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Research Article

FEATURES OF CARDIOVASCULAR COMPLICATIONS DEVELOPMENT IN COVID-19

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ABSTRACT

This article provides a review of data on the prevalence and impact of cardiovascular disease on the course and outcomes of the new coronavirus infection COVID-19. A new strain of coronavirus, the COVID-19 (CoronaVirus Disease-2019) pandemic caused by SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2) is causing a rapid increase in cases and high death rates worldwide. Despite the effect of SARS-CoV-2 on the pulmonary system, it leads to the development of multiple organ failure, including due to damage to the cardiovascular system (CVD).

KEYWORDS

Cardiovascular system (CVS), coronavirus infection COVID-19, myocardial infarction, myocarditis, arrhythmia, chronic heart failure (CHF), acute coronary syndrome (ACS).

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INTRODUCTION

Coronavirus infection (COVID-19) and cardiovascular diseases remain one of the global comorbid pathologies in the current period of the pandemic, that is, the prevalence of diseases over each other, exacerbate the level and course of the disease and increase the risk of death of patients.

The management of such patients requires doctors to know the characteristics of a viral infection, its clinical appearance when combined with cardiovascular diseases. In about 50% of patients infected with SARSCoV-2, multimorbidity is detected, the frequency of which increases to 72% in severe COVID-19 [2, 3]. Cardiovascular disease (CVD) and cardiovascular risk factors such as obesity and diabetes mellitus (DM) are common in patients with COVID-19.

According to a retrospective analysis of data (n = 1,590) obtained in 575 hospitals in China, 25% of patients with COVID-19 were diagnosed with comorbidities [4]. Arterial hypertension (AH) occurred in 16.9% of patients, other CVDs - in 53.7% and diabetes - in 8.2%. In an Italian cohort of COVID-19 patients (n = 22 512, of whom 355 died), concomitant coronary heart disease (CHD) was present in 30%, atrial fibrillation - in 24.5%, stroke - in 9.6%, and diabetes mellitus - in 35.5% [5]. An analysis of the database of 5,700 COVID-19 patients admitted to 12 hospitals in New York showed the presence of hypertension in 56.6%, ischemic heart disease - in 11.1%, obesity - in 41.7% and diabetes - in 33.8% [6]. A retrospective analysis of the clinical and demographic indicators of 1,007 COVID-19 patients admitted to hospitals (in the intensive care unit, ICU) of the Russian Federation with acute respiratory distress syndrome (ARDS) found CVD in 61.4% [7]. AH (in 56.3% of patients) and IHD (in 16.3%) were common; less often - a stroke (in 7.1%) and atrial fibrillation (in 9.3%). 26.1% of patients were obese and 25% of patients with type 2 diabetes. The incidence of CVD increased with age, reaching 80% in the group over 60 years old.

A meta-analysis of six clinical studies, including 1527 cases of coronavirus infection, demonstrated the presence of arterial hypertension in 17.1% of patients, coronary heart disease and / or cerebrovascular disease - in 16.4%, type 2 diabetes mellitus - in 9.7% of patients [5]. Moreover, most of the most severe patients admitted to intensive care units were characterized by the presence of concomitant pathology. According to a Chinese study (Wuhan), comorbid pathology significantly increased the risk of an unfavorable outcome: there was a significant increase in mortality among patients with CVD up to 10.5%, with type 2 diabetes mellitus - up to 7.3%, with isolated arterial hypertension - up to 6.0%, while mortality in this population as a whole was 2.3% [6]. The data of Italian colleagues confirm these indicators [7]. According to the Chinese protocol WHO-China, mortality from COVID-19 against the background of CVD was 13.2% (compared to 1% among people without comorbid pathology), and higher

