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# Dermatological manifestations induced by ADHD pharmacotherapy

## *Manifestacje dermatologiczne wywołane farmakoterapią ADHD*

ABSTRACT

Epidemiological data indicate a growing recognition of attention deficit hyperactivity disorder (ADHD) in adults. Pharmacotherapy used in its treatment, in addition to its effects on the central nervous system, also affects the structure and physiological condition of the skin.

This study aimed to analyze the cutaneous adverse reactions associated with pharmacological treatment of ADHD and to explain the mechanisms of their development in the context of clinical and cosmetic practice.

A literature review was conducted. The best-documented dermatological manifestation is peripheral vasculopathy, including Raynaud's phenomenon, associated with increased noradrenergic activity and vasoconstriction. Disturbances in sleep, thermoregulation, sweating, hydration, and nutritional status are also potentially significant. Dermatillomania may result from both ADHD characteristics and individual response to treatment.

The analysis conducted indicates that ADHD pharmacotherapy should be included in the dermatological and cosmetic interview, especially in patients with vasomotor symptoms, healing disorders or mechanical skin damage.

**Keywords:** ADHD, pharmacotherapy, dermatoses, Raynaud's phenomenon, xeroderma, epidermal barrier disorders, dermatillomania.

STRESZCZENIE

Dane epidemiologiczne wskazują na rosnącą rozpoznawalność zespołu nadpobudliwości psychoruchowej z deficytem uwagi (ADHD) u osób dorosłych. Stosowana w jego leczeniu farmakoterapia, poza działaniem na ośrodkowy układ nerwowy, wpływa również na strukturę oraz kondycję fizjologiczną skóry.

Celem pracy była analiza skórnych objawów niepożądanych związanych z leczeniem farmakologicznym ADHD oraz wyjaśnienie mechanizmów ich powstawania w kontekście praktyki klinicznej i kosmetologicznej.

Przeprowadzono przegląd piśmiennictwa. Najlepiej udokumentowaną manifestacją dermatologiczną jest obwodowa waskulopatia, w tym fenomen Raynauda, związana z nasileniem aktywności noradrenergicznej i wazokonstrykcją. Potencjalne znaczenie mają także zaburzenia snu, termoregulacji, potliwości, nawodnienia i stanu odżywienia. Dermatillomania może wynikać zarówno z cech ADHD, jak i indywidualnej odpowiedzi na leczenie.

Przeprowadzona analiza wskazuje, że farmakoterapia ADHD powinna być uwzględniana w wywiadzie dermatologicznym i kosmetologicznym, szczególnie u pacjentów z objawami naczynioruchowymi, zaburzeniami gojenia lub mechanicznym uszkodzeniem skóry.

**Słowa kluczowe:** ADHD, farmakoterapia, dermatozy, fenomen Raynauda, kserodermia, zaburzenia bariery naskórkowej, dermatillomania.

## INTRODUCTION

The estimated prevalence of attention deficit/hyperactivity disorder (ADHD) in adults depends on the diagnostic criteria and research methodology used; however, it most often ranges from approximately 2.5% to 3.5% globally [1]. The intensification of diagnostic processes, based on the American Psychiatric Association's diagnostic criteria for mental disorders (DSM-5-TR, *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision*), results in a growing number of people undergoing long-term pharmacotherapy, primarily involving psychostimulants such as methylphenidate and amphetamine derivatives, as well as non-stimulant medications, including atomoxetine and alpha-2-adrenergic receptor agonists [2].

From a clinical dermatology perspective, this population constitutes a group requiring special attention with regard to the medication history. Medications used to treat ADHD, particularly psychostimulants, can affect sympathetic nervous system activity, peripheral vascular tone, thermoregulation, sweating, appetite, sleep quality, and compulsive behaviors. All of these factors may directly or indirectly modify skin homeostasis [3]. Failure to account for the pharmacological component in the clinical history may lead to an incomplete interpretation of skin changes, which in some patients may be iatrogenic in nature or exacerbated by the administered therapy.

## PATHOMECHANISM OF PHARMACOTHERAPY-INDUCED VASCULAR DISORDERS

One of the best-documented dermatological consequences of ADHD pharmacotherapy is the effect of psychostimulant medications on the vascular system. By blocking reuptake or enhancing the release of neurotransmitters, primarily dopamine and norepinephrine, these compounds lead to an increase in their availability in the synaptic cleft [4].

