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DEBUT OF SYSTEMIC LUPUS ERYTHEMATOSUS IN CHRONIC GOUT ARTHRITIS (PRACTICAL CASE)

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ABSTRACT

Systemic lupus erythematosus (SLE) is an autoimmune systemic inflammatory disease with a variety of clinical manifestations. Gout is a systemic tophi disease characterized by the deposition of crystals of sodium monourate in various tissues and developing in connection with this inflammation in persons with hyperuricemia (HU) caused by environmental and / or genetic factors. Currently, the number of young patients is growing steadily with an increase in the number of patients with gout among women. The combination of SLE and gout is rare, and there are very few descriptions of such clinical situations.

The article contains literature data concerning the combination of systemic lupus erythematosus (SLE) and gout. Peculiarities of pathogenetic mechanisms and therapeutic tactics in combination of two nosological forms are discussed. Based on the analysis of literature data, the article analyzes the features of the onset and clinical picture of SLE against the background of gout, discusses aspects of diagnosis verification, taking into account the need for early prescription of effective therapy and improvement of prognosis. The paper presents our own clinical observation of a combination of SLE and gouty arthritis in a young patient.

Key-words: systemic lupus erythematosus, gouty arthritis, diagnosis, treatment

INTRODUCTION

Gout is a systemic inflammatory disease associated with impaired uric acid (UA) metabolism, clinically manifested by recurrent arthritis and various extra-articular manifestations [5].

The incidence of gout ranges from 5 to 70 per 1000 population per year among men and 1–10 among women. Thus, the ratio of cases of pathology between men and women is on average 7: 1. The symptoms of gout in women are less pronounced due to the low level of uric acid in the blood and the higher level of estrogen in the blood [9].

Gout is manifested by impaired purine metabolism. It is expressed in the accumulation of uric acid and urate and the formation of crystals with their deposition in target organs. Violation of purine metabolism also entails a disorder of carbohydrate and fat metabolism. Genetic predisposition also plays a role in the etiology of gout, since there are a number of genes encoding UA metabolism [5].

The most important risk factor for developing gout is hyperuricemia; there was a positive correlation between the level of serum UA (SUA) and the frequency of seizures. The SUA level reflects the balance between its intake with food, synthesis and excretion, while in 90% of cases, gout is caused by insufficient excretion [15].

Causes of primary gout include isolated renal tubular defects with inadequate UA clearance and rare congenital metabolic disorders.

Secondary gout occurs in older people; it is caused by acquired conditions that affect the processing of nucleic acids or renal excretion of UA. These include myelo- and lymphoproliferative diseases, the use of cytotoxic drugs, fructose consumption, and metabolic syndrome [12].

Systemic lupus erythematosus (SLE) is a systemic autoimmune rheumatic disease of unknown etiology, characterized by overproduction of organ specific autoantibodies to various components of the cell nucleus and the development of immune-inflammatory damage to internal organs [10, 16]. SLE, a prototype of human systemic autoimmune pathology, is one of extremely heterogeneous diseases in terms of both clinical manifestations and genetic predisposition, and pathogenic mechanisms, which often complicates early diagnosis and does not allow personalizing therapy [17, 18].

A characteristic feature of SLE is the variety of debuts of the course and clinical manifestations. Usually, the disease begins with one or more symptoms: unexplained fever, weight loss, anemia, arthritis, skin lesions, Raynaud's phenomenon, serositis, renal disease, neurological disorders (convulsions or chorea), recurrent thrombosis. The clinical picture at the onset of the disease can be strikingly different from the "classical" descriptions of SLE, which often causes diagnostic difficulties not only for general practitioners, but also for rheumatologists. It is no coincidence that SLE is called the "chameleon disease" or "the great disease simulator": there are about fifty diseases requiring differential diagnosis with SLE, especially in the initial stages [11].

The relationship between purine metabolism disorders and immune system disorders is currently being widely discussed by specialists in various fields. Primary importance is given to the products of purine metabolism in the differentiation and proliferation of immunocytes. We have summarized numerous literature data concerning the development of immunodeficiency due to a lack of

enzymes of purine metabolism [17]. Thus, the problem of combining two nosological forms, their pathogenetic mechanisms are currently insufficiently studied, descriptions of clinical situations are rare, therefore the combination of SLE and gouty arthritis is of particular interest.

