

## **CLINICAL FEATURES OF POSTCOVID SYNDROME**

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### **Abstract**

There is no common understanding of the clinical picture of post-ovoid syndrome. The American regulator CDC suggests highlighting (A) persistent symptoms and conditions that begin during acute COVID-19 disease; (B) first-time late complications after an asymptomatic disease or a period of acute symptom relief or remission; (C) the evolution of symptoms and conditions that include some persistent symptoms (for example, shortness of breath) with the addition of new symptoms or conditions over time (e.g. cognitive difficulties). Some manifestations may have similarities with other post-viral syndromes, such as myalgic encephalomyelitis/chronic fatigue syndrome, dysautonomia (for example, postural orthostatic tachycardia syndrome) or mast cell activation syndrome.

**Key words:** *post-covid syndrome, course, symptoms, signs, observation.*

Postcovid syndrome is a long—lasting (for 3 months or more) pathological manifestations after an acute period of the disease. The main signs include pronounced weakness, heaviness in the chest, a feeling of incomplete inhalation, headache, joint and muscle pain, sleep disorders, depression, decreased cognitive functions, thermoregulation disorder, etc. The mechanism of postcovid syndrome may be associated with the emerging chronic inflammation of blood vessels, which negatively affects the work of the nervous system in the first place, as well as disrupts the work of internal organs. SARS-CoV-2 destroys the inner surface of blood vessels, which increases the risk of developing microthrombs in the microcirculatory bed. COVID-19 can also have a direct destructive effect on body cells, as well as cause an excessive immune response and provoke autoimmune diseases.

The aim of the study was to study the features of cerebral hemodynamics and microhemocirculation in patients with postcovid syndrome.

Materials and methods of research. The study included 100 young and middle-aged people from 30 to 55 years old, with a verified diagnosis of the effects of COVID-19 coronavirus infection, confirmed by laboratory research methods and after negative results (PCR, ELISA) on SARS-CoV-2, 15 to 35 weeks after the onset of the disease, who made up the main group. The patients of the main group were divided into subgroup I – 70 people who suffered from COVID-19 disease in mild form and subgroup II- 30 patients who suffered from moderate to severe disease complicated by pneumonia. The control group consisted of 20 healthy subjects of the appropriate gender and age composition who had not suffered from coronavirus infection or

other viral infection over the past 6 months and did not have acute and decompensated chronic pathology at the time of observation.

The results of the study and their discussion.

The study of cerebral blood flow in patients with postcovid syndrome at rest revealed statistically significant differences in Vmax (maximum systolic velocity) blood flow through the main extracranial vessels of the carotid artery system and intracranial vessels of the carotid and vertebral-basilar systems.

In a background study of cerebral hemodynamics in patients of subgroups 1 and 2, a multidirectional change in linear blood flow velocity (LSC) at the extracranial level was observed.

The resting blood flow rate along the common carotid artery (CCA) was significantly lower in patients of the first subgroup and was combined with venous dyshemia, relative to the control group ( $p < 0.001$ ). Thus, the blood flow along the right OCA in patients of subgroup 1 was  $86.95 \pm 1.95$  cm/s and  $91.1 \pm 3.16$  in the control group ( $p < 0.001$ ); along the left OCA, the blood flow rate in subgroup 1 was  $86.8 \pm 1.96$  cm/s and  $92.53 \pm 3.35$  cm/s in the control group ( $p < 0.001$ );

In patients of the second subgroup, there was an increase in the velocity indices for the OSA -  $95.4 \pm 1.54$  cm/s significantly relative to the indicator of the control group ( $p < 0.001$ ); for the left OSA, the blood flow rate in the second subgroup was  $95.1 \pm 1.81$  cm/s ( $p < 0.01$ ), with the control group.

