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Хроническая индуцированная крапивница: классификация, современные аспекты диагностики и терапии

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АННОТАЦИЯ

Хроническая индуцированная крапивница — группа заболеваний, характеризуемая возникновением зудящих волдырей и/или ангиоотёков в ответ на специфические триггеры, такие как механическое раздражение, воздействие высоких и низких температур, вибрация, а также ультрафиолетовое излучение различного спектра, в течение 6 недель и более. Данная группа заболеваний характеризуется длительным течением, существенным снижением качества жизни пациентов, а также риском развития тяжёлых жизнеугрожающих реакций.

Диагностика индуцированной крапивницы — безопасное для пациента воспроизведение симптомов путём воздействия триггера (температурного, механического и т.д.). В настоящий момент в Российской Федерации нет зарегистрированных медицинских приборов для пороговой диагностики большинства физических форм крапивниц, что затрудняет постановку диагноза и динамическое наблюдение за состоянием данных групп пациентов и эффективностью лечения. Открытым остаётся также вопрос о терапии хронической индуцированной крапивницы ввиду недостаточной эффективности рекомендуемых и доступных методов терапии.

В статье представлен обзор актуальных литературных данных.

Ключевые слова: хроническая индуцированная крапивница; провокационное тестирование; антигистаминные препараты; омализумаб.

Как цитировать

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Chronic-induced urticaria; classification, actual aspects of diagnosis and therapy

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ABSTRACT

Chronic-induced urticaria is a group of diseases characterized by the occurrence of itchy wheals and/or angioedema in response to specific triggers, such as mechanical irritation, exposure to high and low temperatures, vibration, and ultraviolet radiation of various spectra, appearing for ⩾6 weeks. This group of diseases is characterized by a long duration, a significant effect on the patient's quality of life, and the risk of severe life-threatening reactions.

The diagnosis of induced urticaria and patient-safe reproduction of symptoms by exposure to a trigger (temperature, mechanical, etc.). Currently, no medical devices have been registered in the Russian Federation for the threshold diagnosis of most physical forms of urticaria. Consequently, diagnosing and dynamic monitoring the state of these patients and their response to treatment are challenging. The question of therapy for chronic-induced urticaria also remains open due to the insufficient effectiveness of the recommended and available treatment methods. This article provides a review of current literature data.

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Keywords: chronic-induced urticaria; provocation testing; antihistamines; omalizumab.

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List of abbreviations

AH: antihistamines
nsH_1-AHs: nonsedating H1 antihistamines
CIndU: chronic inducible urticaria
DLQI: Dermatology Life Quality Index

INTRODUCTION

Chronic inducible urticaria (CIndU) is a subtype of chronic urticaria characterized by recurrent itching wheals and/or angioedema lasting for 6 weeks or appearing in response to specific triggers such as mechanical irritation, exposure to high, or low temperatures, vibration, and UV radiation of various spectra.

CIndU is prevalent in up to 0.5% of the general population [1]. CIndU is diagnosed in 5%–25% of patients with chronic urticaria; its peak incidence occurs at a young age [2]. Studies revealed that the prevalence of CIndU was previously underestimated. Based on recent data, approximately 20% of patients with chronic spontaneous urticaria suffer from CIndU [3].

As described in the results of the international multicenter study AWARE [4], the quality of life of patients with a combination of chronic spontaneous urticaria and CIndU is significantly lower than those of patients suffering from only one type of chronic urticaria. CIndU has longer disease duration than chronic spontaneous urticaria [1, 5]. However, no current statistical data are available on the prevalence of induced forms of urticaria in the Russian Federation.

TYPES OF CINDU

Dermographic urticaria

Dermographic urticaria is the most common type of CIndU, and it is characterized by linear urticarial rashes on the skin appearing in response to mechanical irritation [2, 6]. A study reported [7] that approximately 25% of patients suffering from chronic spontaneous urticaria also have symptoms of dermographic urticaria.

This type of CIndU differs from simple red dermographism by the presence of itch (with simple red dermographism, the wheals do not itch). White dermographism, often associated with atopic diseases, is not associated with dermographic urticaria.

In dermographic urticaria, angioedema develops extremely rarely. This type of CIndU most often recurs over several years, followed by spontaneous remission [8, 9]. According to the review by Zuberbier and Maurer, which was published in 2007 [10], the average duration of dermographic urticaria is 6.5 years.

The diagnosis of dermographic urticaria is most often established based on medical history. Patients complain of a "creeping" itch without wheals, occurring only after mechanical irritation (scratching). Wheals can also appear with other types of mechanical influence, such as in the sites of friction of belts, bandages, upon wearing dense fabrics, washing with a washcloth, massage, skin allergy testing, etc. The diagnosis can be established, among other things, by objective examination (existing and/or provoked linear urticarial wheals on the skin) or analysis of photographs provided by the patient.

After taking a patient's history, provocative testing must be conducted. Typically, such testing consists of mechanical irritation of the skin with a wooden spatula. Calibrated dermographometers (Frick test, not registered in the Russian Federation) can be used to standardize the results. A positive test result is the appearance of pruritic wheals after the provocation, which persists for an average of 10 min (Figs. 1–3).

In the Russian Federation, the management of patients with CIndU is conducted in accordance with the clinical guidelines for the treatment of urticaria adopted by the Russian Association of Allergists and Clinical Immunologists in 2019 [11]. All patients with dermographic urticaria should be informed of the importance of avoiding triggers. Nonsedating H1 antihistamines (nsH1-AHs) in standard doses are recommended as first-line therapy. The dose of AHs can be increased up to fourfold with a lack of control over the symptoms of dermographic urticaria against the background of ongoing therapy [12–15].

In a retrospective study, Krause et al. [16] reported that AH therapy led to complete control of symptoms in 23% of patients and significant improvement in 49%, and only 4% of patients reported no effect. Patients resistant to AH therapy may receive off-label omalizumab treatment [17–19].

