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ОПЫТ ПРИМЕНЕНИЯ КОЛХИЦИНА ПРИ БОЛЕЗНИ СТИЛЛА, РЕФРАКТЕРНОМ К СТАНДАРТНОЙ ТЕРАПИИ

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Experience Of Colchicine Administration in Refractory Still's Disease

Резюме

Болезнь Стилла (БС) — это редкое хроническое аутовоспалительное заболевание, проявляющееся развитием высокой пиковой лихорадки, поражением суставов в виде артралгий и артритов и возникновением макулопапулезной сыпи лососевого цвета. В рекомендациях EULAR (The European Alliance of Associations for Rheumatology) 2024 г. унифицированы критерии диагностики БС, включающие лихорадку ≥39°C (102,2°F), периодическое появление эритематозной сыпи, поражение опорно-двигательного аппарата, нейтрофильный лейкоцитоз, повышение острофазовых показателей — С-реактивного белка (СРБ) и скорости оседания эритроцитов (СОЭ) и ферритина. При возможности определения уровней S100 или интерлейкина (ИЛ) 18, их повышенные значения будут указывать в пользу БС. Также изменены подходы к лечению БС с назначением генно-инженерных биологических препаратов (ГИБП) групп ингибиторов ИЛ-1 или ИЛ-6 при неэффективности глюкокортикоидов (ГК), применение метотрексата рассматривается в случае невозможности инициации терапии ГИБП.

Представленный клинический случай посвящен своевременной диагностике БС и резистентности к стандартной терапии ГК (включая пульс-терапию) и метотрексатом (МТХ). В связи с недостаточной доступностью ГИБП на основании имеющегося опыта назначения колхицина при БС в научной литературе, пациентке модифицирована терапия с добавлением колхицина в дозе 1 мг внутрь в сутки, после назначения которого отмечена регрессия клинический проявлений и нормализация острофазовых показателей.

Данный клинический опыт демонстрирует возможность использования колхицина в качестве альтернативы ГИБП для снижения активности заболевания при рефрактерности БС к терапии ГК и МТХ.

Ключевые слова: *болезнь Стилла, генно-инженерные биологические препараты, колхицин, глюкокортикоиды, метотрексат, воспаление*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

Still's disease (SD) is a rare chronic autoinflammatory disease manifested by the development of high peak fever, joint involvement (arthralgias and arthritis), and the appearance of a salmon-colored maculopapular rash. The 2024 EULAR guidelines unified the diagnostic criteria for Still's disease including fever ≥39°C (102.2°F), recurrent erythematous rash, musculoskeletal involvement, neutrophilic leukocytosis, and elevated CRP and ferritin. If S100 or interleukin (IL)18 levels can be determined, their elevated values will point in favor of SD. Also, treatment strategy have been modified with the administration of biologics of IL-1 or IL-6 inhibitors if glucocorticoids (GCs) are ineffective, and the use of methotrexate (MTX) is considered if biologic therapy cannot be initiated.

This case report focuses on the situation of timely diagnosis of SD and resistance to standard therapy with GCs (including pulse therapy) and MTX. Due to insufficient availability of biologics, based on the existing experience of colchicine prescription in SD in the scientific literature, the patient's therapy was modified with the addition of colchicine at a dose of 1 mg orally per day, after the administration of which regression of clinical manifestations and normalization of acute-phase markers were noted.

This clinical experience demonstrates the feasibility of colchicine administration as an alternative to biologics to reduce disease activity if SD is refractory to GC and MTX therapy.

Key words: *Still's disease, biologics, colchicine, glucocorticoids, methotrexate, inflammation*

Conflict of interests

The authors declare no conflict of interests

Sources of funding

The authors declare no funding for this study

Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article « Experience Of Colchicine Administration in Refractory Still's Disease » for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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ABT — antibiotic therapy, BP — blood pressure, ALT — alanine aminotransferase, ANA — antinuclear antibodies, ANF — antinuclear factor, AST — aspartate aminotransferase, ACPA — anti-citrullinated peptide antibodies, SD — Still's disease, DMARDs, disease-modifying antirheumatic drugs, AOSD — adult-onset Still's disease, >ULN — over the upper limit of normal, SBHI CR — State Budget Healthcare Institution of the Crimea Republic, BAs — biological agents, CSs — corticosteroids, IL — interleukin, CT — computed tomography, LDH — lactate dehydrogenase, MTX — methotrexate, NSAIDs — non-steroidal anti-inflammatory drugs, AC — abdominal cavity, PC — pelvic cavity, PIPs — proximal interphalangeal joints, RCH — Republican Clinical Hospital, RCT — randomized clinical trial, RF — rheumatoid factor, MAS — macrophagal activation syndrome, ESR — erythrocyte sedimentation rate, CRP — C-reactive protein, US — ultrasound, TNF-α — tumor necrosis factor α, RR — respiratory rate, HR — heart rate, JIA — juvenile idiopathic arthritis, JSIA — juvenile systemic idiopathic arthritis, GS — gastroscope, EULAR — The European Alliance of Associations for Rheumatology, ILAR — International League of Associations for Rheumatology, CARRA — The Childhood Arthritis and Rheumatology Research Alliance, PRINTO — Pediatric Rheumatology International Trials Organization, ACR30 — American College of Rheumatology 30, SDAS — Still's Disease Activity Score, SMS — Systemic Manifestation Score, SFS — Systemic Feature Score, mPoS — Modified Pouchot-Activity Score, JAK — Janus kinase

Introduction

Still's disease (SD) is a rare chronic autoinflammatory disease manifesting with high peaking fevers, joint involvement (arthralgia or arthritis), and salmon-pink maculopapular rash. Heterogenous disease signs are associated with a variable combination of clinical manifestations [1, 2].

