Review article / Artykuł przeglądowy

Agnieszka Surgiel-Gemza 0009-0005-8573-6985
Julita Zdrada-Nowak 0000-0003-3255-457X

Department of Cosmetology and Medical Biology, Dr. Władysław Biegański Collegium Medicum, Jan Długosz University in Częstochowa
ul. Armii Krajowej 13/15, 42-200 Częstochowa
+48 694 777 407 *agnieszka.surgiel.gemza.asg@gmail.com*

Sposób cytowania / Cite Surgiel-Gemza A, Zdrada-Nowak J. Impact of dermatoses on patients' mental health. *Aesth Cosmetol Med*. 2024;13(6):255-262. https://doi.org/10.52336/acm.2024.007

Impact of dermatoses on patients' mental health

Wpływ dermatoz na zdrowie psychiczne chorych

ABSTRACT

The skin serves not just a protective function, but also significantly influences body image and social relationships. Consequently, dermatological issues, including acne, eczema, psoriasis, and atopic dermatitis, can yield substantial psychological repercussions for individuals impacted.

This study aimed to investigate the correlation between dermatological issues and mental health, emphasising the influence of emotions, stress, and body image on the psychological well-being of individuals. The significance of a comprehensive strategy in the management of dermatological patients was underscored.

Findings indicate that people with chronic dermatological conditions are more likely to experience anxiety disorders, depression and reduced self-esteem. Skin problems can lead to social isolation and exacerbate negative emotions such as shame and frustration. Stress, as a reaction to these challenges, can further exacerbate skin symptoms, creating a vicious cycle between physical and psychological conditions. The results indicate a necessity for enhanced cooperation between dermatologists and psychologists in the management of patients with persistent skin disorders.

Keywords: psychodermatology, quality of life, scars, stress, mental health

STRESZCZENIE

Skóra pełni nie tylko funkcje ochronne, ale również odgrywa ważną rolę w kształtowaniu wizerunku ciała oraz interakcjach społecznych. Dlatego problemy skórne, takie jak trądzik, egzema, łuszczyca i atopowe zapalenie skóry, mogą mieć znaczące konsekwencje psychologiczne dla osób, u których one występują.

Celem pracy było zbadanie związku między problemami skórnymi a zdrowiem psychicznym, podkreślając rolę emocji, stresu i postrzegania własnego ciała w kształtowaniu dobrostanu psychologicznego osób chorych. Podkreślono znaczenie podejścia holistycznego w leczeniu pacjentów dermatologicznych.

Wyniki badań wskazują, że osoby z przewlekłymi schorzeniami dermatologicznymi częściej doświadczają zaburzeń lękowych, depresji oraz obniżonego poczucia własnej wartości. Problemy skórne mogą prowadzić do izolacji społecznej, a także pogłębiać negatywne emocje, takie jak wstyd i frustracja. Stres, będący reakcją na te wyzwania, może dodatkowo nasilać objawy skórne, tworząc błędne koło pomiędzy stanem fizycznym a psychicznym. Wnioski sugerują konieczność zwiększenia współpracy między dermatologami a psychologami w opiece nad pacjentami cierpiącymi na przewlekłe choroby skórne.

Słowa kluczowe: psychodermatologia, jakość życia, blizny, stres, zdrowie psychiczne

INTRODUCTION

Skin diseases (dermatoses), due to their chronic nature, are significant medical as well as psychological problems. It has been proven that there are interconnections between most dermatoses and psychological factors. High emotional load, and uncontrolled, severe and permanent stress affect the proper functioning of the entire human body. Prolonged

states of stress affect the course of most diseases of internal organs. Stress is recognised as a contributing component in the aetiology of diseases such as hypertension, diabetes, depression, and many other different dermatoses [1]. Severe stress is identified as an underlying cause to the development or exacerbation of skin lesions in a certain dermatological condition. Examples of such disorders include acne vulgaris, rosacea, atopic dermatitis (AD), psoriasis, and vitiligo. Dermatoses can exacerbate stress and disturb proper psychological functioning, resulting in diminished self-esteem [2].

Chronic skin diseases usually do not pose an immediate threat to a person's life. However, by causing changes in appearance can influence both self-esteem and the perceptions of others towards an individual. Such alterations cause fear of interacting with other people, leading to social phobia or exclusion by society. It has been observed that these reactions often come from both the affected person and his or her environment, which consequently significantly affects the patient's mental state [3, 4]. Dermatological diseases, due to the manifestation of symptoms on visible parts of the skin such as face, neck, hands, can cause reduced comfort in the patient's life. As a consequence, dermatoses can contribute to the limitation of self-realization and fulfillment of certain social roles [5, 6].

Psychodermatology, an emerging medical discipline at the convergence of dermatology, psychology, and psychiatry, examines the impact of emotional and stress-related factors on the progression of skin disorders. Psychocosmetology, emphasising the multidisciplinary management of skin health, is increasingly gaining popularity [7].

