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Chronic heart failure and COVID-19

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Abstract

The coronavirus pandemic has caused a rapid increase in the number of cases and high deaths worldwide. A new coronavirus infection in the presence of an initial cardiac pathology can provoke decompensation of chronic heart failure. The article shows current information on the pathogenesis of COVID-19 and organ-specific lesions that develop in this disease is presented. Data on inflammation and its biochemical markers, on the features of coagulopathy, endothelial damage and microthromboses has been reflected in detail in this article. Particular attention is paid to the role of type 2 angiotensin-converting enzyme receptors and type 2 transmembrane serine protease in the development of organ-specific lesions in COVID-19. The pathogenesis of damage to the cardiovascular system is considered in detail with the presentation of data from latest literature on changes in the myocardium in patients who underwent COVID-19.

Keywords: Chronic heart failure; COVID-19; Cardiovascular disease; Uzbekistan

1 Introduction

As known from literature, a new coronavirus (SARS-CoV-2) in December 2019 became the etiological agent of an outbreak of pneumonia in the Chinese city of Wuhan [1]. The infection was named "COVID-19" and in March 2020 the World Health Organization declared it a global pandemic [2]. In most cases, COVID-19 presents clinically as an acute respiratory infection of the upper respiratory tract, in some cases with additional and characteristic symptoms such as hypo-/anosmia, hypo-/ageusia. It is also possible to involve the lower respiratory tract with the development of viral pneumonia, and in severe cases - respiratory distress syndrome (RDS) in adults, up to death due to multiple organ failure, especially in elderly patients and people with a number of concomitant diseases (diabetes mellitus) , obesity, diseases of the cardiovascular system - CVS, oncological diseases, etc.) [3]. As part of a systemic inflammatory response against the background of uncontrolled hyperproduction of cytokines - interleukin (IL)-1ß, IL-6, monocytic chemoattractant protein 1, etc. - in combination with suppression of the function of NK cells [4], there is a high risk of developing cytokine storm syndrome. Cytokine storm syndrome, which unfolds as part of infection with pathogens of the human coronavirus family, has a number of characteristics reminiscent of macrophage activation syndrome (MAS), also known as secondary hemophagocytic lymphohistiocytosis (sHLH) [5], a life-threatening condition characterized by uncontrolled proliferation of activated lymphocytes and macrophages with a massive release of pro-inflammatory cytokines, which is also associated with rheumatic diseases, such as systemic lupus erythematosus and juvenile idiopathic arthritis with a systemic onset. It is this feature that served as a prerequisite for the use of anticytokine drugs in severe COVID-19 pneumonia, as well as in other conditions accompanied by MAS [6]. Interestingly, the severe course of COVID-19 pneumonia differs from classical sHLH (or MAS), and it is more correct to speak of MAS-like pulmonary immunopathology. The key features of sHLH/MAS are hemophagocytosis and acute consumption coagulopathy with the development of disseminated intravascular coagulation (DIC) accompanied by hypercytokinemia and hyperferritinemia. Hemophagocytosis has been described in some patients with severe SARS (no definitive data on COVID-19 has yet been published), and DIC may develop in some patients with COVID-19 pneumonia, but usually

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shortly before terminal illness [7]. Hypercytokinemia in MAS is accompanied by extremely high ferritin levels (≥10,000–100,000 ng/mL), while in COVID-19, ferritin typically ranges from 500–3,000 ng/mL (rarely higher). It is also relatively atypical for COVID-19 to have hepatic dysfunction with secondary coagulopathy. It is believed that massive pulmonary infiltration by macrophages and other immunocytes in severe COVID-19 pneumonia leads to diffuse inflammation, alveolar damage involving the adjacent microvascular bed [8].

With the daily rise in the number of confirmed cases and the accumulation of clinical data, COVID-19 infection has become a major concern of the medical community. Various studies have determined that immune system dysregulation, increased metabolic demand, and procoagulant activity caused by coronavirus are likely to be responsible for the increased risk of poor outcomes in people with cardiovascular disease [9].

The increasing incidence of myocardial injury, vascular dysfunction, and thrombosis in patients with COVID-19, including those with asymptomatic or minimal early infection, raises important questions about potential long-term cardiovascular events that may include heart failure, life-threatening arrhythmias and conduction disorders, sudden cardiac death, myocardial blood flow disturbance due to damage to the microvascular bed, aneurysms of the coronary arteries and aorta, arterial hypertension, lability of heart rate and blood pressure response to physical activity, accelerated development of atherosclerosis, as well as venous and arterial thromboembolism [10].

