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PROFILE OF PRO-INFLAMMATORY INTERLEUKINS IN SYMPTOMATIC EPILEPSY OF VARIOUS ETIOLOGIES

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In recent years, various researchers have identified the relationship of epilepsy with immune disorders, which led to the determination of this pathology to the group of immunosaviable diseases with progredent current [1,2].

Thus, the role of inflammation mediators in the development and progression of epilepsy is actively studied. There are numerous experimental and clinical data on the role of pro-inflammatory factors in the pathogenesis of epilepsy, in particular, on the connection of increased levels of pro-inflammatory cytokines and acute phase proteins with the risk of convulsive seizures [3]. When analyzing the immunopathogenesis of neurogenic diseases it is necessary to understand the role of immune system factors in formation, development and progression of pathological processes. It is known that in various diseases of nervous system, on the one hand, destructive changes take place in brain tissues which are under immunological control, and on the other hand, nervous regulatory mechanisms are disturbed [4].

Among a wide range of studies in these diseases, priority may be given to studies of the cytokine profile with an assessment of the balance of pro- and anti-inflammatory cytokines, and it is important not only to measure serum content of cytokines but also to assess their productive activity. This makes it possible to assess the potential ability of the body to develop and progress the disease, i.e., may be a certain prognostic criterion of the course of the immunopathological process. The study of cytokines, especially their productive capacity, is an important criterion of non-specific immunity.

Objective: to determine the cytokine profile of spontaneous and induced production in patients with symptomatic epilepsy of different etiologies.

Research materials and methods: The research was carried out in the laboratory of immunopathology and immunopharmacology of the Republican Scientific Research Centre of the Ministry of Health of the Republic of Uzbekistan in 2016-2017. The level of spontaneous and induced products of pro-inflammatory cytokines and anti-inflammatory cytokines in plasma was determined by ELISA-method using commercial test-systems "Vector-Best" (Novosibirsk, Russia) on ELISA-analyzer "StatFax-2100". Blood was taken from the elbow vein in the amount of 5 ml and plasma was separated from the form elements.

We studied and analyzed the level of cytokines IL-1βTNF-α, IL-2 and IL-6 in 38 children of the study groups. The cytokines were determined in 10 (26.3%) patients with the consequences of neuroinfections, 8 (21.1%) with the consequences of Acute Brain Circulation Disturbance (ABCD), and 6 (15.8%) children with the consequences of perinatal CNS lesions, 5 (13.2%) patients with brain abnormalities and 9 (23.7%) patients with nonlesional epilepsy. The

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control group consisted of 8 children without chronic diseases or any acute somatic diseases in the last 2 months.

Cytokine levels in serum and plasma are known to reflect the current state of the immune system, while in situations where there is a shortage or imbalance of regulatory factors, the ability of blood cells to secrete cytokines must be assessed. At the same time, the spontaneous production indicates the extent to which cells are already activated invitro, mitogen-induced allows assessing the potential ability of cells to secrete cytokines. For these purposes, the "cytokine incentive" kit was used, which is a kit whose main components are the sterile medium and complex mitogen - a mixture of lyophilized polyclonal activators, which are used for induction of cytokines invitro. The sensitivity of the methods for cytokines is up to 1 pg/ml.

Written consent for the study was obtained from the patients' parents. Statistical processing of the obtained data was carried out by the SPSS Statistics 22 (IBM) software package,

The results of the research: Our studies have shown that the IL-1β both spontaneous and induced production is significantly reduced in all groups of children with different neurological nosologies. Thus, the lowest value of spontaneous production was revealed in the group of children with ABCD consequences (4.43±0.31 pkg/l), with brain development abnormalities (4.98±0.3 pkg/l) and in patients with nonlesional epilepsy (4.85±0.51 pkg/l) (Table 1).

Table 1.

Analysis of spontaneous and induced production of essential cytokines of the immune system in children with symptomatic infections.

