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Case report

Experience of abrocitinib in atopic dermatitis

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ABSTRACT

Atopic dermatitis is an autoinflammatory, genetically determined, itchy disease characterized by a long, relapsing course and a sharp decrease in the patient's quality of life. Atopic dermatitis is caused by a complex interaction of immune dysregulation, epidermal gene mutations, and multi-environmental factors that affect the skin, causing intense itchy rashes. The disease often occurs among young children and subsequently has age-related clinical features.

Currently, the treatment of atopic dermatitis has a wide range of options, including drugs from the group of small molecules recently registered in the Russian Federation. The drugs are intended for patients with moderate to severe atopic dermatitis. Baricitinib, abrocitinib and upadacitinib as Janus kinase inhibitors from the small molecule group have already demonstrated good tolerability, safety and a pronounced clinical effect in the vast majority of patients with atopic dermatitis in available clinical trials.

The first clinical experience with the use of abrocitinib in adult patients with atopic dermatitis is presented. The interest of the presented clinical cases lies in the demonstration of high clinical efficacy and safety of mono- and combination therapy with the Janus kinase inhibitor abrocitinib. Despite the short period of treatment and observation (4 weeks), objective severity of atopic dermatitis manifestations (SCORAD index >60 points) and sharp decrease in the patients' quality of life (DLQI index >20 points), a reduction of clinical parameters exceeding 60% was achieved at a dose of 200 mg daily. During the whole observation period no adverse clinical reactions or changes in laboratory parameters were registered against the background of abrocitinib administration.

The pilot results of abrocitinib use are encouraging and provide grounds for conducting longer and larger-scale studies to investigate the efficacy of the drug for its inclusion in the combination therapy of patients with moderate to severe atopic dermatitis.

Keywords: atopic dermatitis; phototherapy; small molecules; abrocitinib; case report.

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Клинический случай

Опыт применения аброцитиниба при атопическом дерматите

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АННОТАЦИЯ

Атопический дерматит — аутовоспалительное, генетически детерминированное зудящее заболевание, которое характеризуется длительным рецидивирующим течением и резким снижением качества жизни больного. Атопический дерматит вызывается сложным взаимодействием иммунной дисрегуляции, мутаций эпидермальных генов и мультифакторов среды, которые поражают кожу, вызывая интенсивные зудящие высыпания. Заболевание часто встречается у детей раннего возраста и в дальнейшем имеет возрастные клинические особенности.

В настоящее время лечение атопического дерматита имеет широкий спектр возможностей, среди которых зарегистрированные в Российской Федерации препараты группы малых молекул, предназначенные для больных атопическим дерматитом средней и тяжёлой степени. Представители из группы малых молекул барицитиниб, аброцитиниб и упадацитиниб как ингибиторы янус-киназ в ходе имеющихся клинических исследований уже продемонстрировали хорошую переносимость, безопасность и выраженный клинический эффект у подавляющего большинства больных атопическим дерматитом.

В статье приводится первый клинический опыт применения аброцитиниба у взрослых больных атопическим дерматитом. Интерес представленных клинических случаев заключается в демонстрации высокой клинической эффективности и безопасности моно- и комбинированной терапии ингибитором янус-киназ аброцитинибом. Несмотря на короткий период лечения и наблюдения (4 недели), объективную тяжесть проявлений атопического дерматита (индекс SCORAD >60 баллов) и резкое снижение качества жизни пациентов (индекс DLQI >20 баллов), при дозе препарата 200 мг в день была достигнута редукция клинических показателей, превышающая в целом 60%. За весь период наблюдения каких-либо нежелательных клинических реакций или изменений лабораторных показателей на фоне приёма аброцитиниба не зафиксировано.

Пилотные результаты применения аброцитиниба обнадёживают и дают основания к проведению более продолжительных и масштабных исследований по изучению эффективности препарата с целью его включения в комбинированную терапию больных атопическим дерматитом средней и тяжёлой степени.