A systematic review of omalizumab therapy in patients with CIndU included seven publications presenting the treatment experience of 72 patients with dermographic urticaria: 54 and 18 patients were treated with omalizumab and placebo, respectively [18]. The most conclusive evidence comes from a randomized, placebo-controlled trial of the efficacy and safety of omalizumab in patients with dermographic urticaria resistant to AH therapy. A cohort of 55 patients with chronic dermographic urticaria was divided into three groups. The first group received omalizumab at a dose of 150 mg, the second group received omalizumab at a dose of 300 mg, and the third group received a placebo for 10 weeks. All patients were assessed for critical friction thresholds and quality of life using the Dermatology Life Quality Index (DLQI) before therapy. The average DLQI scores (11.1) before treatment initiation indicated a significant decrease in the quality of life in this group. During omalizumab therapy at doses of

Fig. 1. Frick-test. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

Fig. 2. Methodic of performing of provocation testing in patient with symptomatic dermographism. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

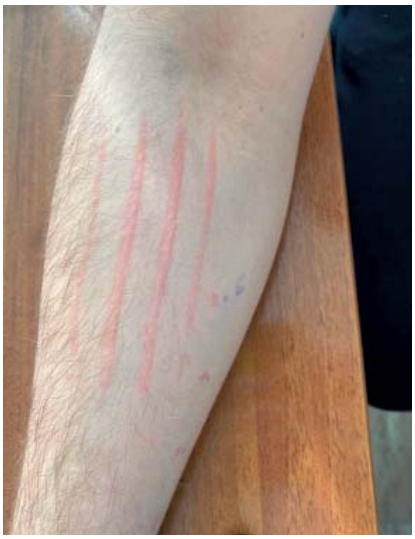

Fig. 3. Positive result of provocation testing in patient with symptimatic dermographism. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

300 and 150 mg, the participants showed clinically significant decreases in critical friction thresholds compared with patients who received a placebo. No significant difference was found in the effectiveness of therapy depending on the omalizumab dose, baseline levels of critical trigger threshold, or disease activity. During therapy, 72% and 58% of patients treated with 150 mg of omalizumab and 300 mg of omalizumab, respectively, improved their DLQI score by ≥4 points (minimum clinically significant difference). In the placebo group, only 32% of the patients experienced a similar improvement. The end point of observation was 10 weeks from the initiation of therapy. No changes in clinical parameters during therapy were observed in 33% of patients who received 150 mg of omalizumab and 42% of patients who received 300 mg of omalizumab. In the placebo group, 83% of patients noticed no clinically significant improvement.

Metz et al. [20, 21] described several clinical cases of successful omalizumab therapy in patients with dermographic urticaria along with chronic spontaneous urticaria and other CIndU types.

Therefore, omalizumab is a reasonable option for patients with chronic dermographic urticaria who do not respond to nsH1-AH therapy.

Cold urticaria

Cold urticaria, a CIndU subtype, is characterized by wheals, and/or angioedema appearing after cold exposure. This disease is a complex clinical problem due to the high risk of cold-induced anaphylaxis [22]. Typical cold urticaria is characterized by the appearance of wheals on the skin after cold exposure, resolving within a few hours [23]. In typical cold urticaria, a local reaction can be replicated using an ice cube test and/or testing with a TempTest device (not registered in the Russian Federation) [24, 25].

Atypical cold urticaria is characterized by an atypical localization of wheals and/or an atypical response to provocative testing [26–29]. Atypical forms of cold urticaria include systemic atypical cold urticaria, localized cold urticaria, localized cold reflex urticaria, delayed cold urticaria, cold-induced cholinergic urticaria, and cold-dependent dermographism.

The incidence of cold urticaria is approximately 0.05% in the population. The incidence of cold urticaria is higher in countries with cold climate. Women experience the disease more often. Cold urticaria can debut at any age; however, it peaks in the second or fourth decade of life [28, 29]. The average duration of cold urticaria is approximately 6 years; however, it can recur for 20 years or longer [24, 30–33].

Cold urticaria often coexists with other CIndU types. In two retrospective studies, 21% and 22% of patients (in each of the two studies) had a combination of dermographic urticaria and cold urticaria, and 8% and 10% of patients had a combination of cold urticaria and cholinergic urticaria, respectively [24, 34]. According to Sánchez et al. [7], cold urticaria occurs in approximately 13% of patients with chronic spontaneous urticaria.

The severity of cold urticaria can vary from local urticaria to systemic manifestations, including broncho-obstructive syndrome, hypotension, dizziness, gastrointestinal symptoms, and anaphylactic reactions [24, 29, 35]. The clinical presentation of cold urticaria depends on the duration of cold exposure, critical threshold temperatures, and other factors that have not yet been determined. Patients with cold urticaria show several individual critical temperatures, ranging from <0°C to >27°C [36].

Potential triggers include low ambient temperature (cold season and air-conditioned rooms), cold water (swimming and showering), contact with cold surfaces, and consumption of cold foods (ice cream) or drinks.

Although cold urticaria symptoms are more severe in the cold season, the frequency of symptoms in 60% of patients, Siebenhaar et al. [37] revealed that it did not depend on the season.

Risk factors for the development of life-threatening reactions can be both the consumption of cold foods (and subsequent development of oropharynx edema) and extensive contact with cold (e.g., swimming in open water, infusion of cold solutions, prolonged surgical interventions; the risk is associated with low temperatures in the operating room, introduction of unheated infusion solutions, and contact with cold surfaces [35, 38–40].

In addition to taking a medical history, provocative testing is performed to diagnose cold urticaria. During the test, an ice cube is placed in a plastic bag and applied to the inner surface of the forearm. The exposure time is 5 min, and the reaction is evaluated within 10 min after the end of exposure. The reaction is positive if an urticarial rash occurs at the site of cold exposure (Fig. 4). The ice cube test only provides a qualitative assessment of the reaction (positive/negative) but does not imply an assessment of the temperature sensitivity threshold. In international practice, the TempTest device is used for the diagnosis of cold and heat types of physical urticaria. TempTest allows the evaluation of the critical time of exposure and critical temperature threshold that induce the occurrence of clinical manifestations in this group of patients; however, this device is not registered in the Russian Federation.

