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Review

The impact of urticaria on the psychosomatic aspects of patient's functioning

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ABSTRACT

Urticaria is classified as a skin disease, yet it exerts significant impact on psychological functioning and behavior of patients. It impairs quality of life, disturbs sleep, and increases the prevalence of anxiety and depressive disorders, all associated with stigmatization and chronic pruritus. Disease-related stress may exacerbate the clinical course through neuroimmunological mechanisms involving interactions between skin cells, mast cells, and neurons. The aim of this review is to present the relationship between urticaria and psychosomatic aspects of patient's functioning. A narrative literature review was conducted using the PubMed database. The analysis included original research articles, review articles, meta-analyses, and case reports published between 2015 and 2025. The analysis demonstrated a significant association between urticaria, particularly its chronic form, and increased prevalence of mental disorders. Stress plays a dual role, acting both as a factor exacerbating disease severity and as a direct trigger for mast cell degranulation via catecholamine release or cholinergic stimulation in specific subtypes, such as adrenergic and cholinergic urticaria. The clinical picture is frequently compounded by anxiety disorders, phobias, and low self-esteem, although severity of psychological symptoms does not always correlate with severity of skin lesions. Urticaria represents a systemic condition with complex pathogenesis, in which psychological and immunological factors interact and reinforce each other, creating a vicious cycle. Effective clinical management requires a holistic, individualized approach that includes psychological support, improving treatment outcomes, and quality of life.

KEYWORDS

quality of life, neuroimmunology, urticaria, behavioural disorders, mental health

INTRODUCTION

Urticaria is a common skin disease characterized by the appearance of itchy wheals, defined as areas of skin redness with central clearing, which typically resolve spontaneously. Its underlying mechanism involves edema of the upper layers of the dermis, caused by mast cell degranulation and the release of histamine [1].

Urticaria is classified as acute when symptoms last up to 6 weeks, usually following allergen exposure or chronic when they persist beyond 6 weeks, often without an identifiable cause. It is chronic urticaria that is more frequently associated with broadly defined psychiatric disorders [2]. Chronic somatic symptoms and the stigmatization that frequently accompanies them often exert a negative impact on the psychosocial functioning of patients.

Given the above, this review article aims to demonstrate the links and discuss the potential causes of the comorbidity between psychiatric disorders and urticaria.

MATERIAL AND METHODS

Literature for this article was gathered using the PubMed database. We included original research, review articles, meta-analyses, and case reports from 2015 to 2025. The search terms included: "urticaria and behavioral disorders," "urticaria and psychosomatic symptoms," "urticaria and mental health," "stigmatization of patients with skin lesions," "urticaria and neuroinflammation," and "neurobiology of urticaria". Results were first evaluated by title and abstract, then by full text, isolating the studies that provided the best insight into the subject. The review encompasses a wide range of topics, from molecular and neurobiological mechanisms of urticaria to epidemiological findings and studies on psychiatric comorbidities and stigma in pediatric patients. Additionally, the bibliographies of these initial papers were manually searched for further relevant literature. This process yielded a final count of 25 publications. Key findings were summarised in Table I.

Table I. Interrelationships between urticaria and psychiatric disorders described in the literature

Reference number	Authorship	Title	Main result
[1]	Peck G, et al.	<i>Global epidemiology of urticaria: Increasing burden among children, females and low-income regions</i>	Up to 70% of children with urticaria were diagnosed with a psychiatric disorder.
[5]	Paller AS, et al.	<i>Stigmatization and Mental Health Impact of Chronic Pediatric Skin Disorders</i>	The stigmatization of children with skin diseases is associated with reduced quality of life, impaired psychological functioning, depression, and, to a moderate extent, anxiety disorders and worsened peer relationships.
[6]	Vivar KL and Kruse L	<i>The impact of pediatric skin disease on self-esteem</i>	Skin diseases significantly impact children's self-esteem. Conditions with an onset before 3 years of age primarily affect the parent-child relationship, whereas those emerging during school age and adolescence influence peer relationships and self-esteem.

[9]	Hergüner S, et al.	<i>Levels of depression, anxiety and behavioural problems and frequency of psychiatric disorders in children with chronic idiopathic urticaria</i>	Compared to their healthy peers, children with chronic urticaria were more likely to suffer from psychiatric disorders, most notably anxiety disorders, separation anxiety, and phobias.
[10]	Zhu CK, et al.	<i>Assessment of Quality of Life in Children with Chronic Urticaria using the Children's Dermatology Life Quality Questionnaire Index (CDLQI): A Retrospective Cohort Study,</i>	Sleep disturbances were observed in 50% of patients with chronic urticaria, twice the rate seen in the control group. In most pediatric cases, allergic urticaria has little to no impact on the quality of life; however, 27% of children with chronic urticaria report a low quality of life, which correlates with a later age of symptom onset, elevated CRP levels, the presence of cold-induced urticaria, and a history of atopic dermatitis.
[14]	Konstantinou GN and Konstantinou GN	<i>Psychological Stress and Chronic Urticaria: A Neuro-immuno-cutaneous Crosstalk. A Systematic Review of the Existing Evidence</i>	The majority of patients with urticaria report experiencing a significant stressor prior to disease onset. The prevalence of psychiatric disorders is higher in the urticaria patient population than in the general population. Chronic skin conditions exacerbate psychiatric disorders, just as chronic stress aggravates dermatological diseases.
[15]	Xiang YK, et al.	<i>Psychological Stress and Urticaria: Pathophysiologic and Therapeutic Updates</i>	Emotional stress is both a trigger and a consequence of chronic urticaria (CU), creating a self-perpetuating cycle. Studies show that one-third of CU patients suffer from at least one psychiatric disorder, most notably anxiety, depression, and PTSD. Insomnia, in