Ключевые слова: атопический дерматит; фототерапия; малые молекулы; аброцитиниб; клинический случай.

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INTRODUCTION

Atopic dermatitis may begin at any age [1], though it is more prevalent in infancy or early childhood. Approximately 50% of cases develop symptoms within the first year of life, and as many as 85% of individuals experience an onset by the age of five. The hallmark symptoms of atopic dermatitis are severe pruritus, xerosis, and eczematous skin lesions [2]. The disease presents a diagnostic challenge, particularly in adult-onset cases [3]. The most prevalent diagnostic markers of atopic dermatitis are the specific clinical presentations, body areas most commonly affected, and pruritic rash [4]. In most cases, the disease is characterized by alternating periods of flare-ups and remissions. Severe pruritus in atopic dermatitis results in considerable distress and significantly compromises the quality of life. The disease may be associated with allergic comorbidities [5, 6].

The pathogenesis of atopic dermatitis can be attributed to multiple factors, including genetic susceptibility, epigenetic modifications, epidermal barrier defects, altered skin microbiome, and dysregulated immune responses [6, 7]. It has been demonstrated that inherited mutations, in particular those affecting the filaggrin gene, are associated with the epidermal barrier dysfunction and the occurrence of skin xerosis [8]. Although filaggrin is essential for maintaining skin hydration, its deficiency contributes to the development of atopic dermatitis through alternative mechanisms. In particular, impaired epidermal keratinization has been shown to trigger Th2- and Th17-mediated immune responses, which play a role in the pathogenesis of atopic dermatitis [9, 10]. Some studies have demonstrated a positive correlation between filaggrin gene mutations and elevated levels of immunoglobulin E (IgE), which may in turn induce the disease progression to the atopic march [11]. A potential link between atopic dermatitis and deficient ceramide 1, a component of the intercellular matrix, has also been suggested [12].

A comprehensive understanding of the disease mechanisms requires an appreciation of immune dysregulation. It is postulated that allergens and pathogens can penetrate readily through a compromised epidermal barrier, thereby triggering a hyperimmune response with the release of proinflammatory cytokines, which in turn give rise to the observed clinical symptoms. It can be reasonably deduced that the primary mechanism underlying the pathogenesis of atopic dermatitis is the disruption of the skin barrier [13].

Atopic dermatitis may be associated with reduced production or qualitative alterations of antimicrobial peptides. Consequently, the proliferation of microbial pathogens, including opportunistic species, may result in recurrent secondary infections [14], predominantly caused by *Staphylococcus aureus* [15].

Lymphoid cells, which are responsible for cytokine production, are involved in regulating both immune and non-immune mechanisms within tissues. They are capable of inducing pro-inflammatory cytokines such as interleukins (IL) 4, 5, 9, and 13. This suggests that innate lymphoid cells can initiate a Th2-mediated immune response, thereby triggering an allergic cascade [16].

In light of the aforementioned considerations, a systematic approach to the treatment of atopic dermatitis is recommended. However, regular topical use of moisturizing, lipid-saving, and emollient agents to restore the skin barrier and control xerosis is essential in the treatment and prevention of flare-ups of any severity [17]. Topical glucocorticoids are a class of potent anti-inflammatory medications that have emerged as the first-line therapeutic option for patients with atopic dermatitis. Calcineurin inhibitors have also demonstrated efficacy as external immunosuppressants in the treatment of atopic dermatitis, with a favorable safety profile [18].

Systemic glucocorticoids are indicated for moderate-to-severe atopic dermatitis, given the potential for adverse effects and the tendency for symptoms to recur rapidly upon discontinuation. For secondary infections, antibacterial agents are indicated. Non-steroidal systemic immunosuppressants, including cyclosporine A and methotrexate, may offer a viable alternative to steroid therapy. These drugs have a specific toxicity profile and diverse therapeutic effects across different patients, necessitating frequent laboratory monitoring [19]. Furthermore, a substantial body of evidence has demonstrated the efficacy of various modalities, including ultraviolet B (UV-B) 311 nm light therapy, transcranial electrical stimulation, and general cryotherapy. As a part of a combination therapy for patients with atopic dermatitis, these modalities have been shown to significantly improve treatment outcomes.