The aim of this work is to describe the case of the development of the onset of systemic lupus erythematosus in combination with gouty arthritis.

Materials and methods. Patient F., 30 years old, has been observed at the Republican Rheumatologic Center in Tashkent since 2013. She was admitted to the Department of Rheumatology of the Multidisciplinary Clinic of the Tashkent Medical Academy (TMA) with a diagnosis of Gouty arthritis. Systemic lupus erythematosus?

Complaints upon admission: fever, recurrent headaches, shortness of breath with little physical exertion, pain in the small joints of the hands and feet, dryness and redness of the skin, especially of the face and hands, general weakness, malaise.

Anamnesis of the disease: it is known that the patient has been complaining of pain in the area of small joints of the hands and feet, knee joints, morning stiffness for 1.5-2 hours for 10 years, periodical appearance of tophus in the area of the metacarpophalangeal joints of the hands and feet, auricles. The first 2-3 years from the onset of the disease was observed by a rheumatologist at the place of residence with a diagnosis of "Rheumatoid arthritis". She was repeatedly treated with various non-steroidal anti-inflammatory drugs and intra-articular injections of glucocorticoid drugs. Since 2013, she first turned to the Republican Rheumatological Center of the TMA Multidisciplinary Clinic, where, after a complete examination, the patient was diagnosed with Gout. The patient was recommended dietary food - table №6 according to Pevzner. She took anti-gout therapy irregularly.

A month ago, the patient developed a fever up to 39 ° C, pain and swelling in the joints of the hands and feet, erythematous rashes on the face and chest, ulceration of the oral mucosa, myalgia. She went to a medical institution at her place of residence, from where she entered the TMA clinic and was hospitalized in the rheumatology department.

Objective status: the condition at the time of admission was regarded as severe due to systemic manifestations of the underlying disease (carditis, nephritis). Consciousness: clear. Position: passive. Physique: normosthenic. Skin: dryish, polymorphic discoid rash on the face, neck and hands.

Osteoarticular system: swelling in the small joints of the hands, limitation of movement. Tofuses were found on the auricles, in the area of the right elbow joint, in the II-IV metacarpophalangeal joints of the left hand, in the II interphalangeal joint of the right hand and in the II interphalangeal joint of the right leg.

Respiratory system: auscultatory breathing is weakened vesicular, in the lower parts of the lungs on both sides, wheezing is not heard.

Cardiovascular system: moderately muffled heart sounds. The rhythm is correct, systolic murmur at the apex. Heart rate 98 beats. per minute, pulse 98, blood pressure 160/100 mm Hg. Art.

Digestive system: tongue moist. The abdomen is soft and painless. The liver along the edge of the costal arch, dimensions 10x9x7 cm, is painless on palpation. Peritoneal irritation symptoms (-). Stool is normal.

Urinary system: positive tapping symptom on both sides. Urination is rare, painless.

Laboratory and instrumental analyzes. Examination in a blood test revealed an increase in ESR (52 mm / h), pancytopenia (Hb 80 g / l, erythrocytes 22.3×10^{12} / l, leukocytes 1.5×10^9 / l, platelets 172×10^9 / l), urine analysis: protein 2.8 g / day, epit. 16-18 / l, leukemia. 20-22 / l, erit. 2-3 / l, cyl. hyaline 3-4 / l, granular 2-3 / l, urates ++++. Biochemical blood test: urea 27.2 mmol / l, creatinine 176.4 μ mol / l, total protein 68.6 g / l, uric acid in the blood 0.82 mmol / l, ALT 24 U / L, AST 13 U / L, blood sugar 3.8 mmol / l, total bilirubin 16.7 mmol / l, calcium 1.9 mmol / l, endogenous creatinine clearance 42.6 ml / min.

