

treatment. Am J Med. 2019; 132 (1): 32-37. DOI: 10.1016

15. Fullam T, Statland J. Upper Motor Neuron Disorders: Primary Lateral Sclerosis, Upper Motor Neuron Dominant Amyotrophic Lateral Sclerosis, and Hereditary Spastic Paraplegia. Brain Sci. 2021 May 11; 11(5): 611. DOI: 10.3390/brainsci11050611.

16. Hudson A.J. Amyotrophic lateral sclerosis: Clinical evidence of differences pathogenesis and etiology. Amyotrophic lateral sclerosis: Concepts in Pathogenesis and Etiology. 1990; 108-143.

17. Liewluck T, David S Saperstein. Progressive Muscular Atrophy. Neurol Clin. 2015 Nov; 33(4): 761-73. DOI: 10.1016/j.ncl.2015.07.005.

18. Longinetti E, Fang F. Epidemiology of amyotrophic lateral sclerosis: an update of recent literature. Curr Opin Neurol. 2019; 32(5): 771-776. DOI:10.1097/WCO.000000000000730

19. Norris F, Shepherd R, Denys E, et al. Onset, natural history and outcome in idiopathic adult motor neuron disease. J Neurol Sci. 1993; 118(1): 48-55. DOI: 10.1016/0022-510x(93)90245-t.

20. Jeffrey M Statland, Richard J Barohn, April L McVey, et al. Patterns of Weakness, Clas-

DOI 10.25789/YMJ.2021.76.20

УДК 61.616-06

sification of Motor Neuron Disease, and Clinical Diagnosis of Sporadic Amyotrophic Lateral Sclerosis. Neurol Clin. 2015 Nov; 33(4): 735-48. DOI: 10.1016/j.ncl.2015.07.006. Epub 2015 Sep 8.

21. Mitsumoto H, Nagy PL, Gennings C, et al. Phenotypic and molecular analyses of primary lateral sclerosis. Neurol Genet. 2015; 1(1): 3. Published 2015 Apr 14. DOI: 10.1212/01. NXG.0000464294.88607.dd.

22. Statland JM, Barohn RJ, Dimachkie MM, et al. Primary Lateral Sclerosis. Neurol-Clin. 2015; 33(4): 749-760. DOI:10.1016/j. ncl.2015.07.007.

23. Fournier ChN, Murphy A, Loci L, et al. Primary Lateral Sclerosis and Early Upper Motor Neuron Disease: Characteristics of a Cross-Sectional Population. J Clin Neuromuscul Dis. - 2016 Mar; 17(3): 99-105. DOI: 10.1097/ CND.000000000000102.

24. Meyer T, Münch C, van Landeghem FK, et al. Progressive muscle atrophy. A rarely diagnosed variant of amyotrophic lateral sclerosis. Nervenarzt. 2007; 78(12): 1383-1388. DOI:10.1007/s00115-007-2288-y.

25. Ramanathan RS, Rana S. Demographics and clinical characteristics of primary lateral sclerosis: case series and a review of literature. Neurodegener Dis Manag. 2018; 8(1): 17-23. DOI:10.2217/nmt-2017-0051.

26. Kim WK, Liu X, Sandner J, et al. Study of 962 patients indicates progressive muscular atrophy is a form of ALS. Neurology. 2009; 73(20): 1686-1692. DOI: 10.1212/WNL.0b013e3181c-1dea3.

27. Swash M, Schwartz M. Neuromuscular diseases: a practical approach to diagnosis and management. Springler. New York. 1988; 456.

28. Gordon PH, Cheng B, Katz IB, et al. The natural history of primary lateral sclerosis. Neurology. 2006; 66(5): 647-653. DOI:10.1212/01. wnl.0000200962.94777.71.

29. Bogucki A, Pigońska J, Szadkowska I, et al. Unilateral progressive muscular atrophy with fast symptoms progression. NeurolNeurochir Pol. 2016; 50(1): 52-4. DOI: 10.1016.

