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DIAGNOSIS OF DERMATOMYOSITIS. CLINICAL CASE

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Annotation: The described clinical case is notable for the early diagnosis of dermatomyositis. Timely diagnosis and correct selection of therapy are fundamental to reducing the incidence of various complications and improving the quality of life of patients with this pathology.

Key words: dermatomyositis, arthralgia, myalgia, autoimmune disease.

Dermatomyositis is a systemic connective tissue disease characterized by autoimmune damage predominantly involving the skin and muscles, accompanied by inflammation of the proximal musculature and muscle weakness [1].

The classic cutaneous manifestations of dermatomyositis include heliotrope rash and erythematous eruptions on the extensor surfaces of the metacarpophalangeal, proximal, and distal interphalangeal joints of the hands, as well as the elbows and knees.

Muscle involvement in the absence of skin manifestations is referred to as polymyositis [10].

A form of dermatomyositis involving only the skin occurs extremely rarely [6]. Dermatomyositis and polymyositis belong to the group of idiopathic inflammatory myopathies [5].

The overall incidence of dermatomyositis is 9.63 cases per 1 million population — 13.98 cases among women and 4.68 among men. Women are affected 2–3 times more often than men. The estimated prevalence of dermatomyositis is about 20 cases per 100,000 people [15].

Dermatomyositis has a bimodal age distribution, with the first peak occurring between 5 and 14 years of age, and the second between 45 and 64 years [7,14].

It should also be noted that dermatomyositis in many patients may be associated with malignancies. A clear relationship has been established between dermatomyositis and the development of malignant neoplasms.

Among patients with idiopathic inflammatory myopathies, the incidence of cancer ranges from 7% to 30% [9].

Patients with dermatomyositis have a higher risk of developing cancer compared to those with polymyositis. It is also important to emphasize that after a diagnosis of dermatomyositis, the overall risk of developing malignancies increases threefold [8].

The etiology of dermatomyositis remains unknown. It is believed to result from an autoimmune process that develops in genetically predisposed individuals in response to environmental

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triggers. The disease is thought to arise due to pathological activation of the complement system under the influence of specific triggers.

Possible triggering factors include hypothermia, excessive sun exposure, drug reactions, intense physical activity, psychological stress, and various infections (both acute and chronic exacerbations), as well as vaccination [6].

Certain seasonal variations have been found to influence the development of dermatomyositis, suggesting that infectious triggers may play a role in its etiology [2,3].

Dermatomyositis is an immunologically mediated disease. In 50–70% of patients with polymyositis and dermatomyositis, antinuclear antibodies (ANA) or other specific autoantibodies circulate in the blood [7,11].

Myositis-specific antibodies (MSAs) are among the most important diagnostic markers of dermatomyositis.

Some of these autoantibodies are specific for myositis (e.g., anti–aminoacyl-tRNA synthetase, anti–Mi-2), while others are nonspecific, though also associated with muscle inflammation (e.g., anti-nRNP, anti-Ro/SSA, anti-Ku, anti-PMS1).

Among patients with dermatomyositis or polymyositis, anti–Jo-1 antibodies are most frequently detected, with a prevalence of 20–30% [13].

Certain antibodies are clearly associated with distinct clinical phenotypes of the disease. For instance, anti–Mi-2 antibodies are linked with heliotrope rash and Gottron's sign [4].

Anti–aminoacyl-tRNA synthetase antibodies are found in patients with interstitial lung disease, non-erosive arthritis, and Raynaud's phenomenon [12].

Anti–HMGCR and anti–SRP antibodies are characteristic of necrotizing autoimmune myositis, whereas anti–cN1A antibodies are more commonly found in patients with inclusion body myositis.

Clinical Case

Patient: D.S.M., born in 1972, was admitted to the Department of Rheumatology at the multidisciplinary clinic of Tashkent Medical Academy on **December 20, 2023**.

Upon admission, the patient complained of **rash in the oral cavity**, **pain and difficulty swallowing**, **general weakness**, **headache**, **fatigue**, **progressive symmetrical weakness in the muscles of the arms and legs**, **muscle pain at rest and during movement**, **difficulty performing any physical activity**, **pain in the joints of the hands and knees**, **fever up to 39.0°C**, **skin rash**, **itching**, and **scaling of the palms**.

According to the patient, she has considered herself ill for the past **six months**. The cause of the disease was not associated with any specific factors. The illness began with **erythematous red rashes** on the face, neck, and various parts of the body, accompanied by **muscle pain**.

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Figure. Appearance of patient D.S.M., born 1972.

