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Comparative analysis of psycho-cognitive status in elderly patients with comorbidities depending on the presence of post-COVID syndrome

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Abstract

Objective. To compare psycho-cognitive status of elderly patients with various comorbidities depending on the presence of post-covid syndrome.

Materials and methods. This observational analytical cohort study included 133 patients with atrial fibrillation and various comorbidities (coronary artery disease, arterial hypertension, obesity, type 2 diabetes mellitus (T2DM)) aged 60–72 years with and without history of SARS-CoV-2 infection and post-covid syndrome (PCS). Patients were divided into 2 groups: group 1 included 123 patients without the history of COVID-19, group 2 — 110 patients with the history of SARS-CoV-2 infection. All study participants underwent general clinical examination; assessment of psycho-cognitive status using the "SPMSQ" and "HADS" scales. Statistical analysis has been performed using RStudio software.

Results. Anxiety and depression have been established in 49–61% of patients with comorbid diseases and were more prevalent among patients after COVID-19. Subclinical anxiety was seen in 29% of patients without COVID-19 and in 27% of patients with PCS; clinically significant anxiety — in 13% of patients from both groups. The analysis of patients' cognitive functions showed that cognitive dysfunction was more prevalent among patients with PCS (p = 0,007); while the prevalence of mild cognitive impairment was comparable between groups, but was higher among patients with PCS – 22% vs. 8% (p = 0,005). Severe cognitive impairment was seen only in patients with PCS – 2%. The analysis of separate groups with various comorbidities showed significant differences in patients with T2DM, 51% in those without PCS compared with 28% among patients with PCS (p = 0,012).

Conclusion. The effects of COVID-19 remain uncertain. Therefore, the assessment of long-term consequences after the infection in patients with various comorbidities is required and can be achieved by reprofiling and initiation of large cohort studies aimed not only to assess long-term outcomes of SARS-CoV-2 infection, but also to investigate psycho-cognitive dysfunction.

Keywords: anxiety, depression, cognition, comorbid diseases, SARS-CoV-2, post-covid syndrome.

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Introduction

Nowadays the prevalence of psycho-cognitive disorders grows in general medical practice and especially among patients with various comorbid diseases. Neuropsychiatric disorders that manifest not only with anxiety or depression but also with cognitive impairment are of special importance since they can significantly aggravate life quality [1, 2]. It is known that viral infections can affect cognitive functions and lead to the development of dementia, and the current coronavirus infection affect both cognitive and psychological functions [3–5]. Exposure to powerful stressor in most cases is associated with higher depression and anxiety levels, both in the short and long term, that can not only impair life quality, but can also affect the prognosis. The increased stress may affect immune system and increase the risk of infectious diseases development, including the new coronavirus infection [6, 7]. According to current literature approximately 1/5 of patients experience cognitive impairment within 12 weeks after COVID-19, that is associated with gender (women), age, the severity of illness and the presence of comorbidities [8]. The study by Premraj L. et al., that included 15530 patients 3 months after COVID-19, have established cognitive impairment, decreased attention, "brain fog" in 1/3 of patients, anxiety and depression in 23% and 17%, respectively. Cognitive impairment did not depend on the course of post-COVID syndrome (cognitive deficit slightly increased rather than disappeared over time), while anxiety and depression were significantly more prevalent among those who recovered from infection more than 6 months ago [9, 10]. Several mechanisms of cognitive impairment are discussed in the literature [11]. MRI usually shows no signs of structural changes; however, positron emission tomography

