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THE INFLUENCE OF HERPES FAMILY VIRUSES ON THE COURSE OF NOVEL CORONAVIRUS INFECTION

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We have analyzed the impact of herpes virus infection on the course of a new coronavirus infection (NCVI). Infection of the examined contingent with herpes family viruses reached 95.3-100%. An association of NCVI with herpes simplex viruses 1, 2 types (HSV 1, 2 types) was found, but no correlation was found between the positivity coefficient (CP) of HSV 1, type 2 and the severity of NCVI. This can be explained by the fact that the sampling was carried out in the remote period after the transferred NKVI. Considering that both herpes viruses and the SARS-CoV-2 virus cause multiple organ damage and can aggravate each other, the study of co-infection seems to be very relevant.

Keywords: viruses of the herpes family (Herpes viridae), herpes simplex virus 1, 2 types (HSV types 1,2), cytomegalovirus (CMV), Epstein-Barr virus (EBV), new coronavirus infection (NCVI).

Introduction. Among the viruses of the herpes family, 8 of the most common are distinguished: herpes simplex virus types 1 and 2 (HSV-1 and HSV-2); shingles virus, or human herpesvirus type 3 (HHV-3); human herpes virus type 4, or Epstein-Barr virus (EBV); human herpes virus type 5, or cytomegalovirus (CMV); human herpes viruses type 6 and 7 (HHV-6 and HHV-7); and herpes virus type 8 or Kaposi's sarcoma-associated virus (HHV-8). The presence of herpes virus infection is a marker of immunodeficiency [1].

HSV-1 and HSV-2 are widespread human pathogens with worldwide prevalence rates of about 67% and 13%, respectively [20]. Diseases caused by HSV types 1, 2, including labial / nasal herpes, genital herpes, herpetic stromal keratitis, herpetic eczema, disseminated disease in newborns, meningitis and herpes simplex encephalitis. There is evidence that there is a direct link between the occurrence of HSV type 1 and type 2 infection and neurodegenerative diseases. [8, 23, 29, 32, 33].

Human cytomegalovirus (CMV) is widespread and affects 40–100% of the population worldwide [5]. Most infected people remain asymptomatic due to a rapid immune response [18]. Primary infection or reactivation viral can cause severe multiple organ damage in the presence of immunodeficiency [15].

More than 90% of the adult population is latently infected with oncogenic EBV[17, 26]. Chronic or recurrent EBV infection of epithelial cells is associated with systemic lupus erythematosus and Sjogren's syndrome. Chronic/recurrent infection of B cells is associated with rheumatoid arthritis, multiple sclerosis, and other diseases [22].

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a highly contagious and virulent virus, an inflammatory respiratory disease called novel coronavirus infection (NCVI, COVID-19), that is having a global impact on the global public health system[30]. According to WHO data, as of October 7, 2022, there were 617,597,680 confirmed cases of COVID-19 in the world, including 6,532,705 deaths [34]. Possible SARS-CoV-2-associated neurological diseases have been described: Guillain-Bare syndrome [13], encephalomyelitis [27], myalgia, and damage to the neuromuscular synapse [27].

The COVID-19 pandemic has caused numerous adverse consequences for people with neurological disorders and neurodegenerative diseases [25], such as Parkinson's disease, Alzheimer's disease, multiple sclerosis [2, 3, 6, 10, 12, 28].

Since reactivation of varicella-zoster virus, EBV, and CMV has already been reported in patients with COVID-19 [7, 14, 16, 19, 21], of particular interest is the study of the interaction of herpes family viruses and SARS-CoV-2 in terms of studying a possible mutually reinforcing pathogenic influence.

Purpose of the study: to assess the relationship between the carriage of herpes family viruses and a new coronavirus infection.

Tasks:

1. Analyze the carriage of herpesvirus infection in the examined contingent
2. To assess the degree of mutual influence of pathogens of herpesvirus infection and a new coronavirus infection in patients with confirmed COVID-19

Materials and methods. The object of the study were 175 people working and living in the cities Aldan and Tommot of the Aldan region, of which 66 (37.7%) men and 109 (62.3%) women. The study program for the adult population included the following sections: informed consent of the respondent to conduct research; blood donation (according to the protocol of the Ethics Committee of the YSC CMP); questionnaire survey to assess the objective state; blood sampling from the cubital vein in the morning on an empty stomach after a 12-hour abstinence from food for an immunological study.

The study protocol was approved at a meeting of the local committee on biomedical ethics of the Federal State Budgetary Scientific Institution "Yakutsk Scientific Center for Complex Medical Problems" (Minutes No. 56 dated March 10, 2022, decision No. 3).