30. Marin B, Boumédiene F, Logroscino G, et al. Variation in worldwide incidence of amyotrophic lateral sclerosis: a meta-analysis. Int J Epidemiol. 2017 Feb 1; 46(1): 57-74. doi: 10.1093/ije/dyw061. PMID: 27185810; PMCID: PMC5407171.

31. Wijesekera LC, Leigh PN. Amyotrophic lat

M.A. Varlamova, T.K. Davydova, L.D. Olesova, V.A. Makarova POST-COVID 19 SYNDROME OF CHRONIC FATIGUE AND EMOTIONAL DISORDERS IN RESIDENTS OF YAKUTSK

The analysis of chronic fatigue syndrome and the level of anxiety and depression in 161 patients aged 20 to 72 years was conducted who had an acute infection with COVID-19 from 3 to 12 months ago. Young and middle-aged women are more susceptible to COVID-19 viral pneumonia in a severe and critically severe form. Men are more severely affected by COVID-19 compared to women aged 32-51 and 61-70. Anxiety-depressive disorders and chronic fatigue syndrome can develop at any time in the post-ovarian period, from 3 months to 12 months.

Keywords: COVID-19, post-COVID 19 syndrome, anxiety-depressive syndrome, chronic fatigue syndrome, HADS scale.

Relevance: In March 2020, the World Health Organization (WHO) announced the global COVID-19 pandemic. Like any major epidemic outbreak, it has caused negative consequences for individuals and society as a whole, covering almost all aspects of life. Neurological disorders caused by human coronaviruses, including SARS-CoV-2, are attracting the attention of researchers.

Thus, chronic angioencephalopathy, structural epilepsy, parkinsonism, leukoencephalopathy, and other progressive forms of neurodegenerative and autoimmune pathology are long-term complications from the central and peripheral nervous system in persons who have undergone COVID-19 [2]. Neurological syndromes that develop during the acute period are also described. diseases and after, which last more than 12 weeks long-COVID hypostolic syndrome (PCS). PMS is included in the new edition of the International Classification of Diseases, revision 10, where it is designated as "post-COVID-19 condition" under the code U09.9 [1, 9].

E.M. Amenta et al. [12], classifying the manifestations of COVID-19, identified residual symptoms that persist after recovery from an acute infection, organ dysfunction that persists after initial recovery, and new symptoms or syndromes that develop after an initial asymptomatic or mild infection. The incidence of postcoid syndrome as a whole is 10-35%, while for hospitalized patients it can reach 85% [10]. The possibility of developing postcoid syndrome in patients with a mild form of the disease or asymptomatic course is very important, which must be taken into account when managing these patients [11]. The clinical picture of

postcoid syndrome is very diverse. Fatigue is the most common symptom after COVID-19, with an incidence of 17.5% to 72% among hospitalized patients, and duration in some cases exceeding 7 months. after the onset of the disease [13]. Up to 40% of patients hospitalized with COVID-19 within 2-4 months. after discharge, a decrease in exercise tolerance is noted [5]. These symptoms, as well as pain in joints and muscles for no apparent reason, headaches, decreased memory and concentration, insomnia, lack of feeling of rest after a full night's sleep, dizziness, can be attributed to chronic fatigue syndrome (CFS), which can develop after suffering viral infection [4]. Also, patients with postcoid syndrome may experience emotional disturbances such as anxiety and depression, which are detected in 40% of patients even after 6 months. after COVID-19 [1]. According to foreign and domestic studies, during the first wave of COVID-19, clinically completed anxiety and depressive disorders (TDR) were diagnosed in 20-40% of the population, in 20-35%

VARLAMOVA Marina Alekseevna – research associate, neurologist of the Clinic of YSC CMP, varlamova.m@yandex.ru; DAVYDOVA Tatyana Kimovna – Candidate of Medical Sciences, associate researcher, head of the lab.; OLESOVA Lyubov Dygynovna –Candidate of Biological Sciences, associate researcher, head of the lab.; MAKAROVA Victoria Alekseevna – chief physician of the Delta Clinic, Yakutsk.

of cases, clinically significant symptoms of post-traumatic stress disorder and an acute reaction to stress, disorders sleep patterns were found in almost 50% of the population [3].

