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Neuro-Immuno-Psychological Aspects of Chronic Urticaria

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Abstract

Urticaria is a condition that causes itchy wheals, angioedema, or both. The pathophysiology of chronic spontaneous urticaria is still unknown. It is proposed that there is no dominant and independent mechanism of CSU; however, there are various immunological and non-immunological abnormalities that act concurrently or/and follow each other, resulting in clinical symptoms. According to the most recent hypothesis, mast cells (MCs) are activated *via* autoantibodies in an autoallergic or autoimmune mechanism, and mediators released from degranulated MCs are responsible for the vasoactive and neurospecific effect in CSU. Many clinical observations suggest that psychological stress can be both a triggering factor in the onset of CSU and a modulating factor in the course of the disease and therapy effectiveness.

Keywords: Urticaria • Psychological stress • Neuroimmunology • Mast cells

Introduction

It is worth noting, however, that a number of inflammatory mediators, neuropeptides, and neurotransmitters facilitate this phenomenon. This review summarises recent research on the neuro-immuno-psychological aspects of CSU, emphasising the emerging role of neuro-immune interactions. It also emphasises the utility of psychological tools in determining the baseline level of perceived stress and the presence of its symptoms. It also suggests the use of non-invasive interventions to reduce psychological stress and anxiety. For CSU, a bio-psycho-social approach that includes psychological support and patient education appears to be just as important as traditional pharmacotherapy. It allows for more effective active disease control and a longer period of remission in this disease. Chronic urticaria is a condition characterised by the recurrence of itchy wheals (hives), angioedema, or both, for more than six weeks. According to various reports, the lifetime prevalence of all types of urticaria is typically less than 10%, whereas CU develops in only about one-fourth of these people. One-third of all CU patients have both hives and angioedema, 30-40% have isolated hives, and approximately 10% have isolated angioedema. The disease's natural history is extremely diverse. Approximately half of the patients experience a three-month self-limited evolution, and the disease resolves in nearly 80% of them within a year. However, more than 10% of patients are expected to have a disease that lasts 5 years or longer.

Literature Review

The pathomechanism of chronic spontaneous urticaria is unknown. It has been proposed that there is no dominant and independent mechanism of CSU; however, there are various immunological and non-immunological abnormalities that act mutually or/and follow each other, resulting in CSU clinical symptoms. Mast cells and mediators released from these cells are undeniably important players in the development of all types of urticaria. Antihistamines remain the first-line therapy in CSU, but they are not equally effective in all cases. Following the appearance of a wheal, light and electron microscopy revealed degranulated MCs in the dermis. According to some reports, CSU and other types of urticaria cause an increase in the number of skin MCs in both lesional and non-lesional skin.

Given the diverse repertoire of MC receptors, it is important to remember that FcRI-mediated stimulation is not the only possible mechanism of MC activation. MC receptors specific to neuropeptides, neurotransmitters, and hormones are particularly important in the stress-induced skin reaction. Other than histamine, there is a growing body of research on the potential role of other factors in the CSU pathological cascade reaction, i.e., mediators other than histamine recruited inflammatory cells and skin-related cells, particularly keratinocytes as a source of proinflammatory cytokines and coagulation system activation. CSU's genetic background was also confirmed. Surprisingly, the urticarial lesion was reported to be the COVID-19's second most common skin-related symptom.

Discussion

The association with psychological stress suggests that the neuroendocrine system may play a role in the etiopathogenesis of urticaria. However, it is still unclear how psychological stress affects the skin immune system in CSU. Although the role of psychological factors in development and aggravation has not been conclusively established, the psychosocial impact of CSU is widely acknowledged. Chronic disease, prolonged treatment, and multiple consultations with different practitioners all contribute to a decline in psychological functioning. Urticaria patients have significantly higher levels of somatization, obsessive-compulsive disorder, interpersonal sensitivity, depression, anxiety, and stress. It is also common to find a link between the severity of the disease and poor psychological wellness or a lower quality of life.

Many clinical studies have found a link between the onset and exacerbation of inflammatory dermatoses and high levels of psychological stress. Increased psychological stress is linked to worsening and more frequent relapses of urticaria, regardless of the type. The authors present bidirectional interactions between psychological stress and chronic inflammatory skin diseases. Patients with CSU are especially vulnerable to stress-induced alternations because the triggering agent of the disease is unknown; thus, signs and symptoms appear unexpectedly and spontaneously during life activities, putting the patient under constant stress. For the patients, this situation creates a vicious circle [1-6].

Conclusion

This paper emphasises the complexities of stress and its relationship to MC activity. Stress affects not only the HPA axis and SAM, but also the gut-brain axis, which allows bidirectional communication between the central and enteric nervous systems, connecting the brain's emotional and cognitive centres with peripheral intestinal functions. Mast cells are important axes effectors. Although FcRI-dependent signalling is the primary pathway in MC activation,

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FcRI-independent mechanisms may also play a role in the CSU pathological cascade. The interaction between MCs and skin sensory nerve fibres as a source of many neuropeptides and neurotransmitters is of particular interest. A better understanding of MC stimulation *via* stress-related neurohormonal receptors may hold the key to a novel therapeutic approach to CSU.

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Conflict of Interest

There are no conflicts of interest by author.

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