Human skin is involved in the transmission of emotions. In situations such as anxiety, the skin pales, during anger it turns red, and in times of excessive stress, increased sweating is observed. Receptors in the skin enable the perception of various stimuli, including touch, pressure, heat, cold, and pain. External signals are conveyed to the spinal cord and subsequently to the brain via receptors [8, 9].

THE EFFECT OF STRESS ON THE PHYSIOLOGY OF THE BODY

Homeostasis is the ability to maintain the constancy of the body's internal environment under all circumstances. The body tries to maintain physiological equilibrium in all situations, which is essential for its proper functioning. Stressful conditions are supposed to represent a transient disruption; nevertheless, this is not invariably true. Walter B. Cannon was the creator of the "fight or flight" theory and viewed stress as a reaction to a threat, an adaptation to the situation and a response to the threat. The sympathetic nervous system stimulates the adrenal medulla to release adrenaline, which sets off a cascade of responses. The body's stress/threat response is the primary affect to danger [10, 11].

Selye et al. observed nonspecific physiological changes during aversive stimuli, which were physiological-endocrine in nature and caused by the action of the hypothalamic-pituitary-adrenal cortex axis [12, 13]. The researcher called this response general adaptation syndrome (GAS).

The course of the stress reaction

1. Emergency response stage
   - Shock phase - the stage when the factor directly affects the body, causing non-specific physiological changes. Mobilization phase - reduces the effect of stress on the body by, among other things, increasing blood pressure.
2. Stage of resistance
   - The phase of relative adaptation - the body adapts to the existing situation, the symptoms from the first stage disappear, the stressful situation continues, but the body copes relatively well, but poorly tolerates other stimuli.
3. Exhaustion stage
   - An increase in the body's arousal at the physiological level, which is not used to combat the threat, physiological functions are disrupted, protein synthesis is reduced, which affects the defence capabilities of the immune system [13].

The stress response is produced by the central parts of the brain (thalamus, amygdala and prefrontal cortex), which form a functional triangle, as well as the central-peripheral centers (sympathetic-adrenergic axis and hypothalamic-pituitary-adrenal axis). The external stressor is received by receptors of the peripheral nervous system, and reaches the thalamus, which is a relay station to the amygdala. The signal is processed into emotions, mostly negative. In response to stimulation of other brain centers, neurotransmitters and neurohormones are released. The prefrontal cortex plays a role in confronting the neural signal with accumulated knowledge and responding to the stimulus. Activation of the sympathetic nervous system occurs immediately after a stressor is acted upon. The adrenal medulla secretes increased amounts of adrenaline and norepinephrine, triggering fight-or-flight readiness responses. The activated hypothalamus releases corticoliberin and vasopressin prompting the pituitary gland to secrete corticotropin, which stimulates the adrenal cortex to synthesize and secrete the stress hormones cortisol, corticosterone and cortisone [14-19].

The long-term effects of stress affect the entire body's functioning in physical, mental and social aspects. It has been observed that stress causes immediate as well as long-term consequences. In addition to systemic changes such as blood glucose abnormalities, headaches, neck pain, chest pain, shallow breathing, etc., the development of stress-related disease complications occurs [20].

Stress also impacts the skin, leading to the formation of stretch marks, breakage of hair and nails, skin atrophy and dryness, increased sweating, and exacerbation of skin disorders such as psoriasis, atopic dermatitis, or acne vulgaris. The deterioration of the skin due to stress impacts an individual's psychological functioning, leading to feelings of humiliation, social isolation, sadness, anxiety, and suicidal ideation [15].

THE IMPORTANCE OF PSYCHODERMATOLOGY IN THE TREATMENT OF DERMATOSES

Psychophysiological disorders

Psychophysiological disorders include dermatoses whose symptoms can be induced or exacerbated by stress. The neuro-immuno-cutaneous-endocrine (NICE) model explains the interaction between different organs, neurotransmitters, hormones and cytokines [21, 22].

The Griesemer Index, a coefficient assessing the influence of emotions on the onset of various skin disorders, was developed following an examination of many patient interviews detailing the conditions preceding lesion formation (Table 1) [23].

The presented relationships between stress and emotions and the regulation of the nervous, immune and endocrine systems, as well as their effects on the physiology of the skin and the course of diseases, have still not been fully elucidated. Studies have been conducted on such disease entities as psoriasis, atopic dermatitis and alopecia areata.

As an example, a study by Gupta et al. found that 39% of patients with psoriasis confirmed the onset of a stressful event within a month preceding the beginning of the disease, compared to only 10% of the control group [25]. In comparison, in the case of atopic dermatitis, the onset of symptoms is preceded by stress in up to 70% of patients. The clear impact of emotions, and stress on the initiation and course of the mentioned dermatological diseases has been proven. One of the key factors in the treatment process should be relaxation methods, behavioral-cognitive techniques or hypnosis. In many cases, consultation with specialists like a psychologist or psychiatrist, and the inclusion of anti-anxiety or antidepressant treatment, should be recommended [26-29].