(PET) reveals zones of decreased metabolism [12]. Neuropsychological changes manifest by impaired memory and ability to memorize [13, 14]. Several studies attempted to search for the evidence of direct effect of the SARS-CoV-2 virus on brain cells. The results of such studies [10, 11], which included patients with various disease severity, did not establish the presence of the virus in the brain tissue or cerebrospinal fluid (CSF) or indirect signs of viral infection. The revealed increase in the level of neurospecific proteins in the CSF of patients with COVID-19 may indicate brain cells damage, although it gives no clue as to the nature of the pathological process. At the same time, the data obtained may indicate that the COVID-19 may initiate mechanisms of delayed brain damage and cause neurodegenerative diseases [15, 16]. It is also considered that SARS-CoV-2 RNA remains in the brain tissue for a long time and, therefore, aggravates the loss of neurons over time [17, 18]. The relationship between inflammation markers and cognitive impairment in patients with post-COVID has been established [19]. Several studies established increased permeability of the blood-brain barrier that can prolong neuroinflammation and cause memory and attention impairment, as well as direct viral invasion into the central nervous system through the olfactory bulb [20–22]. It is also noteworthy that hypoxia caused by insufficient oxygen supply to the brain due to impaired lung function and endothelial dysfunction that occurred during COVID-19 may manifest as cognitive dysfunction in the long-term period [23–25]. The increase in the frequency and severity of psycho-cognitive disorders together with the limitation of its diagnosis highlights the importance of psycho-cognitive status assessment at early stages of the disease, including the assessment of anxiety and depression. This study aimed to assess mentioned above psychopathological symptoms.

Objective. To compare psycho-cognitive status in elderly patients with various comorbidities, depending on the presence of post-COVID syndrome.

Materials and methods

This observational analytical cohort study included 223 patients with atrial fibrillation and various comorbidities (coronary heart disease (CHD), arterial hypertension (AH), abdominal obesity (AO), type 2 diabetes mellitus (T2DM)) aged 60–74 years, with and without the history of documented SARS-CoV2 infection with post-COVID syndrome. Patients were divided into two groups: group 1 included 123 patients (64 [62; 69.5] years) without the history of COVID-19 and group 2–110 patients (65 [62; 68] years) with the history of COVID-19. Group 2 included 55.5 % of men and 44.5 % of women, group 2–52.8 % and 47.2 % men and women, respectively. Each group has been divided into two subgroups depending on the presence of various comorbidities: T2DM and obesity. The subgroup with obesity included patients under 64 [61; 67] years old, and had more women (53.8 %). The inclusion criteria were: signed voluntary informed consent to participate in research; age from 60 to 74 years old; stage 3 arterial hypertension; CHD, I–II functional class of angina pectoris; atrial fibrillation (all types, without cardiac embolism); T2DM with HbA1c level under 8.5 %; obesity stages I and II; the presence or absence of COVID-19 with SARS-CoV-2 RNA identification. In those who had documented history of COVID-19 caused by SARS-CoV-2, the duration of the disease over 12 weeks (for the group of patients with post-COVID syndrome). Exclusion criteria: symptomatic hypertension; CHD, III–IV FC of angina pectoris, microvascular, vasospastic, unstable angina; stages 4–5 of chronic kidney disease; T2DM with HbA1c level over 8.5 %; type 1 and other specific types of diabetes; chronic obstructive pulmonary disease; anemia (hemoglobin level below 130 g/l in men and below 120 g/l in women); malignant neoplasms; acute stages of other chronic diseases; mental disorders; alcohol consumption over 21 standard drinks per week for men and over 14 standard drinks for women.

All study participants underwent general clinical examination, biochemical blood test, lipid panel, glucose, glaciated hemoglobin, potassium, estimation of glomerular filtration rate, C-reactive protein, NT-proBNP level; Echo-CG according to the standard protocol; assessment of systolic and diastolic blood pressure (SBP and DBP) (office and self-measurement monitoring with the calculation of average level); assessment of cognitive status using the SPMSQ (Short Portable Mental Status Questionnaire) — a portable mental status questionnaire for cognitive deficits; HADS (Hospital Anxiety and Depression Scale) to identify and assess the severity of depression and anxiety among study participants.

The study was conducted in accordance with the standards of good clinical practice and the principles of the Declaration of Helsinki, the protocol was

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approved by the ethics committee of the Novosibirsk State Medical University of the Ministry of Healthcare of Russian Federation (protocol No. 148). All included patients signed voluntary informed consent to participate in research.

Statistical analysis

Statistical data analysis was performed using the RStudio software (version 2021.09.2 Build 382 — © 2009–2022 RStudio, Inc., USA, URL https://www.rstudio.com/) and the R language (version 4.0.2, URL https://www.R-project.org/). Descriptive characteristics are presented as median [first quartile; third quartile] for numerical data, percentages with confidence interval (CI) [lower bound of 95 % CI; upper bound of 95 % CI] for categorical data. The Mann-Whitney U-test with the calculation of 95 % CI was used to assess the statistical difference between the numerical characteristics of compared groups. The level of statistical significance was set as $p < 0.05$.