			Lesional epilepsy			
	Control n=8	Non-leasional epilepsy n=9	Group 1 n=10	Group 2 n=6	Group 3 n=8	Group 4 n=5
Spont. IL-2	17,22±0,85	**8,43±1,34****	**10,79±1,3****	**7,73±0,94***	**7,7±1,1***	**12,8±0,74***
Induc. IL-2	24,4±2,65	**13,62±2,27^^**	**14,65±2,26^^**	**9,71±1,1****	**13,53±4,15^^**	19,96±1,2
Spont.IL-6	14,48±4,39	15,47±3,73	16,69±4,92	**43,2±12,65^^**	16,25±5,56	31,8±9,24
Induc. IL-6	195,25±0,58	**158,82±15,23^**	**145,4±12,7^**	**155,06±8,11***	**162,2±11,7^^**	**132,2±18,75****

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Spont.IL-1β	20,17±9,25	4,85±0,51	8,68±3,22	5,93±1,85	4,43±0,31	4,98±0,3
Induc. IL-1β	69,02±10,2	60,07±7,74	**41,78±6,11^^**	45,15±6,96	54,8±8,45	44,28±11,06
ΦНО-α	25,8±5,6	17,53±1,96	25,07±7,05	39,9±12,45	16,48±2,5	38,36±15,75

Note: significance level in relation to the control * - P=0,000; ** - P≤0,001; ^ - P≤0,005; ^ - P≤0,05;

Moreover, the spontaneous products of IL-1β were not reliably reduced in all groups compared to the reference values.

The analysis of induced IL-1β products revealed a reliably reduced production in vitro IL-1β in patients with the consequences of inflammatory brain disease, which indicates the exhaustion of potential reserves of innate immunity, which in turn may be manifested by the functional failure of the immune response. Thus, in patients of this group the indicators of induced IL-1β were 41.78±6.11 pkg/l (t=2.39; df=16; P=0.029). In other groups of patients, the reduced spontaneous production of IL-1β was unreliable.

Thus, in patients with nonlesional epilepsy the IL-2 spontaneous production index was 17.22±0.85 pkg/l (t=5.36; df=15); P=0,000), the most significant decrease in serum concentration of IL-2 was observed in patients in the groups of patients with the consequences of perinatal brain lesions (2nd group) and with the consequences of ABCD (3rd group) and made up 7,73±0,94 pkg/l (t=7,38; df=12; P=0,000) and 7,7±1,1 ng/l (t=6,7; df=14; P=0,000), respectively.

Given the above, it should be noted that the low values of IL-2 in spontaneous and induced products prove immunodeficiency in the specific link of immunity, which is expressed by the suppression of T-cell immunity.

Further, the values of TNF-α were analyzed, which showed that spontaneous production of it was not reliably reduced in groups of children with nonlesional epilepsy (t = 1.46; df=15; P = 0.166), and the effects of ABCD, both hemorrhagic and ischemic (t = 1.5; df=12; P = 0.162), with unreliable increases in children with the effects of Perinatal lesion of the central nervous system (PLCNS) (t = 1.13; df=12; P = 0.281) and brain abnormalities (t = 0.9; df=11; P=0,38). Elevated TNF-α values are known to indicate the extent of tissue damage against the background of the inflammatory response. As can be seen, from the statistical data processing data, the results obtained were unreliable compared to the control group.

The study of IL-6 level showed that its spontaneous production was increased in all groups of patients. Reliable increase of the spontaneous IL-6 content was noted in children with the consequences of cerebrospinal birth traumas (t=2.4; df=12; P=0.03) and the upward tendency in

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patients with brain development abnormalities (t=1.9; df=11; P=0.083), which also testified to the presence of a pronounced damage to brain tissue. Induced production of IL-6 showed that the production potential of IL-6 was reduced, which again indicates a cytokine deficiency. Thus, in patients with nonlesional epilepsy this index was 158.82±15.23 pkg/l (t=2.24; df=15; P=0.04), in patients with the consequences of inflammatory brain diseases it was 145.4±12.7 pkg/l (t=3.46; df=16); P=0,003), in patients with the consequences of cerebrospinal birth traumas of 155,06±8,11 pkg/l (t=5,77; df=12; P=0,000), in patients with the consequences of ABCD 162,2±11,7 pkg/l (t=2,8); df=14; P=0,014), in patients with brain development abnormalities 132,2±18,75 pkg/l (t=4,36; df=11; P=0,001) relative to the control group 195,25±0,58 pkg/l.