Mental disorders secondary to dermatological diseases

Such disorders include those that occur as a consequence of a primary dermatosis, e.g. acne, psoriasis, atopic dermatitis, alopecia. In case of these conditions, the disease symptoms and prolonged treatment, or the location of skin lesions in a conspicuous place can secondarily cause increased stress, affect image perception, lower self-esteem, shame or fear

Table 1 Griesemer Index - an coefficient of the influence of emotions on the occurrence of various dermatological diseases

Diagnosis	Percentage of cases resulting from emotions	Biological incubation - the time between the onset of stress and the appearance of symptoms.
Profuse sweating	100	seconds
Increased scratching	98	seconds
Local itching	98	several days, up to 2 weeks
Local hair loss	96	2 weeks
Acne rosacea	94	2 days
Pruritus	86	seconds
Lichen planus	82	several days, up to 2 weeks
Hand eczema (dyshidrosis)	76	2 days, until blisters appear
Atopic eczema	70	seconds, to the onset of itching
Self-harm	69	seconds
Urticaria	68	minutes
Psoriasis	62	several days, up to 2 weeks
Traumatic eczema	56	seconds
All eczema (except contact)	56	several days
Acne	55	2 days, until red lumps appear
Hair loss	55	2-3 weeks

Source: [21, 23, 24]

of exclusion from a social group. Another accompanying symptom in dermatological diseases is pruritus, which is a cause of increased tension, can disrupt concentration, and lead to insomnia or excessive nervousness [23].

The visible symptoms of the disease become a cause of anger, frustration, rebellion, depression and even an increased risk of suicide. It is observed that more than 60% of patients with acne suffer from depression or have anxiety symptoms. Adolescents with acne perceive themselves as worse ones compared to healthy peers. The identical issue pertains to patients with atopic dermatitis. A significant degree of behavioural dysregulation has been noted, similar to that observed in individuals with acne vulgaris [30, 31]. In a study conducted by Sleep et al. in a group of 30 patients with psoriasis, atopic dermatitis and alopecia areata, the deterioration requiring hospitalization caused patients to experience significantly elevated levels of anxiety and depression. Additionally, it was found that women experienced more severe anxiety and depressive symptoms compared to men [32].

Primary psychiatric disorders with cutaneous manifestation

The primary condition is a psychological disorder that causes skin lesions. Skin-manifesting illnesses include parasitic lunacy (Ekbom syndrome), classified under the ICD-10 category of hallucinations according to the International Classification of Mental and Behavioral Disorders.

Other examples of this type of diseases found in the new classification of mental disorders (DSM-V, Diagnostic and Statistical Manual of Mental Disorders), listed under one group of obsessive compulsive and related disorders (OCRD), are dysmorphophobia, trichotillomania, onychophagia or dermatillomania. Non-suicidal self-injury (NSSI) resulting from emotional states is an increasingly serious and growing problem [33]. It was observed that one in every two young patients at the Department of Adolescent Psychiatry at the Medical University of Lodz showed a history of NSSI [22].

Dermatoses caused or aggravated by psychotropic drugs and psychiatric effects of dermatological treatment

Drugs from the psychotropic group can potentially cause a specific reaction on the skin, such as allergic reaction, psoriasis, hair loss problem or acne. During treatment with normothymic agents (e.g., lithium, anticonvulsants), most severe dermatological disorders are observed [34].

The subsequent effects of psychotropic drugs on the onset or severity of dermatological conditions are as follows:

- **anticonvulsants:** rash, alopecia, pruritus, hyperpigmentation, erythema multiforme, Stevens-Johnson syndrome, exfoliative dermatitis, excessive sweating, psoriasis, acne lesions;
- **lithium:** psoriasis, acne, alopecia, folliculitis, urticaria, rash;
- **antipsychotic drugs:** hypersensitivity to light (phenothiazine derivatives) and changes in pigmentation (thioridazine and chlorpromazine), local reactions after injections of depot drugs;
- **antidepressants:** hyperhidrosis, bleeding, hypersensitivity to light, discoloration (tricyclic antidepressants);
- **therapies with other psychotropic drugs:** risk of allergic reaction and rash [35].

Dermatological treatment affects the patient's psyche, and the therapies and drugs used in dermatoses can cause or exacerbate psychological disorders such as:

- **isotretinoin:** depression,
- **interferon-α:** depression, suicidal thoughts, psychosis, cognitive impairment,
- **ultraviolet radiation:** mood changes, hypomania,
- **methotrexate:** mood disorders,
- **dapson:** psychosis,
- **antihistamines:** delirium, central nervous system depression,
- **glucocorticosteroids:** post-steroid mood disorders or psychosis, anxiety, mania, delirium, depression [35].