Results

The average age of study participants was 64 [62; 68] years. Since blood pressure instability is one of the main causes of cognitive deficit, especially in patients with post-COVID syndrome, all the patients underwent the assessment of hemodynamic parameters. Patients with the history of COVID-19 infection had lower DBP (66 [60; 72.75] mmHg) compared with patients from group 1 (80 [70; 88] mmHg ($p < 0.001$)). At the same time median SBP did not differ between groups — 156 [143.25; 165] mmHg ($p = 0.668$). Target SBP values were exceeded in 80 % [70 %; 87 %] of the elderly (⩾ 140 mm Hg) patients from group 1 versus 79 % [68 %; 86 %] from group 2 ($p = 0.862$). Elevation of DBT ⩾ 80 mm Hg was noted in 42 % [32 %; 53 %] of patients without the history of COVID-19 and in 12 % [6 %; 21 %] of patients from group 2 ($p < 0.001$), and decreased DBP < 70 mm Hg was found in 30 % [21 %; 41 %] and 57 % [46 %; 68 %] of patients from groups 1 and 2, respectively ($p = 0.001$). Pulse blood pressure ⩾ 60 mmHg was registered in 78 % [68 %; 86 %] of patients from the group 1 vs. 89 % [80 %; 94 %] — from group 2 ($p = 0.082$). In group 2 the median heart rate was 77 [74; 80] beats per minute versus 76 [67; 78] beats per minute in group 1 ($p < 0.001$). The comparative analysis of the coronary heart disease (CHD)/ arterial hypertension (AH)/atrial fibrillation (AF)+ T2DM subgroups depending on the history of COVID-19 revealed high SBP, heart rate and low DBP in patients after viral infection, DBP ($p < 0.001$) and heart rate ($p = 0.002$) differences were statistically significant. SBP in the group without and with PCS was 152 [143; 165] mmHg and 154 [141.5; 164.25] mm Hg, DBP was 78 [69; 82] mmHg and 68 [60; 76] mmHg, heart rate was 75 [67; 77] and 77 [73.75; 80] beats per minute, respectively. Similar data were obtained in the subgroups with CHD/AH/AF + AO depending on the presence of post-COVID syndrome.

Thus, average SBP values were increased in all study participants, and the subgroups with CHD/AH/AF + AO + PCS had the highest values — 162 [145.25; 168] mm Hg, as well as the lowest DBP values — 60 [60; 70] mm Hg, while patients with abdominal obesity without PCS had the highest DBP values — 82.5 [78; 88] mmHg. In general, SBP target values in elderly was ⩾ 140 mmHg, and DBP ⩾ 80 mmHg, as well as DBP decrease < 70 mmHg was also detected (Table 1).

It is known that psychological state change in the elderly, people of senile age and centenarians predominantly with the development of anxiety and depression. In this study we assessed psychological state using the HADS (Hospital Anxiety and Depression Scale) that is widely used in clinical practice by general practitioners, cardiologists, geriatricians, and rehabilitation specialists. In current study the median anxiety score in both patients without and with the history of coronavirus infection and PCS was 7 [6; 8] and 8 [6; 9] ($p = 0.031$), respectively, which can be interpreted as slight anxiety increase in patients with PCS without statistical significance, but with the development of subclinical anxiety in patients after COVID-19. It was found that 0–7 points or the absence

Table 1. **Hemodynamic parameters in groups with various comorbidities**

Group	SBP, mmHg	DBP, mmHg	HR, beats per minute
CHD/AH/AF + T2DM	156 [143,25; 165]	80 [70; 88]*	76 [67; 78]*
CHD/AH/AF + T2DM + PCS	156 [143,25; 165]	66 [60; 72,75]*	77 [74; 80]*
CHD/AH/AF + AO	161.5[145,25; 68]*	82.5 [78; 88]*	76[68; 80]
CHD/AH/AF + AO + PCS	162 [145,25; 168]*	60 [60; 70]*	78 [74; 80]

Comment. * — difference is statistically significant.