Moreover, the smallest value was found in the group of children with developmental abnormalities, which confirms congenital changes in cytokine regulation.

Analysis of the data for the presence of correlation between the level of interleukin content in blood, the severity of convulsive seizures according to NHS3 and ECHESS scales, seizure frequency and duration of the disease showed the following results. In patients with ***nonlesional epilepsy,*** a strong direct correlation between the content of spontaneous and induced IL-2 in the blood (r=0.73; P=0.026) and the content of induced IL-1β and IL-6 (r=0.8; P=0.009) was reliably determined.

The analysis of proinflammatory interleukins content in serum depending on seizure frequency showed that patients with daily seizures had the level of spontaneous IL-2 (t=2,8; P=0,018) and induced IL-6 (t=2,7); P=0,02) was authentically below the control level, in patients with weekly seizures there was an authentically decreased level of spontaneous IL-2 (t=9,5; P=0,000), induced by IL-2 (t=3,7; P=0,004), and an authentically decreased level of induced by IL-6 (t=4,37; P=0,001) in relation to the control group.

The analysis of interleukin content depending on the disease duration showed that the number of spontaneous IL-2 (t=2,89; P=0,018) in patients with "0-1 year" seizures was authentically decreased in relation to the control group. In patients with the attacks of "1-3 years" duration there was a reliable reduction of the content of induced IL-2 (t=3,4; P=0,016) and induced IL-6 (t=2,79; P=0,021) and made 11,03±2,88 pkg/l and 132,5±40,6 pkg/l against control, respectively, 24,4±2,6 pkg/l and 195,2 pkg/l. In patients with the "length of service" seizures of "3-5 years" there is a decrease of both spontaneous IL-2 (t=3.1; P=0.017) and induced IL-2 (t=27.3; P=0.000) relative to control. In the group of children with epileptic seizures more than 5 years' old there was also a significant decrease of spontaneous IL-2 (t=6,98; P=0,003) and induced IL-2 (t=3,5; P=0,007) relatively to control.

It should be noted that children with epileptic seizures of more than 1-year-old had an unreliable increase in the content of spontaneous IL-6 in relation to control. No statistically significant differences in interleukin levels depending on the duration of the disease were revealed.

In patients with the ***consequences of inflammatory brain diseases (group 1)***, reliable direct strong correlations were found between the IL-1β induced and the duration of seizures (r=0.634; P=0.049), spontaneous and induced IL-2 (r=0.785; P=0.007), and induced IL-6 and IL-1β (r=0.65; P=0.04). It should be noted that the content of induced IL-2 (t=3.05; P=0.019), induced IL-6 (t=6.37; P=0.000) and induced IL-1β (t=3.12; P=0.019) was significantly reduced concerning control. In patients with weekly seizures, there was a significant decrease in the

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spontaneous IL-2 (t=4.09; P=0.004) and induced IL-2 (t=4.07; P=0.002), and the content of induced IL-6 (t=5.3; P=0.000) relative to control.

The content of induced IL-6 (t=2,48; P=0,038) was also reduced in patients with daily seizures relatively to control.

At the same time, a reliable difference depending on the frequency of seizures within this group was determined only in the content of induced IL-1β, so in patients with weekly seizures, the content of induced IL-1β was 50.97±8 pkg/l, while in patients with daily seizures this index was 25.05±4.9 pkg/l (t=2.75; P=0.036). That is, the more often epileptic seizures were observed, the less IL-1β content was.

Also, there is a reliable higher content of induced IL-2 in children with the duration of attacks "3-5 years" - 37.7±0.1 pkg/l, compared to children with the duration of attacks "1-3 years". - 11.95±1.36 pkg/l (t=5.5; P=0.003), at the control level of 24.4±2.65 pkg/l.

In patients with the duration of attacks "0-1 year" there was a significant decrease in the content of induced IL-6 (t=3.08; P=0.013) in relation to control; in patients with the duration of attacks "1-3 years" there was a significant decrease in the level of spontaneous IL-2 (t=4.78; P=0.001), induced IL-2 (t=3.77; P=0.003) and induced IL-6 (t=4.9; P=0.000).