TempTest is a device with a U-shaped piezoelectric rash that generates a temperature range from 4°C to 44°C (Fig. 5). For testing, the patient places their forearm on the device so that the skin fits snugly against the piezoelectric rash. The exposure time is 5 min. The reaction is evaluated within 10 min, and the result is positive if an urticarial rash appears at the site of cold exposure. TempTest makes it possible to measure the critical temperature threshold with an accuracy of ±1°C [28] (Fig. 6).

All patients with cold urticaria should be warned about the avoidance of triggers and the risks of developing severe life-threatening reactions. Patients with a history of cold-induced anaphylaxis or life-threatening edema should be made aware of the action plan in the case of systemic reactions. The patients or their immediate relatives should be trained in the administration of emergency drugs (adrenaline and systemic corticosteroids).

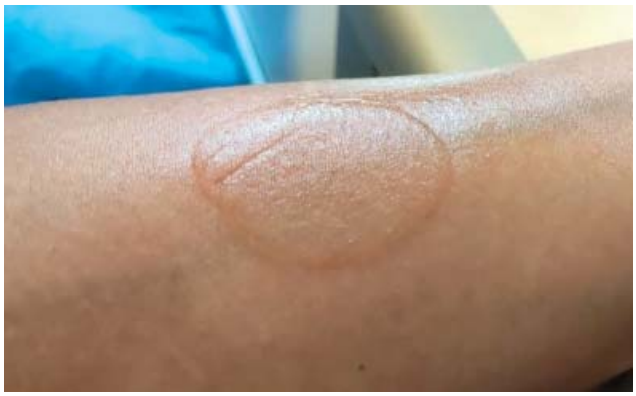

Fig. 4. Positive result of the ice cube testing. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

Fig. 5. TempTest. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

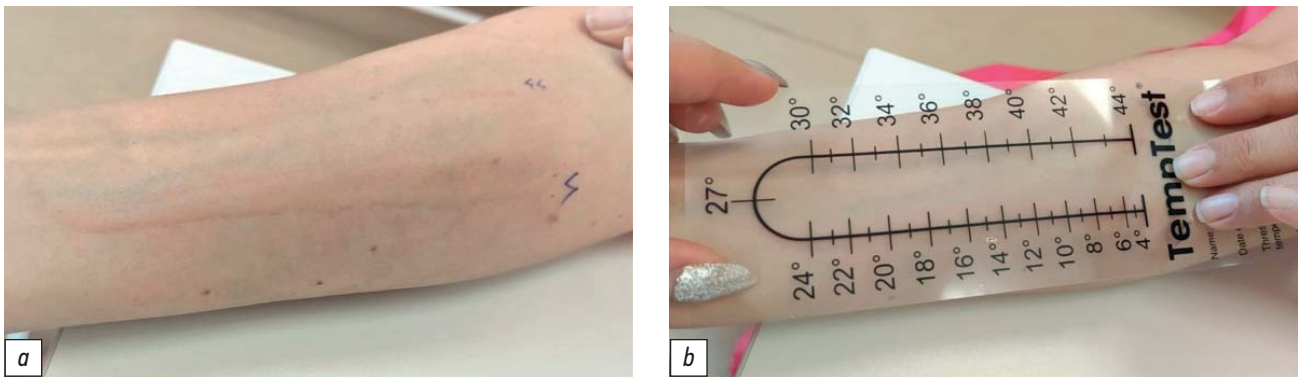

Fig. 6 (*a*, *b*). Positive result of provocation testing by TempTest device in patient with cold urticarial. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

The first and second lines of therapy for cold urticaria include nsH1-AHs in standard and escalated doses, respectively.

A meta-analysis of nine randomized clinical trials demonstrated the efficacy of AHs in patients with cold urticaria [41]. The dose escalation of AHs (bilastine, desloratadine, and rupatadine) demonstrated a significant reduction in the critical temperature threshold compared with the group of patients who received standard doses of AHs or placebo [42].

Given the individual response to therapy, nsH1-AHs choice, and daily dose can be personalized. It is also possible to switch from one AH to another until a satisfactory effect is obtained. Positive clinical experience with this approach has been described; however, no evidence-based scientific data are available [43]. Up to 30% of patients do not report cold urticaria symptoms upon treatment with high doses of nH1-AHs; however, such therapy does not reduce the risk of anaphylaxis with extensive cold exposure in these patients. Note that approximately 20% of patients did not show a decrease in the critical temperature threshold even upon treatment with high doses of AHs [41].

Currently, various hypotheses of the reasons for AH therapy failure are being considered. This phenomenon may be due to the polymorphism of histamine receptor genes or genes of histamine mobilizing enzymes and involvement of other groups of histamine receptors (H2 and H4 receptors) and other groups of receptors other than histamine in the pathogenesis of urticaria [44, 45].

In patients who are resistant to AH therapy, off-label treatment with omalizumab can be initiated, targeting free circulating immunoglobulin E (IgE) and affecting the functions of basophils/mast cells [24].

The efficacy of omalizumab in patients with cold urticaria was confirmed by Maurer et al. through a meta-analysis [20]. In a placebo-controlled, randomized clinical trial, the response to 150, and 300 mg of omalizumab was observed as early as 4 weeks after initiation. Dosages of omalizumab varied individually from 150 to 600 mg/month [46].

However, data on the efficacy of ciclosporin are limited [46]. Alternative treatment regimens also include tricyclic antidepressants (doxepin) and immunosuppressive drugs (azathioprine and mycophenolate mofetil) [24].

Cholinergic urticaria

Cholinergic urticaria is characterized by itching, redness, and urticarial rashes that occur after exercise and passive heating (hot bath, hot and spicy food, and stress reactions). Generally, rashes are short term (15–60 min), i.e., "flashing" in nature. Urticarial rashes are small and occur mainly on the skin of the trunk and extremities [2, 24, 47–52].

A pattern has been noted in the development of cholinergic urticaria in people predisposed to atopy [53], especially in patients with early disease onset [54].