In addition to the approaches outlined in the clinical guidelines, monoclonal antibodies, a class of biopharmaceuticals (e.g., dupilumab, an IL-4 and IL-13 inhibitor), and small-molecule drugs targeting signaling pathways are emerging as a current standard of care in atopic dermatitis. These drugs are indicated for patients presenting with moderate-to-severe atopic dermatitis. A review of the available clinical studies indicates that small-molecule drugs, namely Janus kinase inhibitors baricitinib, abrocitinib, and upadacitinib, have demonstrated high efficacy in the treatment of atopic dermatitis [20].

In a recent study, Reich et al. [21] evaluated the efficacy and safety of abrocitinib. Their findings suggest that abrocitinib 200 mg daily is an effective and safe treatment option for adults with moderate-to-severe atopic dermatitis. The drug was well tolerated over a 26-week period and demonstrated a significant clinical effect in the majority of patients. Bieber et al. [22] also showed that abrocitinib was more efficacious than placebo or dupilumab in reducing the symptoms of atopic dermatitis.

In their study, Blauvelt et al. [23] evaluated the long-term efficacy of abrocitinib 200 mg/day in 1,233 patients over 12 weeks. Subsequently, the patients were randomized to

one of three groups: abrocitinib 200 mg, abrocitinib 100 mg, and a placebo group that did not receive any active drug over 40 weeks. The authors observed no episodes of flare-ups among the subjects who received abrocitinib 200 mg. In 80% of cases, patients who discontinued the therapy subsequently experienced a recurrence of the disease. The combination of repeated rehabilitation treatment and local therapy for atopic dermatitis yielded a favorable therapeutic response.

The U.S. Food and Drug Administration (FDA) has approved abrocitinib for the treatment of moderate-to-severe atopic dermatitis in adolescents aged 12–17 years and adults who have not responded to other systemic drugs [24]. The expanded use is based on data from JADE TEEN, a phase III, randomized, double-blind, placebo-controlled clinical trial [25]. The previous three phase III studies (JADE MONO-1, JADE MONO-2, JADE COMPARE) also demonstrated favorable outcomes. Abrocitinib 100 mg or 200 mg is also included in the current version of the Russian clinical guidelines for the treatment of atopic dermatitis. The drug may be used either alone or in combination with topical therapy for moderate-to-severe disease, including in refractory cases where other standard treatments have proven ineffective [26].

Therefore, the present situation requires further investigation into the efficacy and safety of small-molecule drugs for the treatment of atopic dermatitis, as the currently available studies are insufficient to conclusively assess the clinical efficacy of this class.

This study describes two cases of atopic dermatitis treated with abrocitinib.

CASE REPORTS

Case report 1

Case description. On December 12, 2023, a male patient (born in 1984) was admitted to the Rakhmanov Clinic for Skin and Venereal Diseases of the Sechenov First Moscow State Medical University (Rakhmanov Clinic). The patient presented with a pruritic rash located inside the elbows, on the neck and chest. The patient was diagnosed with atopic dermatitis upon admission.

Medical history. The patient reported that the initial presentation of symptoms occurred approximately 3 or 4 years before, when he experienced persistent inflammatory lesions affecting the groin, hands, buttocks, and dorsal feet. Although a similar rash had occasionally occurred before, it was transient, mild in severity, and required no medical attention. Following phototherapy, there was a notable worsening of the skin eruptions observed on the patient's chest and back (Fitzpatrick skin type I). Subsequently, the rash extended to other regions of the body. The therapy included intravenous sodium thiosulfate, ozonized physiological saline solution, and topical applications of Dermovate cream. This resulted in a mild therapeutic effect and a two-month remission. In 2022, the patient presented to the Rakhmanov Clinic, where he received a standard topical therapy comprising Tetraderm, Dermovate and Triderm creams, which demonstrated a transient efficacy. Subsequently, the patient was treated at several clinics, where he was administered dexamethasone, prednisone, antibiotics, antihistamines, and systemic oxygen ozone therapy, resulting in transient partial remissions. On December 12, 2023, he was admitted to the Rakhmanov Clinic. Phototherapy (311-nm UV-B, 15 seconds) resulted in a flare-up with more intense rash color, crusting, dry and itchy eruptions.

