

**A CASE OF CUTANEOUS SARCOIDOSIS.****A.B.Raxmatov****X.I.Nasimov****B.R.Madreyimov****S.F.Shoximardonova**

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Abstract

Cutaneous sarcoidosis is a specific manifestation of a systemic granulomatous disease of unknown etiology, occurring in approximately one-quarter of patients with sarcoidosis and characterized by exceptional clinical polymorphism. This article reviews current data on the epidemiology, etiopathogenesis, classification, histopathology, differential diagnosis, and treatment of cutaneous sarcoidosis, and presents an original clinical case — a papulo-plaque form of the disease in a female patient with widespread, symmetric lesions of the face, trunk, and limbs requiring inpatient treatment; the diagnosis was histologically confirmed by the detection of non-caseating epithelioid-cell granulomas in a skin biopsy. The effectiveness of stepwise therapy, including systemic glucocorticosteroids, antihistamines, topical agents, and physiotherapy, is demonstrated, and the need to exclude systemic involvement in newly diagnosed cutaneous sarcoidosis is emphasized.

Key words: skin sarcoidosis, clinical features, diagnostics, treatment.

Annotatsiya

Teri sarkoidozi: adabiyotlar shari va klinik kuzatuv

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Teri sarkoidozi — etiologiyasi noma'lum tizimli granulematoz kasallikning o'ziga xos ko'rinishi bo'lib, sarkoidozli bemorlarning taxminan to'rttdan bir qismida uchraydi va o'ziga xos klinik polimorfizmi bilan ajralib turadi. Maqolada teri sarkoidozining epidemiologiyasi, etiopatogenezi, tasnifi, gistopatologiyasi, differensial diagnostikasi va davolash usullari bo'yicha zamonaviy ma'lumotlar sharhi keltirilgan, shuningdek o'z klinik kuzatuvimiz — yuz, tana va oyoq-qo'llarda tarqalgan, simmetrik toshmalar bilan namoyon bo'lgan papulo-plakali shakldagi kasallik holati tasvirlangan bo'lib, bemor statsionar davolanishga muhtoj bo'lgan; tashxis teri biopsiyasida kazeosiz epitelioid-hujayrali granulyomalar aniqlanishi orqali gistologik jihatdan tasdiqlangan. Tizimli glyukokortikosteroidlar, antigistamin preparatlar, mahalliy vositalar va fizioterapiyani o'z ichiga olgan bosqichma-bosqich davolashning samaradorligi ko'rsatilgan, shuningdek birinchi marta aniqlangan teri sarkoidozida tizimli shikastlanishni istisno qilish zarurligi ta'kidlangan.

Kalit so'zlar: teri sarkoidozi, klinik belgilari, diagnostikasi, davolash.

Аннотация

Клинический случай саркоидоза кожи.

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Саркоидоз кожи — специфическое проявление системного гранулематозного заболевания неизвестной этиологии, выявляемое приблизительно у четверти пациентов с саркоидозом и отличающееся исключительным клиническим полиморфизмом. В статье представлен обзор современных данных об эпидемиологии, этиопатогенезе, классификации, гистопатологии, дифференциальной диагностике и лечении кожного саркоидоза, а также описано собственное клиническое наблюдение — случай папуло-бляшечной формы заболевания у пациентки с распространёнными симметричными высыпаниями на лице, туловище и конечностях, потребовавший стационарного лечения; диагноз был гистологически подтверждён обнаружением неказеозных эпителиоидно-клеточных гранулём в биоптате кожи. Показана эффективность поэтапной терапии, включающей системные глюкокортикостероиды, антигистаминные препараты, топические средства и физиотерапию, а также подчёркнута необходимость исключения системного поражения при впервые выявленном кожном саркоидозе.

Ключевые слова: саркоидоз кожи, клиника, диагностика, лечение.

Sarcoidosis is a multisystem disease of unknown etiology characterized by the formation of non-caseating granulomas in affected organs and tissues; the lungs, with bilateral hilar lymphadenopathy, are most frequently involved. The first clinical description of a cutaneous lesion later attributed to sarcoidosis is credited to Jonathan Hutchinson (1875); the concept of the disease subsequently broadened from an isolated cutaneous process to a multisystem granulomatous disease capable of affecting the lungs, lymph nodes, eyes, heart, and nervous system [9,11].

The skin is the second most commonly affected organ after the lungs in sarcoidosis; specific cutaneous lesions occur in approximately 25% of patients with systemic sarcoidosis and may arise in isolation, precede the systemic process, accompany it, or appear only after the diagnosis has been established, which gives them independent diagnostic significance [11,14].