			particular, is a significant predictor of the disease's onset. The severity of psychological distress, including impaired academic performance and poor sleep quality, correlates directly with the frequency of stressful life events and the clinical severity of urticaria.
[22]	Slater KN, et al.	<i>Adrenergic Urticaria: An Updated Review,</i>	Some patients with adrenergic urticaria suffer from psychiatric disorders or exhibit emotional lability.
[23]	Vadas P, et al.	<i>Cholinergic Urticaria with Anaphylaxis: An Underrecognized Clinical Entity</i>	For patients suffering from cholinergic urticaria, symptoms may develop following stress, anxiety, or embarrassment.
[24]	Kotera A	<i>General anesthetic management in two patients with an anaphylaxis history cholinergic urticaria</i>	Cholinergic urticaria can be triggered by psychological stress, including surgery-related stress (such as anxiety, pain, and nausea).
[25]	Fukunaga A, et al.	<i>Cholinergic Urticaria: Subtype Classification and Clinical Approach</i>	Cholinergic urticaria negatively impacts daily functioning, work, and learning.

DISCUSSION

Psychosomatic aspect

The primary cutaneous manifestation of urticaria is a transient wheal, accompanied by an unpleasant, persistent sensation of pruritus. Prolonged scratching leads to the formation of more visible and persistent skin lesions: erosions and excoriations [3]. Persistent pruritus compels the patient to scratch constantly, leading to sleep disturbances, impaired concentration, and fatigue [4]. In the pediatric population, skin lesions frequently stigmatize patients, diminish their self-esteem and impair interpersonal relationships [5]. Children, particularly girls with skin lesions, are often victims of school bullying [6,7]. A range of psychiatric comorbidities has been described in other skin diseases, such as atopic dermatitis, hidradenitis suppurativa, acne vulgaris, vitiligo, and psoriasis. These include anxiety disorders (including separation anxiety), phobias, depressive disorders, and even suicidal ideation [8,9]. Such disorders can also occur in patients with urticaria. Furthermore, no correlation has been demonstrated between the severity of urticaria and the

degree of psychological impairment in children [9]. It remains an undisputed fact that urticaria significantly reduces the quality of life. Patients affected by this condition often require psychosocial support and lifestyle education [10].

Molecular aspect

Mast cells are part of immune system that play a key role in urticaria. They are located near cutaneous sensory nerve endings, where they interact via the CADM1 protein. Their presence has also been confirmed in the central nervous system (CNS), particularly in the hippocampus and spinal cord, as they are capable of crossing the blood-brain barrier. Research has shown that mast cells can exacerbate neuropathic pain by both modulating nociceptors and enhancing the transmission of C-fibers responsible for pain stimuli. Furthermore, they release cytokines that increase vascular permeability, including the blood-brain barrier, triggering localized neuroinflammation within the CNS [11,12,13].

The complex interactions between neurons, mastocytes, and skin cells is not yet fully understood, and it remains unclear whether stress acts as a cause or a consequence of urticaria [14]. It is known, however, that patients under chronic stress experience a more severe and treatment-resistant disease course, likely driven by the secretion of immunomodulating neuropeptides that intensify neuroinflammation [15].

Similar, yet more extensively studied, mechanisms have been described in atopic dermatitis (AD). Skin pruritus in AD is a much more molecularly complex phenomenon; beyond histamine, it involves numerous interleukins, cytokines, leukotrienes, neuromediators and opioids. This creates a vicious cycle: itching leads to scratching, which damages the skin barrier and triggers the release of pro-inflammatory cytokines and neuromediators. These substances only make the itching worse. Instead of bringing relief, scratching actually intensifies the sensation, while altered signaling in the spinal cord helps the condition become chronic. Eventually, the imbalance of these mediators and constant nerve stimulation leads to neuroinflammation [16]. This process has been tied to various behavioral, emotional, and cognitive issues in children, such as ADHD [17,18]. While it is well-established that neuroinflammation in adults is linked to depression and anxiety, we still lack enough similar studies focusing specifically on children [19,20].

Urticaria as a manifestation of stress

Stress itself may act as a trigger for urticaria. This is observed in adrenergic urticaria, in which beta-blockers are used therapeutically. It is often accompanied by symptoms of catecholamine release, such as tachycardia, elevated blood pressure, and wheezing. The skin lesions in this type of urticaria are surrounded by a pale halo caused by vasoconstriction and are short-lived, typically lasting from a few minutes up to half an hour. They may be triggered by emotional stress, but also

by physical trauma, hot baths, physical exertion, or coffee consumption. The exact cause of this phenomenon has not yet been fully elucidated. According to one hypothesis, stress induces the release of catecholamines from the adrenal glands, which leads to mast cell degranulation and vasoconstriction in the skin [21,22].