In patients with ***PLCNS (2nd group***), a reliable direct strong correlation was noted between the blood content of spontaneous IL-2 and induced IL-2 (r=0,887; P=0,018) and induced IL-1β and IL-6 (r=0,997; P=0,000). No significant correlations between seizure severity according to NHS3 and ECHESS scales and interleukin levels were determined.

Analysis of interleukin content dependence on seizure frequency showed a reliable increase of spontaneous IL-6 in patients with daily seizures (t=17.7; P=0.036) compared to monthly seizures. Thus, the level of spontaneous IL-2 (t=4,1; P=0,004) and induced IL-6 (t=27,78; P=0,000) was authentically lower than the control level of such parameters in patients with seizures 1-3 times per month. Patients with weekly seizures also showed a significant decrease in the level of spontaneous (t=6.35; P=0.004) and induced (t=3.6; P=0.005) IL-2, and a significant decrease in induced IL-6 (t=3.55; P=0.006). In patients with daily seizures, the level of spontaneous (t=3.54; P=0.005) and induced (t=2.1; P=0.06) IL-2 and induced IL-6 (t=5.85; P=0.000) was significantly decreased, while the level of spontaneous IL-6 (t=38.9; P=0.000) was significantly increased.

Analysis of interleukin content dependence on the duration of attacks showed that in patients the concentration of spontaneous (r=0,78; P=0,067) and induced (r=0,8; P=0,05) IL-2 directly correlated with the duration of the disease, that is, the longer the disease, the higher the IL-2 content in the blood, but nevertheless, significantly lower the level of control. In patients with the duration of seizures "0-1 year" the content of IL-2 both spontaneous (t=5,8; P=0,05) and induced (t=5,4; P=0,002) was authentically decreased for control, at the same time the content of spontaneous IL-6 was authentically increased for control (t=2,5; P=0,038).

In patients with the "experience" of attacks "1-3 years" a reliable decrease of spontaneous (t=6,8; P=0,000) and induced (t=9; P=0,014) IL-2 level and decrease of induced IL-6 (t=8,8; P=0,000) relative to control was also noted.

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In patients with the duration of attacks "more than 5 years" a reliable decrease of spontaneous IL-2 (t=2,5; P=0,039) and IL-6 (t=27,03; P=0,000) as well as a reliable increase of spontaneous IL-6 (t=4,2; P=0,004) and TNF-α (t=3,14; P=0,016) was registered. When comparing these indices, a reliable increase of induced IL-1β (t=12.09; P=0.007) and TNF-α was observed in patients with seizure duration "more than 5 years" in relation to patients with seizure duration "1-3 years".

In children with ***ABCD consequences (group 3)*** direct correlation relations were reliable between spontaneous IL-2 concentration, severity (r=0,78; P=0,038) and frequency of convulsive seizures (r=0,746; P=0,034), i.e., the more severe and more frequent the epileptic seizures are, the more inflammatory reaction is, but at the same time, the duration of the seizures leads to the exhaustion of potential immunity reserves. Strong direct correlation links are reliably noted between the severity of seizures and the content of spontaneous IL-6 in the blood (r=0.777; P=0.04). There is also a strong direct correlation between spontaneous and induced IL-2 (r=0.869; P=0.005) and induced IL-1β and IL-6 (r=0.751; P=0.032). The inverse strong correlation is reliably observed between induced IL-1β and spontaneous IL-2 (r=-0.8; P=0.017), as well as induced IL-2 and IL-1β (r=-0.90; P=0.002).

The analysis of interleukin levels in blood as a function of seizure frequency showed that the content of spontaneous IL-2 (t=8.5; P=0.001) and induced IL-2 (t=3.8; P=0.004), as well as induced IL-6 (t=3.6; P=0.006) relative to the control group, was significantly reduced in patients with epileptic seizures with frequency 1-3 times per month.

