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Clinical and immunological features of urticarial vasculitis

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Introduction: Urticarial vasculitis is an eruption of erythematous wheals that clinically resemble urticaria but histologically show changes of leukocytoclastic vasculitis that may be divided into normocomplementemic and hypocomplementemic variants [1, 2]. The exact frequency of urticarial vasculitis is not known in the United States or worldwide.The frequency of diagnosis is 45 cases per 1,000,000 population of new cases per year (2020). The male-to-female ratio for urticarial vasculitis is 1 : 2 and the median age of involvement is 43 years [2, 3]. Patients with hypocomplementemic urticarial vasculitis are more likely to show autoantibodies to C1q and vascular endothelial cells [4]. Clinically, urticarial vasculitis is manifested by rashes on the skin (dense blisters, papules, hemorrhagic rash, nodules, which are often layered) [5]. The reasons for the development are not clearly defined, however, one of the reasons of the disease development is hyper activity of the immune response. The population of so-called regulatory T cells with the CD4+CD25+ phenotype limits the intensity of any immune response and prevents the development of autoaggression [3]. Regulatory T cells do not decrease the normal immune response, but could suppress the immune reaction by the threat of massive self-harm. Another causes of urticarial vasculitis are infectious factors, especially Epstein-Barr virus (EBV) and human herpes virus type 6 (HHV6) that belong to the family of herpesviruses.

Material and methods: We have analyzed the immunological histories of 146 patients with systemic autoimmune diseases, including 24 (14 women and

10 men, aged 36 ±7 years) patients with urticarial vasculitis. The diagnosis was established according to the American College of Rheumatology (ACR, 2016). The diagnostic search involved the study of blood and saliva samples, which determined the viral load of EBV and HHV6 DNA by polymerase chain reaction (PCR). The activity of immune system/the T-lymphocytes phenotyping, e.g. T-regulatory cells/ was performed using flow cytometry.

Results: Among 24 patients with urticarial vasculitis, it was diagnosed clinically active phase of the disease in 13 cases, and an inactive – in 11 patients. EBV and/or HHV6 DNA with a mean viral load of 16324 ±1423.1 copies/ml and 10114 ±1722.3 copies/ml, respectively, was detected in saliva and/ or blood in all 13 patients with active urticarial vasculitis. Between patients with severe urticarial vasculitis and DNA "+" EBV/HHV6 in the blood, the number of T-regulatory cells with phenotype CD45+CD4+CD25brightCD127neg was 3.9 ±1.2% and 7.3 ±2.1% in patients with DNA "+" EBV/ HHV6 in saliva ($p < 0.05$). The ratio of CD4+/CD8+ in patients with urticarial vasculitis and active EBV/HHV6 replication in the blood was higher (5.9 ±1.4%) compared to the patients with urticarial vasculitis and active EBV/ HHV6 replication in the saliva 2.95 ±0.4% ($p < 0.05$).

Conclusions: It was found that the viral load of EBV and HHV6 was significantly higher in patients with active urticarial vasculitis compared to clinically inactive course of the disease that may indicate a significant role of herpesviruses in the manifestation and exacerbation of this pathology. Significant reduction of the T-regulatory cells level in patients with active clinical and laboratory manifestations of urticarial vasculitis could help us to use this indicator as a biomarker of process activity. It may be helpful as significant target option for the treatment of patients with urticarial vasculitis.

References

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