ACNE VULGARIS

Acne vulgaris is a dermatological condition that occurs in various age groups, with adolescents most commonly affected. Treatment of moderate acne has traditionally involved topical preparations, which often cause side effects (skin irritation and dryness) [36]. Antibiotic therapy, both topical and oral, is also a popular therapeutic approach due to the increased colonization of the *Cutibacterium acnes* bacterium [37]. This microorganisms play a key role in the pathogenesis of inflammatory acne, mainly through their ability to activate complement components and metabolize sebaceous triglycerides to fatty acids, which chemotactically attract neutrophils. *C. acnes* is a component of the symbiotic flora of human skin as long as the conditions favourable to the development of inflammation are not created. The bacteria can be detected in both inflammatory lesions and blackheads. In addition to *C. acnes*, other bacteria of the skin microbiome may be involved in the development of acne lesions, such as aerobic species of the *Staphylococcus* genus, e.g. *Staphylococcus aureus*, which is often involved in superficial infections of sebaceous skin units [38, 39].

Of particular note in the etiopathogenesis of acne is the composition of sebum, especially the reduction in linoleic acid levels. In addition, squalene, one of the main components of sebum, undergoes oxidation by lipoperoxidase due to reduced vitamin E content. This induces the production of pro-inflammatory cytokines, stimulates keratinocyte proliferation and activates peroxisome proliferator-activated receptor (PPAR). Monounsaturated fatty acids can also affect changes

in keratinocyte proliferation and differentiation [40, 41]. The cause of abnormal keratinization of hair follicles is likely to be the irritation of hair follicle walls by sebum of altered composition and the presence of bacteria in the sebaceous glands' exit ducts, leading to excessive production and accumulation of corneocytes. Keratinocyte hyperproliferation can also be driven by increased androgen levels, changes in sebum lipid composition and excessive growth of *C. acnes* bacteria. The accumulation of sebum and dead skin cells in the hair follicle promotes the growth of microorganisms that damage the follicle walls, leading to acne lesions [42, 43].

Acne significantly affects patients' quality of life, causing discomfort and psychological stress. Acne lesions can hinder social communication, especially in young people, and studies have shown that quality of life decreases especially in women under the age of 18 whose acne and scars involve the face. Several studies have confirmed that regardless of the severity of skin lesions, acne leads to a deterioration in patients' quality of life. In addition to the visible cutaneous lesions, patients often complain about the ineffectiveness of therapy, which, despite temporary improvement, fails to prevent recurrence. The long-term struggle with acne often leads to frustration, prompting patients to seek alternative treatments on their own, such as tanning beds and taking high doses of vitamins [25, 44, 45].

Complications following inflammatory lesions in the course of acne vulgaris are hyperpigmentation and acne scars. Their presence on exposed areas of the body such as the face and the inability to camouflage them are a cause of lowered self-esteem, decreased attractiveness, lack of self-confidence, social isolation, as well as anxiety and depression [46].

SCARS

A scar (*cicatrix*) is a skin lesion resulting from the healing process of a wound. Wounds can arise from a variety of causes, such as mechanical, chemical or thermal trauma, or as a result of chronic inflammation of the skin, for example in the course of acne vulgaris. A scar consists mainly of fibrous connective tissue [47].

Wound healing is a physiological tissue response involving the closure of the defect through the process of scarring. Superficial wounds heal by epidermis without producing a scar. Injury to deeper layers of skin, i.e., the dermis, heals either by epidermis or by granulation with the formation of a scar. Wound healing involves four major overlapping phases: hemostatic and inflammatory, proliferation and remodelling (maturation). It is a complex process consisting of a sequence of catabolic and anabolic phenomena involving different cell types and the course of many biochemical reactions [48].

At sites of injury, the resulting scar tissue can cause local or generalized problems. Local problems include: a change in the appearance of the skin, e.g. retractions, bulges, hyperpigmentation, hypopigmentation, swelling, but also functional problems, e.g. sensory disturbances, tingling, itching, numbness, and pain. On the contrary, the analysis of generalised problems has, until recently, overlooked the significance of scar issues regarding their influence on the overall condition of the body, the tensions conveyed by the organ itself, or adhesions with fascial tissue, which may be classified as pathological alterations. The transmission of tension or tensile forces through fascial tissue from the site of injury to the entire body is observed [49].

Classification of scars

The wound-healing process begins when the tissue is damaged and can take up to 2 years. Types of scars can be considered by appearance, time and cause of their formation.

Scars, according to the chronological criterion, are divided into immature and mature.

- Immature scars are characterized by a duration of less than 1 year after formation, and a thick and hard structure. The scar is convex and extends above the level of the surrounding tissues, not very flexible with a red or pinkish colour, tends to fade when pressed.
- Mature scars are scars more than 1 year after formation, are soft, flat and flexible, and have normal vascularization, meaning that the tissue does not fade when pressed [50, 51].