There are a large number of theories explaining the pathogenesis of the development of postcoid disorders. Undoubtedly, one of the leading roles is played by respiratory failure with the development of hypoxia. Endothelial damage caused by either viral invasion or inflammation, increased blood clotting, a tendency to coagulopathy and thromboembolic complications, damage to the microvasculature are also associated with the development of postcoid disorders [8]. The virus can directly infect peripheral neurons or olfactory sensory neurons and thus use axonal transport to gain access to the central nervous system (CNS). The molecular mechanism underlying the cellular invasion of SARS-CoV-2 is related to its ability to selectively bind to receptors for angiotensin-converting enzyme 2. These receptors are highly expressed in both glial cells and neurons, making the CNS a potential target for SARS-CoV- 2 [7]. At the same time, experimental studies have shown a special vulnerability of the hippocampus with a large loss of neurons in the CA1 and CA3 regions, which may be directly related to cognitive impairment (CI) [15]. SARS-CoV-2 induces a significant immune response up to the development of cytokine storms, which have both acute and delayed effects on the central nervous system. Neuroinflammatory processes can cause changes in the metabolism of neurotransmitters, dysregulation of the hypothalamus-pituitary-adrenal axis, activating microglia, affecting neuroplasticity and causing structural and functional changes in the brain. It was suggested that proinflammatory cytokines are the basis for the disruption of all these systems, becoming the pathogenetic basis for the development of CI and affective disorders (AR) [14].

Persistent or re-emerging symptoms, functional disorders associated with COVID-19, in> 50% of cases, according to questionnaires and analysis of disease outcomes in hospitals and clinics, negatively affect the quality of life, mobility and independence of patients seeking medical care [2] ... The quality of life of patients who have undergone COVID-19 suffers primarily from chronic fatigue syndrome and disorders of the psycho-emotional sphere, and knowledge of not only the delayed long-term consequences of coronavirus infection is necessary to develop therapeutic strategies and prevent these complications.

The aim of the study is to study chronic fatigue syndrome and anxiety-depressive disorders in patients after suffering COVID-19 3-12 months after the disease and to identify their relationship with the degree of lung damage.

Materials and methods. The study was approved by the local committee on biomedical ethics at the Federal State Budgetary Scientific Institution YSC CMP N52 dated March 24, 2021 and was conducted subject to the voluntary informed consent of the participants.

Object of study. The study involved 161 people aged 20 to 72 years who had had COVID-19 from 3 to 12 months. ago, which were divided into 4 age groups: young age from 18-44 years old, average age from 45-59 years old, elderly from 60-74 years old, senile from 75-89 years old.

The materials of the study were medical records of patients with CT protocols of the lungs, questionnaires that included complaints of patients in the postcoid period and data of neurological status.

Inclusion criteria:

1. Patients who have undergone COVID-19, confirmed by medical records;

2. Patients in the postcoid period from 3 to 12 months or more;

3. The age of the patients is from 18 to 72 years;

4. Patients who voluntarily agreed to participate in this study.

Exclusion criteria:

1. Patients in the early skovidny period up to 3 months;

2. The age of the patients is less than 18 years;

3. Patients who refused to participate in this study.

Research methods.

