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# NORMAL AND PATHOLOGICAL PHYSIOLOGY

# A CLINICAL CASE IN THE PRACTICE OF A RHEUMATOLOGIST: SYSTEMIC LUPUS ERYTHOMUS WITH INFLAMMATION OF ORGANS AND SYSTEMS

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#### **Abstract**

Systemic lupus erythematosus (SLE) is one of the most serious systemic connective tissue diseases that develops more often in young women and girls, although it can occur at any age. Patients with SLE still have a high risk of death (3 times higher than in the general population). Early diagnosis, intensification of basic therapy and minimization of the dose of glucocorticoids (GC), assessment of cardiovascular risk and treatment of cardiovascular diseases are considered to be the basis for improving the prognosis in SLE.

**Keywords:** systemic lupus erythematosus, facial erythema, polyarthritis, pneumosclerosis, myocardiofibrosis.

#### Introduction

Rheumatology is one of the most diagnostically complex medical disciplines, which is at the intersection of many specialties. A rheumatologist needs a high level of professional skills, because he often has to diagnose conditions that can hide under the mask of rheumatological symptoms. One of the most difficult groups of rheumatological diseases to differentiate is systemic lupus erythematosus.

Systemic lupus erythematosus (SLE) is a systemic autoimmune polyetiological disease characterized by hyperproduction of autoantibodies to various components of the cell nucleus with the development of immunoinflammatory damage to tissues and internal organs [1, 7]. In all age groups, the incidence is higher among women, especially those of reproductive age, when the ratio of the incidence of women to men is 8:1-15:1, while this pattern is least pronounced in prepubertal age — 4:3 [2, 11] According to over the past 60 years, the 5-year survival of patients with SLE has increased from 50 to 91–97% [16, 18]. Such an improvement is primarily associated with timely diagnosis, and therefore early initiation of therapy, and management of patients based on the principle of "treatment until the goal is achieved" - stable clinical and laboratory remission [12].

SLE is accompanied by multiple organ damage and the development of various complications, including both typical and rare ones. In particular, one of the main reasons for the unfavorable prognosis of the disease is kidney damage [5]. Despite progress in the diagnosis and treatment of this pathology, kidney damage remains the leading cause of death in patients with SLE. Many such patients develop chronic kidney disease (CKD), as well as complications, concomitant diseases and conditions that are a consequence of immunosuppressive therapy: infections, osteoporosis, damage to the cardiovascular and reproductive systems, etc. [3, 4]. Regarding methods of treatment of lupus nephritis in SLE, many studies are being conducted, new promising

drugs are appearing [9, 15]. CKD is also associated with the development of vitamin D deficiency, systemic osteoporosis, and secondary hyperparathyroidism [6].

According to the literature, there are 5 main types of soft tissue calcification: dystrophic, metastatic, idiopathic, iatrogenic, and calciphylaxis [10, 17]. Most often, in systemic connective tissue diseases, a dystrophic process is noted, which consists in secondary tissue damage and the formation of calcifications [8]. It is generally accepted that soft tissue calcifications are detected in dermatomyositis, systemic scleroderma, CREST syndrome, overlap syndrome, but they are rare in SLE [13, 14].

We bring to your attention a clinical case of the development of myocardial cardiomyopathy, pneumosclerosis, polyarthritis and dyscirculatory encephalopathy in a patient with SLE.

**Purpose:** to describe a clinical case of the development of damage to all organs and systems in a patient with SLE and to prescribe combined treatment depending on the damage to the relevant body systems.

### Materials and methods

Patient V., 50 years old, complains of difficulty in self-care, pain and stiffness in the joints of the hands and feet, periodic aching pain in the area of the heart, palpitations, rapid fatigue, coldness of the hands, suffocation during physical exertion.

She considers herself a patient for 11 years. He is periodically treated as an inpatient. Systematically takes GC (diprospan, medrol). In connection with deterioration of well-being and strengthening of the abovementioned symptoms, she was sent for inpatient treatment in the rheumatology department of the OKL.

**Objectively** upon admission: the general condition of the patient is of moderate severity. Normosthenic. Swelling of the feet. Body temperature is 37.1°C. Pulse 82 per minute, satisfactory properties. Blood pressure 140/90 mm Hg. The rhythm of cardiac activity is correct, the ratio of tones is preserved. In the

lungs, percussion is clear pulmonary sound, auscultation - vesicular breathing, basally weakened. Respiratory rate - 22 per minute. The tongue is wet, covered with a white coating. The abdomen is soft, sensitive in the epigastric region, in the left hypochondrium, the edge of the liver is sensitive to palpation. Symptom Pasternacki is negative on both sides. Limitation of movements in the radiocarpal joints: right - dorsiflexion  $0^0$ , palmar flexion -  $25^0$ , left - dorsiflexion  $0^0$ , palmar - 25°. Left shoulder joint: flexion 90°, abduction 90°. Knee joints: right flexion 40°, extension 180°, left flexion 45°, extension 175°. Ankle-foot joints: right dorsiflexion - 30<sup>0</sup>, plantar flexion - 10<sup>0</sup>, left dorsiflexion 30°, plantar 10°. Contractures of both radiocarpal joints. Contracture of the left shoulder joint. Extensor contracture of the right knee joint with impaired function. Flexion-extension contracture of the left knee joint without impaired locomotor function. Contractures of both ankle-foot joints with impaired function. Pain along the paravertebral points of the thoracic spine.