For the first time juvenile systemic idiopathic arthritis (JSIA) was described by Sir George Still at the end of the 19th century, while the adult-onset SD (AOSD) was first described in 1971 by Eric Bywaters. The incidence of SD is 1 per 100,000 population, with the peak incidence in persons aged 16 to 35 years [3, 4, 5].

Due to the common pathogenetic mechanisms, these disease form were defined as a single pathological process, but were also considered specific nosological units with the variable age onset.

According to the 2024 European Alliance of Associations for Rheumatology (EULAR) guidelines, JSIA and AOSD have been compiled into a single disease form of SD [1, 5, 6, 7]. Due to heterogenous clinical manifestations and rarity of this syndrome, quick SD diagnosis is difficult, which has been an obstacle for unification and compilation of diagnostic SD criteria.

Classification criteria of ILAR (International League of Associations for Rheumatology), CARRA (The Childhood Arthritis and Rheumatology Research Alliance), PRINTO (Pediatric Rheumatology International Trials Organization) were known earlier in rheumatological practice. To optimize and accelerate SD diagnosis, 2024 EULAR recommendations have included supportive clinical manifestations for the identification of patients with SD:

- Fever (usually undulating) with body temperature reaching ≥39 °C (102.2 °F), lasting at least 7 days;
- Periodic rash usually coinciding with fever peaks, localized predominantly on the torso. The rash is usually erythematous (salmon-pink), although

other rash types (e.g., urticarial) may be observed — this does not contradict the diagnosis;
- Musculoskeletal lesions (arthralgia/myalgia). Arthritis is an additional, but not mandatory diagnostic criterion — it can develop during the disease [1, 7];
- Neutrophilic leukocytosis, elevated serum C-reactive protein (CRP) and ferritin levels.

It has been additionally defined that significant elevation of interleukin (IL) 18 and/or S100 inflammatory protein (e.g., calprotectin) levels in the blood serum significantly enhances the accuracy of SD diagnosis, thus it is recommended to determine them if possible. The examination aiming to exclude malignancies, infections, other immune-mediated and monogenic autoinflammatory disorders should be arranged in parallel [1, 8].

Updated 2024 EULAR guidelines on SD patient management also reflect changes in the therapeutic tactics, i.e.:

1) to decrease the duration of systemic corticosteroid (CS) use, it is essentially recommended to use IL-1 and IL-6 inhibitors to achieve and maintain treatment targets due to the potent efficacy evidence.
2) IL-1 or IL-6 inhibitors should be started as soon as possible after establishing the diagnosis;
3) Clinical remission should be achieved within 3–6 months without using CSs to discontinue biological agents (BAs) [4].

Based on the EULAR expert opinion, the efficacy of synthetic disease-modifying anti-rheumatic drugs (DMARDs) has been studied insufficiently in SD. In a single randomized clinical trial (RCT) (arranged among patients with juvenile idiopathic arthritis (JIA)) methotrexate (MTX) was not superior to placebo even at the low response threshold based on American College of Rheumatology 30 (ACR30) criteria [9]. Traditionally DMARDs are used in patients with severe joint lesions [10]. In several observational studies patients treated with MTX or MTX combinations with BAs demonstrated response [11]. MTX may be used with steroid-sparing purposes [12]. Despite the fact that IL-1 and IL-6 inhibitors are currently designated as the drugs of choice, MTX should be considered if the former drugs are not available [13]. However, the real anti-inflammatory MTX effect in SD remains debatable, which requires further search for new anti-inflammatory agents to decrease the severity of clinical manifestations accounting for the financial and logistic feasibility in general [12].

In some patients (17–32 %) insufficient treatment response was achieved with the decreased clinical and laboratory activity after administering high CS doses and traditional DMARDs [2, 4, 14]. In such scenario, SD is considered resistant to standard DMARD treatment. Accounting for the heterogenous clinical SD manifestations, the probability of inflammation spread with the involvement of vital organs and systems (pleurisy, pericarditis, hepatomegaly, splenomegaly, myocarditis), as well as the possibility of SD progression to life-threatening conditions, including macrophagal activation syndrome (MAS), timely administration of adequate inflammatory treatment is required with the achievement of low SD activity or remission [15]. Due to financial and logistic difficulties, treatment with IL-1 or IL-6 inhibitors is not always possible, which requires the search for efficient, safe, and economically feasible alternative for BAs [16].

A collision with the issue of mandatory decrease in SD activity, achievement of disease activity control and preventive measures concerning the emergence of MAS within the setting of deficient BA availability has led to the decision on treatment intensification with the administration of colchicine in addition to MTX and CS in the clinical case presented.

Objective: demonstration of colchicine efficacy with the resistant SD course as an alternative to BAs.