The **affected skin area** covered approximately **15%** of the body surface. There were **multiple eruptions in the oral cavity**, causing difficulty in swallowing. On palpation, there was **marked tenderness** in the **proximal muscles** of both upper and lower extremities. **Muscle strength** was significantly reduced — the patient could raise her arms only to shoulder level and could barely lift her legs while lying down. **Assistance was required** to get out of bed. She had difficulty walking and could move independently for only about **20 meters** without external support.

The **joints** appeared unchanged externally, with **full range of motion**. **Palpable lymph nodes** were not enlarged.

Heart sounds were clear and rhythmic; **heart rate** was 120 beats per minute. **Blood pressure:** 100/60 mmHg.

Cardiac dullness boundaries were within normal limits. **Nasal breathing** was unobstructed. **Auscultation** of the lungs revealed **vesicular breath sounds**, with no rales. **Percussion** revealed clear lung sounds. **Respiratory rate:** 20 breaths per minute.

The **abdomen** was of normal shape, soft, and painless upon superficial palpation, actively participating in respiration.

Liver size by Kurlov: 9×8×7 cm. The **spleen** was not enlarged. **Stool** was normal. The **kidney area** showed no visible changes; **Pasternatsky's sign** was negative on both sides. **Urination** was free and painless; **diuresis** was normal. **Edema**was noted on the **feet and lower legs**.

The patient had previously received both **inpatient and outpatient treatment** multiple times and had been seen by various specialists.

In **2017**, she was diagnosed with **bronchial asthma** and has been receiving regular therapy. She takes **Ebem 5 mg once daily**. However, her condition progressively worsened — skin rashes increased, muscle weakness intensified, and muscle pain became more severe.

Due to the deterioration of her health and exacerbation of symptoms, the patient underwent examination at the **Arthrology Department of the Tashkent Medical Academy** and was subsequently hospitalized in the **Department of Internal Medicine Rehabilitation** for further evaluation and treatment.

Past medical history: recurrent respiratory infections.

Allergic history: unremarkable.

Family history: negative — no similar diseases among relatives.

Objective examination at admission:

General condition — **moderate severity**. The patient was **conscious**.

Skin: pale pink. A **heliotrope rash** was present on the face, accompanied by **edema** of the cheekbones, nasolabial folds, nasal wings, and upper eyelids. On the **neck, hands, forearms, and trunk**, there were multiple **erythematous, scaly lesions** forming the typical **"shawl sign"** (see figure).

Laboratory and instrumental findings:

- **Complete blood count (CBC):**

Hb – 110 g/L, RBC – 3.9×10^{12}/L, MCV – 0.9 μm, Platelets – 290×10^{9}/L, WBC – 11.4×10^{9}/L; Neutrophils – 78%, Lymphocytes – 13%, Monocytes – 4%, Eosinophils – 2%, Bands – 3%.

ESR – 26 mm/h.

- **Rheumatic tests (21.12.2023):**

C-reactive protein – 30 mg/L; Rheumatoid factor – 24 IU/mL; Antistreptolysin-O – 630 IU/mL.

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- **Urinalysis:**

Volume – 90.0 ml, color – yellow, appearance – cloudy, specific gravity – 1.032, protein – 0.066 g/L.
Epithelium: squamous – 13–15, transitional – 10–11, renal – 9–10.
Leukocytes – 14–15 per field of view, unchanged – 0–1.
Mucus +, salts +.

- 

Wassermann Reaction (21.12.2023): Negative.

Biochemical Blood Analysis (21.12.2023):
Total protein – 74.0 g/L;
Glucose – 5.4 mmol/L;
Urea – 4.9 mmol/L;
Creatinine – 59.7 µmol/L;
Total bilirubin – 14.1 µmol/L;
ALT – 37 U/L;
AST – 20 U/L.

Coagulogram:
Hematocrit – 50;
Erythrocytes – 49–58;
Fibrinogen – 466 mg%;
Plasma tolerance to heparin – 4'00";
Standard test – negative;
Thrombotest – V (Level V).

Echocardiography (EchoCG):
Findings: Left ventricular cavity not dilated —
End-diastolic diameter (EDD) – 4.0 cm,
End-diastolic volume (EDV) – 72.0 ml,
Ejection fraction (EF) – 68.0%,
Left atrium (LA) – 3.0 cm.
Right heart chambers not dilated.
Mitral valve — no structural changes.
Aortic pulsation preserved; aortic root diameter – 3.0 cm.