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proportions of diabetes mellitus (9.2%) and arterial hypertension (8.4%) among deceased patients [2, 8]. Considering the possible mechanisms of such a high prevalence of the combination of coronavirus infection and CVD, one should point out the potentially common risk factors for these diseases. For example, age, exerting a suppressive effect on the immune system, increases the susceptibility to viral infection and ensures its more severe course. Previously, a decrease in the titer of protective antibodies by about 50% was noted in persons over 65 years of age after vaccination against influenza; in younger patients, such dynamics was not observed [9]. Other cardiovascular risk factors, O.L. Barbarash et al. 19, such as diabetes mellitus and dyslipidemia, also cause impaired immune response, increasing the risk of viral infection [10, 11]. In addition, CVD is considered as a marker of accelerated immune aging with a negative impact on the course and prognosis in COVID-19 [4]. Currently, there is no accurate information about the long-term results of treatment, but there is evidence of the development of cardiac complications after the relief of acute symptoms of COVID-19 and their occurrence even after patients recover [12]. Italian colleagues report a case of fulminant myocarditis in a convalescent one week after the resolution of all respiratory symptoms [13]. Later cardiovascular events are extremely important as they can significantly affect the long-term prognosis. Thus, the association of SARS-CoV with disturbances in the regulation of lipid and carbohydrate metabolism, as well as the chronic damaging effect of this viral agent on the cardiovascular system, is assumed [14].

The combination of COVID-19 with the clinic of acute coronary syndrome (ACS) is relevant from several points of view. First, like any acute inflammatory disease, COVID-19 can be a provoking factor in the destabilization of an atherosclerotic plaque and the implementation of an atherothrombotic ACS scenario. In addition, hemodynamic disturbances inherent in severe intoxication (tachycardia, hypo- or hypertension) can act as a provoking factor in the development of myocardial infarction, myocardial infarction.

Do not forget that cardiotoxicity against the background of infection or the appointment of antiviral therapy, manifested by appropriate clinical symptoms and an increase in the concentration of troponins, brain natriuretic peptide, necessitates a careful differential diagnosis of virus-induced myocardial damage with classic ACS against the background of atherothrombosis. Patients with severe symptoms of COVID-19 can develop classic atherothrombotic ACS with subsequent death. In this category of patients, the cause of death should be discussed and correctly determined when formulating the underlying disease in the pathological diagnosis. Noteworthy is the publication of Chinese colleagues [18], which examines the phenomenon of a significant increase in troponin T levels in patients with COVID-19 without an established history of CVD and clinical signs of myocardial infarction. The authors explain this effect by the so-called phenomenon of virus-induced myocardial

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damage, which is closely associated with an increase in C-reactive protein (a factor of systemic inflammation), natriuretic peptides (markers of myocardial distress), the development of life-threatening arrhythmias and acute heart failure. Moreover, the likelihood of death at high troponin T values was increased both in patients with and without cardiovascular comorbidity. In this regard, the issues of correct differential diagnosis of virus-induced myocardial damage and myocardial infarction should be given close attention in the management of patients with COVID-19 [30].

The world's leading medical journals, contrary to established practice, willingly accept descriptive cases with the morphological picture of patients who died from COVID-19. The authors thoroughly analyze postmortem macro- and microscopic histological changes in the lungs and myocardium, and also pay attention to the presence of thrombosis of the coronary arteries, the state of atherosclerotic plaques, since the mechanisms of the negative effect of coronavirus infection on the course of CVD remain the subject of discussion [19].