Cholinergic urticaria is classified based on the proposed pathogenesis and clinical manifestations:

- Type 1: common cholinergic urticaria associated with sweating without angioedema.
- Type 2: cholinergic urticaria of the follicular type with a positive autologous serum test.
- Type 3: cholinergic urticaria with angioedema and reaction to sweating.
- Type 4: cholinergic urticaria with acquired anhidrosis/hypohidrosis [55–60].

In type 1 cholinergic urticaria, it is assumed that rashes occur due to blocked ducts of the sweat glands because of which sweat accumulates in the duct of the gland. Then, the sweat antigen penetrates the skin and binds to mast cell receptors, which subsequently leads to mast cell degranulation and emergence of clinical symptoms. In this type of cholinergic urticaria, the urticarial rash has no connection with the hair follicles. For type 1 cholinergic urticaria positive reaction to sweating, the administration of acetylcholine and a negative test with autologous serum are definitive [58, 61].

In the follicular type of cholinergic urticaria, mast cell degranulation leads to the appearance of rashes caused by an increase in body temperature and involvement of serum factors (autoantibodies). Urticarial lesions are localized around the hair follicles. For type 2cholinergic urticaria, a negative test with acetylcholine and a positive test with autologous serum are definitive [62, 63].

Type 3 cholinergic urticaria is characterized by angioedema associated with exercise, which occurs without the involvement of food triggers (omega-5 gliadin). Currently, the causative allergens, or clinical characteristics of this type have not been described. According to several reported clinical cases in this cohort of patients, two skin diseases are often combined, such as atopic dermatitis, and cholinergic urticaria. Autosensitization to sweat components was assumed to occur with increased sweating, subsequently causing both urticarial rashes and eczema-like rashes characteristic of atopic dermatitis. These patients respond poorly to AH therapy [64].

In type 4 cholinergic urticaria, rashes accompany acquired anhidrosis and/or hypohidrosis, an acquired disorder characterized by reduced amounts of sweat, which occurs without a specific cause and is not associated with any neurological disorders. This condition often develops in men and is characterized by pain and paresthesia of the skin in cases accompanied by sweating and rashes characteristic of cholinergic urticaria. Psychogenic sweating in anhidrosis/hypohidrosis is not affected [65].

Exercise-associated anaphylaxis is the most common type of severe life-threatening reactions in cholinergic urticaria. It usually debuts with skin symptoms on the distal parts of the body (palms, soles, and earlobes) with subsequent spread and fusion of erythematous and/or urticarial rashes, whereas classic cholinergic urticaria is characterized by the appearance of small urticarial rashes throughout the skin.

Passive heating (e.g., hot water) does not trigger the development of exercise-associated anaphylaxis, although it can also cause symptoms of cholinergic urticaria [1].

Provocative testing is recommended to confirm the diagnosis of cholinergic urticaria in these patients. Given the high risk of developing severe life-threatening reactions during provocative testing, the test should be conducted in a hospital setting with subsequent monitoring of the patient's condition during the day.

The first type of test was performed on a stationary simulator (bicycle ergometer or treadmill) (Fig. 7). The patient should perform physical exercises in such a way that during 30 min of the test, the initial heart rate increased by three beats every minute. Wearing warm clothes or high room temperature may make it easier to perform provocative tests. The test is considered positive if exercise causes a typical rash within 10 min (Fig. 8). The faster the positive response to exposure, the more active the course of cholinergic urticaria. If the test result is positive, the patient should be monitored for 24 h.

In addition, a passive heat test may be performed to differentiate between exercise-induced anaphylaxis and cholinergic urticaria. The test was carried out in a bath of water heated to 42°C. The patient continued to be in the bath until the body temperature rises by 1°C compared with the initial temperature measured before testing. The result is evaluated during the test and within 10 min after its completion [66].

In all patients with cholinergic urticaria, the importance of avoiding excessive exercise and passive overheating should be explained; however, preventive measures are not always possible.

Fig. 7. Tredmill using to providing provocation testing in patient with cholinergic urticarial. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

The main symptomatic drugs in these patients are nsH1-AHs in standard [67, 68] and escalated [69] doses; however, this treatment is effective only in some patients.

Currently, clinical experience with omalizumab therapy in patients with cholinergic urticaria is reported. Randomized placebo-controlled trials have not been conducted; however,