1. The clinical method included the study of the demographic data of patients, age, duration of the disease and its clinical manifestations from the nervous system;

2. Method of questioning. The questionnaire included questions about fatigue during the day, disturbances in the rhythm of sleep, muscle pain, headache, chills, visual impairment, impaired sensitivity, impaired smell and taste, unsteadiness of gait, dizziness, memory impairment, blood pressure drops, gastrointestinal upset;

3. Research on hospital scale of anxiety and depression Hospital Anxiety and Depressoin Scale (HADS) on a point system (0-21 points). Patients with anxiety and depression were divided into 2 groups, respectively: subclinical severe anxiety / depression syndrome (from 8-10 points) and severe anxiety / depression syndrome (11 points or more) on the HADS scale.

4. Method of neuroimaging - computed tomography of the chest organs (CT); According to the CT lung protocols, the patients were divided according to the degree of lung involvement into V groups: Group I "CT-0" (zero) - no signs of viral pneumonia; II group "KT-1" (light) - the presence of a zone of compaction of the type of "frosted glass", the involvement of less than 25% of the lung volume; III group "KT-2" (moderate) - the presence of a zone of compaction of the type of "frosted glass", the involvement of 25 to 50% of the lung volume; IV "KT-3" (heavy) - zones of compaction of the "frosted glass" type, involving from 50 to 75% of the lung volume; V "CT-4" (critical) - diffuse compaction of the lung tissue like "ground glass" and consolidation in combination with reticular changes. Involvement of more than 75% of the lung volume.

5. Statistical research method: The accumulation, correction, systematization of the initial information and visualization of the results were carried out in Microsoft Office Excel 2016 spreadsheets. Statistical analysis was carried out using the STATISTICA 13.3 program (developed by StatSoft.Inc). Quantitative indicators were assessed for compliance with the normal distribution, for this, the Shapiro-Wilk test was used (with the number of subjects being less than 50) or the Kolmogorov-Smirnov test (with the number of subjects being more than 50). Aggregates of quantitative indicators, the distribution of which differed from normal, were described using the values of the median (Me) and the lower and upper quartiles (Q1-Q3). Ratings were described with absolute values and percentages. The Mann-Whitney U test was used to compare independent populations in cases where there were no signs of normal data distribution. When comparing several samples of quantitative data with a distribution other than normal, the Kruskal-Wallis test was used, which is a nonparametric alternative to one-way ANOVA. The comparison of nominal data was carried out using the Pearson x2 test, which allows us to assess the significance of differences between the actual number of outcomes or qualitative characteristics of the sample falling into each category and the theoretical number that can be expected in the studied groups if the null hypothesis is valid. The critical value of the significance level was taken equal to 0,05.



Results and discussion. All patients (n = 161) were divided into 4 groups according to the terms of the post-covid syndrome:

from 3 to 6 months; from 6 to 9 months; from 9-12 months; more than 12 months. 159 patients (98.7%) belonged to the Yakut ethnic group, 2 patients were Russians (1.3%). Of these women - 93 (57.5%), men - 68 (42.5%). Of these, 125 are employed (77.6%).

In this study, no statistically significant differences in gender were found among the examined patients. The median (Me) age in the general group of the surveyed was 53 years (42.0-61.0), while in men Me of age was 51 years (40.0-60.5), in women Me of age was 54 years (42, 0-62.0). Thus, men and women were comparable in age, which made it possible to carry out a comparative analysis of CT data by age in the general group (Table 1).

It follows from Table 1 that the mild form of COVID-19 (CT-1) was experienced by patients at a young and middle age by 42.86% (n = 48), respectively, in both groups compared with elderly patients, whose share was 25%. (n = 12). In old age, CT-1 was not registered. The largest proportion of patients with moderate viral pneumonia (CT-2) was in elderly patients, which amounted to 43.75% (n = 21) compared with other age groups. At the same time, severe COVID-19 pneumonia (CT-5) was experienced by young and middle-aged patients compared to elderly patients (p =, 026077).

