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## Changes of cellular immunological parameters in patients with psoriasis and activated chronic herpes simplex virus infection during treatment

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**Introduction & Objectives:** The possible role of viral persistence as an epigenetic factor in the development of psoriasis is discussed when a specific antigen (virus, especially type 1,2 (HSV 1,2)) is considered as a trigger factor for direct or indirect action on immunocompetent cells.

**The purpose** To evaluate the peculiarities of blood lymphocytes phenotyping in patients with psoriasis and activated chronic Herpes simplex virus infection compared to patients with psoriasis, activated chronic Herpes virus infection, and healthy persons during treatment.

**Materials & Methods:** 120 patients with psoriasis and/or activated chronic, HSV types 1,2 were examed.

**Results:** In patients with psoriasis and activated HSV types 1,2, there was an increase in the number of T-lymphocytes and NK-lymphocytes, helper and cytotoxic subpopulations of T-lymphocytes in comparison with healthy persons. Also, in this group, the number of cytotoxic lymphocytes and T-helper cells was higher compared to patients with activated HSV 1,2. The functional capacity of T-lymphocytes and the regulatory activity of T-helpers were lower in patients with psoriasis and activated HSV 1,2 compared to healthy persons and patients with psoriasis only. As a result of the antiviral therapy (acyclovir, inosine pranobex) on the background of basic therapy in patients with psoriasis and HSV 1,2, it has been found a decrease in the number of cytotoxic lymphocytes, helpers, NK-cells. It has been found restoring the moderate correlation between the number of T-helper cells and cytotoxic lymphocytes in the studied group of patients after antiviral therapy (r = -0.51) and the number of B-lymphocytes and cytotoxic lymphocytes r = 0.41. A multifactorial relationship between the number of B- and T-lymphocytes and their activated population has been found. In the mild activated HSV 1,2 on the background of psoriasis, a decrease in T-helper cells and an increase in activated and regulatory lymphocytes, when using inosine pranobex as antiviral therapy, have been found. When using basic therapy in this group, there was a further decrease in the number of regulatory T-lymphocytes. In severe activated HSV 1,2, when using combined antiviral therapy with acyclovir and inosine pranobex in patients with psoriasis and HSV 1,2 activated, there have been detected a normalization of NK-cells and an increase of regulatory lymphocytes that were not found in the group which used basic therapy with acyclovir.

**Conclusion:** The phenotyping of lymphocytes in patients with psoriasis and activated HSV 1,2 was characterized by an increase of NK cells (CD16+56+, CD45+; p=0.0151), cytotoxic T-lymphocytes (CD3+CD8+, CD45+; p= 0.0019), T-helper (CD3+CD4 +, CD45+; p=0.0011) and decreasing activity of their regulatory subpopulation (CD4+, CD25+; p=0.0528) compared to patients with psoriasis. In patients with psoriasis and activated HSV 1,2, the multifactorial relationship between the number of CD19+, CD45+-lymphocytes, CD3+CD8+, CD45+-lymphocytes, and CD3+HLA+-lymphocytes has been detected. The antiviral treatment on the background of basic therapy caused a decrease in the number of T-helpers (p=0.0518), increased activity of T-lymphocytes (p=0.0251) and regulatory cells (p=0.0365) in mild HSV 1,2 using inosine pranobex in patients with psoriasis, and a decrease of NK-cells (p=0.0412) and increasing activity of regulatory lymphocytes (p=0.0351) in moderate and severe HSV 1,2 in patients with psoriasis using inosine pranobex and acyclovir.