Clinical Case Report

The female patient O., 32 years old, was hospitalized into the Department of Rheumatology of the State Budget Healthcare Institution of the Crimea Republic (SBHI CR) N.A. Semashko Republican Clinical Hospital (RCH) complaining of severe pain in metacarpophalangeal, proximal interphalangeal joints of hands (mostly in the right hand), wrist, ankle, and both shoulder joints; pain in the cervical spine; swelling of small joints in both hands, crepitation during motions in joints of lower extremities; morning stiffness for over 2 hours, significant general weakness, body temperature elevation to 37.2–38.0 °C (without signs of acute respiratory viral infections), periodic rash on lower extremities and buttocks.

Medical history: the disease started on January 22, 2025 with the emergence of erythematous & macular rash, sore throat, scratchy feeling in the throat. 4 days later, the patient noted more severe fever (~38 °C) and visited the general practitioner at place of residence; she was counseled by the otorhinolaryngologist and diagnosed with catarrhal tonsillitis. The following antibiotic therapy (ABT) was administered: azithromycin 500 mg/day, ceftriaxone 1.0 g intramuscularly twice daily. During 5 days of therapy, febrile fever persisted,

while pain and swelling emerged in the small joints of hands (Fig. 1), wrist, shoulder, and ankle joints; maculopapular rash spread to the skin of legs, thighs, buttocks, and forearms — it was accompanied by mild itching (Fig. 2). Sore throat and swallowing discomfort significantly improved, although lymphadenopathy of anterior cervical lymph nodes emerged. The common blood count detected leukocytosis (16×10^9/L) with neutrophilic left shift and elevated erythrocyte sedimentation rate (ESR) to 35 mm/h, C-reactive protein (CRP) to 71 mg/L. ABT was adjusted, with one drug switched to levofloxacin 500 mg twice daily orally. No clinical effects were observed within 3 days of therapy; laboratory tests demonstrated worsening, with leukocytosis elevation to 26×10^9/L and ESR to 51 mm/h, CRP — to 171 mg/L; elevated transaminase levels (aspartate aminotransferase (AST) — 82 U/L, alanine aminotransferase (ALT) — 96 U/L). The urinalysis demonstrated transient proteinuria (~0.3 g/day).

Due to the treatment inefficacy, the patient on her own visited the outpatient rheumatologist at place of residence on February 5, 2025 — the diagnosis of undifferentiated arthritis was established, and the following drugs were administered: methylprednisolone 16 mg once daily orally; meloxicam 15 mg once daily orally. The patient noted positive effects during the treatment administered — the rash disappeared, the joint syndrome improved, and the fever diminished to subfebrile values. Laboratory monitoring (February 13, 2025) revealed positive changes, with CRP levels dropping to 40.26 mg/L; blood test for rheumatoid factor (RF) — 14.1 IU/mL (N<14 IU/mL), antinuclear factor (ANF; HEp2-line) — 1:320 with the granular nuclear fluorescence, AC-2,4 (N<1:80). On February 21, 2025, the patient was counseled by the outpatient rheumatologist of SBHI CR N.A. Semashko RCH, where SD was suspected for the first time; the blood test for IL-18 was proposed as a clarifying SD marker. While adjusting treatment, it was recommended to increase methylprednisolone dose to 20 mg, MTX was administered in the initial dose of 10 mg weekly parenterally in combination with folic acid 5 mg weekly and laboratory parameter monitoring; the inpatient treatment was scheduled in the Department of Rheumatology SBHI CR N.A. Semashko RCH for the diagnosis verification and therapeutic management correction.

Upon the admission into the inpatient department on February 25, 2025, physical examination demonstrated mildly impaired general condition, with the body temperature of 37.3 °C. The skin was of physiological color, dry, without rash. Mobile, moderately tender anterior cervical lymph nodes up to 2 cm in diameter were palpated, not adhering to surrounding tissues. The thyroid gland was not enlarged. Vesicular breathing with no rales was auscultated in lungs; respiratory rate (RR) 16/min, SaO_2 97 %. Body weight: 69 kg.