Noteworthy, the patient's mother had a history of food allergies.

Status localis (on admission). The patient presented with chronic inflammatory skin eruptions. The abundant, acute inflammatory rash was found to have spread to the armpits, neck, inner elbows, chest, and abdomen. The eruptions were represented by diffuse, erythematous lesions with poorly defined borders and varying shapes. Additionally, there were multiple, severely scaly papules rising above the skin level. Furthermore, the affected area exhibited punctate and linear excoriations, shallow cracks, and punctate erosions coated with yellow-brown crusts. Lichenifications and crusts were observed inside the elbows and around the neck, with white dermographism on the visually intact skin. Additionally, the patient reported that the skin lesions were intensely pruritic. The patient's Scoring Atopic Dermatitis (SCORAD) and Dermatology Life Quality Index (DLQI) scores were 58 and 19, respectively (Figure 1).

Clinical and diagnostic findings. Hematology (December 14, 2023): hemoglobin (Hb) 160 g/L, red blood cells (RBC) 5.39×10^{12}/L, platelets 239×10^{9}/L, white blood cells (WBC) 9.41×10^{9}/L, neutrophils 74.8%, lymphocytes 15.6%, monocytes 9%, and erythrocyte sedimentation rate (ESR) 5 mm/h.

Urinalysis (December 14, 2023): transparent, light-yellow urine, pH 6.5, specific gravity 1.016.

Blood chemistry (December 14, 2023): total protein 68 g/L, uric acid 394.8 μmol/L, cholesterol 6.44 mmol/L, triglycerides 2.72 mmol/L, bilirubin 14.2 μmol/L, aspartate aminotransferase (AST) 15 U/L, alanine aminotransferase (ALT) 24 U/L, creatinine 102 μmol/L, and glucose 5.44 mmol/L.

A diagnosis of severe atopic dermatitis was based on the patient's symptoms, medical history, and clinical presentation.

Treatment. On December 15, 2023, a 2-week course of abrocitinib 200 mg once daily was initiated. This treatment decision was based on the severity and extent of the inflammatory process, failure of previous treatments, and a notable decline in the quality of life.

Treatment outcomes and follow-up. At 12 days of the in-hospital treatment, the patient's symptoms improved significantly with a reduction in the intensity of the rash color and the resolution of lichenifications, crusting, and pruritus. The SCORAD and DLQI scores improved to 26 and 7,