Indicators of the linear velocity of blood flow in the internal carotid artery (ICA) had similar trends to the LSC for OCA. The blood flow rate was significantly lower in patients of subgroup 1, relative to the control group ( $p < 0.001$ ). Thus, the blood flow along the right ICA in patients of subgroup 1 was  $52.58 \pm 2.07$  cm/s and  $62.6 \pm 4.57$  in the control group ( $p < 0.001$ ); along the left ICA, the blood flow rate in subgroup 1 was  $52.75 \pm 2.2$  cm/s, and  $63.6 \pm 4.1$  cm/s in the control group ( $p < 0.001$ ); In patients of the second subgroup, an increase in the rate of ICA was observed -  $65.2 \pm 1.73$  cm/s ( $p < 0.01$ ) relative to the indicator of the control group; according to the left LSA, the blood flow rate in subgroup 2 was  $65.77 \pm 1.28$  cm/s, without significant differences with the control group.

When assessing the blood flow rate in the vertebral artery (PA), it was also significantly lower in patients from subgroup 1, relative to the control group. Thus, the blood flow along the right PA in patients of subgroup 1 was  $12.72 \pm 0.84$  cm/s ( $p < 0.001$ ) with the control group  $13.93 \pm 1.53$ ; along the left PA, the blood flow rate in subgroup 1 was  $12.74 \pm 0.84$  cm/s and  $14.40 \pm 1.28$  in the control group ( $p < 0.001$ ).

In patients with postcovid syndrome who had pneumonia in the acute period of the second subgroup, an increase in velocity parameters was observed on the right PA-  $13.17 \pm 1.06$  cm/s without significant differences with the control group; on the left PA, the blood flow rate in the 2nd subgroup was  $13.39 \pm 1.18$  cm/s without significant differences with the control group.

Resistance indices (RI) were significantly reduced in the subgroup of patients of the first subgroup in OSA and PA and amounted to  $0.69 \pm 0.01$  and  $0.63 \pm 0.01$ , respectively ( $p < 0.01$ ). In patients from the second subgroup, an increase in RI in PA was observed to  $0.67 \pm 0.01$  in the 2A subgroup, which was significantly higher than the control group ( $p < 0.01$ ).

During the study, a violation of vascular anatomy was noted in patients of the 2nd subgroup, with early formation of vascular disorders (C, S vessels), and the formation of initial signs of atherosclerotic lesion the vessel is a violation of differentiation into layers of the intima-media complex.

At the intracranial level, the linear velocity of blood flow in the medial cerebral artery (SMA) was estimated. A decrease in blood flow rates was revealed in both the group of patients of the first and second observation subgroups relative to the control group ( $p < 0.001$ ),

When assessing venous outflow in the observation groups, all patients showed signs of an increase in the velocity along the rectus sinus to  $50 \pm 0.8$  and  $57 \pm 0.6$  cm/s, respectively, reliable relative to the control group ( $p < 0.001$ ). According to the Rosenthal veins, the discharge acceleration is up to  $27 \pm 0.98$  and  $32 \pm 0.55$  cm/s, respectively ( $p < 0.001$ ). The veins of the vertebral plexuses were expanded to 5-6 mm, the discharge reached  $30 \pm 0.58$  cm/sec in both subgroups. In subgroup II, discharge along the medial cerebral vein was accelerated to  $20 \pm 0.78$  cm/sec, and in subgroup I to  $17.3 \pm 0.4$  cm/sec ( $p < 0.001$ ).

In the control group, the rate of discharge through venous vessels remained within the age limits.

Thus, the study of cerebral hemodynamics in patients of the studied groups showed a significant effect of the venous component on the autoregulation of cerebral blood flow. Thus, in patients who had a mild coronavirus infection, the velocity indices of the venous link had significantly high values with reduced velocity indices of arterial vessels. Thus, a decrease in the rates of linear blood flow through the main cerebral arteries, a decrease in the tone of arterial vessels, which is a compensatory reaction in case of difficulty in venous outflow, was revealed, however, under these conditions, perfusion cerebral insufficiency is possible, which leads to transient hypoxia of the brain and, as a result, impaired autoregulation of blood flow.

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