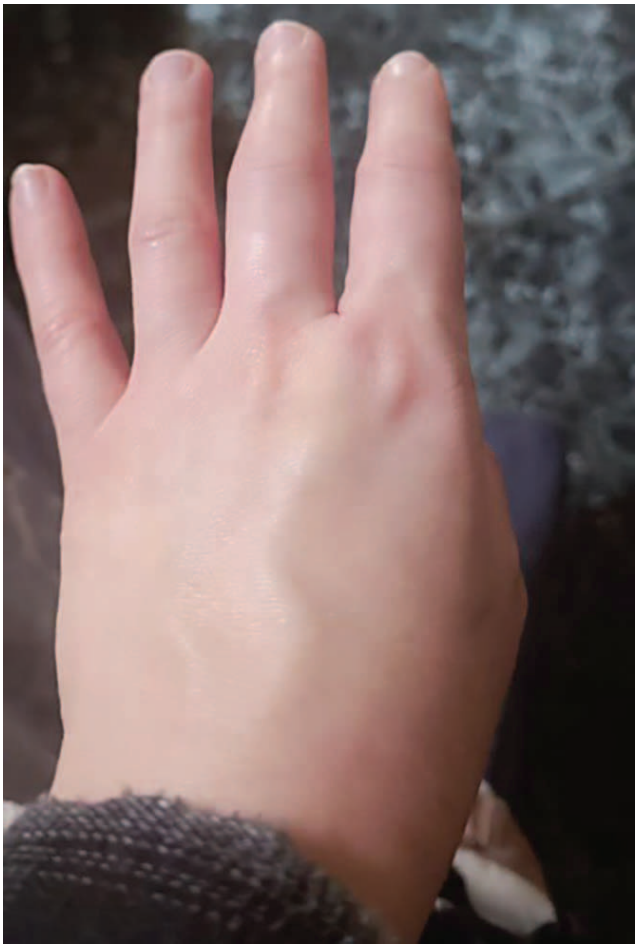


Figure 1. *Arthritis of the small joints of the hands*

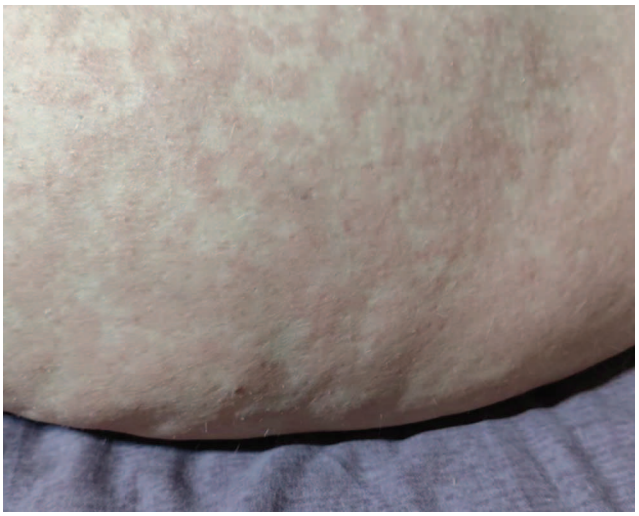


Figure 2. *Maculopapular rash on the thighs*

The chest percussion revealed normal cardiac sizes within the age limits. Pathological murmurs were not auscultated, although cardiac tones were muffled. The cardiac rhythm was regular. Blood pressure (BP) 130/70 mm Hg in both arms. Heart rate (HR) 80 beats per minute. The tongue was moist and clean. The abdomen was soft, not enlarged, non-tender on palpation. Peritoneal irritation signs were negative. The liver was not enlarged. The spleen was palpated and enlarged (14×7 cm). The lumbar punch sign was negative bilaterally. No peripheral edema was detected. Urination and defecation were normal. The urination was free, not difficult or painful. The stool was normal, without pathological inclusions, with bowel movements once or twice daily. Meningeal signs were negative.

Examination of joints in upper extremities: tenderness and swelling during the palpation of right shoulder, both wrist joints, second proximal interphalangeal joints (PIPs) in both hands; positive hand compression test bilaterally; positive Dowborn arch test in the upper third of the right arm. Examination of joints in lower extremities: tenderness and defiguration of both ankle joints.

Blood tests at the moment of hospitalization: Hb 98 g/L, white blood cells 13.6×10^9/L (74.7 % neutrophils), platelets 275×10^9/L, ESR 35 mm/h, AST 37.1 U/L, ALT 19.9 U/L, CRP 48.2 mg/L, RF 12.8 IU/mL, ANF (HEp-2) 1:320, anti-cyclic citrullinated peptide antibodies (ACPA) <5 IU/mL (N<5 IU/mL). Urinalysis parameters were within the reference range.

Before hospitalization, malignancies were excluded in the patient using the ultrasound (US) of abdominal cavity (AC) and kidneys, which demonstrated echographic signs of moderate splenomegaly; US of pelvic organs did not reveal pathological alterations. Computed tomography (CT) of the chest did not demonstrate focal and infiltrative pulmonary lesions. X-ray of feet (anteroposterior view): signs of polyarthritis of distal feet joints, Grade 1 hallux valgus. X-ray of hands: signs of periarticular osteoporosis in 2nd to 5th metacarpophalangeal and 2nd to 5th PIPs, corresponding to signs of Grade 1 arthritis. Echocardiography: no signs of pericarditis, valvular lesions detected. Gastroscopy: signs of congestive gastrobulbopathy, duodenogastric reflux, Grade 1–2 cardia insufficiency, chronic distal reflux esophagitis.

The patient was examined by the hematologist — Grade 1 chronic iron deficiency anemia was diagnosed, lymphoproliferative processes were excluded.

Based on the clinical & laboratory data, the preliminary diagnosis was established:

“Main disease: Still’s disease, early stage, high activity (DAS28-CRP=5.94, DAS28-ESR=6.07), X-ray stage 1, FC II.

Concomitant diseases: Chronic gastritis, remission. Grade 1 chronic iron deficiency anemia”.

Due to the insufficient efficacy, methylprednisolone dose was increased to 24 mg once daily orally; with the purpose of further CS dose decrease, the patient had her methotrexate dose increased to 15 mg weekly in combination with folic acid 5 mg weekly orally in inpatient conditions.

Next day after the hospitalization, the patient had a recurrent body temperature spike to 38.5 °C, worsening pain in peripheral joints, and the emergence of erythematous-macular rash on the skin of the face and neck (Fig. 3, 4). It was decided to assess the profile of antinuclear antibodies and antiphospholipid antibodies, C3/C4 complement components to exclude the systemic connective tissue disorder, as well as to modify the treatment with methylprednisolone pulse therapy (250 mg No. 3 intravenously).