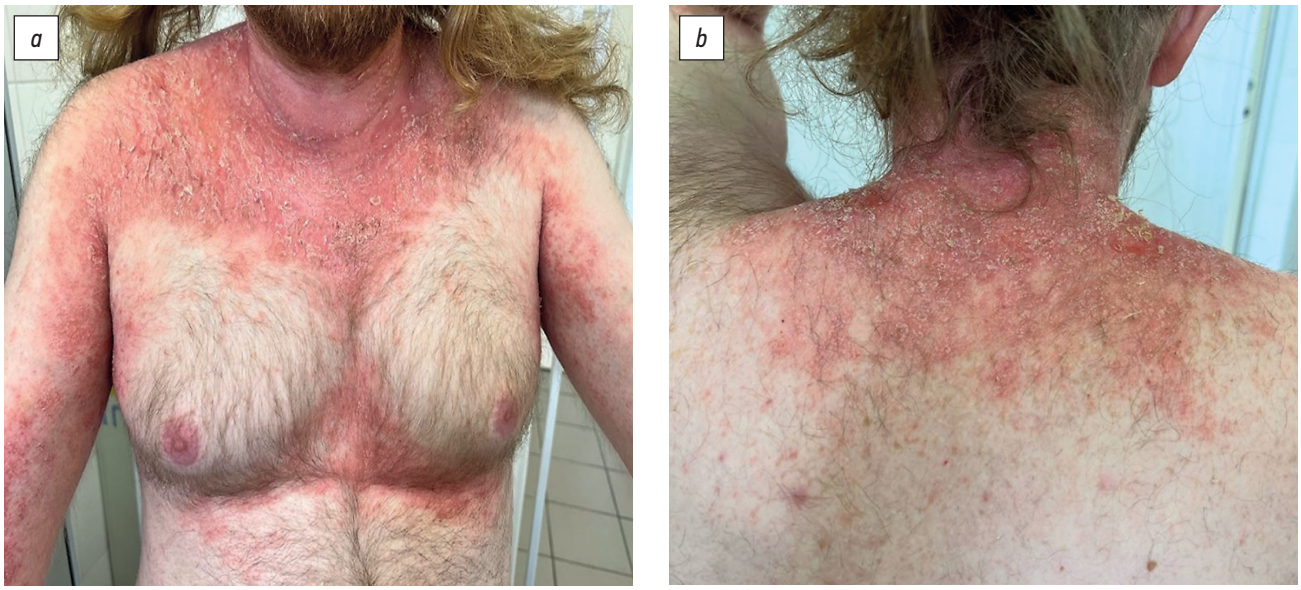

Fig. 1. Patient Ch., 39 years old, with atopic dermatitis before treatment: itchy rashes on elbows, chest (*a*) and back of neck (*b*).

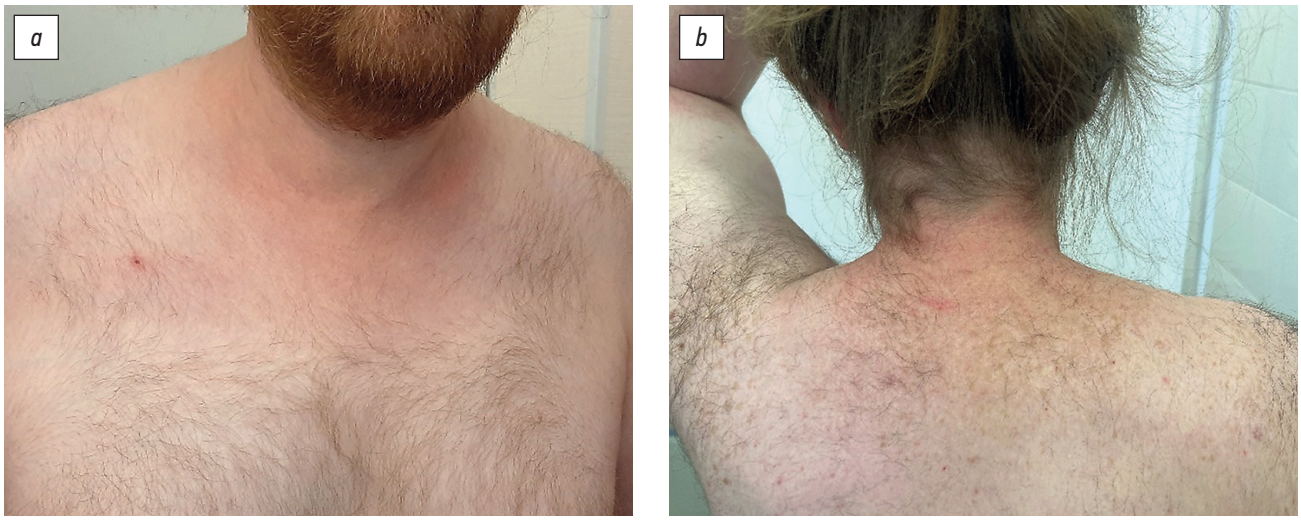

Fig. 2. Patient Ch. after two weeks of abrocitinib (11.01.2024): significant regression of skin manifestations in the chest (*a*) and posterior neck (*b*).

respectively. The drug was well-tolerated, and no laboratory abnormalities were reported. Upon discharge, the patient continued abrocitinib 100 mg once daily (Figure 2).