The subjects were divided into two groups: group 1 (main) consisted of 132 people (132/175, 75.5%) who had undergone NCVI of varying severity (the severity was assessed on a specially developed scale; the form of the disease was taken into account according to hospital discharges and outpatient cards); group 2 (control) included 43 people (43/175, 24.5%) without a history of NCVI. In group 1 there were 49/132 men, 83/132 women; on a racial basis, the distribution

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was as follows: 118/132 people - Caucasians, 14/132 people - Asians; the average age was 43.4±11.3 years. In group 2 17/43 people - men, 26/43 people - women, racially 40/43 - Caucasians, 3/43 - Asians, the average age of the subjects in the control group was 45.3±11.6 years. Both groups were comparable in terms of gender and age characteristics.

Analysis of the level of immunoglobulin IgM and IgG (the analysis took into account PP - the coefficient of positivity) to HSV 1, type 2, CMV, EBV, SARS-CoV-19 was carried out in blood serum by enzyme-linked immunosorbent assay (ELISA) on a Uniplan photometer using standard kits "Vector best" (Russia), according to the manufacturer's instructions. In addition, the level of avidity of antibodies to antigens of HSV 1, type 2, CMV, EBV viruses was determined.

The obtained data were analyzed using the SPSS 22 program (IBM, USA). Descriptive statistics for quantitative data are given as the median and the 25th and 75th quartiles (Me [Q25; Q75]). To compare two independent groups, the analysis was carried out using the Mann-Whitney U-test, for three independent groups - using the Kruskal-Wallis test. When comparing qualitative data, $\chi 2$ and Fisher's exact test were used. The critical level of statistical significance for the two groups was determined at $p \leq 0.05$.

Research results. In 120/175 (68.6 ± 7.9%) of the examined, clinical manifestations of chronic herpes virus infection in history were detected, such as: labial, genital, herpes.

As a result of the study, it was found that IgG to HSV type 1.2 was found in 95.4% (125/132) in group 1 and in 100% (43/43) of the examined - in group 2 without statistically significant differences. The positivity coefficient (PC) in group 1 was 16.12 [15.91; 16.29], in group 2 - 16.06 [15.84; 16.23] c.u., without statistically significant differences between the groups. HSV-IgM was found in 2/132 (1.5%) of those examined in group 1 and in 1/43 (2.3%) in group 2 (Table 1). The median avidity of antibodies to type 2 HSV-1 antigens was 100.0 [97.0; 100.0] and 100.0 [96.5; 100.0] in the first and second groups, respectively (U = 2120, p=0.839).

CMV-IgG were detected in 100% in both examined groups (132/132 in group 1 and 43/43 in group 2). CMV-IgM in group 1 is positive in 5 (3.8%), in group 2 - in 4 (9.3%) people (table 1). The median avidity of antibodies to CMV antigens was 95.0 [82.0; 99.0] and 94.5 [77.5; 99.0] in the first and second groups, respectively (U = 1139.5; p=0.997).

EBV-IgM was positive in 7/132 people (5.7%) in group 1 and in 2/43 people (4.7%) in group 2. EBV-IgG was positive in 129/132 people (98.5%) in group 1 and in 41/43 people (95.3%) in group 2, without a statistically significant difference. The positivity ratio (CP) of EBV-IgG to the nuclear antigen in group 1 was 12.78 [11.93; 13.03], in group 2 - 12.76 [9.5; 13.0] c.u., without statistically significant differences between the groups (table 1). The median avidity of antibodies to EBV antigens was 99.67 [98.21; 100] and 99.32 [98.32; 100.0] in the first and second groups, respectively (U = 960; p=0.888).

Of the 132 people who recovered from the, 63/132 (47.7%) had a mild disease, 53/132 (40.2%) had a moderate degree, 12/132 (9.1%) had a severe degree, the severity of NCVI is unknown in 4/132 people (3.0%). On average, the period from the moment of NCVI was 6.97+6.5 months. 35/132 people (26.5%) had post-COVID syndrome. Of the main complaints in persons with post-covid syndrome, the following were noted: general weakness and increased fatigue - in 15/35 people (42.9%), violation of smell, taste, vision - in 10/35 people (28.6%), memory loss in 5/35 people. (14.3%), myalgia - in 4/35 people (11.4%).

We analyzed the relationship between class M and G antibody titers and the severity of NCVI (Table 2). The CP of antibodies to SARS-CoV-2 in mild infection was at the level of 11.64 [11.31; 11.82] c.u., with a moderate course - 11.66 [11.57; 11.83] c.u., in severe cases – 11.66 [11.13; 11.78] (p = 0.487, H = 1.44).