The overall global incidence of sarcoidosis is estimated at approximately 16.5 per 100,000 in men and 19 per 100,000 in women, with a peak age of onset of 25–40 years; the lifetime cumulative risk is higher in women and in individuals of African-American descent compared with the Caucasian population [11,13]. The epidemiology of cutaneous sarcoidosis specifically has been studied less thoroughly: data on sex and age distribution are inconsistent, and racial differences in disease risk and severity have been documented but not fully explained [14,16].

The etiology of sarcoidosis remains unknown; a combined effect of genetic predisposition (HLA class II genes, antigen-presentation genes) and environmental or infectious triggers in a susceptible host is postulated. Its pathogenesis is attributed to dysregulation of the Th1- and Th17-mediated immune response with hyperactivation of inflammatory cascades and granuloma formation; foreign bodies and mycobacterial antigens are considered possible triggers — molecular diagnostic methods have detected mycobacterial DNA and proteins within sarcoid granulomas, which has provided the rationale for studying antimycobacterial therapy [6,11,17].

Cutaneous manifestations of sarcoidosis are divided into specific lesions — showing non-caseating granulomas on biopsy (papules, plaques, subcutaneous nodules, lupus pernio, scar infiltration, and rarer forms) — and non-specific lesions, i.e., reactive processes without

granulomas on biopsy (most commonly erythema nodosum). Specific lesions are found in 20–30% of cases of cutaneous sarcoidosis; the extremely wide variability of its morphology has earned cutaneous sarcoidosis the reputation of a “great imitator” [10,11,14].

The morphological substrate of specific lesions is the sarcoid non-caseating granuloma — an aggregate of epithelioid histiocytes and giant cells with a sparse lymphocytic “cuff” (“naked granulomas”), which frequently contain asteroid bodies and Schaumann bodies. A typical histological picture is not found in every case, which complicates diagnostic verification and requires exclusion of other granulomatous processes [14].

The differential diagnosis of cutaneous sarcoidosis includes infectious granulomatous processes — primarily lupus vulgaris and tuberculoid leprosy, as well as non-tuberculous mycobacterioses and deep mycoses — and non-infectious conditions: foreign-body reactions (diagnosed by polarization microscopy of the biopsy specimen), drug-induced sarcoid-like reactions, and granuloma annulare. At the clinical level, the scope of differential diagnosis depends on the morphology of the lesion: papular forms are differentiated from rosacea and lichen planus, plaque forms from psoriasis and cutaneous lymphomas, and lupus pernio from chilblains and discoid lupus erythematosus [14,19].

The most common forms are the papular and plaque forms: skin-colored, yellow-brown, or violaceous papules with a characteristic “apple-jelly” hue on diascopy, located predominantly on the face; the papular form is associated with an acute course and a favorable prognosis, whereas the plaque form is associated with a chronic course [8,12,14]. Darier-Roussy subcutaneous sarcoidosis presents as firm, mobile nodules on the extremities and is usually accompanied by mild systemic involvement [1]. Lupus pernio is the most specific variant, presenting with indurated violaceous plaques on the nose and cheeks, closely associated with upper respiratory tract involvement and a torpid, treatment-resistant course [18]. Scar sarcoidosis presents as infiltration and discoloration of pre-existing scars [14].

The diagnosis is based on correlating the clinical picture with histological demonstration of non-caseating granulomas in a skin biopsy (with mandatory polarized-light examination to exclude foreign material) and the sequential exclusion of other granulomatous diseases. Once the diagnosis is confirmed, systemic work-up is indicated to assess extracutaneous involvement: chest radiography, pulmonary function testing, ECG and echocardiography, ophthalmologic examination, and laboratory parameters (calcium, liver function tests, vitamin D). No laboratory marker specific to cutaneous sarcoidosis exists; the serum ACE level is non-specific and is used mainly for monitoring disease activity over time [4,14].