Mustoe's classification of scars, based on their appearance and texture, is as follows:

- **a normal scar** is flat with a light colour;
- **an abnormal scar** is red, may cause itching or pain, and is slightly raised above the surface of the surrounding tissue. Many such scars heal properly over time, become flat and take on a colour similar to the surrounding area;
- **a linear hypertrophic scar** is red, slightly raised, sometimes giving an itching sensation or pain, and does not extend beyond the surgical wound area. It usually develops within the first week and may enlarge over the next 3-6 months. The scarring process can take up to 2 years, and ends in a convex, tightening scar of varying width. Regression of the lesion is possible;
- **a hypertrophic scar with a large area** is a planar, convex scar, sometimes there is itchiness. An example is a burn scar;
- **a small keloid** is a limited, convex scar less than 0.5 cm in diameter, characterized by itching and extending beyond the original wound area;
- **a large keloid** is a large, convex scar, more than 0.5 cm in diameter, painful or itchy, extending beyond the outline of the wound, and may enlarge over the following years [52].

Acne scars form as a result of skin damage during the healing process of inflammatory lesions. There are two basic types of scars: atrophic scars, resulting from collagen loss, and hypertrophic scars, resulting from an excess of collagen.

Between 80% and 90% of individuals with acne scars present atrophic scars resulting from collagen depletion, whereas a few display hypertrophic scars and keloids [53].

Types of atrophic scars

Atrophic scars most often appear as a consequence of an acute inflammatory process that leads to collagen destruction and skin atrophy. They are characterized by a sunken structure, lying below the level of the surrounding skin. A significant proportion of them arise as a result of acne lesions or after smallpox (Figure 1).

There are the following types of atrophic scars:

- scars of the ice-pick type (the shape of a pick) - are narrow with a depth reaching even to the lower layer of the dermis bordering the subcutaneous tissue. In this type of scars, sharp edges and constriction running deep into the skin are observed, and the cross-section of the scars is V-shaped. Scars of this type are common and account for 60 to 70% of atrophic scars;
- boxcar (carriage shape) scars - have a round or oval shape, are quite wide and flat, the cross-section of the scars is U-shaped or square. They are characterized by well-defined edges and a sharply demarcated edge. Scars of this type are not contiguous, and their depth varies from 0.1 to 0.5 mm. They occur mainly on the cheeks and jaw area, accounting for 20% to 30% of all atrophic scars;
- rolling (cylindrical) scars - wide (about 4-5 mm), shallow, M-shaped in cross-section, with soft edges and fine margins. They give the skin a wavy appearance, usually appear on large, extensive areas of skin, and are caused by chronic inflammation. They can vary in size, plus they often merge with other cylindrical scars to form clusters. These scars account for 15% to 20% of atrophic scars.

Gan et al. proposed expanding the classification of acne scars to include papular scars, which are hypertrophic scars that also occur as a complication of acne [55, 56].

Types of hypertrophic scars

Hypertrophic and keloid scars are characterized by excessive collagen deposition and concomitant reduced collagenase activity. Hypertrophic scars are pink, convex and hard, with thick collagen bundles that remain within the boundaries of the original injury site. The histology of hypertrophic scars is similar to that of other cutaneous scars. In contrast, keloids form as reddish-purple nodules that spread beyond the boundaries of the original wound. Hypertrophic and keloid scars are more common in people with high phototypes, i.e., grade 4-6 on the Fitzpatrick scale, and form mainly on the thorax [53, 57].

THE IMPACT OF SCARS ON PSYCHOLOGY AND PAIN COMPLAINTS

In the digital era, physical attractiveness is a significant factor for many people regarding self-acceptance and societal acceptability. Adolescents frequently aspire to emulate individuals featured on magazine covers or social media platforms. They adhere to the belief in an ideal appearance and impeccable skin; any perceived beauty fault leads them to feel inferior to their peers, resulting in social exclusion and stigmatisation. Undoubtedly, any injury due to unfortunate accidents, and the consequent formation of various types of scars, have the greatest negative impact on the psyche of the victim. However, scars can also arise from other causes, such as planned surgeries, various types of dermatoses, such as acne, and other diseases that cause disfigurement, which often leads to suffering, depression and further psychological conditions. The statement that a scar on the skin often leaves a mark on the psyche as well is undoubtedly accurate. In the context of scarring, not only psychological problems are observed, but also the effect of scarring due to trauma on the state of tension in the muscular and fascial systems is observed. In such a situation, there is immobilization or adhesion in and around the scar area, which leads to a forced body position, resulting in pain symptoms, and functional