The mechanism of vasoconstriction may play an important role in the vascular consequences of dermatological pharmacotherapy for ADHD. Psychostimulant medications, by enhancing catecholaminergic transmission, may increase the activity of the sympathetic noradrenergic system. In the cutaneous circulation, norepinephrine interacts with postsynaptic α-adrenergic receptors, including $\alpha_1$ and $\alpha_2$ receptors, located in vascular smooth muscle. Their activation increases intracellular calcium ion concentrations and leads to muscle contraction, narrowing of the vascular lumen, and reduced blood flow in the skin. In susceptible patients, this may clinically manifest as Raynaud's phenomenon, acrocyanosis, or *livedo reticularis* [5, 6].

From a clinical perspective, this condition may be associated with symptoms such as a sensation of cold extremities, episodic pallor or cyanosis of the fingers, hypersensitivity to cold, and symptoms consistent with Raynaud's phenomenon. In the differential diagnosis, other manifestations of microcirculatory disorders may also be considered, such as marbled skin discoloration or reticular cyanosis; however, the best-documented drug-induced manifestation remains vasculopathy, including Raynaud's syndrome [5].

Chronic or recurrent restriction of blood flow may potentially reduce the supply of oxygen and nutrients to the dermis. In clinical practice, this does not automatically indicate vascular insufficiency of the skin, but it should prompt more careful patient selection for invasive or thermal procedures. In individuals reporting vasoconstrictive symptoms, the possibility of prolonged healing, increased skin reactivity, post-procedural hypersensitivity, and potentially poorer tolerance of procedures that disrupt the integrity of the epidermis should be considered [5, 7].

## EPIDERMAL BARRIER DYSFUNCTION AND SECONDARY XERODERMA

The effect of ADHD pharmacotherapy on homeostasis of the epidermis is indirect and results from activation of the sympathetic nervous system, sleep disturbances, reduced appetite, changes in the body's water balance, and disturbances in neuroendocrine regulation. In this context, some individuals may exhibit increased skin reactivity, reduced tolerance to external stimuli, and symptoms of dryness or skin discomfort. It should be emphasized, however, that a direct link between medications used to treat ADHD and permanent damage to the skin barrier requires further clinical research [7].

Psychostimulant medications may affect the autonomic regulation of skin function, including sweating and thermoregulation. Some patients experience increased sweating, dry mouth, decreased thirst, or dehydration that is not due to limited fluid intake. These disturbances may indirectly affect the functioning of the skin's hydrolipid barrier, whose physiological pH typically ranges from 4.5 to 5.5. A shift in pH toward a more alkaline state may disrupt the activity of enzymes responsible for exfoliation and barrier regeneration, making the epidermis more susceptible to irritation and colonisation by microorganisms [8].

A key component of the integrity of the epidermal barrier is the proper composition of the intercellular cement, particularly ceramides, cholesterol, and free fatty acids. Chronic stress, sleep disturbances, and activation of the neuroendocrine axis may adversely affect keratinocyte function and barrier repair processes. In the case of patients treated for ADHD, indirect factors such as reduced appetite and deficiencies in protein, zinc, B vitamins, or fatty acids, which are essential for proper epidermal regeneration [9].

As a result of the destabilisation of the skin barrier, transepidermal water loss (TEWL) may increase. Clinically, this manifests as a subjective sensation of tightness, dryness,

burning, discomfort, and a reduced tolerance threshold to external stimuli. The skin may become more reactive, prone to microcracks, and less resistant to irritants [9].

From a clinical practice perspective, skin exhibiting signs of dehydration and a compromised barrier requires a more cautious approach to treatment. This applies in particular to exfoliating acids, retinoids, microneedling, laser therapy, and other techniques that may temporarily exacerbate inflammation. In such patients, it is advisable to prepare the skin in advance by restoring the hydrolipid barrier, reducing irritating ingredients in skincare products, and optimizing hydration and nutrition [9].