We also conducted a separate study depending on the gender of the surveyed. In men, the highest median age was revealed at CT-4 and amounted to 67.0 (61.0; 70.0) years. At the same time, 50% of men in the CT scan group - 5 were under the age of 39.5 (32.0; 51.0). Thus, men aged 32 to 51 years and from 61 to 70 years in our study were most susceptible to the development of severe viral pneumonia (H = 14.65; p = 0.006).

When examining the relationship between lung lesions and age by CT scan groups among women, it was found that the highest median age was found at CT-3 and amounted to 61.5 (57.0; 70.0) years. The lowest median age was found at CT-4: 50% of women were younger than 43.5 (24.0; 63.0) years. At the same time, in the groups with CT-1 and CT-2, there were isolated cases of the disease over the age of 70 years. Half of the cases with CT-5 were women younger than 50 years. Thus, younger women were more likely to be affected by severe and critically severe COVID-19 viral pneumonia. And for an older age, mild and modDistribution by age group and CT scan group

		Total:				
age group	1	2	3	4	5	10(a).
18-44	24 42.86	9 16.07	9 16.07	1 1.79	13 23.21	56
45-59	24 42.86	11 19.64	9 16.07	1 1.79	11 19.64	56
60-74	12 25.00	21 43.75	8 16.67	4 8.33	3 6.25	48
75-89	0 0.00	1 100.00	0 0.00	0 0.00	0 0.00	1
Total:	60	42	26	6	27	161

Pearson Chi-square: 23,1998, df=12, *p*=,026077.

Table 2

Table 1

CT scan groups in men and women

floor		Total:				
	1	2	3	4	5	Total.
Men	19 27.94	15 22.06	20 29.41	4 5.88	10 14.71	68
Women	41 44.09	27 29.03	6 6.45	2 2.15	17 18.28	93
Total:	60	42	26	6	27	161

Pearson Chi-square: 18,0689, df=4, p=,001196.

erately severe forms of pneumonia are characteristic (H = 13.21; p = 0.01).

Table 2 shows that when comparing the CT groups, CT-1 is much more common in women: in 44.09% of cases, no signs of viral pneumonia were found. While in men the proportion of CT-1 was only 27.94%; CT - 3 was more common in men: 29.41% versus 6.49% in women. Thus, men were statistically significantly more severely affected by COVID-19 than women. The results obtained correlate with the data of other researchers [5].

The timing of the development of symptoms of CFS and TDR in the postcoid period. We investigated the following symptoms of CFS: fatigue, not dependent on physical activity, decreased performance, memory impairment, dizziness, pain in muscles and joints, unsteady gait, headache, sleep disturbance, swollen lymph nodes. When establishing the diagnosis of chronic fatique syndrome, the time period for the onset of symptoms of 3 months or more was taken into account, as well as the presence of at least four of the symptoms listed above, although some authors recommend using the main 3 criteria and an additional 1 criterion for CFS with a time period of 6 or more months when making a diagnosis. contract. [4]. TDD was determined using the HADS scale.

Emotional disorders of 161 patients with postcoid syndrome had 46 (27.9%) people, including 12 men (26.1%) and 34 women (73.9%). Depressive disorders were observed in 40 (24.8%) patients. At the same time, in 30 (18.6%) patients. subclinical severe depression was noted, in 10 (6.2%) patients - clinically significant. Anxiety disorders were detected in 39 (24.2%) patients. At the same time, 25 (15.5%) of the examined had subclinically expressed anxiety, and 14 (8.7%) had clinically significant anxiety. At the same time, the degree of depression and anxiety did not depend on the degree of lung damage (p = 0.6).

CFS was detected in 40 (24.8%) patients, including 18 (45%) men and 22 women (55%). As a result of the study, in the general group (n-161), in 46.5% (n = 75) of cases, the patients were not diagnosed with CFS and TDR in the postcoid period. Of these, 38 were women and 37 were men. In addition, this group included patients who noted feelings of anxiety and fear that appeared before the illness and were associated with the fear of COVID-19 disease by the patient and his close environment, as well as other reasons, although these patients have clinically significant anxiety and depression on a background of low mood dominated after suffering COVID-19. This group also included patients who were bothered by

complaints of headaches, fatigue, memory loss, sleep disturbance before the development of COVID-19, which were associated with various neurological manifestations of other diseases (hypertension, diabetes mellitus, brain trauma, cervical osteochondrosis) ... We carried out a separate study of the development of CFS and TDR, depending on the timing of the postform period.