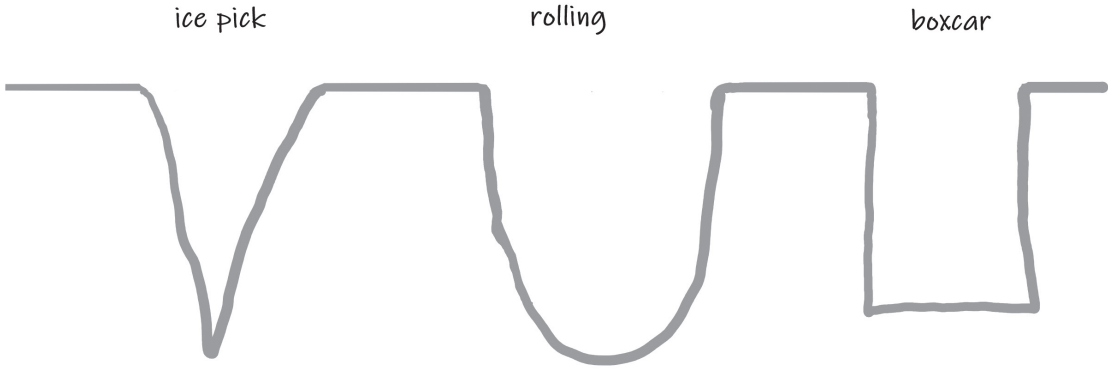

Figure 1 Types of atrophic scars. **Source**: own elaboration based on [53, 54].

limitations. Another symptom can be dysfunction of the surrounding joint or organ, with or without the involvement of the fascial participation system, which can affect the posture change. When the cause of a patient's abnormal posture, resulting from the presence of scar tissue, is not properly diagnosed and then addressed, damage on a structural level, known as a chain of leisings, can occur. The management of such cases, that is, the treatment of diseases and pain syndromes, is based primarily on understanding the functioning of the fascial system in the context of adhesions and taking therapeutic measures that involve more than just the area of the body where the complaints occur [58-60]. Therefore, the collaboration of a group of specialists such as a cosmetologist, dermatologist, surgeon, vascular surgeon, plastic surgeon, physiotherapist, psychologist and psychiatrist in scar reduction therapy is invaluable.

CONCLUSIONS

Studies and observations indicate that chronic skin diseases and their consequences, such as scarring, significantly affect the psychological state of patients. At the same time, emotions play an important role in the course of dermatoses, affecting periods of remission and exacerbation. Such patients require interdisciplinary therapy involving a dermatologist, psychologist, psychiatrist and, depending on the disease entity, other specialists such as an endocrinologist, physiotherapist or cosmetologist. Integrating non-pharmacological therapies, such as relaxation methods or behavioral-cognitive techniques, into the treatment of skin diseases, can improve the effectiveness of medical intervention. Public education regarding chronic dermatoses, including psoriasis, atopic dermatitis, acne, alopecia areata, and vitiligo, should encompass their etiopathogenesis, disease progression, treatment choices, and the fact that these conditions are non-contagious. Various social projects are organized, such as 'I am not Ladybug' concerning psoriasis, '(not) reversible' by artist Joanna Bury, who invited women with vitiligo to a photo shoot, or the 'Understanding AD' campaign under the auspices of the Polish Atopic Diseases Society, with photographs by Jacek Poremba. These projects are designed to bring the problems of those suffering from the aforementioned dermatoses closer to the public. The relationship between skin and psychological conditions and their interaction should be the subject of further research and observation.