It was found that COVID-19 caused heart failure in 23% of 191 patients treated in hospital in Wuhan, China. Cases of severe myocarditis with decreased systolic function have also been reported after a coronavirus infection [11]. Among 68 deaths out of 150 patients with COVID-19, 7% were associated with myocarditis and circulatory failure [12]. But it remains unclear whether heart failure is a consequence of exacerbation of pre-existing left ventricular dysfunction or new cardiomyopathy [13].

Cardiac pericytes with high expression of ACE2 can act as target cells for SARS-CoV-2 [14]. Patients with underlying heart failure showed increased expression of ACE2 at both mRNA and protein levels, meaning that if infected with the virus, these patients may be at higher risk of heart attack and serious illness [15].

Thus, patients with chronic heart failure (CHF) constitute a special risk group for severe COVID-19 and a very high risk of complications. It is possible to worsen the course of CHF against the background of a coronavirus infection due to the addition of respiratory failure typical for this disease, fibrotic changes in the lung tissue and aggravation of cardiopulmonary insufficiency. In this regard, it is optimal to organize dispensary observation of patients with CHF in high-risk rooms.

In the convalescent period after COVID-19, symptoms may persist for a long time, including subfebrile condition, dyspepsia, so it is necessary to continue monitoring the volume of fluid drunk and excreted and the patient's body weight, remembering the need to increase fluid intake with an increase in its loss (significant sweating, fever body, diarrhea, etc.). In the presence of appropriate indications, monitoring of blood electrolytes and the state of kidney function is necessary. It must be remembered that increased dyspnea may be associated with both decompensated CHF and pulmonary embolism or with the development of respiratory failure.

Compared with healthy controls and risk factor-matched controls, patients recently recovered from COVID-19 had lower ejection fraction, higher volumes and higher left ventricular mass, and elevated T1 and T2 on MRI. But none of these measures correlated with time at diagnosis of COVID-19 [16].

In terms of radiographic imaging, in addition to ground glass syndrome and interlobular septal thickening, the ratio of central to gradient distribution was higher in patients with heart failure than in patients with COVID-19. In patients with heart failure, the degree of small pulmonary vein dilatation was higher, and the regression of lung lesions was significantly accelerated after effective treatment of heart failure [17].

Adjusted for age, pre-existing cardiovascular disease (hypertension, coronary artery disease, and congestive heart failure), cerebrovascular disease, diabetes mellitus, chronic obstructive pulmonary disease, renal failure, cancer, ARDS, creatinine levels above 133 μmol/L, and NT -proBNP above 900 pg/mL, Cox's multivariate adjusted proportional hazards regression model showed a significantly higher risk of death in patients with heart injury.

Chronic heart failure (CHF) retains its leading position among the causes of cardiovascular mortality and is an epidemic among therapeutic diseases in the modern medical world. The prognosis remains unfavorable in the group of patients with functional class III-IV (FC) of heart failure (HF). The cost of treating this category of patients is very high due to the

large number of repeated hospitalizations, the ever-increasing cost of drug treatment and the high probability of disability [18].

During the pandemic of a novel coronavirus infection (COVID-19) caused by SARS-CoV-2, it has been proven that the life prognosis of a patient with COVID-19 worsens if the patient has cardiovascular diseases, diabetes mellitus, or chronic lung diseases [19]. The presence of CHF significantly worsens the prognosis both during the period of the disease and during the period of early convalescence after suffering COVID-19 [20]. Thus, according to Bhatt AS, et al almost every fourth patient with CHF died during hospitalization. Few works provide data on a longer prognosis of patients with CHF after undergoing COVID-19. The COVID-19 pandemic has affected the organization of medical care for patients with CHF. A study by the UK National HF Audit (NHFA) showed a 47% reduction in HF hospitalizations during the COVID-19 pandemic compared to 2018-2019, accompanied by an increase in HF deaths at home, in nursing homes and hospices [10]. For the Russian Federation, it is also relevant to analyze the survival of patients with CHF who did not have COVID-19 and who were in self-isolation, which could help reduce the intensity of monitoring them.

Lessons from the previous coronavirus and influenza epidemics suggest that viral infections can exacerbate a preexisting HF, with multiple studies showing an increase in HF re-hospitalization during influenza-like illness seasons [8]. With the more aggressive COVID-19 infection, HF patients are at a considerable higher risk of acute exacerbations, and multiple mechanisms may be responsible for triggering and aggravating this process.