## IMMUNOMODULATORY EFFECTS OF PSYCHOSTIMULANTS ON SKIN TISSUE

The skin is an active immune organ in which immune cells interact closely with peripheral nerve endings and local neuroendocrine mediators. The nervous, endocrine, and immune systems form an integrated regulatory network, and prolonged activation of stress mechanisms, including the sympathetic nervous system, can modulate the inflammatory response and the skin's immune microenvironment [10, 11].

Immune system cells present in the epidermis and dermis, including Langerhans cells and T lymphocytes, possess receptors for neurotransmitters and stress mediators. Pharmacologically increasing catecholaminergic activity may therefore modify the immune response, although the clinical significance of this phenomenon in patients treated with standard doses of medications used for ADHD has not yet been clearly established [12].

The potential effects of this phenomenon may include changes in the balance of the inflammatory response, modulation of lymphocyte activity, and an impact on the course of immunologically based diseases. Population-based studies have demonstrated a correlation between ADHD and an increased prevalence of selected autoimmune diseases. However, this association is a characteristic of the disorder itself, rather than a direct result of the pharmacological treatment used [13]. Currently, there is insufficient evidence to consider standard ADHD treatment as a factor leading to skin immunosuppression.

Pharmacotherapy for ADHD may also affect nervous system tension and exacerbate sensations of itching, burning, or skin discomfort in predisposed individuals.

Under conditions of chronic neurogenic stress, there may be an increased release of neuropeptides, such as substance P, from peripheral sensory fibers. This phenomenon may promote mast cell activation and the release of inflammatory mediators, which could potentially exacerbate the course of inflammatory skin conditions, such as atopic dermatitis or psoriasis [11, 14]. However, this association should be interpreted with caution, as a possible mechanism exacerbating symptoms rather than an independent cause of the disease.

## RAYNAUD'S SYNDROME AS AN IATROGENIC EFFECT OF PHARMACOTHERAPY

In the population of patients undergoing long-term pharmacotherapy for ADHD, there is an increased risk of symptoms of peripheral vasculopathy, including secondary Raynaud's syndrome. This condition, resulting from vasomotor hyperreactivity, is a significant consideration in the differential diagnosis of skin lesions in the distal parts of the body [5, 15-17].

The iatrogenic mechanism results primarily from increased noradrenergic activity and secondary stimulation of adrenergic receptors in vascular smooth muscle. A pharmacologically induced increase in catecholamine availability may lower the reactivity threshold of peripheral vessels, whereby stimuli such as exposure to cold, emotional stress, or a sudden temperature change can trigger excessive constriction of the afferent arterioles [5, 6, 17].

The clinical manifestation of Raynaud's phenomenon may include paroxysmal perfusion disturbances with a characteristic - though not always complete - sequence of skin color changes. The ischemic phase is characterized by pallor, which may be followed by a cyanotic phase associated with slowed blood flow and an increase in the proportion of deoxygenated blood. During reperfusion, reactive hyperemia occurs, which may be accompanied by a burning sensation, pain, numbness, or paresthesia. Symptoms most commonly affect the fingers and toes, and patients may also report increased sensitivity to cold [17].

From the perspective of eligibility for cosmetic procedures, particularly those that disrupt skin integrity, it is essential to consider the severity of vasospastic symptoms. Most cases of Raynaud's phenomenon described during ADHD pharmacotherapy were mild and reversible. However, the presence of ulcers, persistent sensory disturbances, severe pain, prolonged cyanosis, or other signs of ischemia should prompt postponement of the planned procedure and medical consultation [5, 17].

In individuals with diagnosed or suspected drug-induced Raynaud's phenomenon, procedures that disrupt the integrity of the epidermis, such as needle mesotherapy, thread lift implantation, deep chemical peels, or intensive laser therapy, should be approached with greater caution. Impaired vascular perfusion may limit proper transport of oxygen, nutrients, and mediators needed for tissue regeneration, which may increase the risk of prolonged healing, increased skin reactivity, or an abnormal post-treatment response. The risk of necrosis should be considered as potential, but rare and primarily associated with severe microcirculatory disorders or concomitant vascular diseases [5, 17].

Thus, a thorough assessment of the patient's medical history regarding episodes of cyanosis of the fingers and toes, cold sensitivity, pain in the extremities, and skin atrophy becomes an essential part of the patient's evaluation for invasive procedures. This allows for a safer differentiation between primary, autoimmune, and iatrogenic causes of vascular disorders [17].