COVID-19 and chronic heart failure characterizing patients with COVID-19, it should be noted the similarity of respiratory and cardiac symptoms, often manifested by shortness of breath, which can create significant difficulties both at the screening stage and at making a definitive diagnosis, especially in patients with chronic heart failure (CHS) [30]. A particularly difficult category is represented by patients with a burdened history of pulmonary and cardiovascular pathology [22]. According to the analysis of patients in Wuhan (China), CHF was present in 1/3 of patients hospitalized with COVID-19, and was detected in more than half of those who died from this disease [23]. As mentioned above, the SARS-CoV-2 viral agent is capable of causing direct damage to cardiomyocytes, leading to CHF decompensation, shock and sudden death. According to a study by G.Y. Oudit et al., Conducted in Toronto (Canada) during the outbreak of SARS in 2008, in 35% of autopsy samples of the heart taken against the background of SARS-CoV lesion, viral RNA of the pathogen was detected [24]. If we cover in more detail the topic of myocardial damage associated with COVID-19, then the polyetiology of this phenomenon should be noted. Currently, there are active discussions in the scientific community about how SARS-CoV-2 can have a cardiolytic effect with a critical increase in the value of troponins, because this often requires differential diagnosis with MI. In a recent review article by French authors [25], it is suggested that cardiomyocyte necrosis can develop as a consequence of several triggering factors: the direct damaging effect of the SARS-CoV-2 viral agent on cardiomyocytes, since type 2 angiotensin converting enzyme (ACE) receptors are expressed on these cells; hypoxic damage to cardiomyocytes against the background of a cytokine storm, local and systemic acidosis, mitochondrial lysis; microcirculatory disorders against the background of hypercoagulation and hyperaggregation of platelets, as well as coronary spasm. In addition, due to the hyperreactivity of the immune system, scenarios of nonischemic

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death of cardiomyocytes (apoptosis) may paradoxically be triggered [30]. As a result, this leads to a decrease in the functional properties of the myocardium and the development of acute heart failure and other life-threatening conditions [30]. At the same time, COVID-19-induced lung damage can lead to significant disturbances in gas exchange, manifested by hypoxemia, macroerg deficiency, and severe acidosis [30]. The developing intracellular acidosis and free radicals destroy the phospholipid layer of cell membranes, and the influx of calcium ions caused by hypoxia leads to damage and apoptosis of cardiomyocytes [26]. The combination of such features of viral invasion in COVID-19 and the initially severe comorbid status of a patient with CHF leads to a high risk of rapid decompensation of heart failure and the development of multiple organ dysfunction. Naturally, such patients are characterized by a high risk of death due to COVID-19 [27]. There is evidence of the possibility of a low-symptom course of viral infection in patients with CVD. For example, the National Health Commission of China reports that some patients consulted a doctor for the first time because of symptoms associated with CVD: heart palpitations, pain and tightness in the chest, and not at all with fever and cough. Nevertheless, after an additional examination, he was diagnosed with COVID-19 [14]. Chinese colleagues describe the observation of four patients with end-stage CHF who were infected with SARS-CoV-2: two of them had a mild course of the disease, others had a severe course. There is evidence of the possibility of a low-symptom course of viral infection in patients with CVD. For example, the National Health Commission of China reports that some patients consulted a doctor for the first time because of symptoms associated with CVD: heart palpitations, pain and tightness in the chest, and not at all with fever and cough. Nevertheless, after an additional examination, he was diagnosed with COVID-19 [14]. Chinese colleagues describe the observation of four patients with end-stage CHF who were infected with SARS-CoV-2: two of them had a mild course of the disease, others had a severe course. So, in a study of 138 patients with COVID-19, antiviral O.L. Barbarash et al. and cardiovascular diseases drugs were prescribed to 89.9% of patients [27]. Antiviral agents are known to cause heart failure, arrhythmias, or other cardiovascular disorders. Therefore, when prescribing antiviral drugs, it is necessary to carefully monitor the development of cardiovascular complications and, if possible, avoid adverse drug interactions [29].

Thus, the simultaneous development of COVID-19 and cardiovascular pathology aggravates the course of the disease, leading to the disability of many patients, as well as the development of multiple deaths.

REFERENCES

1. Guan W.J., Ni Z.Y., Hu Y., Liang W., Ou Ch., He J. et al. Clinical Characteristics of Coronavirus Disease 2019 in China. N Engl J Med. 2020; 2020 Feb 28. doi: 10.1056/NEJMoa2002032.
2. Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19). 16-24 February 2020. Available at:

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https://wwwwhoint/docs/default-source/coronaviruse/who-chinajoint-mission-on-covid-19-final-reportpdf. (accessed March 9, 2020.