Inpatient laboratory tests revealed preserved CRP (45.6 mg/L), lactate dehydrogenase (LDH) (664 U/L; N=135–214 U/L), and ferritin (285 μg/L; N=13.00–150.00 μg/L) levels. The blood test (immunoblotting) did not reveal antinuclear (ANA) and antiphospholipid antibodies; C3 and C4 levels were within the reference range. Significantly elevated IL-18 levels were demonstrated: >1000 pg/mL (0–270 pg/mL). The patient underwent the follow-up USAC: splenomegaly (140×70 mm) with the normal spleen structure was confirmed. The repeated echocardiography did not reveal pathological valvular lesions.

Based on the detected clinical symptoms (sore throat and scratchy feeling in the upper airways, non-erosive arthritis, erythematous-macular rash, relapsing febrile fever, splenomegaly) and results of laboratory tests (significantly elevated acute-phase reactants — ESR, CRP, ferritin, leukocytosis), as well as a significantly elevated Still's disease marker (IL-18), the following final clinical diagnosis was established:

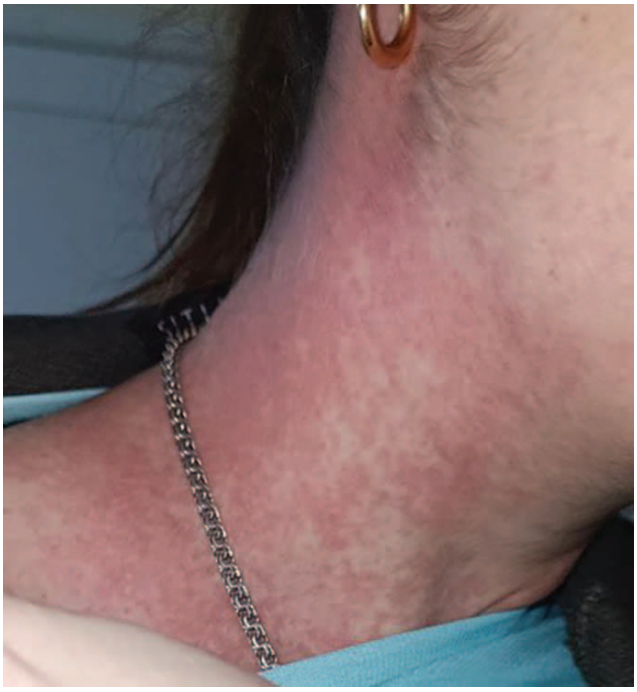
"Main disease: Still's disease, early stage, high activity (DAS28-CRP=5.94, DAS28-ESR=6.07, SDAS=7, mPoS=8, SMS=5, SFS=7), X-ray stage 1. FC II.

Concomitant diseases: Chronic non-erosive gastritis, remission. Congestive gastrobulbopathy. Duodenogastric reflux. Grade 1-2 cardia insufficiency. Chronic distal reflux esophagitis. Grade 1 chronic iron deficiency anemia".

According to the EULAR-2024 guidelines, the Still’s syndrome was evaluated using the validated scales (adapted from [4, 17]) (Table 1).

The following indices were determined in the patient: SDAS=7, mPoS=8, SMS=5, SFS=7 (i.e. high disease activity).

With the following treatment administered: CS 24 mg/day, methotrexate 15 mg weekly, pulse therapy with methylprednisolone (250 mg No. 3, IV infusion) — the patient's condition improved with the body temperature normalization (36.8 °C), rash and joint syndrome regression. After the pulse therapy, positive laboratory changes were noted in the patient: CRP levels decreased to 20.2 mg/L, hemoglobin levels elevated to 116 g/L, ESR decreased to 40 mm/h, ALT and AST levels normalized (23 and 30 U/L, respectively).