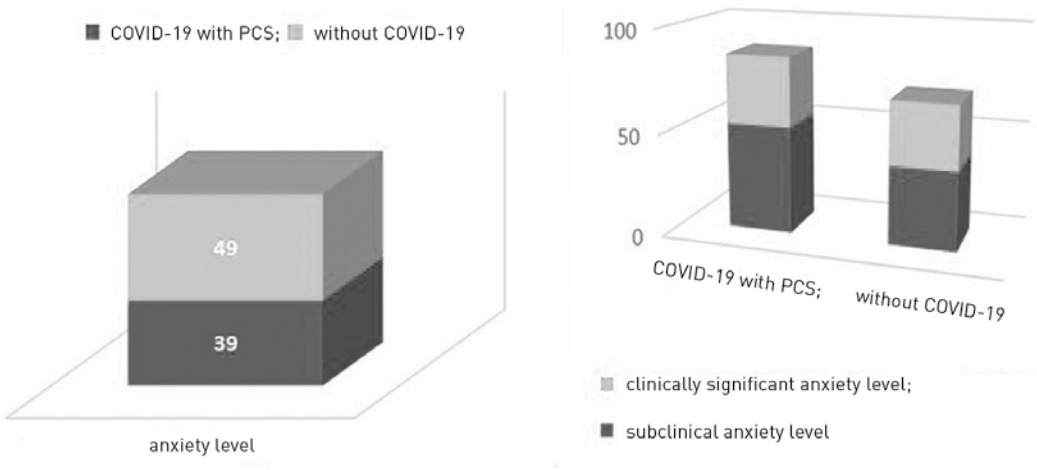

Figure 1. Anxiety level depending on the presence of COVID-19 with post-COVID syndrome

of anxiety, was noted in 61 % [52 %; 69 %] of patients without the history of coronavirus infection and in 49 % [40 %; 58 %] of patients with PCS (p = 0.086), 8–10 points or subclinical anxiety — in 26 % [19 %; 34 %] and 27 % [20 %; 36 %] of patients (p = 0.882), 11 points and above or clinically significant anxiety in 13 % [8 %; 20 %] and 13 % [8 %; 20 %] of patients (p = 0.041), respectively. In the subgroup of patients with comorbid pathology and type 2 diabetes, similar results were obtained — 7 [6; 9] points against 8 [7; 11] points (p = 0.037). The comparative analysis of anxiety level showed greater number of patients with subclinical and clinically significant anxiety after coronavirus infection — 8–10 points scored 25 % [16 %; 37 %] of patients after viral infection and 22 % [13 %; 33 %] without the history of COVID-19 (p = 0.681), 11 or more points — 30 % [20 %; 42 %] and 17 % [10 %; 28 %] (p = 0.099), respectively (Fig. 1). The level of anxiety did not differ depending the presence of obesity, the median HADS anxiety scores were similar and within the normal range.

The presence and the severity of cognitive impairment were assessed depending on the presence of PCS in patients with cardiovascular diseases (CVD) with metabolic syndrome. The median error rate in patients without the history of COVID-19 was 3 [1; 3] errors, and in those with PCS — 3 [2; 4] errors (p < 0.001) that can be interpreted as the absence of cognitive impairment in patients with PCS.

The assessment of cognitive state and the comparison of the degrees of cognitive impairment established that normal cognitive functioning was preserved in less patients after coronavirus infection (27 % [20 %; 36 %] compared with those without the history of infection 45 % [36 %; 54 %] (p = 0.007)) (Figure 2).