#### **Conducted examination:**

PRS from 06.22.23 - negative. Blood group B(III), Rh-positive

General blood test from June 21, 2023 - erythrocytes-  $3.4 \times 10^{12}$ /l, Hb - 106 g/l, k.p. - 0.9, platelets-  $216 \times 10^9$ , leukocytes-  $5.1 \times 10^9$ /l, eosinophils - 2%, rod nuclear - 2%, segment nuclear- 70%, lymphocytes - 25%, monocytes - 1%; erythrocyte sedimentation rate -34 mm/h.

Biochemical analysis of blood from July 1, 2023. protein 76.0 g/l, urea – 6.8 mmol/l, total bilirubin – 11.0  $\mu$ m/l, blood glucose – 3.6 mmol/l.

Biochemical analysis of blood from July 9, 2023. protein 77.0 g/l, urea – 6.7 mmol/l, total bilirubin – 18.0  $\mu$ mol/l, blood glucose – 5.1 mmol/l, AsAT – 0.2  $\mu$ mol/l, AlAT – 0.41  $\mu$ mol/l, thymol test – 1.0 units, urea – 6.6 mmol/l l, cholesterol - 4.2 mm/l.

Blood glucose as of March 7, 2023 - 6.4 mmol/l. Rheumatic tests from June 23, 2023 - ASLO - 200; CRP - 42 mg/ml, sialic test - 328 units, sulfur mucoid -280 units.

General urine tests from 01.07.23 - 100 ml, light yellow, transparent, specific weight- 1012, weakly acidic, single transitional epithelium, single leukocytes, single bacteria, protein, glucose - not found.

Daily glucosuria 06/21/23 1.00 l

Fecal analysis from June 21, 2023 – no helminth eggs were found.

Fluorography of the chest organs from June 29, 2023 – lungs and heart within normal limits.

Electrocardiography from July 1, 2023 – sinus rhythm, regular, heart rate 87 beats per minute. Changes in the myocardium of the back wall of the left ventricle, hypertrophy of the myocardium of the left ventricle.

Ultrasound diagnostics of the abdominal cavity from June 24, 2023 - vertical size of the right lobe of the liver - 152 mm, increased echogenicity. The gallbladder is oval in shape, contains echogenic bile, the walls are compacted. Pancreas of increased echogenicity. Spleen 110x41mm, homogeneous. The right kidney is moderately lowered, N sizes. In both kidneys,

the calyces are compacted, casts of salts, the parenchyma is preserved. Signs of chronic cholecysto-pancreatitis.

Ultrasound diagnostics of the thyroid gland (thyroid gland) - both lobes of the thyroid gland are of normal size, the tissue of the thyroid gland is homogeneously heterogeneous, more on the right. Signs of diffuse goiter.

Consultation of a gynecologist on November 6, 2023, she is healthy.

Consultation of an endocrinologist - Diffuse non-toxic goiter II st. Autoimmune thyroiditis.

The diagnosis was made: Systemic lupus erythematosus, chronic course, activity of the 1 degree, with lesions of the skin (erythema of the face, décolletage, reticular liver), heart (myocarditic myocardiofibrosis, arrhythmic variant of Heart failure IIA, functional class II), lungs (pneumosclerosis Pulmonary failure I), joints (Polyarthritis Ro II stage, Joint functional insufficiency II stage, contractures of carpal, knee, ankle-foot joints), nervous system (dyscirculatory encephalopathy), RES (lymphoadenopathy).

And the treatment carried out: diet No. 10, medrol, dexamethasone, panangin, renalgan, corvitin, tivortin, reosorbilact, metamax, thiotriazoline, neocardil, aspecard, megafen, calcium-D3 nicomed. Passed health school No. 6.

On July 6, 2023, she was discharged home to continue outpatient treatment.

#### **RECOMMENDED** in the future for the patient:

- 1. "D" registration with a rheumatologist, endocrinologist, gastroenterologist at the place of residence.
- 2. Methylprednisolone 20 mg (according to the scheme).
  - 3. Plaquenil 1 tablet at night- 6 months
- 4. Calcium D3 Nikomed 1 tablet x 2 r per day during the period of taking GC.
- 5. Pantaprozole 40 mg 1 tablet x 1 per day during the period of GC reception.
  - 6. Curantyl 1 tablet x 2 times (2 weeks).
- 7. Panangin 1 tablet x 3 times a day the entire period of taking GCS
- 8. Sedative therapy (barboval, motherwort, sedative herbal preparation).
  - 9. Observe the regime of work and rest.

### CONCLUSION

A large number of pathological conditions unrelated to rheumatology can be hidden under the mask of rheumatological symptoms. Systemic lupus erythematosus, which is characterized by polymorphism of manifestations, requires special attention. The presence of comorbid pathology in patients with systemic lupus erythematosus creates problems for verifying the diagnosis of this disease and conducting high-quality multisymptom treatment.

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