REFERENCES / LITERATURA

1. Szepietowski J, Pacan P, Peich A, Grzesiak M. *Psychodermatologia*. Wrocław: Akademia Medyczna im. Piastów Śląskich; 2012.
2. Kieć-Świerczyńska M, Dudek B, Kręcisz B, et al. *Rola czynników psychologicznych i zaburzeń psychicznych w chorobach skóry*. Łódź: CYBRA Łódzka Regionalna Biblioteka Cyfrowa; 2006.
3. Studzińska MM, Lewicka M, Sulima M, Pietrzak D. Występowanie depresji u kobiet chorych na łuszczycę w okresie okołomenopauzalnym a potrzeba wsparcia – przegląd literatury. *Psychiatr Psychol Klin*. 2012;12:120-124.
4. Cepuch G, Wojas K, Zych B, Matuszewska B. Assessment of emotional state of psoriasis patients and the degree of acceptance of the disease. *Fam Med Prim Care Rev*. 2014;16:85-87.
5. Tyc-Zdrojewska E, Trznadel-Grodzka E, Kaszuba A. Wpływ przewlekłych chorób skóry na jakość życia pacjentów. *Clin Dermatology/Dermatologia Klin*. 2011;13(3):155-160.
6. Kowalewska B, Krajewska-Kułak E, Wrońska I, et al. Samoocena jakości życia przez pacjentów z problemami skórnymi. *Dermatologia Kliniczna*. 2010;12(2):106-113.
7. Zalewska-Janowska A. Psychodermatologia w praktyce – studium przypadków ze zmianami skóry. *Sztuka leczenia*. 2011;1-2:69-70.
8. Caputa M. Dlaczego skóra twarzy zdradza nasze emocje? Termoregulacja mózgu. *Kosmos*. 1993;42:347-363.
9. Swider-Al-Amawi M, Marchlewicz M, Kolasa A, et al. Neuroendocrine Function of Skin. *Postep Biol Komorki*. 2010;37:795-806.
10. Landowski J. Neurobiologia reakcji stresowej. *Neuropsychiatria i Neuropsychologia*. 2007;2:26-36.
11. McCarty R. The Fight-or-Flight Response: A Cornerstone of Stress Research. In: Fink G, ed. *Stress: Concepts, Cognition, Emotion, and Behavior*. San Diego: Academic Press; 2016:33-37.
12. Cunanan AJ, DeWeese BH, Wagle JP, et al. The General Adaptation Syndrome: A Foundation for the Concept of Periodization. *Sport Med*. 2018;48:787-797.
13. Selye H. The general adaptation syndrome and the diseases of adaptation. *J Clin Endocrinol Metab*. 1946;6:117-230.
14. Musiała N, Hołyńska-Iwan I, Olszewska-Słonina D. Kortyzol – nadzór nad ustrojem w fizjologii i stresie. *Diagn Lab*. 2018;54:29-36.
15. Polak D, Teległów A, Piotrowska A. Wpływ czynników psychologicznych na powstawanie i przebieg wybranych chorób skóry oraz znaczenie zaburzeń dermatologicznych w dobrostanie psychicznym. *Aesth Cosmetol Med*. 2020;9(5):455-460.
16. Nagalski A, Kiersztan A. Fizjologia i molekularny mechanizm działania glikokortykoidów. *Postepy Hig Med Dosw*. 2010;64:133-145.
17. Skulimowska K. Mutual infuence of somatic and psychical state of patients with somatic disease and neurotic disorder. *Psychoterapia*. 2011;158:41-59.
18. Rozpędek W. Podłoże neurobiologiczne zespołu stresu pourazowego. *Neuropsychiatria i Neuropsychologia/Neuropsychiatry and Neuropsychology*. 2015;10:27-39.
19. Gołyszny M. „Stare" i „nowe" neuropeptydy jako modulatory czynności osi stresu (podwzgórze-przysadka-nadnercza). *Psychiatry*. 2018;3:135-147.
20. Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. *Nutrition*. 2007;23:887-894.
21. Shenefelt PD. Psychodermatological disorders: recognition and treatment. *Int J Dermatol*. 2011;50:1309-1322.
22. Makowska I, Gmitrowicz A. Psychodermatologia – Pogranicze dermatologii, psychiatrii i psychologii. *Psychiatr i Psychol Klin*. 2014;14:100-105.
23. Mojs E. Choroby skóry w ujęciu psychosomatycznym, *Nowiny Lekarskie*. 2010;79(6):483-486.
24. Mavrogiorgou P, Juckel G. Dermatological diseases and their importance for psychiatry. *Nervenarzt*. 2017;88:254-267.
25. Gupta MA, Gupta AK. Psychiatric and psychological co-morbidity in patients with dermatologic disorders: epidemiology and management. *Am J Clin Dermatol*. 2003;4:833-842.
26. Gupta MA, Gupta AK. A practical approach to the assessment of psychosocial and psychiatric comorbidity in the dermatology patient. *Clin Dermatol*. 2013;31:57-61.
27. Taylor R, Bewley A, Melidonis N. Psychodermatology. *Psychiatry*. 2006;5:81-84.
28. Filaković P, Petek A, Koić O, et al. Comorbidity of depressive and dermatologic disorders – Therapeutic aspects. *Psychiatr Danub*. 2009;21:401-410.
29. García-Hernández MJ, Ruiz-Doblado S, Rodriguez-Pichardo A, Camacho F. Alopecia areata, stress and psychiatric disorders: a review. *J Dermatol*. 1999;26:625-632.
30. Gordon-Elliott JS, Muskin PR. Managing the patient with psychiatric issues in dermatologic practice. *Clin Dermatol*. 2013;31:3-10.
31. Behnam B, Taheri R, Ghorbani R, Allameh P. Psychological impairments in the patients with acne. *Indian J Dermatol*. 2013;58:26-29.
32. Śpila B, Jazienicka I, Pucuła J. Analiza czynników psychogennych u chorych na schorzenia skóry. *Dermatol Klin*. 2004;6:137-141.
33. Gupta MA, Guptat AK. The use of antidepressant drugs in dermatology. *J Eur Acad Dermatol Venereol*. 2001;15:512-518.