Immunological analyzes: LE cells 12.2%, antibodies to native DNA (titer 1: 146). *Chest fluoroscopy:* increased pulmonary pattern, compaction of the roots of the lungs, heart without features. *ECG:* Sinus tachycardia with a heart rate of 96 beats. at 1min. EOS is not rejected. Violation of repolarization in the myocardium in the apical, lateral and posterior walls of the left ventricle. *EchoCS:* The global contractility of the left ventricular myocardium is normal (EF 62%), mitral regurgitation 1 degree, systemic changes, fluid in the pericardial cavity ~ 150 ml. *Ultrasound of the kidneys:* Diffuse changes in the renal parenchyma. Uric acid diathesis. Hypoplasia of the left kidney. *X-ray of the joints of the hands and feet:* no pathological changes. *Puncture biopsy of the kidneys* revealed focal segmental mesangiocapillary glomerulonephritis with a pronounced tubulo-interstitial component. A examination of the *synovial fluid* revealed crystals of sodium monourate.

Results

Taking into account the presence of 7 (erythema of the facial skin, articular syndrome, serositis, kidney involvement, hematological changes, immunological changes, the presence of antinuclear antibodies) diagnostic criteria for systemic lupus erythematosus, the diagnosis was made: *Main:* Systemic lupus erythematosus subacute, activity 2 tbsp., Lupus nephritis, lupus dermatitis, lupus carditis. *Concomitant:* Gout, chronic gouty arthritis with tophi, moderate course. *Complication:* Chronic kidney disease stage 3 b (GFR 42.6 ml / min.).

Corticosteroids (prednisolone) were prescribed with a gradual decrease in the dose of the drug with positive clinical and laboratory dynamics, anti-gout therapy was also carried out and the dose of allopurinol was selected. Currently, the patient has no active complaints, abnormalities in the general urine analysis persist (persistent proteinuria up to 1.0–2.0 g / l, single erythrocytes, hyaline casts - 0–1 in the field of view), general blood test within normal range values, ANF - 1:32.

Conclusion. Considering the pathogenesis of gout, it should be noted that there is a deficiency of a number of enzymes of purine metabolism, which lead to hyperuricemia [1,2,4].

Hypoxanthine guanine phosphoribosyltransferase catalyzes the synthesis of inosine monophosphate from hypoxanthine and guanosine monophosphate from guanine and is important for the differentiation and functional activity of T-lymphocytes [7,15]. Adenosine deaminase promotes deamination of adenosine to inosine and deoxyadenosine to deoxinosine. With a lack of this enzyme, severe combined immunodeficiency occurs due to changes in molecular processes, which ensures the proliferation of immunocompetent cells [14,16].

Purine nucleotide phosphorylase catalyzes the formation of hypoxanthine from deaminated inosine deoxyribose, as well as guanosine from deoxyguanosine. Its deficiency is manifested by T-lymphopenia with suppression of the proliferative activity of cells when stimulated with antigens [7,8,3,15].

From the above it follows that enzyme disorders can program an imbalance in the immune system with the subsequent development of autoimmune disorders. Therefore, the study of the components of purine metabolism in systemic lupus erythematosus is promising and relevant, which contributes to the development of new methods in the treatment of combined pathology.

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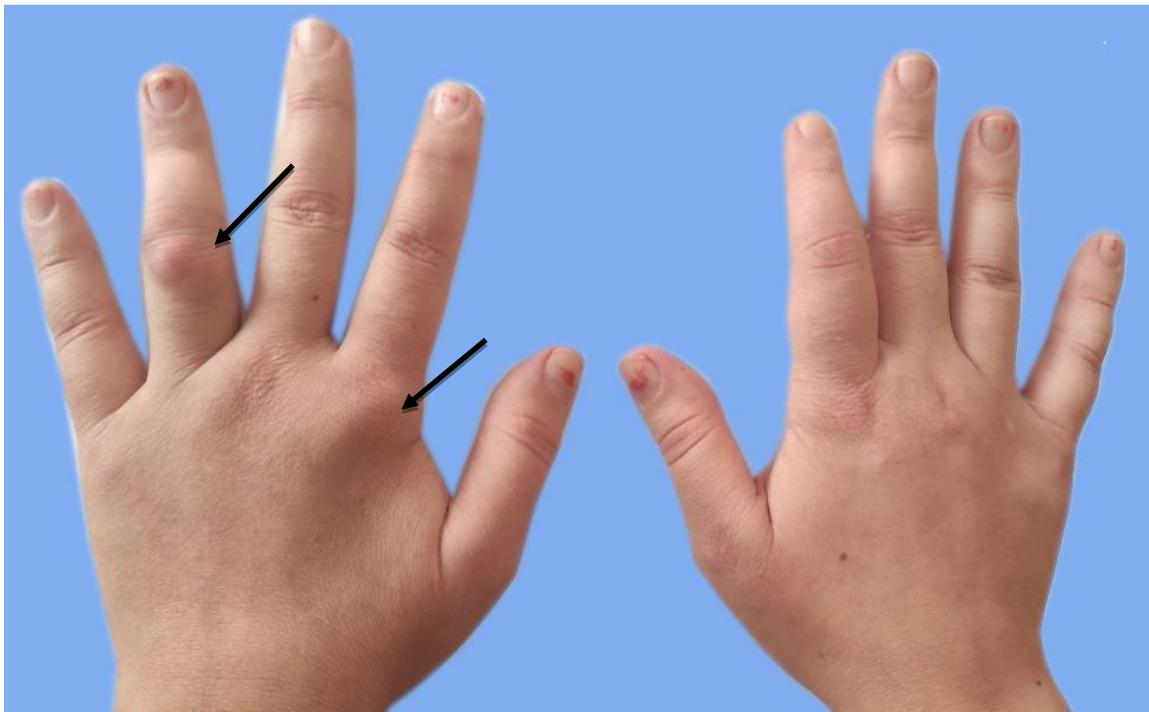