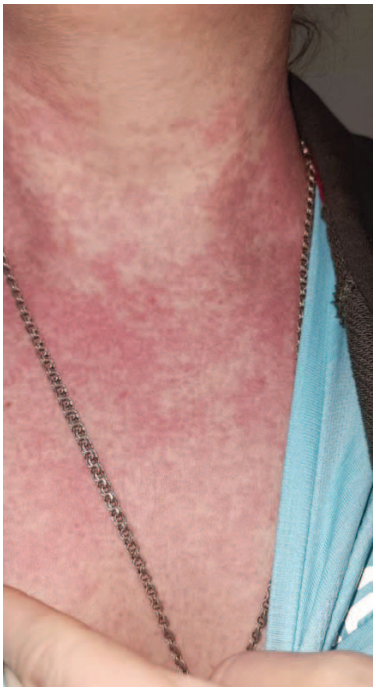


Figure 3, 4. *Erythematous and macular rash on the neck*

Table 1. *Approved scales for assessing BD activity*

Manifestations	SDAS[1]	mPoS[2]	SMS[3]	SFS[4]
Daily or night fever of 37.5–38°C (99.5-100.3°F)	-	-	1	1 ✓
Daily/nighttime fever of 38–39°C (100.4-102.1°F)	-	1 ✓	2 ✓	
Daily/nighttime fever of 39–40°C (102.2-103.9°F)	1		3	
Daily/nighttime fever above 40°C (>104°F)			4	
Muscle pain (myalgia)	-	1 ✓	-	-
>2 swollen joints (inflammatory synovitis)	1 ✓	1 ✓	-	-
Rash in Still's disease (not on the face, confirmed by a doctor)	1 ✓	1 ✓	1 ✓	1 ✓
Weight loss (>10 % of body weight)	1	-	-	-
Sore throat (current or within the past 2 weeks)	1 ✓	1 ✓	1 ✓	-
Pleurisy or pleural effusion		1		
Pericarditis or pericardial effusion	1	1	1	1
Pneumonitis (according to X-ray data)		1		
Peritonitis		-		
Generalized lymphadenopathy	1 ✓	1 ✓	1 ✓	1 ✓
Hepatomegaly or splenomegaly		1 ✓	1 ✓	1 ✓
Elevated liver enzymes (AST[5] or ALT[6] > 1,5 times above the LNL[7])	1 ✓		-	-
Elevated CRP[8] level (>20 mg/l)		-	-	1 ✓
Elevated ESR[9] (>50 mm/h)	1 ✓	-	-	1 ✓
Elevated ferritin (>500 μg/mL)		1 ✓		-
Increased platelet count (>600 × 10^3/μl)	-	-	1	1
Increased white blood cell count (>12,5 × 10^3/μl)	1 ✓	1 ✓	-	1 ✓
Anemia (haemoglobin <9 g/dl)	-	-	1	1

***Notes:** [1] SDAS — Still's Disease Activity Score, [2] SMS — Systemic Manifestation Score, [3] SFS — Systemic Feature Score, [4] mPoS — Modified Pouchot-Activity Score, [5] AST — aspartate aminotransferase, [6] ALT — alanine aminotransferase, [7] LNL — limit of the normal level, [8] CRP — C-reactive protein, [9] ESR — erythrocyte sedimentation rate.

Table 2. *Chronological scale of clinical and laboratory changes*

Data	Clinical changes	Laboratory and instrumental studies of indicators
22.01.2025	• Maculopapular rash • A feeling of tickling in the throat	
25.01.2025	• Body temperature rise to 38C • Contacting a therapist • Catarrhal angina has been diagnosed • ABT[1] is prescribed: azithromycin 500 mg 1 r/day orally and ceftriaxone 1.0 g 2 r / day by injection intramuscularly	
30.01.2025	• Body temperature preservation • Arthritis of small joints of hands, wrist, shoulder and ankle joints • The spread of maculopapular rash on the lower legs, thighs, forearms in combination with itching § • Replacement of ABT with Levofloxacin 500 mg 2p/day with subsequent use within 3 days	• Leukocytes — 16×10⁹/l (N=4-9×10⁹/l) • ESR[2]-35 mm/h (N=2-15 mm/h) • CRP[3]-71 mg/l (N<5,0 mg/l)
03.02.2025	• Lack of positive dynamics	• Leukocytes — 26.4×109/l (N=4-9×10⁹/l) • ESR-51 mm/h (N=2-15 mm/h) • CRP -171 mg/l (N<5,0 mg/l) • ALT[4] — 96 U/l (N<34 U/l), AST[5]-82 U/l (N<31 U/l) • Proteinuria-0,3 g/l (N<0,14 g/l)
05.02.2025	• Consultation with a rheumatologist • "Undifferentiated arthritis" was diagnosed • Treatment: methylprednisolone 16 mg/day, Meloxicam 15 mg/day	
13.02.2025	• Positive dynamics • The disappearance of the rash • Reducing the severity of arthritis	• Leukocytes — 12,3 ×10⁹/л (N=4-9×10⁹/l) • ESR — 38 mm/h (N=2-15 mm/h) • CRP — 40,2 mg/l (N<5,0 mg/l) • RF[6] — 14,1 IU/ml (RF<14,0 IU/ml) • ANA-Hep2[7] — 1:320 (AC-2,4)
21.02.2025	• Rheumatologist's consultation, dose increase • Correction of therapy: – escalation of the dose of methylprednisolone to 20 mg 1 r / day orally – initiation of MTX[8] 10 mg once a week parenterally	
25.02.2025	• A condition of moderate severity • Fever 37.3 C • Lymphadenopathy of the anterior cervical lymph nodes • Splenomegaly • Arthritis of the right shoulder, both wrist joints, II PIP[9] of both hands, both ankle joints • Preliminary diagnosis: "Still's disease, early stage, high degree of activity (DAS28-CRP=5.94[12], DAS28-ESR[13]=6.07), radiological stage 1, FC II» Correction of therapy: escalation of methylprednisolone to 24 mg per day orally, MTX — up to 15 mg once a week parenterally in combination with folic acid 5 mg	• Leukocytes — 13,6×10⁹/l (neutrophils — 74,7 %, pratelets — 275×10⁹/l) • ESR — 35 mm/h (N=2-15 mm/h) • CRP — 48,2 mg/l (N<5,0 mg/l) • RF — 12,8 IU/ml (RF<14,0 IU/ml) • ACPA[10] — <5 IU/ml (N<5,0 IU/ml) • AST — 37,1 U/l (N<31 U/l), ALT — 19,9 U/l (N<34 U/l) • IL-18[11] — >1000 pg/ml (<270 pg/ml) • Renal/abdominal ultrasound: moderate splenomegaly • X-ray of the hand bones: signs of stage 1 arthritis • Hematologist: chronic iron deficiency anemia grade 1, lymphoproliferative process excluded
26.02.2025	• Fever up to 38.5°C • Increased joint pain • Appearance of erythematous-macular rash on the face • Diagnostics: assessment of the antinuclear antibody and antiphospholipid antibody profile, complement components C3 and C4 to exclude systemic connective tissue disease • Modification of therapy: pulse therapy with methylprednisolone 250 mg № 3 intravenously by drip • The final clinical diagnosis: • "Primary: Still's disease, early stage, high activity (DAS28-CRP=5.94, DAS28-ESR=6.07, SDAS=7, mPoS=8, SMS=5, SFS=7), radiological stage 1. FC II.	• CRP — 45,6 mg/l (N<5,0 mg/l) • LDH[14] — 664 U/l (N=125-220 U/l) • Ferritin — 285 mkg/l (N=10-150 mkg/l) • Abdominal ultrasound: splenomegaly
03.03.2025	• Sore throat • Recurrence of rash in the décolleté, upper and lower extremities • Fever 37.8°C • Therapy adjustment: add colchicine at a dose of 1 mg orally once daily	• CRP — 20,2 mg/l (N<5,0 mg/l) • CBC[15]: haemoglobin — 116 g/l (N=120-140 g/l), ESR — 40 mm/h (N=2-15 mm/h) • ALT — 23 U/l (N<31 U/l), AST — 30 U/l (N<34 U/l)
07.03.2025	• Fever — 36.8C • Regression of maculopapular rashes and arthritis, reduction in the severity of sore throat, leveling of lymphadenopathy	• CRP — 8,82 mg/l (N<5,0 mg/l) • CBC: ESR — 26 mm/h (2-15 mm/h), leukocytes — 10,1×10⁹/l
04.04.2025	• No complaints • Treatment: MTX 15 mg once a week parenterally, methylprednisolone 16 mg orally with subsequent de-escalation to 4 mg, colchicine 1 mg once a day orally	• CRP — 3,39 mg/l (N<5,0 mg/l) • ESR — 15 mm/h (N=2-15 mm/h) • Ferritin — 139 ng/ml (N=11,0 до 306,8 ng/ml)