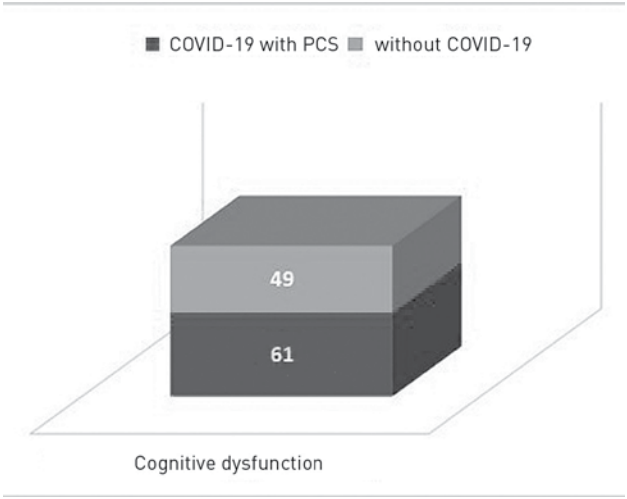

Figure 2. Cognitive impairment depending on the presence of COVID-19 and post-COVID syndrome

The levels of mild cognitive impairment did not differ significantly between compared groups, however patients from the group with PCS showed more pronounced cognitive impairment, moderate cognitive impairment was higher in patients with PCS — 22 % [15 %; 30 %] vs. 8 % [4 %; 14 %] (p = 0.005), severe cognitive impairment was found only in those with PCD — 2 % [1 %; 6 %] (p = 0.001) (Figure 3). The subgroups with obesity had the same number of errors — 3 [1; 4] errors in patients without PCS and 3 [2.25; 5] errors in those with PCS (p = 0.020).

After dividing study participants by the severity of cognitive impairment, statistical significance was achieved only in those who made 5–7 errors, which corresponds to moderate cognitive impairment, thus,

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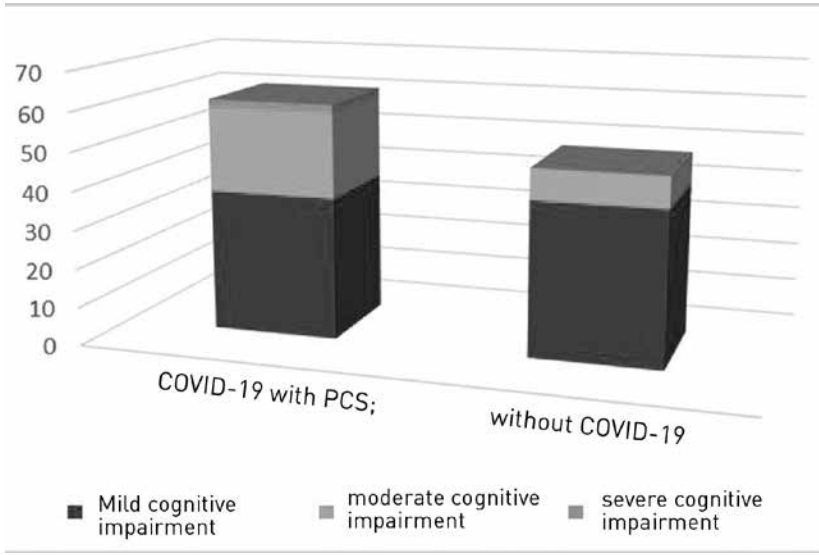

Figure 3. The severity of cognitive impairment depending on the presence of COVID-19 and post-COVID syndrome

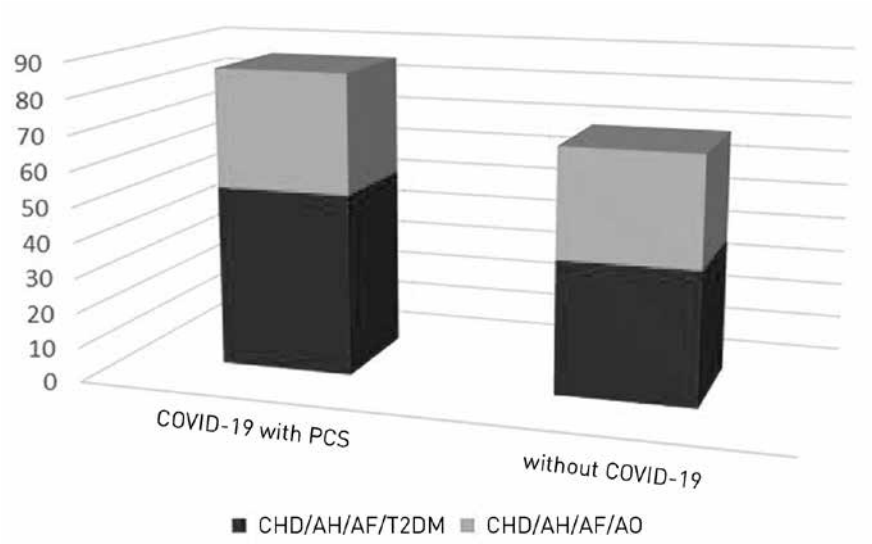

Figure 4. Cognitive impairment in clinical subgroups depending on the presence of COVID-19 and post-COVID syndrome