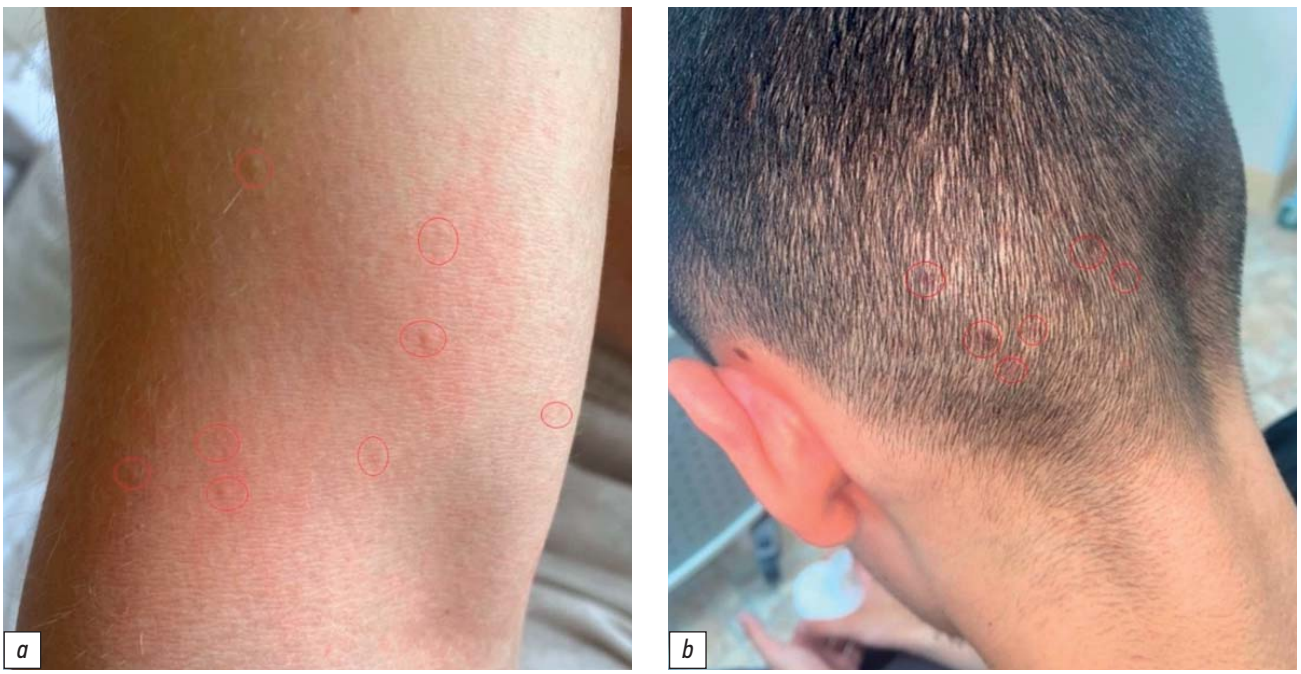

Fig. 8 (*a*, *b*). Positive result of provocation testing in patient with cholinergic urticarial. (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

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a series of clinical cases of omalizumab therapy in patients with cholinergic urticaria involving eight patients [20] and six isolated clinical cases [21, 70–74] have been described. In a retrospective analysis, Metz et al. [17] showed that 62% of patients achieved a complete response to treatment (no rash, negative results of the provocative test), 13% noted a significant improvement (>50% reduction in the provocation threshold/>50% reduction in symptoms), and 25% did not observe improvement during this treatment. Most patients received 150 mg of omalizumab, and one patient received 300 mg omalizumab.

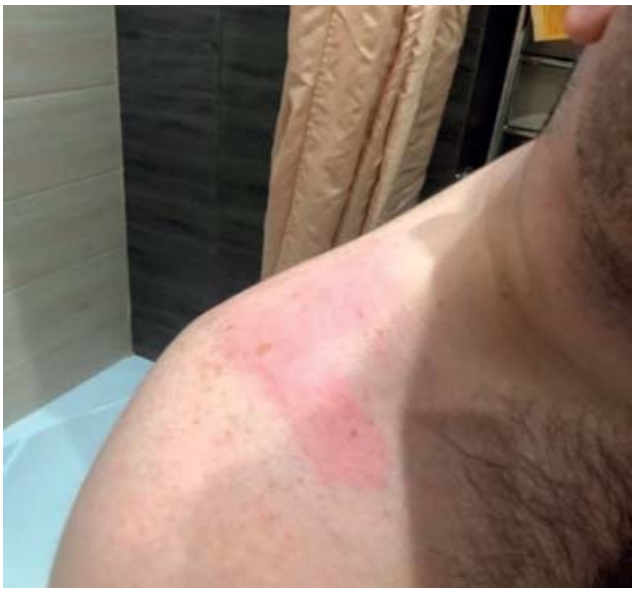

Fig. 9. Positive result of provocation testing in patient with delayed pressure urticaria (test with sand bag). (Photo from the archive of the Moscow City Reseach and Practical Center of Allergology and Immunology Ministry of Healthcare of Moscow).

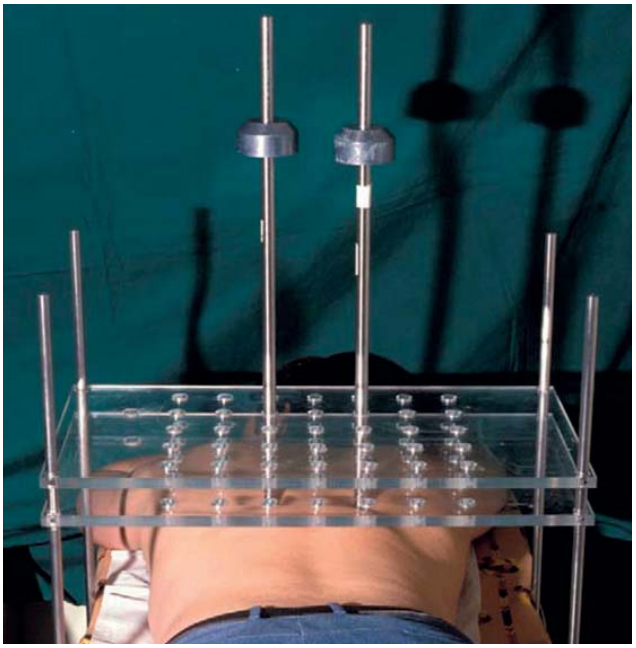

Fig. 10. Provocation testing in patient with delayed pressure urticaria by standartisated cillnders [82].

In cases not responding to AH therapy, cases of effective treatment with scopolamine butyl bromide [75], methanthelinium bromide [76], a combination of propranolol, AHs, and montelukast [77] and administration of botulinum toxin [78] have been described. The use of high doses of danazol (600 mg daily) has also been described with a positive effect; however, the low safety profile of the drug limits its use [79–81].

Delayed pressure urticaria

Delayed pressure urticaria is characterized by delayed erythema and subcutaneous edema that develop several hours after pressure exposure. When distal parts of the body (hands and feet) are affected, these clinical symptoms are indistinguishable from angioedema. Pressure-related symptoms occur on average 4–6 h after exposure; however, a manifestation period of 30 min to 48 h was also described [82].

Most frequently, symptoms occur after wearing heavy bags (wheals on the hands), cycling (wheals on the crotch and feet), and sites of clothing pressure (wheals in sites of contact with straps and elastic bands). Foot swelling can occur after long walks and jogging, wearing narrow shoes, and prolonged foot pressure on the pedal or shovel blade, upon using construction tools. The pathogenesis of delayed pressure urticaria is currently unclear. In the skin (both affected and unaffected) of patients, high levels of tumor necrosis factor alpha and interleukin-8 were registered [83].

The main method of provocative testing for delayed pressure urticaria is the sandbag test.