In patients with weekly seizures, the content of induced IL-6 (t=2.5; P=0.034) relative to the control group was significantly reduced. A reliable decrease of spontaneous IL-2 (t=3,6; P=0,034) and induced IL-2 (t=3,48; P=0,007) content relative to the control parameters was revealed in patients with daily seizures.

Analysis of the correlation between the level of pro-inflammatory interleukins showed that in patients with the duration of the disease "0-1 year" a reliable decrease in the blood content of spontaneous IL-2 (t=6,85; P=0,006) and induced IL-2 (t=7,47; P=0,000), as well as induced IL-1β (t=3,1; P=0,017), was revealed in comparison with the control group. In patients with the duration of attacks "1-3" years, there was also a significant decrease in the level of the above indicators - spontaneous IL-2 (t=10.2; P=0.001) and induced IL-2 (t=3.3; P=0.01), as well as induced IL-6 (t=2.57; P=0.033). In patients with the disease duration of "3-5 years" there was a similar tendency, i.e. a decrease of spontaneous IL-2 (t=6,36; P=0,036), induced IL-2 (t=6,03; P=0,000) and induced IL-6 (t=2,55; P=0,034) in relation to control. A significant reduction in induced IL-6 (t=2.56; P=0.033) was observed in patients with seizure periods longer than 5 years compared to the control group.

Patients with ***brain abnormalities (4 group)*** showed a direct strong correlation between spontaneous IL-1β and IL-2 (r = 0.897; P = 0.03) induced by IL-2 and IL-6 (r = 0.897; P = 0.03) as well as induced IL-1β and IL-6 (r = 0.911; P = 0.03), TNF-α and spontaneous IL-6 (r = 0.903; P = 0.03), inverse strong correlation between TNF-α and spontaneous IL-1β (r = -0.891; P=0,04). In all study groups, a direct strong correlation of spontaneous and induced IL-2 levels in blood was reliably established in patients, while in a group of children with brain abnormalities, this correlation was reversed, which confirms the depletion of the immune response of patients of this group.

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In patients with weekly seizures, there was a significant decrease in the level of induced IL-6 (t=2.58; P=0.03) relative to control, in patients with more frequent daily seizures there was a decrease in the content of spontaneous IL-2 (t=5.7; P=0.000) and induced IL-6 (t=7.8; P=0.000) relative to control group.

The level of some interleukins was also reduced depending on the duration of the disease, so the level of induced IL-6 (t=2,58) was significantly reduced in patients with the duration of the disease "1-3 years"; P=0,03) in relation to control, also its decrease was noted in the group of children with "3-5 years" and "more than 5 years" experience and made 135,2±0,01 pkg/l (t=34,4; P=0,000) and 118,75±26,45 pkg/l (t=2,5; P=0,04) in relation to control group 195,25±0,58 pkg/l, respectively.

In children with the disease duration of "3-5 years," there was also a reliable decrease of spontaneous IL-6 (t=2,87; P=0,02), and the level of TNF-α (t=2,5; P=0,04). In the group of patients with the duration of attacks "more than 5 years," there was a reliable decrease in the level of spontaneous IL-2 (t=5,24; P=0,004) and induced IL-1β (t=3,08; P=0,017) in relation to the control group. No statistically significant differences were found between these subgroups.

Discussion. In the pathogenesis of symptomatic epilepsy in children, a decrease in and/or oppression of the induction products of the main regulatory cytokines of the monocyte-macrophagal profile (TNF-α, IL-1β, IL-6) and adaptive immunity (IL-2) can be traced. Failure of congenital immunity factors in children was revealed, which was expressed in suppression of basic regulatory cytokines even with in vitro stimulation, which naturally contributed to suppression of IL-2, which is an essential cytokine of adaptive immunity. Therefore, the chronic course of the above neurological conditions, which is a manifestation of the established secondary immunological failure syndrome, is important and demonstrative. The revealed changes are important pathogenetic criteria of the course of the disease and its prognosis, which can become the basis of the pathogenetic interpretation of the disease and further target for the creation of targeted immunotherapy in neurological diseases.