IgM titers to CMV did not differ in different degrees of severity of NCVI: 0.29 [0.21; 0.38] - with a mild degree, 0.29 [0.21; 0.46] - with an average degree, 0.24 [0.16; 0.29] – in severe course of the disease (p=0.187; H=4.81). The level of IgG to CMV also did not differ depending on the severity of NCVI: 10.93 [9.91; 11.03] with mild course, 10.78 [9.63; 10.96] with moderate and 10.86 [8.7; 11.1] in severe infection (p=0.472; H=2.52). IgM to EBV to the capsid antigen (VCA) in mild NCVI was 0.18 [0.16; 0.26], with moderate NCVI - 0.24 [0.16; 0.32], in severe cases - 0.21 [0.16; 0.29] (p=0.379; H=3.08). IgG to the EBV nucle-

Table 1

The level of antibodies to viruses of the Herpes family in two examined groups

	Main group, N = 132	Control group, N = 43	p-level
HSV-IgM Negative Positively Doubtful	123 (93.9) 2 (1.5) 6 (4.6)	40 (93.1) 1 (2.3) 2 (4.6)	p=0.933; χ
HSV-IgM, CP c.u.	0.32 [0.25; 0.43]	0.3 [0.23; 0.41]	p=0.47; U = 2528.5
HSV 1,2-IgG Negative Positively	6 (4.6) 125 (95.4)	0 43 (100)	p=0.16; χ
HSV 1,2-IgG, CP c.u.	16.12 [15.91; 16.29]	16.06 [15.84; 16.23]	p=0.47; U = 2528.5
Avidity HSV-1,2	100.0 [97.0; 100.0]	100.0 [96.5; 100.0]	p=0.839; U = 2120
CMV-IgM Negative Positively	126 (96.2) 5 (3.8)	39 (90.7) 4 (9.3)	p=0.15; χ
CMV-IgM CP c.u.	0.29 [0.21; 0.37]	0.33 [0.21; 0.66]	p=0.97; U = 2743.5
CMV-IgG Positively	131 (100)	43 (100)	-
CMV-IgG, CP c.u.	10.82 [8.97; 11.0]	10.75 [9.6; 11.0]	p=0.28; U = 2446
CMV avidity	95.0 [82.0; 99.0]	94.5 [77.5; 99.0]	U = 1139.5; p=0.997
EBV-IgG Negative Positively	2 (1.5) 129 (98.5)	2 (4.7) 41 (95.3)	p=0.23; χ
EBV-IgG to nuclear, CP c.u.	12.78 [11.93; 13.03]	12.76 [9.5; 13.0]	p=0.153; U = 2348
EBV-IgM negative Positively Doubtful	114 (93.5) 7 (5.7) 1 (0.8)	41 (95.3) 2 (4.7) 0	p=0.86; χ
EBV-IgM VCA, CP c.u.	0.21 [0.16; 0.29]	0.18 [0.13; 0.26]	p=0.11; U = 1807.5
Avidity VEB	99.67 [98.21; 100]	99.32 [98.32; 100.0]	U = 960; p=0.888

Table 2

Relationship between herpes family viruses and NCVI

	Mild disease of the NCVI	The average severity NCVI	Severe disease NCVI	p-level
HSV1,2 type IgM	0,3 [0,24; 0,42]	0,35 [0,28; 0,47]	0,3 [0,28; 0,43]	p=0,382; H = 3,06
HSV 1,2 type IgG	16,18 [15,96; 16,42]	16,07 [15,83; 16,23]	16,29 [16,12; 16,5]	p=0,025; H = 9,34 p1,2=0,021 U = 1296 p1,3=0,228 U = 276 p2,3=0,008 U = 142,5
CMV IgM	0,29 [0,21; 0,38]	0,29 [0,21; 0,46]	0,24 [0,16; 0,29]	p=0,187; H = 4,81
CMV IgG	10,93 [9,91; 11,03]	10,78 [9,63; 10,96]	10,86 [8,7; 11,1]	p=0,472; H = 2,52
EBV to nuclear IgG	12,91 [12,27; 13,03]	12,74 [9,19; 13,03]	12,86 [12,44; 13,24]	p=0,477; H = 2,49
EBV VCA IgM	0,18 [0,16; 0,26]	0,24 [0,16; 0,32]	0,21 [0,16; 0,29]	p=0,379; H = 3,08

ar antigen in mild NCVI was at the level of 12.91 [12.27; 13.03], with moderate severity - 12.74 [9.19; 13.03], with severe NCVI - 12.86 [12.44; 13.24] (p=0.477; H=2.49)

IgM to HSV 1, type 2 amounted to 0.3 in mild NKVI [0.24; 0.42], with a moderate course - 0.35 [0.28; 0.47], in severe cases - 0.3 [0.28; 0.43] (p=0.382; H=3.06).