35 % [23 %; 49 %] of patients with PCS compared with 9 % [4 %; 19 %] of patients without PCS (p = 0.001) made from 5 to 7 errors by the SPMSQ. Cognitive impairment positively correlated with and anxiety and depression groups (r = 0.345, p = 0.033).

When comparing the subgroups of patients with CVD and T2DM depending on the of COVID-19 infection, patients with PCS showed statistically significantly more errors — 3 [2; 4] errors vs. 2 [1; 3] errors (p = 0.006). In addition, statistically significantly less patients did not score more than 2 errors and more patients without PCD showed normal cognitive functions — 51 % [39 %; 63 %] compared with 28 % [19 %; 40 %] in those with PCS (p = 0.012) (Figure 4).

Thus, despite almost equal median of errors made by patients by short portable mental status questionnaire (SPMSQ) that was used to assess the presence and severity of cognitive deficit through the number of errors, patients after new coronavirus infection more often had mild, moderate and severe cognitive impairment and less often showed normal cognitive status.

Discussion

To this day there are data indicating the relationship between arterial hypertension (AH) and cognitive impairment [26]. At the same time, it is known that endothelial dysfunction leads to renin-angiotensin-al-

dosterone system imbalance and causes not only transformation of the vascular bed, but also contributes to the blood-brain barrier dysfunction that eventually cause cognitive impairment [27, 28]. The data of cognitive state assessment can be used as marker for the progression of cerebral damage, especially in the elderly, which was highlighted in our study.

According to the World Health Organization, in 2019 every eight person on the planet suffered from mental disorder, with anxiety and depression being the most prevalent. In 2020 during the pandemic of COVID-19 infection, the number of patients with anxiety and depression has increased significantly. Within one year, the prevalence of these disorders increased up to 28% [29]. Long-term psychological consequences after COVID-19 are still being investigated [30]. Several researchers noted the increase in the number of patients with mental disorders in the post-COVID period [31]. In our work, we have found that anxiety and depression were more prevalent among patients with the history of COVID-19, cognitive impairment with the predominance of moderate and severe levels was also more often observed in patients after COVID-19, which confirms the data of previous studies.

Conclusion

The effects of COVID-19 remain uncertain, and if COVID-19 will continue to circulate for many years, its long-term outcomes could grow exponentially. In this regard, it is necessary to follow up patients after COVID-19 to assess the long-term consequences, especially those with comorbid pathologies that serves as poor prognostic factor. This could be achieved by reprofiling and initiation of large cohort studies aimed not only to assess long-term outcomes of SARS-CoV-2 infection, but also to investigate psycho-cognitive dysfunction.

Conflict of interest: None declared.