A bag (strap width 3 cm, weight 7 kg) or a weighted strap (strap width 1.5 cm, weight 2.5 kg or width 6, 5 cm, weight 5 kg) is hung at the test site (shoulder, upper back, thigh, or forearm) for 15 min. The result is evaluated within 24 h. A reaction is positive if wheals or edema occur at the site of pressure [84–86] (Fig. 9). Another diagnostic method involves the vertical placing of metal cylinders of various cross-sectional areas and weights on the skin of the back, thigh, or forearm (Fig. 10, [82]). The mass and cross-sectional area of the cylinders vary among researchers [82–86]. Currently, no tests are standardized for the diagnosis of delayed pressure urticaria, and this method is not registered in the Russian Federation.

Patients with delayed pressure urticaria are encouraged to avoid wearing tight shoes and clothing. Other potential factors that trigger the development of wheals must be avoided.

The first line of therapy for these patients is nsH1-AHs; however, as in other forms of CIndU, their effectiveness is limited. Dapsone, systemic corticosteroids, montelukast, sulfasalazine, escitalopram, oral coumarins, tranexamic acid, theophylline, intravenous immunoglobulins, and tumor necrosis factor inhibitors have been described as alternative therapies [87–100].

No randomized placebo-controlled studies of the efficacy of omalizumab in patients with delayed pressure urticaria

have been conducted; however, several clinical cases have been described (11 publications). The clinical case of a patient with a 10-year course of urticaria and a partial response to therapy (540 mg of fexofenadine and 100 mg of azathioprine per day) by Bindslev-Jensen and Skov [98] was the first to be published. The starting dose of omalizumab was 150 mg, and complete symptom alleviation was noted within 2 days from the start of treatment. After the publication of this article, a series of clinical cases with a complete response to omalizumab after the first injection was described [17, 101]. Metz et al. [21] reported that 7 of 8 (88%) patients achieved complete symptom control, whereas 1 (12%) showed significant improvement after the treatment. In another study, 3 (60%) of 4 patients showed a complete response to omalizumab [70]. Two cases with mixed results were also published: two patients with a combination of chronic spontaneous urticaria and delayed pressure urticaria who had complete remission after the first injection of omalizumab. In the third patient with severely delayed pressure urticaria and chronic spontaneous urticaria who had not responded to standard therapy for 20 years, omalizumab improved the symptoms of chronic spontaneous urticaria but aggravated the symptoms of delayed pressure urticaria; thus, the treatment was discontinued after the second injection [101].

Solar urticaria

Solar urticaria is characterized by recurrent episodes of urticaria on areas of the skin exposed to sunlight. Although life-threatening reactions in this type of CIndU are extremely rare, the disease can significantly limit the daily living activities of patients and reduce their quality of life [102–105].

The wavelength of light that induces the development of solar urticaria rashes ranges from class B UV radiation to visible light (wavelength from 300 to 500 nanometers) [106].

Solar urticaria is a rare type of urticaria. It accounts for <0.5% of all cases of chronic urticaria and 7% of all photodermatoses. The disease debuts at a young age (mean patient age of 35 years); however, cases that debut in newborns or older people have been reported. In population, women predominate with no ethnic differences. Concomitant atopic diseases occur in <30% of patients. The combination of solar urticaria with other types of urticaria is registered in 16% of cases [107, 108].

Itchy urticarial wheals occur within minutes of sunlight exposure, predominantly on the exposed skin and under thin and white clothing that allows sunlight to pass through. Wheals are often accompanied by angioedema of the periobitual area and/or mucous membranes. Some patients have red dermographism on the sun-exposed skin. Systemic symptoms such as nausea, shortness of breath, or loss of consciousness may develop, which most often occur when large skin areas are isolated for a long time. Life-threatening reactions such as anaphylaxis are extremely rare. Typically, wheals disappear spontaneously, without a trace, within 24 h.

Currently, no devices are standardized for testing patients with solar urticaria in the Russian Federation. The diagnosis of solar urticaria is made based on the patient's complaints, history of present disease, and objective examination. Provocative testing can be also performed using UV lamps with rays of the A and B spectrum (UVA, UVB) and visible light sources. Phototesting intends to determine the spectrum of action (to determine the wavelength) and minimum urticarogenic dose (minimal dose that induces the emergence of wheals). Light sources are placed at a distance of 10–15 cm from the patient's back, and then irradiation of various durations and intensities is carried out. The clinical response is assessed every 10 min for 1 h. Erythema and wheals appear immediately after phototesting and disappear within a few minutes after irradiation (Fig. 11) [109]. However, in many cases, phototesting with artificial light sources does not provide positive results because artificial lights cannot simulate the radiation characteristic of natural light. In some patients with solar urticaria, a positive phototest result is noted only after a second attempt at testing.

Currently, no recommendations are generally accepted for treating solar urticaria. Patients are advised to avoid sun