The relationship between the titers of class G antibodies to HSV type 1,2 viruses and the severity of NKVI turned out to be statistically significant. Thus, in mild NKVI, the IgG level was 16.18 [15.96; 16.42] c.u., with an average severity of the disease - 16.07 [15.83; 16.23] c.u., in severe course - 16.29 [16.12; 16.5] (p=0.025; H=9.34). At the same time, he draws attention to the fact that the level of titers did not have a noticeable effect on the severity of the course of NCVI, but indicates the activity of herpes virus infection.

The influence of herpes viruses on the development of post-COVID syndrome was assessed. We did not find any statistically significant differences between patients with and without post-COVID syndrome.

Discussion. Thus, the infection of the examined contingent with herpes family viruses reached 100%: HSV 1,2-IgG was positive in 95.4% in group 1 and 100% in group 2; CMV-IgG were detected in 100% of cases in both groups; EBV-IgG was found in 98.5% of cases in the first group and in 95.3% in the second group. A high level of avidity of antibodies to antigens of herpes viruses indicates a long-standing infection process in the main and control groups.

When assessing the level of antibodies to herpes viruses at different degrees of severity of NCVI, a connection was established with HSV types 1, 2, however, we did not find a correlation between the level of CP HSV types 1, 2 and the severity of NCVI. This can be related to the fact that the sampling was carried out in the remote period after the transferred NCVI. In general, our results are consistent with previously published information on the relationship between NCVI and herpesvirus lesions.

So, in the study of Weber S. et al. (2022) found that patients under 60 years of age with severe COVID-19 had a very high prevalence of CMV seropositivity, while the distribution of CMV status in those with mild disease was similar to that in the German population. Predictive models support the hypothesis that CMV serostatus, unlike HSV, may be a strong biomarker for identifying younger individuals at higher risk of developing severe COVID-19, particularly in the absence of other comorbidities [9].

Katz J. et al. (2021) found that the prevalence of herpes simplex virus-1 in the COVID-19 patient population was 2.81% compared to 0.77% in the hospital population with an odds ratio of 5.27 (adjusted for sex, race and age odds were 5.18, 4.48, and 4.61, respectively); the prevalence of varicella zoster virus in patients with COVID-19 was 1.8% compared to 0.43% among inpatients, odds ratios of 5.26 before adjustment and 5.2, 5.47 and 4.76 after adjusting for sex, age and race, respectively [24].

Some studies have reported skin manifestations of COVID-19, such as rashes, petechiae, and urticaria, which are non-specific and may be associated with herpes viruses rather than the SARS-COV-2 strain [4, 19]. Shingles virus may even be an indicator of latent COVID-19 infection [19]. Reactivation of herpes viruses due to an immunosuppressive condition associated with COVID-19 can be potentially life-threatening [11]. Therefore, a number of authors recommend studying the prevalence of these cases and taking them into account in the differential diagnosis of human herpes viruses, even if COVID-19 is confirmed [24]. The prevalence of both HSV-1 and zoster virus was significantly higher in the COVID-19 group [31]. It can be assumed that COVID-19 lowers the threshold for reactivation of herpes viruses, which requires further research in this direction.

Conclusion. Thus, the overlaying of herpesvirus infection on NKVI can probably contribute to the aggravation of the course of COVID-19. It should be remembered that both herpes viruses and SARS-Cov-2 lead to multiple organ damage, as well as to the development of neurodegeneration. Considering that severe forms of herpetic lesions are similar to the COVID-19 clinic, it is important in such cases to include studies on the herpes viral load in the differential diagnosis. Of particular interest is the study of co-infection with herpes viruses and SARS-Cov-2 in the long term, since there is an assumption that herpes viruses lead to the development of latency of the NKVI pathogen. In particular, this is important in assessing the risks of developing neurodegenerative diseases.

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LIPID IMBALANCE IN YAKUTSK RESIDENTS WHEN INFECTED WITH THE SARS-COV-2 VIRUS AND IN THE POST-COVID PERIOD

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A study of lipid metabolism was carried out in 161 residents of Yakutsk aged 20 to 72 years who had a disease with a new coronavirus infection SARS-CoV-2 from 3 to 12 months ago. The aim of the work was to assess the lipid profile after recovery from COVID-19, depending on the post-COVID period and severity of the disease, as well as the level of total cholesterol in inpatients at admission, discharge and in the post-COVID period. According to the results of the study, when infected with the SARS-CoV-2 virus, lipid metabolism is disturbed: in the acute period of infection, the level of total cholesterol decreases, in the post-COVID period, its level significantly increases. The most pronounced shift in the lipid profile towards atherogenicity occurs in patients who recovered from COVID-19 three months ago due to a decrease in the antiatherogenic fraction of lipids. Dyslipidemia is most pronounced in patients who have had an infection with severe lung damage (50-75%) due to an increase in atherogenic lipid fractions that create a risk of atherosclerosis.

Keywords. SARS-CoV-2, COVID-19, lipids, dyslipidemia.