References

1. Biagianti B., Di Liberto A., Nicolò Edoardo A., Lisi I., Nobilia L., de Ferrabonc G.D., Zanier E.R., Stocchetti N., Brambilla P. Cognitive Assessment in SARS-CoV-2 Patients: A Systematic Review. Front Aging Neurosci. 2022;1; 14:909661. https://10.3389/fnagi.2022.909661
2. Kamchatnov P.R., Cheremin R.A., Skipetrova L.A. and others. Cognitive impairment in patients who underwent COVID-19. BC. 2022; 4:33–37. Russian. doi: 10.17116/jnevro20221220317
3. Dantzer R. Neuroimmune Interactions: From the Brain to the Immune System and Vice Versa. Physiological Reviews. 2018;1;98Q):477–504. https://10.1152/phys-rev.00039.2016
4. Kanberg N, Ashton NJ, Andersson LM, Yilmaz A, Lindh M, Nilsson S, Price RW, Blennow K, Zetterberg H, Gisslen M. Neurochemical evidence of astrocytic and neuronal injury commonly found in COVID-19. Neurology. 2020;22;95(12):e1754-e1759. https://10.1212/WNL. 0000000000010111
5. Harris S.A., Harris E.A. Herpes simplex virus type 1 and other pathogens are key causative factors in sporadic Alzheimer's disease. J Alzheimers Dis. 2015;48:319–353. doi: 10.3233/jad-142853
6. Damiano R.F., Guedes B.F., de Rocca C.C., de Pádua Serafim A., Castro L.H.M., Munhoz C.D., Nitrini R., Filho G.B., Miguel E.C., Lucchetti G., Forlenza O. Cognitive decline following acute viral infections: literature review and projections for post-COVID-19. Eur Arch Psychiatry Clin Neurosci. 2022 Feb;272(1):139–154. https://10.1007/s00406-021-01286–4
7. Crivelli L., Palmer K., Calandri I., Guekht A., Beghi E., Carroll W., Frontera J., García-Azorín D., Westenberg E., Winkler A.S., Mangialasche F., Allegri R.F., Kivipelto M. Changes in cognitive functioning after COVID-19: A systematic review and meta-analysis. Alzheimers Dement. 2022;18(5):1047–1066. https://10.1002/alz.12644
8. de Lorenzo R, Conte C, Lanzani C, et al. Residual clinical damage after COVID-19: A retrospective and prospective observational cohort study. PLOS ONE. 2020; 15(10). doi: 10.1371/journal.pone.0239570
9. Premraj L., Kannapadi N.V., Briggs J., Seal S.M., Battaglini D., Fanning J., Suen J., Robba C., Fraser J., Cho S.M. Mid and long-term neurological and neuropsychiatric manifestations of post-COVID-19 syndrome: A meta-analysis. J Neurol Sci. 2022;(15)434:120162. https://10.1016/j.jns.2022.120162
10. Goh Y, Beh DL, Makmur A, et al. Pearls and oysters: facial nerve palsy as a neurological manifestation of Covid-19 infection. Neurology. 2020;10.1212/ WNL.0000000000009863. https://10.1212/WNL.0000000000009863.
11. Hugon J., Msika E.F., Queneau M., Farid K., Paquet C. Long COVID: cognitive complaints (brain fog) and dysfunction of the cingulate cortex. J Neurol. 2022 Jan; 269(1): 44–46. https://10.1007/s00415–021-10655-x
12. Guedj E., Campion J.Y., Dudouet P., Kaphan E., Bregeon F., Tissot-Dupont H., Guis S., Barthelemy F., Habert P., Ceccaldi M., Million M., Raoult D., Cammilleri S., Eldin C. 18 F-FDG brain PET hypometabolism in patients with long COVID. Eur J Nucl