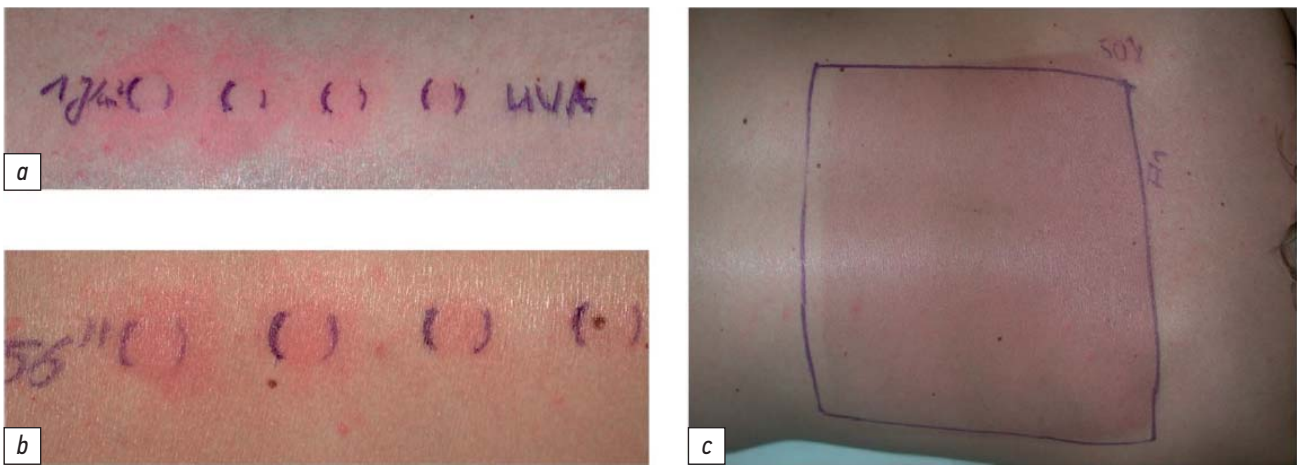

Fig. 11. Positive results of provocation testing in patient in patient with solar urticaria: *a* — positive results of tesring with UVA specter; *b* — positive results of tesring with UVB specter; *c* — appearing of immediate erythema after UVA specter impact [109].

exposure, use a broad-spectrum sunscreen, and wear dark clothing [110, 111].

The first line of therapy is nsH1-AHs in standard doses (loratadine, cetirizine, and fexofenadine). As with other forms of chronic urticaria, dose escalation is often required.

Phototherapy (UVA, UVB, and visible light) and photochemotherapy (PUVA) can be used to induce sunlight tolerance. Phototherapy protocols should be based on the spectrum of the causative radiation and minimum urticarogenic dose.

Cases of successful use of immunoglobulins, plasmapheresis, cyclosporine, and afamelanotide in patients tolerant to AH therapy were described [112].

The efficacy of omalizumab in patients with solar urticaria has been described in many clinical cases. Successful omalizumab therapy in patients with solar urticaria up to complete disease remission has been described [17, 20, 21, 113–119]. Starting doses of omalizumab in these patients ranged from 150 mg to 375 mg; however, dose escalation to 450 mg was needed to control the symptoms in some patients [116, 120]. Symptoms often recurred 2–8 weeks after treatment discontinuation but disappear again after injections of omalizumab [21]. A case of omalizumab therapy (after approval by the ethical committee) in a 6-year-old child was also reported. The initial dose of 75 mg did not provide visible improvements and was gradually increased to 300 mg two times per month, followed by remission of urticaria. No side effects were registered [121].

A case of omalizumab therapy failure was also described. In a patient who received four doses of omalizumab 150 mg once per 4 weeks, neither clinical improvement, nor a decrease in the photosensitivity threshold was noted. A study reported worsening of symptoms after three doses of 150 mg omalizumab, indicating the need for individualized therapy [122].

Heat urticaria

Heat urticaria is a rare type of physical urticaria that is characterized by pruritic urticarial rashes appearing within minutes of exposure to a heat trigger [123, 124]. Most often, this type of urticaria affects women aged 20–45 years. Typically, heat urticaria is a long-term disease. At the time of diagnosis, the average disease duration is approximately 2 years [124].

The pathogenesis of heat urticaria may be based on both the release of histamine from mast cells (immediate-type reactions) and some genetic aspects (delayed type reactions). Clinically, heat urticaria can manifest in both localized and generalized forms, depending on whether the skin area is directly heated (localized forms) or whether the conduction of heat from distant skin areas upon their heating (generalized forms) occurred.

Systemic symptoms such as weakness, bronchospasm, headache, nausea, vomiting, and tachycardia can develop. The diagnosis of heat urticaria is made based on the patient's complaints, disease history, and provocative test results.

A hot tube test (50–55°C) is performed. The tube is applied to the inner surface of the forearm. The exposure time is 5 min, and the reaction is evaluated within 10 min after the exposure. A reaction is positive if an urticarial rash appears at the exposure site. The average threshold temperature is approximately 44°C [124]. The hot tube test gives only a qualitative assessment of the reaction (positive/negative) but does not allow an assessment of the temperature sensitivity threshold. In international practice, the TempTest device (not registered in the Russian Federation) is used to evaluate the critical time of exposure and critical temperature threshold, inducing clinical symptoms in these patients.

Treatment options for heat urticaria are limited as for other types of induced urticaria. Owing to the frequent ineffectiveness of nsH1-AHs, various drug combinations (e.g., combinations of H1 and H2 AHs) can be used [5, 15, 22, 125].

Data on omalizumab therapy in patients with heat urticaria are limited at the moment, and four publications involving five patients are available. Two patients had severe heat urticaria, in which standard methods failed to control the symptoms. After the first dose of 300 mg omalizumab, a significant improvement was registered [126–128]. One case recorded a negative provocative test result. In another observation, nearly complete control over the symptoms was obtained after administration of four doses of omalizumab (alternating 300 mg and 450 mg) one dose per 2 weeks. The effect of the 450 mg-dose therapy was better than of the 300 mg-dose therapy: the patient received 38 doses of omalizumab with no side effects and no need for additional therapy [126].

Two other reports described either no effect [20] or minor improvement in patients with heat urticaria resistant to standard therapy (AHs and montelukast) [17].

Vibratory urticaria

Vibratory urticaria (or vibratory angioedema) occurs after the exposure of the skin to a vibration trigger [118, 128]. Clinically, this type of urticaria manifests by local itching and edema without urticarial wheals and occurs after the interaction of the skin with the vibrating object (most frequently affecting the hands).

Triggers can include working with a lawnmower or other similar machinery, riding a bicycle, horse, or motorcycle. High-risk professions are drivers, impact hammer operators, carpenters, and machine operators.

Currently, there is a hypothesis on the pathogenetic significance of ADGRE2 receptor mutation in familial cases of vibratory urticaria. This mutation leads to the destabilization of mast cell autoinhibitors, which leads to mast cell IgE-independent degranulation upon exposure to vibration [127].

Symptoms of vibratory urticaria reach their peak 4–6 h after exposure to the trigger and typically resolve within 24 h. Symptom severity and duration most often correlate with the area of exposure [128].