D.A.Levit's scientific works with co-authors present 5 stages of development of the systemic inflammatory reaction [5,6]. The decrease in the content of pro-inflammatory cytokines in epilepsy can be explained by the fourth stage of the inflammatory reaction, i.e., development of compensatory anti-inflammatory reaction, and so-called "immune paralysis." The essence of this stage is that in response to the systemic inflammatory reaction there is an ejection of anti-inflammatory cytokines - IL-4 and IL-10, which inhibit the secretion of inflammatory mediators, such a phase of massive production of anti-inflammatory cytokines is called CARS syndrome (Compensatory Anti-inflammatory Response Syndrome). This syndrome significantly reduces the production of TNF-α and IL-6 monocytes in response to the heteration. According to the authors, the formation of this syndrome leads to immunodeficiency and further development of the infection process.

IL-2 stimulates the proliferation and differentiation of activated T-lymphocytes into effector cytotoxic T cells. The main producers of IL-2 are T-helpers. An important result of IL-2 action on resting or mitogenic-stimulated cells is to ensure their proliferation. The significant function of IL-2 is to control immune reactions and maintain immunological tolerance, and its absence leads to defective control of effector cells, which leads to autoimmune aggression. Research in recent years has shown that when IL-2 is insufficient, autoimmune T-lymphocytes are

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spontaneously formed in the immune system, having an amplified homing to the brain, where they interact with brain cells, violating their function. The deficiency of IL-2 leads to stimulation of proinflammatory cytokines production in the hippocampus [7], disrupts hippocampus architecture and is associated with behavioural disorders in adult mice [8]. In case of violation of IL-2 production only by brain cells (without deficiency of this cytokine in the immune system), there is a doubling of the number of T cells entering all brain areas.

Thus, although IL-2 is not currently characterized as a cytokine with anti or proconvulsant activity, studies of its action (or lack thereof) on the structure of certain brain regions have shown its important role in creating and maintaining normal hippocampal architecture. Thus, according to our study, it can be argued that the deficit of IL-2 causes the autoimmune process in the brain, actually in the hippocampus area and thus supporting the alteration of this area leads to the development of resistant forms of epilepsy. The advanced theory clearly proves the effectiveness of anti-inflammatory and immunomodulatory drugs (immunoglobulins, corticosteroids) in resistant forms of epilepsy [9].

TNF-α increases the production of IL-1β and IL-6, previously it was believed that TNF-α has a destructive effect on the brain [10, 11], but the knowledge accumulated in recent years suggests that TNF-α plays a significant role in the recovery of the nervous system [12,13].

It should be noted that the actions of IL-6 are extremely numerous: first of all, it is participation in the realization of inflammatory and immune reactions, hematopoiesis. Moreover, IL-6 contributes to both the exacerbation of chronic and chronic acute inflammatory processes. There is information in the literature that IL-6 deficiency causes a decrease in the level of neuroprotective antioxidants that contribute to the oxidative stress increase in inflammation [14].

Conclusions.

1. In the pathogenesis of symptomatic epilepsy in children a reliable decrease and/or oppression of both spontaneous and mitogen induced production of the main regulatory cytokines of the IL-1β monocyte-macrophagous profile in patients with the consequences of inflammatory brain diseases ($P<0,05$) is traced, which indicates the development of immunodeficiency status in this category of patients;

2. Reliable decrease of IL-2 production in all children of the study group confirms the exhaustion of adaptive immunity, which proves that the autoimmune process plays a vital role in epilepsy genetics.

3. The reliable increase ($P<0,05$) of IL-6 level in patients with the consequences of birth cerebrospinal traumas, testifies in favor of the fact that the main damaging factor in patients of this group was the fetal asphyxia during childbirth. A reliable decrease in mitogen induced by IL-6 production in all study groups proves the presence of the process of continued oxidative stress in patients.

4. The level of TNF-α in the study groups was ambiguous, tending to decrease in patients with NLE ($P=0,199$) and patients with ABCD consequences ($P=0,17$), at the same time an increase of its level was observed in patients with PCST ($P=0,28$) and brain abnormalities ($P=0,38$). The inaccuracy of the results can be explained with insufficient sample size and

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properties of the group of conditionally healthy children. We believe that in this case, we should expand the sample of study groups.

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