analysis were very different, and in some cases even hypocoagulable states were noted. .Signs of coagulation activity: The amount of soluble fibrin-monomer

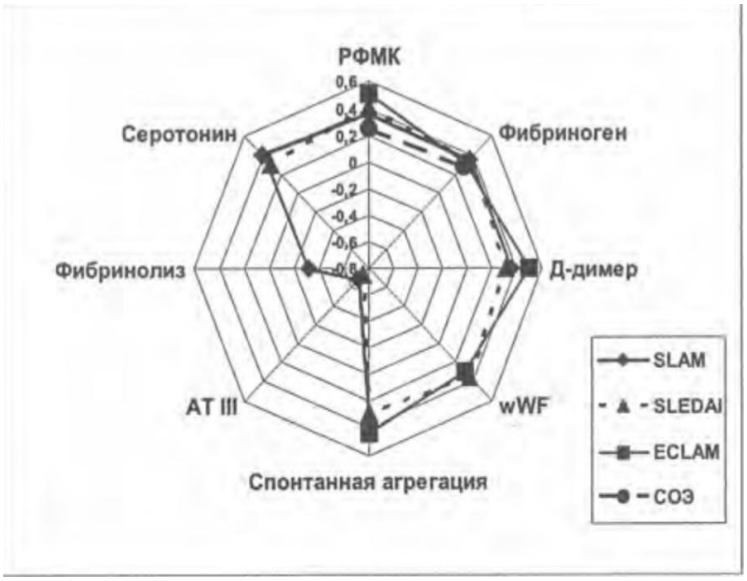
complexes (RFMK): increased by 81% ($p<0.01$), which is the main indicator of coagulation activity. The level of D-dimer was increased in 53.3% of patients. The correlation analysis proved that these indicators are associated with thrombotic complications: RFMK and livaedo: $r=0.46$ ($p<0.01$), RFMK and trophic ulcers: $r=0.25$ ($p<0.01$), RFMK and headache: $r=0.38$ ($p<0.01$), RFMK and thrombophlebitis: $r=0.22$ ($p<0.01$). Conclusion:SQYThe tendency to thrombosis in is probably the result of a subclinical intrinsic coagulation state associated with endothelial damage, as evidenced by the high level of FV:Ag.

Changes in the anticoagulant system

Antithrombin III (AT III) and protein S levelsSQYdecreased in patients with ($p<0.05$). The spontaneous fibrinolysis index decreased almost 2-fold ($p<0.01$). Patients with decreased AT III levels had a higher frequency of the following clinical signs:

Relationship between activity indices and hemostasis indicators

SQY The role of hemostasis disorders in the pathogenesis of CKD, the relationship between immune factors, coagulation and platelet hemostasis disorders, was proven by correlation analysis. The analysis was carried out with SQY activity indices – SLAM, SLEDAI, ECLAM – and SOE (erythrocyte sedimentation rate). Monitoring levels of erythrocyte sedimentation rate (ESR), D-dimer, fibrinogen, spontaneous platelet aggregation, FV: Ag, and AT III is important for predicting thrombotic disorders in SQY and determining treatment strategies. Reliable correlations between hemostasiological indicators and SLE (systemic lupus erythematosus) activity.



Conclusion

It is common for patients with SQY to have abnormalities in the coagulation system, which corresponds to the characteristics of persistent intravascular coagulation syndrome (DVS) grade 3 [28]. In particular, hypofibrinogenemia and thrombocytopenia are observed, especially in patients with secondary AFS. Activation of the blood coagulation system in SQY is mainly

accompanied by a sharp increase in platelet-vascular hemostasis, and to a lesser extent by activation of the coagulation system. These changes are accompanied by: the acquisition of procoagulant properties of endothelial cells as a result of immune aggression, the weakening of fibrinolysis, and a decrease in the activity of the anticoagulant system.

References

1. Nasonov Ye.L. Evaluation of the role of digital immunity and inflammation // Rheumatic diseases, 1997.
2. Dixit K., AN R. Nitrate-modified DNA and the etiology of SQY // Lupus, 2004.
3.Nasonova BA, Folomeeva OM Mortality in SQY // Nauchno-prakticheskaya rheumatologiya, 2001.
4. Manger K. et al. Risk factors in a cohort of 338 patients with SQY // Ann. Rheum. Dis., 2002.
5. Cervera R. et al. Analysis of 1000 patients // Medicine (Baltimore), 2003. Manger K.
6. Becker BF, Heindl B., Kupatt C., Zahler S. Endothelial function and hemostasis. Z. Cardiol., 2000, 89(3), 160-167
7. Ekdahl KN, Ronnblom L., Sturfelt G., Nilsson B. Increased phosphate content in complement component C3, fibrinogen,vitronectin, and other plasma proteins in systemic lupus erythematosus: covariation with platelet activation and possible association with thrombosis. Arthr. Rheum., 1997, 40(12), 2178-2186
8. Rajagopalan S., Somers EC, Brook RD et al. Endothelial cell apoptosis in systemic lupus erythematosus: a common pathway for abnormal vascular function and thrombosis propensity. blood, 2004,-103(10), 3677-3683
9. Esmon CT, Fukudome K., Mather T. et al. Inflammation, sepsis, and coagulation. Hematolog., 1999, 84(3), 254-259
10. Ekdahl KN, Bengtsson AA, Andersson J. et al. Thrombotic disease in systemic lupus erythematosus is associated with a maintained systemic platelet activation. Br. J. Haematol., 200411. Esmon CT Does inflammation contribute to thrombotic events?
Haemostas., 2000, 30 (suppl. 2), 34-40
12. Inoh M., Tokuda M., Kiuchi H. et al. Evaluating systemic lupus erythematosus disease activity using molecular markers of hemostasis. Arthr. Rheum., 1996, 39(2), 287-291 preliminary classification criteria for definite antiphospholipid syndrome. Arthr. Rheum., 1999, 42, 1309-1311 and atherosclerosis in rheumatoid arthritis. J. Rheumatol., 2002, 29(5), 875-882
13.Bokarev IN Differential diagnosis and treatment vnutrennix bolezney: Blood clots, ili hemorrhagichesky syndrome. Differential diagnosis and treatment. Moscow, 2002, 76.