Another type of stress-related urticaria is cholinergic urticaria, which may be triggered by elevated temperature, physical exertion, as well as stress and strong emotions. It can also be induced by the intradermal administration of acetylcholine, from which it derives its name [23,24]. It is characterized by the presence of small urticarial wheals associated with sweating. Its pathomechanism has not yet been fully clarified. However, it is known to be related to an abnormal response of the sweat glands to increased body temperature, with stress being one such trigger. Several hypotheses have been proposed to explain this phenomenon, including the presence of antibodies directed against sweat glands, obstruction of sweat ducts, damage to sweat glands with leakage of sweat into the dermis, or even hypersensitivity to sweat itself [25]. As outlined above, adrenergic and cholinergic urticaria represent clear evidence of the close interplay between psychological factors and skin condition. Due to their similar clinical presentation and a shared triggering factor, namely intense stress, their differentiation often poses a diagnostic challenge. However, both conditions clearly demonstrate that the same psychological stimulus can induce cutaneous reactions through distinct neurobiological pathways [25]. The comparison below summarizes the main pathophysiological and clinical differences (Table II).

Table II. Differential characteristics of urticaria associated with psychogenic factors and stress

Characteristic	Adrenergic urticaria	Cholinergic urticaria
Trigger factor	Stress, as well as a hot bath, injury, physical exercise, coffee, ginger, chocolate, aubergine, spicy foods, and tea.	High air temperature, strenuous physical activity, stress, emotionally distressing events, a hot, spicy meal.
Proposed pathophysiology	Stress stimulates activation of the locus coeruleus in the brainstem and the release of noradrenaline, which activates the beta-1-adrenergic receptors in the amygdala. This leads to adrenergic stimulation of the adrenal medulla, the release of, and an increase in, the levels of adrenaline and noradrenaline in the blood, which activate and cause degranulation of the skin's mast cells.	Several theories have been proposed 1. An immediate-type allergy to autologous antigens contained in sweat, e.g. the MGL_1304 protein from <i>Malassezia globosa</i> 2. Acetylcholine stimulates mast cells around the hair follicle 3. Penetration of sweat into the dermis and IgE-mediated mast cell activation 4. An-/hypohidrosis with reduced expression of the CHRM3 receptor in sweat glands, leading

		to degranulation of neighbouring mast cells by acetylcholine.
Symptoms	Tachycardia, tachypnoea, elevated blood pressure, paraesthesia, wheezing.	Dysphagia, dysphonia, stridor, wheezing, cough, chest tightness, shortness of breath, nausea, vomiting, colicky abdominal pain, diarrhoea, light-headedness, fainting, palpitations, symptoms of anaphylaxis.
Skin lesions	Transient, scattered skin lesions: hives, papules, macules and plaques surrounded by a white halo.	Small urticarial wheals and papules associated with sweating, surrounded by erythema, accompanied by intense pruritus.
Duration	A few minutes - half an hour	Symptoms usually begin within a few minutes to 20 minutes and typically subside after 1 hour.
Diagnostics	An intradermal injection of 5-15 ng of adrenaline or 3-10 ng of noradrenaline in 0.02 ml of saline solution.	The appearance of small urticarial wheals within 10 minutes of spending 15 minutes in warm water (42 °C) (such that the body temperature rises by ≥ 1 °C above the baseline temperature).
Therapy	Beta-blockers (propranolol 10-60 mg orally, 2-3 times daily)	Antihistamines, followed by steroids, danazol, scopolamine, a combination of propranolol, antihistamines and montelukast, self-administered desensitisation therapy for sweating, and regular physical activity.

CONCLUSIONS

Urticaria, although classified as a dermatological condition, significantly impacts the daily functioning of patients and their families. The pathomechanism of this disorder is highly complex and warrants further investigation. The fact that stress acts as both a trigger and a consequence of urticaria creates a vicious cycle of cutaneous lesions to induce severe psychological distress, which in turn exacerbates the clinical course of the disease. Breaking this cycle requires a holistic approach. Conventional therapies, such as standard antihistamines or glucocorticoids, are not always sufficient. In certain cases, personalized treatment and psychotherapy are essential. Patients with chronic urticaria, particularly pediatric patients, require comprehensive care from a multidisciplinary team.

Use of AI tools statement

The Gemini AI tool was utilized for grammatical correction, and stylistic optimization to ensure the text meets professional and formal communication standards.

Authors' contribution

Study design – A. Borek, G. Brożek

Data collection – P. Szastok, M. Tarsa

Manuscript preparation – M. Farnik

Literature research – A. Pawlik, F. Drzymala

Final approval of the version to be published – A. Borek, P. Szastok, M. Tarsa, A. Pawlik, M. Farnik, F. Drzymala, G. Brożek

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