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FEATURES OF THE CYTOKINE PROFILE IN PATIENTS WITH PRIMARY HIV INFECTION: A COMPARATIVE ANALYSIS WITH A CONTROL GROUP

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Abstract. The aim of this study was to assess serum cytokine profile parameters in individuals with primary HIV infection in comparison with healthy controls.

Materials and Methods. A total of 783 patients aged 21 to 60 years with newly diagnosed HIV infection were examined. The control group consisted of 20 healthy donors. Serum levels of IL-1β, IL-6, IL-4, and IL-10 were measured using enzyme-linked immunosorbent assay (ELISA). Statistical analysis included calculation of mean values, standard deviations, and significance testing ($p<0.05$).

Results. Patients with primary HIV infection showed significantly elevated levels of IL-1β (46.36±2.67 pg/mL vs. 8.06±1.23 pg/mL), IL-6 (10.20±0.76 vs. 6.80±1.06), IL-4 (36.33±1.88 vs. 5.85±0.99), and IL-10 (33.49±2.13 vs. 17.31±3.00) compared to the control group (all $p<0.05$).

Conclusion. Primary HIV infection is associated with activation of both pro-inflammatory and anti-inflammatory cytokines, reflecting systemic immune activation and dysregulation characteristic of the early stage of the disease.

Keywords: HIV, cytokines, IL-1β, IL-6, IL-4, IL-10, immune status, primary infection

Introduction. Human immunodeficiency virus (HIV) remains one of the most pressing global public health challenges. Following infection, the immune system undergoes rapid and extensive activation involving both innate and adaptive responses. Cytokines, as key mediators of intercellular communication, are central to the inflammatory response and play a crucial role in the pathogenesis of HIV infection [1,2]. Assessing changes in the cytokine profile during the early stages of HIV enables evaluation of immune system activation and the direction of the immune response — whether pro-inflammatory or regulatory. These parameters have prognostic significance and may serve as biomarkers for disease progression risk [3].

Materials and Methods. The study included 783 patients with newly diagnosed HIV infection, aged 21 to 60 years. Among them, 59.51 ± 1.75% (n=466) were male and 40.49 ± 1.75% (n=317) were female. Age distribution was as follows: 21–30 years: 11.24 ± 1.13%, 31–40 years: 39.08 ± 1.74%, 41–50 years: 33.46 ± 1.69%, Over 50 years: 16.22 ± 1.38%.

The control group consisted of 20 apparently healthy individuals without signs of viral or chronic inflammatory diseases.

Laboratory Analysis: Serum concentrations of IL-1β, IL-6, IL-4, and IL-10 were determined using solid-phase ELISA kits (Vector-Best, Novosibirsk, Russia).

Statistical processing was performed using standard software. Differences were considered statistically significant at $p<0.05$ based on Student's t-test.

Results. Significant differences in cytokine levels were found between HIV-infected individuals and the control group:

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Cytokine	Control (n=20)	HIV (n=90)	p-value	Change
IL-1β (pg/mL)	8,06 ± 1,23	46,36 ± 2,67	<0,001	↑ в 5,8 раза
IL-6 (pg/mL)	6,80 ± 1,06	10,20 ± 0,76	<0,001	↑ в 1,5 раза
IL-4 (pg/mL)	5,85 ± 0,99	36,33 ± 1,88	<0,001	↑ в 6,2 раза
IL-10 (pg/mL)	17,31 ± 3,00	33,49 ± 2,13	<0,001	↑ в 1,9 раза

The most pronounced increases were observed in IL-1β and IL-4, suggesting systemic inflammatory activation and a shift from Th1- to Th2-type immune response.
Discussion. Elevated IL-1β and IL-6 levels reflect robust activation of innate immunity, typical of the acute phase of HIV replication. IL-1β enhances the expression of adhesion molecules and chemokines, promoting leukocyte recruitment, while IL-6 further activates B cells and contributes to systemic inflammatory responses [4,5]. IL-4 is a key cytokine in Th2 responses, promoting humoral immunity and IgE class switching. Its marked elevation implies a skewing from Th1 to Th2 dominance, thereby weakening cellular immunity critical for viral control [6]. IL-10, an anti-inflammatory cytokine, inhibits pro-inflammatory cytokine production and downregulates antigen presentation. While early IL-10 elevation may limit immune hyperactivation, excessive levels are associated with T-cell and dendritic cell suppression, facilitating viral persistence [7].
Thus, the early cytokine response in HIV is characterized by a complex pattern of concurrent inflammation, immune suppression, and Th1/Th2 imbalance.
Conclusion. Primary HIV infection is associated with marked alterations in cytokine profiles, with increases in both pro-inflammatory (IL-1β, IL-6) and anti-inflammatory (IL-4, IL-10) mediators. These changes reflect pronounced immune activation and dysregulation, contributing to the pathogenesis of immune suppression. Cytokine profiling may serve as an additional tool for early diagnosis, monitoring, and prognosis of HIV infection.

Conflict of Interest: The authors declare no conflict of interest.
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