## THE NEUROBIOLOGICAL BASIS OF DERMATILLOMANIA

In the neuropsychiatric context, a significant issue is the disruption of skin integrity resulting from compulsive or impulsive skin picking, referred to as dermatillomania or skin-picking disorder. In patients with ADHD, this phenomenon may stem from the clinical presentation of the disorder itself, including impulsivity, reduced inhibitory control, psychomotor agitation, or the search for sensory stimulation, as well as from the effects of the prescribed pharmacotherapy [18-20]. The effects of psychostimulant medications, which lead to increased synaptic availability of dopamine and norepinephrine, may influence psychomotor activity and internal tension in predisposed patients and motor stereotypes. The data regarding the effect of psychostimulants on dermatillomania are inconclusive. Bernardes et al. described a female patient with ADHD and comorbid skin-picking disorder in whom the use of methylphenidate was associated with an improvement in skin-damaging behaviours [19]. Kara and Akaltun presented a different observation, describing the onset of this disorder after starting methylphenidate therapy [20]. The varying nature of the observed changes suggests that the effect of treatment may depend on the patient's individual response, and the available data. Based primarily on case reports, do not allow for the determination of either the frequency of this phenomenon or a clear causal relationship. At the same time, it should be emphasized that in other patients, effective treatment of ADHD may improve impulse control and indirectly reduce self-destructive behaviours. For this reason, dermatillomania should not be treated exclusively as a iatrogenic manifestation, but rather as a phenomenon requiring individual clinical assessment [19, 20].

Clinically, skin picking manifests as habitual, often partially unconscious mechanical trauma directed at minor imperfections of the epidermis, such as bumps, blackheads, scabs, or skin irregularities. This leads to the formation of abrasions, erosions, minor wounds, and scabs, as well as inflammatory changes secondary to repeated trauma to the skin [18, 21].

Prolonged inflammation at the site of injury may contribute to the persistence of tissue defects in the form of atrophic or hypertrophic scars, post-inflammatory hyperpigmentation, and dystrophic changes. In cases of concomitant skin dryness, a compromised epidermal barrier, or a tendency toward secondary infections, the healing process may be further prolonged [18, 21].

A key element of the differential diagnosis is the topographic analysis of the lesions. Lesions associated with skin picking are often located in areas easily accessible to the patient's hands, such as the face, arms, chest, thighs, or scalp. They may tend to be symmetrical or align with the patient's dominant hand [21].

The psychological and behavioural components must also be taken into account. Standard dermatological interventions may be ineffective if the mechanism of recurrent skin damage is not identified. In such cases, effective management requires a combination of barrier care, treatment of inflammatory lesions, patient education, and, if necessary, collaboration with a psychiatrist or psychotherapist [18, 19].

## OXIDATIVE STRESS AND CELLULAR AGING PROCESSES

In the context of aesthetic medicine and anti-aging, it is important to ask whether long-term pharmacotherapy of ADHD can affect the skin's regenerative processes and the rate of skin aging. Currently available data do not allow for a definitive conclusion that standard psychostimulant treatment directly accelerates skin aging. However, several indirect mechanisms can be identified that could potentially worsen skin quality in some patients [22, 23].

One of these is chronic activation of the sympathetic nervous system and the adverse effects of oxidative stress. Prolonged neuroendocrine stress, sleep disturbances, and increased stress reactivity can affect cortisol metabolism. Cortisol, as one of the main mediators in the stress response, can influence the metabolism of the skin's extracellular matrix. Detailed data are provided by a study by Chae et al. conducted on human fibroblasts. The cells were exposed for 24 hours to cortisol at concentrations of 0.1, 10, and 100 nM. The expression of the COL1A1 gene, responsible for the synthesis of type I collagen, was then assessed, and the amount of type I procollagen. The following methods were used for the analysis: quantitative real-time polymerase chain reaction (qRT-PCR) and the enzyme-linked immunosorbent assay (ELISA). Cortisol decreased the expression of type I collagen, and the observed effect was associated with the activation of the glucocorticoid receptor and the inhibition of transforming growth factor beta (TGF-β) signalling [24]. This result points to a biologically plausible mechanism by which chronic hormonal stress affects the skin's extracellular matrix; however, the study was experimental in nature and did not involve patients being treated for ADHD. It should be noted, however, that the effect of medications used for ADHD on skin collagen degradation has not been unequivocally confirmed in clinical trials. Another mechanism considered in the context