When studying the timing of the development of symptoms of CFS and TDS in the postcoid period, no statistically significant results were obtained in terms of gender. In the rest of the subjects (n = 86), the symptoms of CFS and TDS were manifested at all periods of the postcoid period, while none of its periods prevailed over the others. We also studied the dependence of the symptoms of CFS and TDR on the degree of lung damage. At the same time, not a single symptom in CFS and TDR depended on CT data, except for a shaky gait. 92 patients complained of unsteadiness of gait. Stiffness in the postcoid period was more common in middle-aged women than in young and old age. It can be assumed that the wobbly gait in middle-aged women is associated with autonomic dysfunction of the nervous system caused by the transferred COVID-19, while in old age this symptom can be explained by concomitant vascular diseases, and at a young age by the absence of concomitant vascular diseases. Pearson Chi-square: 10.1883, df = 3, p =, 017032.

Conclusions:

1. Men aged 32 to 51 years and from 61 to 70 years in our study were most susceptible to the development of severe viral pneumonia.

2. In women with previous viral pneumonia of moderate severity, it was found in elderly patients compared with other age groups. At the same time, young and middle-aged patients suffered from COVID-19 viral pneumonia in a severe and critically severe form.

3. Men were statistically significant-

ly more severely affected by COVID-19 than women;

4. Anxiety-depressive disorders and chronic fatigue syndrome can develop at any time in the post-covid period from 3 months to 12 months.

5. Stiffness in the postcoid period was more common in middle-aged women than in young and old age.

This study was carried out as part of the initiative project of the Yakutsk Scientific Center for Complex Medical Problems "Comprehensive assessment of the health of patients who have undergone a new coronavirus infection (COVID-19)."

Reference

1. Возможные подходы к терапии астенических и когнитивных нарушений при постковидном синдроме / А. Н. Боголепова, Н. А. Осиновская, Е. А. Коваленко, Е. В. Махнович // Неврология, нейропсихиатрия, психосоматика. – 2021. – Т. 13. – № 4. – С. 88-93. – DOI 10.14412/2074-2711-2021-4-88-93. [Bogolepova AN, Osinovskaya NA, Kovalenko EA, Makhnovich EV. Possible approaches to the therapy of asthenic and cognitive disorders in postcoid syndrome. Nevrologiya, nejropsihiatriya, psihosomatika. 2021; 13 (4): 88-93. – DOI 10.14412 / 2074-2711-2021-4-88-93 (In Russ.).]

2. Постковидные неврологические синдромы / В.В. Белопасов, Е.Н. Журавлева, Н.П Нугманова [и др.]. - Клиническая практика. -2021. - Т. 12. - №2. - С. 69-82. doi: 10.17816/ clinpract71137 [Belopasov VV, Zhuravleva EN, Nugmanova NP, et al. Postcovid neurological syndromes. Klinicheskaya practika. 2021; 12 (2): 69-82. doi: 10.17816 / clinpract71137 (In Russ.).]