Case report 2

Case description. A 43-year-old male patient presented to the Rakhmanov Clinic with pruritus, skin rash, sleep disturbance, and irritability.

Medical history. As reported by the patient's mother, he has been experiencing dermatological problems since birth. These included a pruritic rash that first emerged on the face, inner elbows, back of knees, and neck. The patient's family history indicates that the patient's father suffered from atopic dermatitis during his childhood. During the entire period, the patient self-medicated with a variety of corticosteroids and Suprastin with a transient improvement in symptoms. He reports a tendency for his symptoms to improve in the summertime. By 2013, the rash had extended to the scalp. In July 2023, the patient's symptoms worsened with the spread of a rash throughout the body, including on the face. On November 22, 2023, additional laboratory testing showed a total IgE level of 6,540 IU/mL. The outpatient treatment, which included antihistamines and topical corticosteroids, failed to provide a sustained therapeutic effect. Given the patient's presentation of a torpid variant of the dermatological process, he was admitted to the Rakhmanov Clinic to receive a combination therapy.

Status localis (on admission). The skin eruptions were indicative of a chronic inflammatory process. The rash exhibited a symmetrical distribution, affecting primarily the inner elbows, lower legs, neck, and back. The eruptions predominantly consisted of pale-pink, flat, rounded papules

0.2–0.5 cm, which randomly merged to form extensive lichenifications. The extensive, pruritic excoriations were primarily localized on the neck, face, inner elbows, back of the hands, and lower legs (Figure 3).

Laboratory results. The hematology and urinalysis values were found to be within the reference ranges. On November 07, 2023, the total IgE level was 6,540 IU/mL. On the same day, the patient was found to be negative for allergen-specific IgE, and ELISA further confirmed the absence of helminth or protozoan invasions. The baseline SCORAD and DLQI scores were 63 and 21, respectively.

The diagnosis of atopic dermatitis was based on the patient's symptoms, past medical history, relevant family history, a history of food allergy in childhood, typical clinical presentations, and laboratory data.

Treatment. On November 07, 2023, narrow-band UV-B light therapy (311 nm; 4 sessions/week, a total of 8 sessions) was initiated for the severe and extensive process. The initial dose for UV-B phototherapy was 0.1 J/cm^2. No contraindications were identified. Medications: abrocitinib 200 mg once daily for 14 days, then 100 mg (1 tablet) once daily for 14 days; loratadine 10 mg (1 tablet) 2 times daily; omeprazole 20 mg (1 capsule) once daily, 30 minutes before meals; azithromycin 500 mg (1 tablet) once daily; fluconazole 150 mg (1 capsule) once every 3 days.

Treatment outcomes and follow-up. At 3 weeks, a favorable response to the combination therapy was documented, with the patient's symptoms of intense erythema color, crusting, and excoriation showing a significant improvement. Significant skin clearance and near-complete resolution of pruritus were reported. The treatment was well-tolerated; no side effects and adverse events were documented. The patient achieved an improvement in the SCORAD and DLQI scores to 32 and 8, respectively (Figure 4).