of skin aging is oxidative stress. Psychostimulant medications, by affecting catecholamine metabolism and neuronal activity, may theoretically promote increased production of reactive oxygen species (ROS). Excessive accumulation of ROS in skin cells, particularly in fibroblasts, may disrupt mitochondrial function, limit the proliferative potential of cells and promote senescence processes [22, 25]. However, with regard to the skin of patients treated for ADHD, this mechanism should be considered a biologically plausible hypothesis rather than a definitively proven clinical effect.

In a study by Pujos et al., which combined clinical assessment with cellular studies, individuals with moderate chronic psychological stress were found to have reduced antioxidant potential and impaired skin barrier parameters. The severity of changes in skin structure, including texture and fine lines, was approximately 32.9% greater compared to the group with lower stress levels. In the experimental part, elevated concentrations of stress hormones also affected the integrity of deoxyribonucleic acid (DNA), extracellular matrix synthesis, healing processes, and barrier parameters [26]. However, the study did not address pharmacotherapy of ADHD; therefore, its results can only provide a mechanistic rationale for the potential role of chronic stress activation, and do not prove that psychostimulants accelerate skin aging. There is currently insufficient evidence to conclude that patients undergoing long-term pharmacological treatment for ADHD exhibit a statistically significant higher rate of collagen degradation compared to a control population.Such a conclusion requires well-designed clinical trials evaluating skin parameters such as collagen density, elasticity, TEWL, ultrasound imaging of the skin, or markers of extracellular matrix degradation [26].

However, indirect factors, often observed in patients taking psychostimulants, may have practical significance: suppressed appetite, irregular meals, reduced protein intake, and deficiencies in zinc, iron, magnesium,B vitamins, and sleep disturbances. All of these factors can affect the skin's repair processes, the synthesis of structural proteins, normal keratinization, and the inflammatory response [27].

At the same time, medication-induced difficulty falling asleep or changes in sleep quality may limit nighttime tissue regeneration. Deep sleep plays a crucial role in repair processes, hormonal regulation, and cellular regeneration. Therefore, in patients reporting a deterioration in skin quality during ADHD treatment, it is advisable to evaluate not only the medication itself but also sleep, diet, hydration, stress levels, skincare, and the presence of compulsive behaviors related to the skin [27].

From the perspective of evidence-based medicine (EBM), it is therefore the safest to view the potential impact of ADHD pharmacotherapy on skin aging as an indirect and multifactorial phenomenon. It does not result solely from the drug's action, but from the combined effects of metabolic, neuroendocrine, nutritional, behavioral, and environmental factors [22, 23].

## SUMMARY

The skin, as an organ highly sensitive to neuroendocrine modulation, may exhibit increased reactivity, vasomotor disturbances, a tendency toward dryness, hypersensitivity, scratching, and prolonged healing in some patients treated for ADHD. The best-documented dermatological aspect of psychostimulant pharmacotherapy remains its effect on peripheral blood vessels, including the potential for symptoms consistent with Raynaud's phenomenon. Other mechanisms, including epidermal barrier dysfunction, immunomodulation, oxidative stress, and accelerated skin aging, should be interpreted with caution, as potential and multifactorial, requiring further clinical studies.

The identified disorders, including systemic dysfunctions, possible impairment of the skin barrier, a neuroinflammatory component, behavioral skin damage, and factors indirectly affecting regeneration, justify the need for a more thorough pharmacological assessment in clinical dermatology and aesthetic cosmetology.

A key element of safe practice is the assessment of medications used, the presence of vasoconstrictive symptoms, blood pressure, diet, hydration, stress levels, and any potential compulsive behaviors toward the skin. When planning invasive procedures, it is advisable to focus on protective strategies aimed at stabilizing the hydrolipid barrier, reducing inflammation, optimizing tissue perfusion, and tailoring the intensity of treatments to the individual.

An interdisciplinary approach, combining knowledge from the fields of clinical dermatology, pharmacology, psychiatry, and aesthetic cosmetology, forms the foundation for the safe care of patients whose biological and behavioral profiles may be modified by the pharmacotherapy they receive.

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