Figure 1. Arrows indicate tophuses



Figure 2. Patient 30 years old. Scaly skin rash on the face

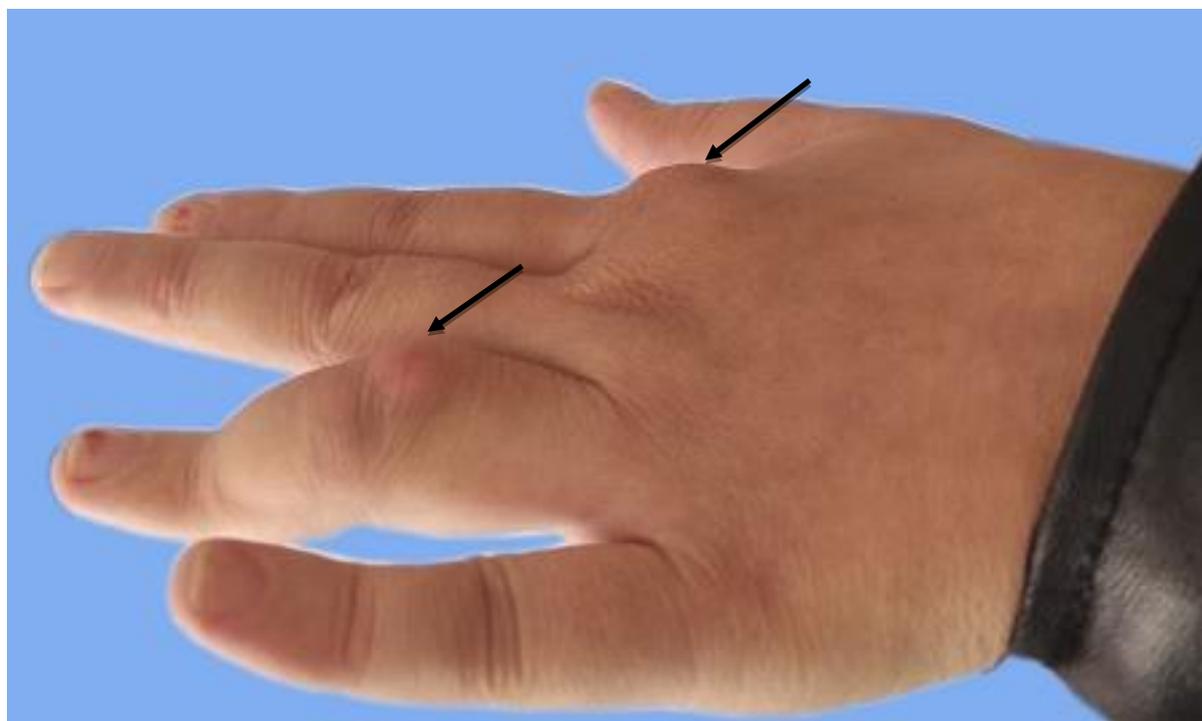




Figure 3,4. Arrows indicate tophuses





Figure 5,6. Arrows indicate tophuses