Treatment follows a stepwise approach: asymptomatic lesions may be observed without therapy given the possibility of spontaneous remission; progressive or disfiguring lesions are initially managed with topical and intralesional glucocorticosteroids, and if these are ineffective, treatment is escalated to hydroxychloroquine and tetracyclines, then to systemic immunosuppressants (methotrexate) and TNF- α inhibitors (adalimumab, infliximab), which are most effective in refractory forms and lupus pernio [2,3,11,20]. Antimycobacterial therapy deserves separate mention: in a randomized, placebo-controlled trial of the CLEAR regimen (levofloxacin, ethambutol, azithromycin, rifampin), a statistically significant reduction in lesion size was observed compared with placebo, supporting a role for mycobacterial antigens in sustaining granulomatous inflammation [5]. As an adjunctive method for lesions resistant to pharmacological therapy, particularly lupus pernio with a prominent vascular component, the

pulsed dye laser (PDL) is used: case series have reported reductions in erythema, infiltration, and lesion size, although the efficacy of this method varies and, according to a systematic review, requires further study [7,15].

We report our own clinical observation. Patient T.M., a 37-year-old woman (case history No. 4664), was admitted to the Republican Specialized Scientific and Practical Medical Center of Dermatovenereology and Cosmetology, Ministry of Health of the Republic of Uzbekistan, on 20.07.2026. On admission she complained of a rash localized to the face, abdomen, lumbar region, arms, and legs; pruritus; hair loss; insomnia; and constipation.

Anamnesis morbi According to the patient, the rash has been present for 2 years; she does not associate the onset of the disease with any particular cause. The rash first appeared on the leg; she has previously been treated for this condition on several occasions but does not recall the medications used, and treatment produced only a temporary effect. Because of rapid spreading of the rash over the past month, the patient presented to the Republican Specialized Scientific and Practical Medical Center of Dermatovenereology and Cosmetology and was referred to the dermatomycology department for inpatient treatment with a diagnosis of "sarcoidosis."

Epidemiological history. Over the past 6 months the patient received outpatient and inpatient injections; she has not received blood transfusions; has not undergone any surgical procedures; has not visited a dentist; and has not travelled abroad.

Anamnesis vitae. She grew up and developed under satisfactory conditions and started school at age 7. She is currently a homemaker. Past illnesses: acute respiratory viral infections. Family status: mother of four children; family members are healthy. She denies a family history of hereditary disease. No allergic reactions to medications or food have been noted. She denies harmful habits. She is not a blood donor.

Status praesens objectivus. General condition satisfactory, active position, clear consciousness, adequate responses to questions. Peripheral lymph nodes not enlarged, non-tender. Normosthenic body build, musculoskeletal system without deformities. Breathing free through the nose; vesicular breath sounds in the lungs, no wheezing, respiratory rate 16/min. Heart sounds clear and rhythmic, blood pressure 120/80 mmHg, pulse 76 bpm. Tongue moist, coated with a white fur. Abdomen soft and non-tender on palpation, participates in the act of breathing; liver and spleen not palpable; costovertebral angle tenderness (Pasternatsky's sign) negative bilaterally. Urination free and regular; stool regular, with a tendency to constipation.

Status localis. The cutaneous pathological process is chronic, widespread, symmetric, and inflammatory in nature, involving the skin of the face, abdomen, lumbar region, arms, and legs. The morphological elements are nodules and plaques. The skin in the affected areas is thickened; the central portion of the lesions shows a shiny, flattened surface of violaceous nodules ranging from 0.3 mm to 0.5 cm, which coalesce to form plaques up to 2 cm in diameter. Peripheral lymph nodes are not enlarged. Hair and nail plates are unchanged.(see Fig. 1)



Fig. 1.
Facial skin lesions.



Fig. 2.
Lesions on the right arm.

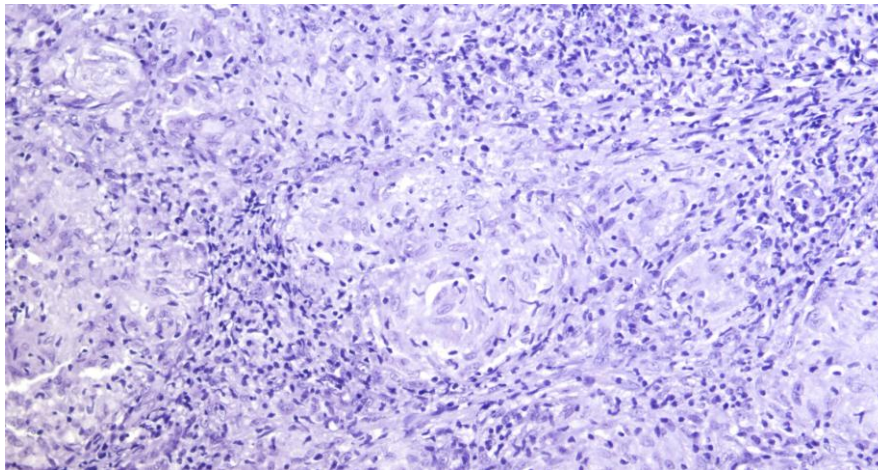


Fig.3.