*** Notes.** [1] ABT — antibacterial therapy, [2] ESR — erythrocyte sedimentation rate, [3] CRP — C-reactive protein, [4] ALT — Alanine aminotransferase, [5] AST — Aspartate aminotransferase, [6] RF — Rheumatoid factor, [7] ANA-HEp2 — Antinuclear Antibodies HEp-2 Substrate, [8] MTX — methotrexate, [9] PIP — proximal inter-phalangeal (PIP) joints, [10] ACPA — anticitrullinated peptide antibodies, [11] Interleukin 18 — IL-18, [12] DAS28-CRP — Disease activity score 28 — CRP, [13] DAS28-ESR — Disease activity score 28 — ESR, [14] LDH — lactate dehydrogenase, [15] CBC — common blood count.

However, 3 days after methylprednisolone administration the patient developed a scratchy feeling in the throat, recurrent rash in the V-neck area, on the skin of upper and lower extremities, fever (37.8 °C). Due to the resistant disease course, the rheumatologist team decided to administer colchicine (1 mg once daily orally) for 28 days.

During colchicine treatment, the patient's body temperature normalized (36.8 °C), maculopapular rash and arthritis regressed, the scratchy feeling in the throat improved, lymphadenopathy disappeared. 14 days after the start of colchicine therapy, laboratory tests demonstrated CRP level decrease to 8.82 mg/L, ESR — to 26 mm/h, white blood cells — to 10.1×10^9/L.

Positive clinical & laboratory trends were also preserved during the next days. Within 28 days after hospitalization, clinical signs did not recur, while levels of CRP (3.39 mg/L; N<5 mg/L), ESR (15 mm/h), and ferritin (139 ng/mL) normalized. The follow-up USAC detected the preserved splenomegaly (~140 mm long). Further methylprednisolone dose tapering was recommended (4 mg every 14 days to 16 mg), followed by the rheumatologist counseling with the purpose of tapering the CS dose to the supportive one. It was also recommended to continue methotrexate 15 mg weekly in combination with folic acid 5 mg, colchicine 1 mg for 3 months with the monitoring of CBC, urinalysis, creatinine, urea, ALT, AST, glucose, CRP.

See Table 2 for changes of clinical & laboratory parameters during the whole treatment selection period.

Discussion

The pathogenesis of autoinflammatory diseases affects the congenital immune system; several pathologies are characterized by the activation of inflammasomes with subsequent IL-1β production [18]. It should be noted that clinical signs of SD are somewhat similar to the manifestations of autoinflammatory diseases.

In its turn, colchicine is widely used in rheumatology in the treatment of gout, familial Mediterranean fever, and Behcet's disease [19]. The drug action principle is based on the inhibition of chemokines, neutrophils, and endothelial cell adhesion molecules [21]. A recent study has shown that colchicine inhibits the assembly of the inflammasome complex by affecting the transport of the apoptosis-associated SPEC-like protein and the protein caspase recruiting domain [18, 20]. These data allow to propose that colchicine may modulate the inflammasome-mediated proinflammatory cascades, which defines the review of the treatment concept for colchicine-sensitive inflammatory conditions.