3. Психоэмоциональные расстройства и нарушения сна у пациентов с COVID-19 / М. А. Самушия, С.М. Крыжановский, А.А. Рагимова (и др.]. // Журнал неврологии и психиатрии им. С.С. Корсакова. – 2021. – Т. 121. - №4. – вып. 2. – С. 49-54 doi.org/10.17116/jnevro202112104249 [Samushia MA, Kryzhanovsky SM, Ragimov AA, et al. Psychoemotional disorders and sleep disorders in patients with COVID-19. Jhurnal Nevrologii i psykhiatrii im. S.S. Korsakova. 2021; 121 (4) 2: 49-54 (In Russ.).] doi.org/10.17116/jnevro202112104249

4. Путилина М.В. Астенические расстройства как проявление синдрома хронической усталости /// Журнал невропогии и психиатрии им. С.С. Корсакова. – 2021. – Т. 121. – №8. – С. 125-130 doi.org/10.17116/jnevro2021121081125 [Putilina MV. Asthenic disorders as a manifestation of chronic fatigue syndrome. Jhurnal nevrologii and psickiatrii im. S.S. Korsakov. 2021; 121(8): 125-130 (In Russ.).] doi.org/10.17116/ jnevro2021121081125

5. Davis HE, Assaf GS, McCorkell L, et al. Characterizing Long COVID in an International Cohort: 7 Months of Symptoms and Their Impact medRxiv preprint. doi: 10.1101/2020.12.24.20248802; this version posted April 5, 2021. Available from: https://www.medrxiv.org/con- tent/10.1101/2020. 12.24.20248802V3

6. Nekaeva ES, Bolshakova AE, Malysheva ES, et al. Characteristics of the Novel Coronavirus Infection (COVID-19) in Middle-Aged Adults Gender. Sovrem Tekhnologii Med. 2021; 13(4): 16-24 (In Russ.).] doi: 10.17691/stm2021.13.4.02.

7. Baig A, Khaleeq A, Ali U, et all. Evidence of the COVID-19 Virus Targeting the CNS: Tissue Distribution. Host-Virus Interaction, and Proposed Neurotropic Mechanisms. ACS Chem Neurosc. 2020 Apr 1; 11(7):995-8. doi: 10.1021/ acschemneuro.0c00122

8. Maltezou HC, Pavli A, Tsakris A. Post-COVID Syndrome: An Insight on Its Pathogenesis. Vaccines (Basel). 2021 May 12;9 (5):497. doi: 10.3390/vaccines9050497

9. Greenhalgh T, Knight M, A'Court M, et al. Management of post-acute COVID-19 in primary care. BMJ. 2020 Aug 11;370:m3026. doi: 10.1136/bmj.m3026

10. Pavli A, Theodoridou M, Maltezou HC. Post-COVID syndrome: Incidence, clinical spectrum, and challenges for primary healthcare professionals Arch Med Res. 2021 May 4. S0188-4409(21)00081-3. doi: 10.1016/j.arcmed.2021.03.010. Online ahead of print.

11. Goërtz YMJ, van Herck M, Delbressine JM, et al. Persistent symptoms 3 months after a SARS-CoV-2 infection: the post- COVID-19 syndrome? ERJ Open Res. 2020 Oct 26;6(4):00542-2020. doi: 10.1183/23120541.00542-2020. eCollection 2020 Oct.

12. Amenta EM, Spallone A, Rodriguez- Barradas MC, et al. Post-acute COVID-19: An overview and approach to classification. Open Forum Infect Dis. 2020 Oct 21;7(12):ofaa509. doi: 10.1093/ofid/ofaa509. eCollection 2020 Dec.

13. Halpin SJ, McIvor C, Whyatt EG, et al. Post-discharge symptoms and rehabilitation needs in survivors of COVID-19 infection: A cross-sectional evaluation . J Med Virol. 2021;93:1013-22. doi: 10.1002/jmv.26368

14. Rhie SJ, Jung EY, Shim I. The role of neuroinflammation on pathogenesis of affec- tive disorders. J Exerc Rehabil. 2020;16:2-9. doi: 10.12965/jer.2040016.008. eCollection 2020 Feb.

15. Ritchie K, Chan D, Watermeyer T. The cognitive consequences of the COVID-19 epidemic: collateral damage? Brain Commun. 2020 May 28;2(2):fcaa069. doi: 10.1093/brain- comms/ fcaa069. eCollection 2020.