The diagnosis is made based on complaints, present disease history, and provocative testing. Provocative testing can be performed using a vortex vibrator. The testing time is 10 min. The reaction is positive if the skin in the affected area becomes hyperemic, and swelling, and itching occur.

The importance of avoiding specific vibrational stimuli should be explained to the patients. Cases of successful nsH1-AH therapy in both standard and escalated doses were described as treatment options. No clinical cases of successful omalizumab therapy in patients with vibratory urticaria have been described [5, 15, 22].

Contact urticaria

Contact urticaria is characterized by the appearance of wheals at the site of the contact of skin and/or mucosa with an exogenous agent[1] [129]. Symptom severity can vary from localized to generalized wheals and anaphylactic reactions.

Contact urticaria has two types: nonimmunologic contact urticaria and immunologic contact urticaria [130]. Nonimmunologic contact urticaria is the most common type of urticaria. The response to a trigger develops as an immediate hypersensitivity reaction. Symptoms usually occur only in the contact area. The most common causes are cosmetics, drugs, balsam of Peru, benzoic acid, cinnamic alcohol or aldehyde, and sorbic acid (a preservative used in many foods — raw meat, fish, and vegetables). Immunologic contact urticaria is an IgE-mediated hypersensitivity reaction that occurs in patients previously sensitized to a particular trigger.

Prior sensitization to the trigger may have occurred through contact with the skin and mucous membranes (respiratory or gastrointestinal). The reaction occurs from a few minutes to a few hours after contact with the causative allergen. Most frequently, these reactions occur after exposure to rubber, latex, antibiotics, metals, parabens, benzoic, and salicylic acids, polyethylene glycol, short-chain alcohols, raw meat, fish, and vegetables [129, 131].

Typically, with contact urticaria, the skin of the face or upper limbs is affected. High-risk professions include medical workers, food workers, hairdressers, bakers, dental technicians, agricultural workers, electricians, veterinarians, and others.

The diagnosis of contact urticaria is established based on the clinical findings. In the history of the present disease, the timing of disease onset, time, and duration of clinical symptoms, affected skin region, family history, recent use of drugs, anaphylactic reactions in the past, systemic reactions to insect stings, and stress reactions must be indicated [129, 130, 132].

Laboratory diagnostics include allergy testing (scratch tests or prick tests, specific IgE levels) and provocative testing. Patch tests are not registered in the Russian Federation.

The first line of therapy is H1-AHs and their combination with H2-AHs [5, 15, 22]. The topical administration of local agents (topical corticosteroids) on the affected area is also an option.

Aquagenic urticaria

Aquagenic urticaria is an extremely rare disease where wheals develop after direct skin contact with water [133–136]. Some patients may experience reactions with tap water or seawater only. Women suffer from aquagenic urticaria more often than men, and the peak incidence of the disease occurs during puberty. Familial cases have been reported.

Several theories of the pathogenesis of aquagenic urticaria have been proposed. First, water promotes the penetration of the antigen through the skin and activates dermis mast cells; second, the interaction of water with skin lipids forms a substance that directly promotes mast cell degranulation. Mast cells can be activated via the cholinergic pathway [137].

Rashes in aquagenic urticaria are typically similar to those in cholinergic urticaria (small urticarial rashes up to 1–3 mm in diameter). Clinical manifestations are observed within 20–30 min with symptom alleviation within 30–60 min. Systemic symptoms are rare; however, such cases have been reported. They are often associated with a combination of other forms of CIndU (dermographic, cold, or cholinergic urticaria) [138].

Aquagenic urticaria is diagnosed based on provocative testing: a wet compress (35°C) is applied to the upper body. The exposure time is 30 min, and ambient water temperature must be used for the differential diagnosis of cold and heat urticaria.

The first line of therapy is nsH1-AHs, as well as their combination with UV light therapy [6, 15, 23, 129, 134].

Only one case of omalizumab therapy for aquagenic urticaria was described [139]. In a patient with a 2-year history of aquagenic urticaria resistant to AHs, montelukast, ranitidine, diphenhydramine, and hydroxyzine, symptoms were relieved after two doses of omalizumab 300 mg.

CONCLUSION

No domestic epidemiological data reflect the prevalence of CIndU and its trends over time. Currently, no medical devices are registered for determining some forms of physical urticaria (cold urticaria and heat urticaria) in the Russian Federation, which makes it difficult to establish a diagnosis with an indication of the trigger threshold dose and continue case follow-up in these patients.

The introduction of innovative examination methods will help in managing patients with physical forms of urticaria, optimizing the medical care delivery to these patients,

[1] UpToDate [web]. Dice JP, Gonzalez-Reyes E. Physical (inducible) forms of urticaria. Available www.uptodate.com/contents/physical-inducibleforms-of-urticaria.

reducing the risk of life-threatening conditions, and preventing the development of severe forms of urticaria. The methods under development can be used in the examination of certain population groups: citizens working in conditions of exposure to high and low temperatures and vibration (employees from the northern regions, transport services workers, builders, etc.), athletes, and career guidance of citizens planning to work in these conditions.

The problem of CIndU treatment remains unresolved. In many cases, treatment with standard, and escalated doses of nsH1-AHs does not fully control disease symptoms. In addition, no randomized controlled trials have focused on the efficacy and safety of escalated doses of H1-AHs in most types of CIndU.

Placebo-controlled studies have demonstrated the effectiveness of omalizumab for treating dermographic and cold urticaria. Several clinical case reports have described positive experiences with omalizumab therapy in other types of CIndU. However, it is not currently registered for the treatment of induced urticaria and can only be prescribed as an off-label therapy. The experience of omalizumab therapy described in the literature is heterogeneous (different dosing regimens, duration of use, and different definitions of therapy efficacy), which greatly complicates the standardization of results and their objective analysis.

Further clinical studies are needed for a more detailed study of the current forms of therapy and development of new treatments. Moreover, further studies of the etiopathogenesis of induced urticaria forms are needed to identify possible prognostic criteria for the severity of CIndU, its expected response to therapy, and development of new treatment methods.

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