3. Шляхто Е. В., Конради А. О., Арутюнов Г. П., Арутюнов А. Г., Баутин А. Е., Бойцов С. А., Виллевальде С. В., Григорьева Н. Ю., Дупляков Д. В., Звартау Н. Э., Козиолова Н. А., Лебедев Д. С., Мальчикова С. В., Медведева Е. А., Михайлов Е. Н., Моисеева О. М., Орлова Я. А., Павлова Т. В., Певзнер Д. В., Петрова М. М., Ребров А. П., Ситникова М. Ю., Соловьева А. Е., Тарловская Е. И., Трукшина М. А., Федотов П. А., Фомин И. В., Хрипун А. В., Чесникова А. И., Шапошник И. И., Явелов И. С., Яковлев А. Н. Руководство по диагностике и лечению болезней системы кровообращения в контексте пандемии COVID-19. Российский кардиологический журнал. 2020;25(3):3801. doi:10.15829/1560-4071-2020-3-3801.
4. Driggin E., Madhavan M.V., Bikdeli B., Chuich T., Laracy J., Bondi-Zoccai G. et al Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the Coronavirus Disease 2019 (COVID-19) Pandemic. J Am Coll Cardiol. 2020 Mar 19. Epublished Doi:10.1016/j.jacc.2020.03.031.
5. Li B., Yang J., Zhao F., Zhi L., Wang X., Liu L., Bi Z., Zhao Yu. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China. Clin Res Cardiol. 2020 Mar 11; 1-8 doi: 10.1007/s00392-020-01626-9
6. Wu Z., Mc Googan J.M. Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72314 Cases From the Chinese Center for Disease Control and Prevention. JAMA. 2020. Feb 24. doi: 10.1001/jama.2020.2648.
7. Porcheddu R., Serra C., Kelvin D., Kelvin N., Rubino S. Similarity in Case Fatality Rates (CFR) of COVID-19/SARSCOV-2 in Italy and China. J Infect Dev Ctries. 2020;14:125- 128. doi: 10.3855/jidc.12600.
8. Chen N., Zhou M., Dong X., Qu J., Gong F., Ya. Han et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020;395:507-513. doi: 10.1016/S0140-6736(20)30211-7.
9. Liu W.M., Vander Zeijst B.A., Boog C.J., Soethout E.C. Aging and impaired immunity to influenza viruses: implications for vaccine development. HumVaccin. 2011; 7 Suppl: 94-8.
10. Zidar D.A., Al-Kindi S.G., Liu Y., Krieger N.I., Perzynski A.T., Osnard M. et al. Association of Lymphopenia With Risk of Mortality Among Adults in the US General Population. JAMA. Netw Open 2019; 2:e1916526 doi: 10.1001/jamanetworkopen.2019.16526.
11. Saltiel A.R., Olefsky J.M. Inflammatory mechanisms linking obesity and metabolic disease. J Clin Invest. 2017;127:1-4
12. Akhmerov A., Marban E. COVID-19 and the Heart. Circulation. 2020 Apr 11. doi: 10.1161/CIRCRESAHA.120.317055.