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Aortic valve — no structural abnormalities.
Pulmonary artery — age-appropriate; root diameter – 2.0 cm.
Left ventricular walls slightly thickened, normokinetic.
Interventricular septum thickness – 0.9 cm; posterior wall thickness – 0.9 cm.
Doppler echocardiography — no pathological flows.
Left ventricular filling pattern — normal.

Conclusion:
No evidence of valvular heart disease or local left ventricular wall motion abnormalities.
Global left ventricular contractility normal.
Tachycardia noted.

ECG:
Sinus tachycardia with HR 110 bpm.
Electrical axis of the heart — vertical.
Signs of impaired repolarization processes in the myocardium of the posterior wall of the left ventricle.

Liver and Gallbladder:
Conclusion: Grade I hepatic steatosis (fatty liver).

Kidneys and Adrenal Glands:
Conclusion: Bilateral pyelonephritis. Left renal cyst.

Chest X-ray:
Conclusion: Enhanced broncho-vascular pattern. Left-sided scoliosis.

Final Diagnosis:
Idiopathic Dermatomyositis, acute course, activity grade III, involving the parotid gland, esophagus, skin, and muscles.

Conclusion:
This clinical case is notable for the **early diagnosis of dermatomyositis**.
Timely recognition of the disease and the correct choice of therapy are **fundamental for reducing the frequency of complications** and **improving the quality of life** of patients with this pathology.

References:

1. Orlova E.V. et al. Dermatomyositis: Clinical Case and Literature Review // Russian Medical Journal. – 2017. – No. 11. – P. 850–852.

2. Beel A.J. et al. Ground-glass opacity heralding invasive lung adenocarcinoma with prodromal dermatomyositis: a case report // J. Cardiothorac. Surg. – 2018. – Vol. 13, No. 1. – P. 20.

3. Bendewald M.J. et al. Incidence of Dermatomyositis and Clinically Amyopathic Dermatomyositis: A Population-Based Study in Olmsted County, Minnesota // Arch. Dermatol. – 2010. – Vol. 146, No. 1. – P. 26–30.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

4. Bernet L.L. et al. Ovoid palatal patch in dermatomyositis: a novel finding associated with anti-TIF1γ (p155) antibodies // JAMA Dermatol. – 2016. – Vol. 152, No. 9. – P. 1049–1051.

5. Buchbinder R., Hill C.L. Malignancy in patients with inflammatory myopathy // Curr. Rheumatol. Rep. – 2002. – Vol. 4, No. 5. – P. 415–426.

6. Cassius C. et al. Biomarkers in adult dermatomyositis: tools to help the diagnosis and predict the clinical outcome// J. Immunol. Res. – 2019. – Vol. 2019. – P. 9141420.

7. Dalakas M.C. Inflammatory muscle diseases // N. Engl. J. Med. – 2015. – Vol. 372, No. 18. – P. 1734–1747.

8. Gunawardena H., Betteridge Z.E., McHugh N.J. Myositis-specific autoantibodies: their clinical and pathogenic significance in disease expression // Rheumatology (Oxford). – 2009. – Vol. 48, No. 6. – P. 607–612.

9. Hill C.L. et al. Frequency of specific cancer types in dermatomyositis and polymyositis: a population-based study // Lancet. – 2001. – Vol. 357, No. 9250. – P. 96–100.

10. Love L.A. et al. A new approach to the classification of idiopathic inflammatory myopathy: myositis-specific autoantibodies define useful homogeneous patient groups // Medicine (Baltimore). – 1991. – Vol. 70, No. 6. – P. 360–374.

11. Marvi U., Chung L., Fiorentino D.F. Clinical presentation and evaluation of dermatomyositis // Indian J. Dermatol. – 2012. – Vol. 57, No. 5. – P. 375–381.

12. Mukhammadieva S.M. Efficacy and safety of anti-inflammatory treatment in ankylosing spondyloarthritis // British View. – 2023. – Vol. 8, Issue 7. – P. 24–31.

13. Nagaraju K., Lundberg I.E. Polymyositis and dermatomyositis: pathophysiology // Rheum. Dis. Clin. North Am. – 2011. – Vol. 37, No. 2. – P. 159–171.

14. Pulatova Sh., Nabiyeva D., Abduazizova N. et al. Clinical and pathogenetic values of disorders of mineral metabolism in ankylosing spondylitis // Philosophical Readings. – 2022. – Vol. XIII, No. 4. – P. 20–28.

15. Tanimoto K. et al. Classification criteria for polymyositis and dermatomyositis // J. Rheumatol. – 1995. – Vol. 22, No. 4. – P. 668–674.