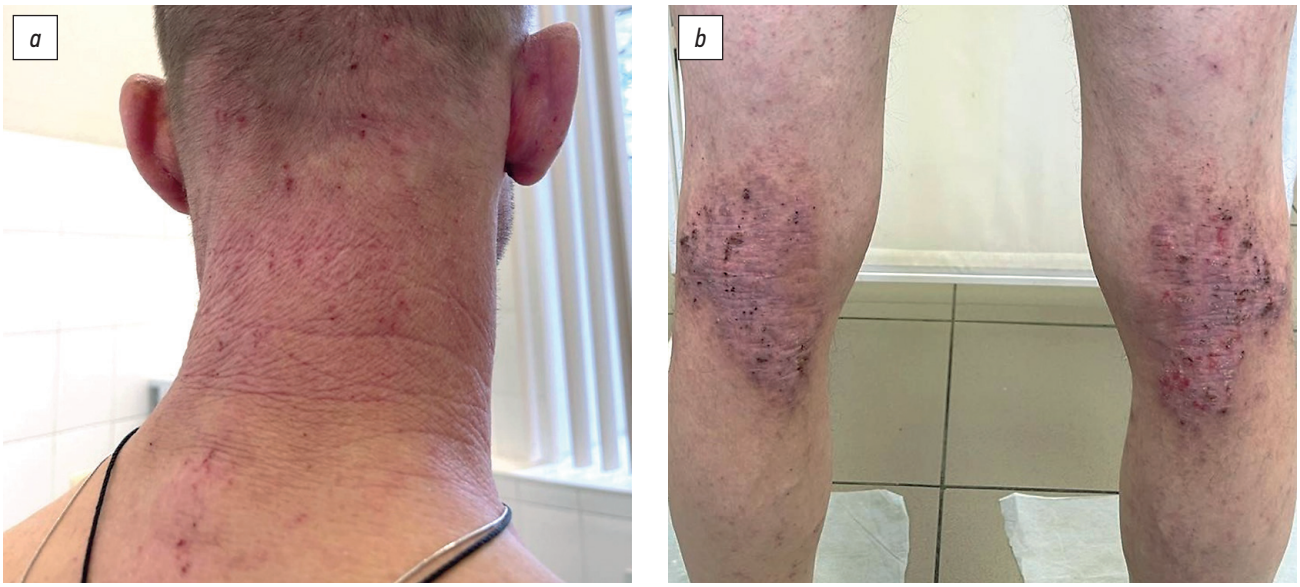

Fig. 3. Patient P., 43 years old, with atopic dermatitis before treatment: rash localised mainly on the neck, back (*a*) and lower limbs (*b*).