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13. Inciardi R.M., Lupi L., Zaccone G., Italia L., Raffo M., Tomasonit D. et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). JAMA Cardiol. 2020; DOI: 10.1001/jamacardio.2020.1096.
14. ZhengY.-Y., MaY.-T., Zhang J.-Y., Xie X. COVID-19 and the cardiovascular system. Nature Reviews Cardiology. 2020. Mar 5. doi: 10.1038/s41569-020-0360-5.
15. Ganatra S., Hammond S.P., Nohria A. The Novel Coronavirus Disease (COVID-19) Threat for Patients with Cardiovascular Disease and Cancer JACC: Cardio Oncology. 2020. doi: 10.1016/j.jaccao.2020.03.001.
16. Mortality G.B.D., Causes of Death C. Global, regional, and national life expectancy, all-cause mortality, and causespecific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet. 2016;388:1459-1544.
17. Liang W., Guan W., Chen Retal. Cancer patients in SARS-CoV-2 infection: a nationwide analysis in China. Lancet Oncol. 2020;21:335-337.
18. Guo T., FanY., Chen M., Wu X., Zhang L., He T., Wang H., Wan J., Wang X., Lu Z. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). JAMACardiol. 2020 Mar 27. doi: 10.1001/jamacardio.2020.1017.
19. Sharon E. Fox, Aibek Akmatbekov, Jack L. Harbert, Guang Li, J. Quincy Brown, Richard S. Vander Heide. Pulmonary and Cardiac Pathology in Covid-19: The First Autopsy Series from New Orleans. medRxiv. https://www.medrxiv.org/content/10.1101/2020.04.06.20050575v1/doi:https://doi.org/10.1101/2020.04.06.20050575.
20. Welt F.G.P., Shah P.B., Aronow H.D., Bortnick A.E., Henry T.D., Sherwood M.W., Young M.N., Davidson L.J., Kadavath S., Mahmud E., Kirtane A.J., American College of Cardiology's (ACC) Interventional Council and the Society of Cardiovascular Angiography and Intervention (SCAI), Catheterization Laboratory Considerations During the Coronavirus (COVID-19) Pandemic: From ACC's Interventional Council and SCAI. J Am Coll Cardiol. 2020 Mar 16; S0735-1097(20)34566-6. doi: 10.1016/j.jacc.2020.03.021..
21. Bettari L., Pero G., Maiandi C., Messina A., Saccocci M., Cirillo M., Troise G., Conti E., Cuccia C., Maffeo D. Exploring personal protection during high-risk PCI in a COVID-19 patient – Impella CP mechanicalsupportduring ULMCA bifurcationstenting. JACC: CaseReports. April 2020 DOI:10.1016/j.jaccas.2020.03.006.
22. Fried J.A., Ramasubbu K., Bhatt R., Topkara V.K., Clerkin K.J., Horn E. et al. The Variety of Cardiovascular Presentations of COVID-19. Circulation. 2020 Apr 3. doi: 10.1161/CIRCULATIONAHA.120.047164.
23. Juneman E. Leading the compassionate charge. Circ Heart Fail. 2020 Apr;13(4):e007085. doi: 10.1161/CIRCHEARTFAILURE.120.007085.

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24. Oudit G.Y., Kassiri Z., Jiang C., Liu P.P., Poutanen S.M., Penninger J.M., Butany J. SARS-coronavirus Modulation of Myocardial ACE2 Expression and Inflammation in Patients With SARS Eur J Clin Invest. 2009 Jul; 39(7):618-25. doi: 10.1111/j.1365-2362.2009.02153.x.
25. Haeck G., Ancion A., Marechal P., Oury C., Lancellotti P. COVID-19 andcardiovascular diseases. Rev Med Liege. 2020 Apr;75(4):226-232.
26. Huang C., Wang Y., Li X., Ren L., Zhao J., Hu Y., Zhang L., Fan G., Xu J., Gu X. et al Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020 Feb 15;395(10223):497-506. doi: 10.1016/S0140-6736(20)30183-5.
27. Wang D., Hu B., Hu C., Zhu F., Liu X., Zhang J., et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. JAMA. 2020 Feb 7. doi: 10.1001/jama.2020.1585.
28. Dong N., Cai J., Zhou Y., Liu J., Li F., End-stage Heart Failure with COVID-19: Strong Evidence of Myocardial Injury by 2019-nCoV, JACC Heart Fail. 2020 Apr 7. pii: S2213-1779(20)30200-6. doi: 10.1016/j.jchf.2020.04.001.
29. Sakabe M., Yoshioka R., Fujiki A. Sick sinus syndrome induced by interferon and ribavirin therapy in a patient with chronic hepatitis C. J. Cardiol. Cases. 2013 Oct 29;8(6):173- 175. doi: 10.1016/j.jccase.2013.08.002.
30. О. Л. Барбараш, В.Н. Каретникова, В.В. Кашталап, Т.Н. Зверева, А.М. Кочергина. Новая коронавирусная болезнь (COVID-19) и сердечно-сосудистые заболевания. Комплексные проблемы сердечно-сосудистых заболеваний. 2020; 9 (2): 17-28. DOI: 10.17802/2306-1278-2020-9-2-17-28