The Practical Guidelines for Rheumatologists concerning SD published by the British Rheumatology Society [21] traditionally include CS, MTX, azathioprine, and hydroxychloroquine as anti-inflammatory treatments used in SD. If the disease is resistant to therapy administered, treatment can be modified with the administration of IL-1 (canakinumab, anakinra) or IL-6 (tocilizumab) inhibitors; tumor necrosis factor α (TNF-α) inhibitors may be considered, although the priority is given to IL-1 and IL-6 inhibitors [21]. Colchicine is not listed as a possible therapeutic option in SD.

A review by Gerfaud-Valentin M. et al. [22] notes that TNF-α inhibitors may be used in chronic polyarticular resistant SD, predominantly in systemic lesions; however, the efficacy of tumor necrosis factor α inhibitors is limited in time, while switching from one drug to another is efficient approximately in 50 % cases. Based on Efthimiou P. et al. [23], patients with arthritis without systemic manifestations, lower IL-18 and ferritin levels in the blood serum are more likely to demonstrate positive effects during the tumor necrosis factor α inhibitor use, while switching inside this drug group does not lead to positive effects.

IL-6 levels are significantly elevated in active SD; this cytokine is considered a target in case of SD resistant to standard treatment [24]. The use of tocilizumab, a humanized anti-IL-6 receptor antibody that blocks membrane-bound and soluble IL-6 receptors in SD resistant to CSs, DMARDs, TNF-α inhibitors, and cyclosporine, has demonstrated positive effects regarding arthritis and systemic manifestations [24].

IL-1 inhibitors play a pivotal role among BAs regarding efficacy. Treatment with IL-1 inhibitors is significantly more efficient in patients with SD resistant to traditional treatment [25]. Quick start of IL-1 blocking therapy is associated with better disease outcomes and optimal patient retaining in the inpatient department [26, 27]. The response to IL-1 inhibitors is quick and stable, which allows patients to taper CS doses. It is important that the inefficacy of the first IL-1 inhibitor does not exclude the achievement of therapeutic effects with the use of another IL-1 inhibitor. Besides, IL-1 inhibitors have demonstrated a satisfactory safety profile in the SD treatment [28, 29].

The use of a Janus-kinase (JAK) inhibitor tofacitinib led to disease remission and CS dose tapering in 14 cases of resistant SD [30].

Despite a large spectrum of BAs that have demonstrated positive effects in resistant SD, IL-1 and IL-6 inhibitors are leading in the efficacy [31]; the results of clinical trials for canakinumab and tocilizumab in systemic SD manifestations have showed that these

treatment methods cannot completely decrease the risk of the macrophagal activation syndrome, even with sufficient disease control [32].

A review of Bindoli et al. [33] analyzed the efficacy and safety of SD and MAS treatment, and colchicine was considered a possible therapeutic option along with CS, intravenous immune globulin based on the experience of V. Myachikova et al. [35]. However, the experience of using colchicine in patients with SD is sparse. The focus of authors' attention was concentrated on the possibility of using BAs, in particular IL-1 (canakinumab, anakinra, rilonacept) and IL-6 (tocilizumab) inhibitors [34].

The first data on the efficacy of colchicine in combination with non-steroid anti-inflammatory drugs (NSAIDs) in patients with SD and serositis were published in the study of V. Myachikova et al. [34]. When analyzing the medical charts of patients that used colchicine as an additional drug in the setting of pericarditis, the effects were achieved not only concerning pericarditis, but also regarding other manifestations, i.e. arthritis/arthralgia, rash, leukocytosis, inflammatory markers. More than 50 % patients achieved complete disease remission, 15 % patients achieved partial response, and approximately 20 % patients did not respond to therapy. Based on the author's view, due to favorable safety profile colchicine may become an evident alternative in the first line of treatment of systemic SD with serositis. Besides, one should note rather low colchicine costs and its availability, thus making it possible to use as an alternative to CSs, MTX, and BAs [35].

Tomoyuki Asano et al. used colchicine in patients with SD resistant to TNF-α or IL-6 inhibitors. In our clinical case colchicine demonstrated efficacy concerning the decreased severity of SD symptoms in a patient resistant to CSs and BAs; thus, it can be also positioned as an alternative to BAs — IL-1 and TNF-α inhibitors [19].

Accounting for the high BA costs and the risk of adverse reactions (increased infection rate), colchicine is a good alternative for decreasing the inflammatory process activity in patients with SD, if the timely BA treatment is impossible due to the time and financial constraints.

Our experience of adding colchicine to MTX and CS in the treatment of SD led to the complete regression of clinical symptoms, including systemic ones, as well as to the persistent control of laboratory inflammatory markers. Due to high CRP levels at the disease onset, systemic manifestations, high SD activity, and resistance to standard treatment, this patient was in an evident risk group for MAS. The addition of colchicine to MTX and CS helped to reach the complete remission along with the prevention of MAS, a life-threatening SD complication. Based on the positive experience of using colchicine as a DMARD in combination with MTX and CS, this therapy regimen may become a robust and efficient BA alternative in resistant SD, if BAs are not available or contraindicated.

Conclusions

This clinical experience of using colchicine in a patient with AOSD has demonstrated colchicine efficacy in SD resistant to CS, as well as the possibility of its use as an alternative to costly BAs (i.e. IL-1 and IL-6 inhibitors).

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