Med Mol Imaging. 2021;48(9):2823–2833. https://10.1007/s00259–021-05215–4

13. Edén A., Simrén J., Price R.W., Zetterberg H., Gisslén M. Neurochemical biomarkers to study CNS effects of COVID-19: A narrative review and synthesis. J Neurochem. 2021;159(1):61–77. doi: 10.1111/jnc.15459
14. Starichkova A.A., Tsygankova O.V., Khidirova L.D., etc. Cardiometabolic features of postcovid syndrome in elderly and senile persons with comorbid pathology. breast cancer. Medical review. 2022;6(9):501–508. Russian. https://10.32364/2587-6821-2022-6-9-501-508
15. Generoso J.S., Barichello de Quevedo J.L., Cattani M., Lodetti B.F., Sousa L., Collodel A., et al. Neurobiology of COVID-19: how can the virus affect the brain? Braz J Psychiatry. 2021; 43:650–664. doi: 10.1590/1516-4446-2020-1488
16. Monti G, Leggieri C, Fominskiy E, et al. Two-months quality of life of COVID-19 invasively ventilated survivors; an Italian single-center study. Acta Anaesthesiologica Scandinavica. 2021; 65(7). doi: 10.1111/aas.13812
17. Ceban F., Ling S., Lui L.M.W., Lee Y., Gill H., Teopiz K.M., Rodrigues N.B., Subramaniapillai M., Di Vincenzo J.D., Cao B., Lin K., Mansur R.B., Ho R.C., Rosenblat J.D., Miskowiak K.W., Vinberg M., Maletic V., McIntyre R.S. Fatigue and cognitive impairment in post-COVID-19 Syndrome: A systematic review and meta-analysis. Brain Behav Immun. 2022; 101:93–135. https://10.1016/j.bbi.2021.12.020
18. Taquet M, Geddes JR, Husain M, Luciano S et al. 6-month neurological and psychiatric outcomes in 236 379 survivors of COVID-19: a retrospective cohort study using electronic health records. The Lancet Psychiatry. 2021; 8(5). doi: 10.1016/S2215-0366(21)00084-5
19. Warren-Gash C, Forbes HJ, Williamson E, et al. Human herpesvirus infections and dementia or mild cognitive impairment: a systematic review and meta-analysis. Scientific Reports. 2019; 9(1). doi: 10.1038/s41598-019-41218-w
20. Zlatar ZZ, Muniz M, Galasko D, Salmon DP. Subjective Cognitive Decline Correlates With Depression Symptoms and Not With Concurrent Objective Cognition in a Clinic-Based Sample of Older Adults. The Journals of Gerontology: Series B. 2018;73(7). doi: 10.1093/geronb/gbw207
21. Kuo CL, Pilling LC, Atkins JL, et al. APOE e4 Genotype Predicts Severe COVID-19 in the UK Biobank Community Cohort. The Journals of Gerontology: Series A. 2020;75(11). doi: org/10.1093/gerona/glaa131
22. Nielsen S.L., Ludwigsen T., Johnsen S., Miskowiak K.W. [Cognitive late sequelae of COVID-19]. Ugeskr Laeger. 2022;11:184(15):V09210730. doi: 10 10.20333/25000136-2022-2-40-48.
23. Madonov P.G., Khidirova L.D., Derisheva D.A. Neuropsychiatric features of current COVID-19 pandemic (analysis of foreign publications in 2020). Medical alphabet. 2020;(33):58–61. Russian. https://10.33667/2078–5631-2020-33-58-61
24. Ostroumova O.D., Kochetkov A.I., Ostroumova T.M. Pulse arterial pressure and cognitive disorders. Russian Journal of Cardiology. 2021;26 (1):4317. Russian. doi: 10.15829/1560–4071-2021-4317
25. Levin RA, Carnegie MH, Celermajer DS. Pulse Pressure: An Emerging Therapeutic Target for Dementia. Front Neurosci. 2020; 14:669. https://10.3389/fnins.2020.00669
26. Thorin-Trescases N, de Montgolfier O, Pinçon A, et al. Impact of pulse pressure on cerebrovascular events leading to age-related cognitive decline. Am J Physiol Heart Circ Physiol. 2018;314(6):H1214-H1224. https://10.1152/ajpheart.00637.2017
27. World Health Organization. Mental disorders. (Electronic resource). https://www.who.int / (date of request: 10.11.2022).
28. Arora T, Grey I, Östlundh L, Lam KBH, Omar OM, Arnone D. The prevalence of psychological consequences of COVID-19: A systematic review and meta-analysis of observational studies. J Health Psychol. 2022;27(4):805–824. https://10.1177/1359105320966639
29. Alonso-Lana S, Marquié M, Ruiz A, Boada M. Cognitive and Neuropsychiatric Manifestations of COVID-19 and Effects on Elderly Individuals With Dementia. Frontiers in Aging Neuroscience. 2020; 12. doi: 10.3389/fnagi.2020.588872
30. Mazza MG, Palladini M, Poletti S, Benedetti F. Post-COVID-19 Depressive Symptoms: Epidemiology, Pathophysiology, and Pharmacological Treatment. CNS Drugs. 2022 Jul;36(7):681–702. https://10.1007/s40263-022-00931-3
31. Daroische R, Hemminghyth MS, Eilertsen TH, Breitve MH, Chwiszczuk LJ. Cognitive Impairment After COVID-19-A Review on Objective Test Data. Frontiers in Neurology. 2021; 12. doi: 10.3389/fneur.2021.699582