Skin biopsy from the lesional area (analysis No. 5605 dated 01.05.2026) shows detachment of the stratum corneum, an unremarkable granular layer, epidermal ridges flattened in places, and focal vacuolar degeneration of basal-layer keratinocytes. Throughout the thickness of the dermis, granulomatous infiltrates composed of lymphocytes, histiocytes, epithelioid cells, and multinucleated giant cells are identified; basophilic degeneration of collagen fibers and dilated vessels with swollen endothelium are noted; skin appendages are preserved (Fig. 3). Pathologist's conclusion: the morphological picture described is most consistent with a diagnosis of cutaneous sarcoidosis.

Diagnosis: Cutaneous sarcoidosis. Plaque form. (histologically confirmed).

ICD-10: cutaneous sarcoidosis, D86.3.

Treatment plan according to the Standard of Dermatovenereology and Medical Cosmetology (Order of the Ministry of Health of the Republic of Uzbekistan No. 180 dated 23.06.2025):

- systemic glucocorticosteroid — 30 mg/day according to the schedule (20 mg at 9:00, 10 mg at 11:00) after meals with plenty of fluids, 10-day course (pathogenetic therapy);
- antioxidant agent (metabolic drug), 3% solution — 10 mL IV drip in 100 mL of 0.9% sodium chloride solution, 6-day course (to stimulate the antioxidant system);
- hepatoprotective agent — 10 mL IV drip in 100 mL of 0.9% sodium chloride solution, 5-day course (to reduce the hepatotoxic effects of medications);
- vitamin preparation (ascorbic acid), 5% solution — 2.0 mL IM, 10-day course (to improve microcirculation and strengthen the vascular wall within lesions);
- antihistamine (H1-receptor blocker) — 5 mg once daily in the evening, 10-day course (for pruritus);
- sedating antihistamine — 25 mg once daily after meals, 10-day course (to reduce pruritus and improve sleep);
- probiotic (bifidobacteria preparation) — 400 mg 3 times daily before meals, 10-day course;
- topical calcineurin inhibitor, 0.1% — applied externally twice daily, 10-day course (to reduce local inflammation);
- topical glucocorticosteroid, 0.05% — applied externally once daily combined with ultrasound therapy, 10-day course (to reduce local inflammation);
- physiotherapy: Vbeam Prima (vascular laser, **wavelength:** 595 nm) ×1 session, general UV irradiation ×5 sessions, ultrasound therapy (with corticosteroid cream), phototherapy.



Fig. 4.

Facial skin lesions after treatment.

The clinical case presented illustrates a papulo-plaque variant of cutaneous sarcoidosis, typical of the disease, with widespread, symmetric involvement of the face, trunk, and extremities, accompanied by pruritus and pronounced general symptoms (insomnia, a tendency to constipation), which complicated the initial clinical diagnosis and necessitated referral of the patient to a specialized dermatovenereologic hospital. The chronic, protracted course with relapses after previous treatment is consistent with the literature data on the torpid nature of the plaque form of cutaneous sarcoidosis and confirms the disease's reputation as a “great imitator” that requires a high level of differential-diagnostic vigilance from the physician. Histological examination of the skin biopsy, which revealed non-caseating epithelioid-cell granulomas with multinucleated giant cells, confirmed the clinical diagnosis and allowed other granulomatous processes to be excluded with a high degree of confidence, which is of decisive importance given the pronounced clinical polymorphism of the disease.

The absence of clinical signs of involvement of other organs at the time of examination does not exclude a systemic process, since cutaneous manifestations frequently precede the onset of extracutaneous sarcoidosis; accordingly, the work-up included in the management plan (imaging of internal organs, consultations with related specialists) appears justified and consistent with accepted diagnostic standards. The stepwise therapy prescribed — a systemic

glucocorticosteroid combined with antioxidant and hepatoprotective support, antihistamines, a topical calcineurin inhibitor, a topical glucocorticosteroid, and physiotherapeutic methods — is consistent with current approaches to the treatment of widespread cutaneous sarcoidosis and, over the course of treatment, achieved stabilization of the cutaneous process with no new eruptions and no adverse events.

This case underscores the need for continued follow-up of the patient with assessment of treatment response, as well as the importance of a multidisciplinary approach for the timely detection of possible systemic involvement in cutaneous sarcoidosis..

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