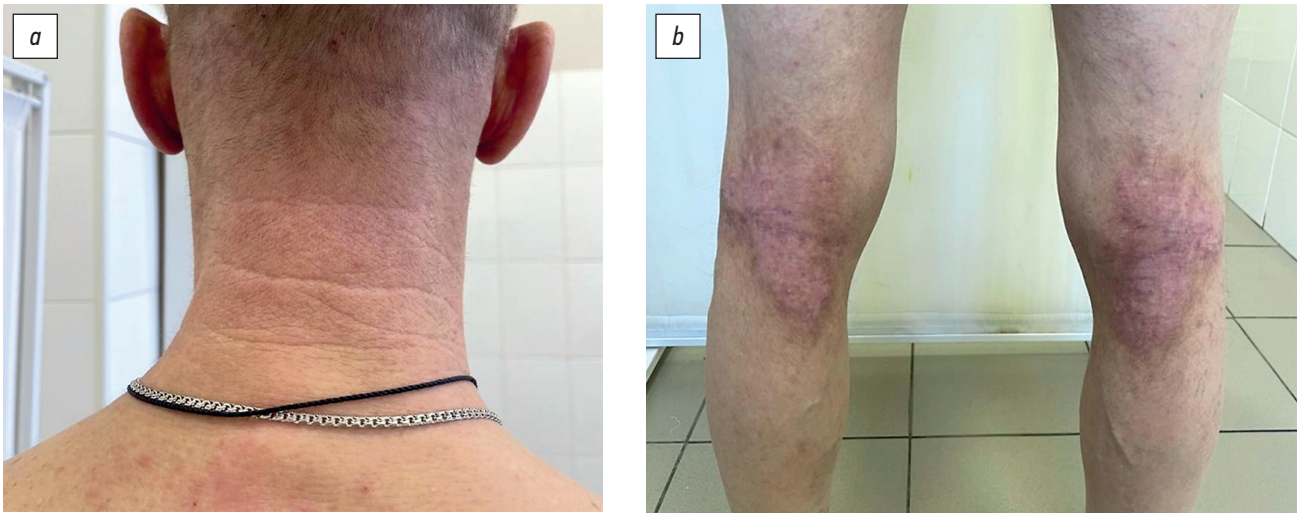

Fig. 4. Patient P. two weeks after therapy with abrocitinib + UVB 311 nm: positive dynamics of the skin process in the back, neck (*a*) and lower limbs (*b*).

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DISCUSSION

The 38-year-old male patient (Case No. 1) presented with the diagnosis of atopic dermatitis, which had been particularly severe for the previous 3 or 4 years. The past medical history was not readily accessible in sufficient detail, with the exception of a documented propensity for food allergies in the patient's mother. As the patient reported, the disease was highly unresponsive to a range of therapeutic regimens prescribed at various private clinics. Since the patient was classified as Fitzpatrick skin type I, the phototherapy sessions proved ineffective and resulted in flare-ups. All other treatments, including detoxification therapy, antihistamines, antibiotics, and systemic and topical corticosteroids, demonstrated only transient and inadequate efficacy. The diagnosis of severe atopic dermatitis was based on typical clinical presentations of significant exudative inflammation. Other diagnostic signs included common locations of the rash, severe pruritus, white dermographism, and significant treatment resistance. The decision to initiate abrocitinib was based on the severity of the clinical symptoms and the lack of efficacy of previous treatments. At 2 weeks, abrocitinib 200 mg once daily resulted in a significant improvement in the clinical symptoms, as evidenced by a reduction in the SCORAD score (from 58 to 26) and the DLQI score (from 19 to 7). Abrocitinib was well-tolerated, and the patient experienced no adverse effects.

The 43-year-old male patient (Case No. 2) experienced first symptoms in early infancy. Prior to his visit to the Rakhmanov Clinic, the patient had been self-medicating with antihistamines and topical glucocorticoid ointments. In 2023, the patient's therapy proved ineffective, resulting in the occurrence of new lesions on the entire body. The most significant eruptions were observed on the inner elbows, back of knees, and hands. The rash was symmetrical and polymorphic in nature. At the Rakhmanov Clinic of the Sechenov First Moscow State Medical University, the diagnosis of atopic dermatitis was based on the findings of a physical examination and the patient's medical history. The patient was subsequently referred for additional testing. The hematological values were found to be within the reference ranges. The elevated level of total IgE in the blood test, at 6,540 IU/mL, was indicative of an IgE-mediated atopic dermatitis. Following a 2-week course of abrocitinib 200 mg followed by 100 mg once-daily dose in combination with 8 sessions of 311-nm UV-B light therapy, the clinical symptoms improved significantly. The patient demonstrated exemplary compliance with the prescribed treatment. At 1 month of the combination therapy, the SCORAD score was 32, demonstrating a 31-point improvement from the baseline. The patient's quality of life also exhibited a notable improvement, as evidenced by a reduction in the DLQI score from 21 (pre-treatment) to 8 (post-treatment). No adverse effects occurred during the treatment.

CONCLUSION

The presented clinical reports illustrate that abrocitinib, a Janus kinase inhibitor, either alone or in combination with other pharmacological agents was highly effective and safe in the treatment of atopic dermatitis. Despite the short-term treatment and follow-up (4 weeks), the objective severity of atopic dermatitis (SCORAD >60), and a dramatic decline in the quality of life (DLQI >20), abrocitinib 200 mg once daily yielded a >60% improvement in the clinical scores in both cases. Throughout the course of treatment, no adverse clinical symptoms or laboratory abnormalities were identified.

The preliminary findings warrant further investigation through larger, longer-term studies to evaluate the efficacy of abrocitinib in combination with other treatments for atopic dermatitis.

ADDITIONAL INFORMATION

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