34. Warnock JK, Morris DW. Adverse cutaneous reactions to mood stabilizers. *Am J Clin Dermatol.* 2003;4:21-30.
35. Locala JA. Aktualne koncepcje w psychodermatologii. *Curr Psychiatry Rep.* 2009;11:60-68.
36. Bergler-Czop B. The aetiopathogenesis of acne vulgaris – what's new? *Int J Cosmet Sci.* 2014;36:187-194.
37. Eichenfield LF, Krakowski AC, Piggott C, et al. Evidence-based recommendations for the diagnosis and treatment of pediatric acne. *Pediatrics.* 2013;131:Suppl:S163-186.
38. Mills OJ, Thornsberry C, Cardin CW, et al. Bacterial resistance and therapeutic outcome following three months of topical acne therapy with 2% erythromycin gel versus its vehicle. *Acta Derm Venereol.* 2002;82:260-265.
39. Dreno B, Dekio I, Baldwin H, et al. Acne microbiome: From phyla to phylotypes. *J Eur Acad Derm Venereol.* 2024;38(4):657-664.
40. Mastrofrancesco A, Ottaviani M, Cardinali G, et al. Pharmacological PPARγ modulation regulates sebogenesis and inflammation in SZ95 human sebocytes. *Biochem Pharmacol.* 2017;138:96-106.
41. Kurokawa I, Nakase K. Recent advances in understanding and managing acne. *F1000Res.* 2020;9(F1000 Faculty Rev):792.
42. Cong T-X, Hao D, Wen X, et al. From pathogenesis of acne vulgaris to anti-acne agents. *Arch Dermatol Res.* 2019;311:337-349.
43. Schmid-Wendtner M-H, Korting HC. The pH of the skin surface and its impact on the barrier function. *Skin Pharmacol Physiol.* 2006;19:296-302.
44. Dreno B, Bordet C, Seite S, et al. Acne relapses: impact on quality of life and productivity. *J Eur Acad Dermatol Venereol.* 2019;33:937-943.
45. Baranowska A, Krajweska-Kułak E, Jankowiak B, et al. Ocena jakości życia pacjentów z trądzikiem pospolitym z wykorzystaniem skal DLQI i CADI. *Probl Hig Epidemiol.* 2014;95(3):713-722.
46. Gebauer K. Acne in adolescents. *Aust Fam Physician.* 2017;46:892-895.
47. Gantwerker EA, Hom DB. Skin: histology and physiology of wound healing. *Facial Plast Surg Clin North Am.* 2011;19:441-453.
48. Witmanowski H, Lewandowicz E, Zieliński T, et al. Hypertrophic scars and keloids Part I. Pathogenesis and pathomechanism. *Advances in Dermatology and Allergology.* 2008;25(3):107-115.
49. Téot L. Structure de la peau et cicatrisation cutanée [Skin structure and cutaneous scarring]. *Rev Infirm.* 2002;80:20-23.
50. Clementoni MT, Azzopardi E. Specific Attention Areas in Scar Management: Management of Atrophic Scars. In: Téot L, Mustoe TA, Middelkoop E, et al. *Textbook on Scar Management: State of the Art Management and Emerging Technologies.* Cham (CH): *Springer*; 2020:353-362.
51. Shin TM, Bordeaux JS. The role of massage in scar management: a literature review. *Dermatol Surg.* 2012;38:414-423.
52. Mustoe TA, Cooter RD, Gold MH, et al. International clinical recommendations on scar management. *Plast Reconstr Surg.* 2002;110:560-571.
53. Fabbrocini G, Annunziata MC, D'Arco V, et al. Acne scars: pathogenesis, classification and treatment. *Dermatol Res Pract.* 2010;2010:893080.
54. Chilicka K, Rusztowicz M, Szyguła R, Nowicka D. Methods for the Improvement of Acne Scars Used in Dermatology and Cosmetology: A Review. *J Clin Med.* 2022;11(10):2744.
55. Gan SD, Graber EM. Papular scars: an addition to the acne scar classification scheme. *J Clin Aesthet Dermatol.* 2015;8:19-20.
56. Jacob CI, Dover JS, Kaminer MS. Acne scarring: a classification system and review of treatment options. *J Am Acad Dermatol.* 2001;45:109-117.
57. Clark AK, Saric S, Sivamani RK. Acne Scars: How Do We Grade Them? *Am J Clin Dermatol.* 2018;19:139-144.
58. González N, Goldberg DJ. Update on the Treatment of Scars. *J Drugs Dermatol.* 2019;18:550-555.
59. Lee HJ, Jang YJ. Recent Understandings of Biology, Prophylaxis and Treatment Strategies for Hypertrophic Scars and Keloids. *Int J Mol Sci.* 2018;19(3):711.
60. Ogawa R. The Most Current Algorithms for the Treatment and Prevention of Hypertrophic Scars and Keloids: A 2020 Update of the Algorithms Published 10 Years Ago. *Plast Reconstr Surg.* 2022;149:79e-94e.

otrzymano / received: 12.09.2024 | poprawiono / corrected: 19.09.2024 | zaakceptowano / accepted: 04.10.2024