New onset of HF was observed in as much as a quarter of hospitalized COVID-19 patients; and in as much as one-third of those admitted to the intensive care unit (ICU) [17, 19], despite not having a history of HF. This could be due to the direct effect of the virus or the systemic inflammation on the heart. Severe acute myocarditis can be a manifestation of the infection resulting in cardiogenic shock, which can then result in multi-organ dysfunction syndrome (MODS) and death [20]. Moreover, the prothrombotic state previously discussed can result in pulmonary embolism and thus acute right ventricular failure. The use of temporary heart pumps such as the Impella may be useful in these scenarios. Stress cardiomyopathy-like picture can also be seen due to the generalized inflammatory response and sympathetic activation, resulting in a more classic acute HF decompensation with elevated filling pressures and pulmonary edema [21].

COVID-19 mechanism of cellular entry has implicated an important class of medications that are part of the Guideline Directed Medical Therapy (GDMT) for heart failure with reduced ejection fraction (HFrEF). Similar to the previously known respiratory coronaviruses, SARS-CoV-2 spike glycoproteins bind to angiotensin-converting enzyme 2 (ACE2) receptors on the cell's outer surface [22]. These receptors are largely in the lungs and small intestines, but have also been reported to be present in the heart [18]. With the emergence of the current pandemic, this known pharmacology led to several editorials and media reports questioning the associated risks in patients taking ACEI/ARB or suggesting that patients should prophylactically stop taking these medications [9]. Such concerns stem from the theory that patients on these medications are likely to have upregulated ACE2 receptors and thus are more prone to viral uptake or worse outcomes if they contract the infection. The interplay of the virus with the ACE2 receptor and ACEI/ARB medications is rather more complex than a pharmacologically induced overexpression of a receptor and a resultant more viral susceptibility. First, while ACE and ACE2 are similar their active sites are different and they do not directly affect each other's activity. Data from animal and human models are conflicting regarding the effect of ACE or even ARBs on the expression and activity of ACE2 [12]. Second, we still do not have data on the effect of these drugs on lung-specific expression of ACE2. In addition, even if we assume that RAAS inhibitors upregulate ACE2 levels, we do not have data to conclude that this will predispose to more entry of the virus in humans. Through converting angiotensin II to angiotensin 1–7, ACE2 in fact partially reverses the effects of the RAAS and its known detrimental outcomes in not only HF (vasoconstriction, myocardial remodeling) but also its potential role in inducing acute lung injury; therefore, attenuating the effects of an overactivated RAAS. This downregulation of RAAS has been used to explain the benefits of ACE2 in protection from severe lung injury in animal models [14]. In a recent case series that included 363 hypertensive patients hospitalized with COVID-19, exposure to ACEI/ARB did not affect worsening disease or mortality [15, 24]. In fact, there are currently ongoing randomized controlled trials to explore a potential benefit for losartan in hospitalized and non-hospitalized COVID-19 patients [21]. A number of physician groups and professional societies recently considered the available data and the well-documented effect of discontinuing ACEI/ARB therapy and advised against changing clinical practice for the purpose of mitigating the pandemic and recommended to continue treatment based on standard indications [23].

2 Conclusion

Thus, to date, a lot of information has been accumulated regarding the variations in the course of COVID-19, often the changes affect not only the lungs and the respiratory tract, but the features of pathogenesis in some cases allow us to conclude that the pathology is multisystemic. Involvement of the CCC is also possible with a mild course of infection,

regardless of the severity of symptoms, which requires further detailed study. It seems reasonable to recommend that all patients who have had a new coronavirus infection undergo echocardiography and follow-up if changes are detected.

Compliance with ethical standards

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Disclosure of conflict of interest

No conflict of interest.

References

[1] Lai CC, Shih TP, Ko WC, Tang HJ, Hsueh PR. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and corona virus disease-2019 (COVID-19): the epidemic and the challenges. Int J Antimicrob Agents. 2020;17:105924.

[2] Karimov, B., Mukhamedova, M., Ubaydullaeva, Z., Uzokov, J., Nosirova, G., & Ubaydullaev, S. (2022, June). Blood coagulation indexes after COVID-19 in patients with coronary artery disease. In European journal of clinical investigation (Vol. 52). 111 RIVER ST, HOBOKEN 07030-5774, NJ USA: WILEY.

[3] Kayumov N. et al. Circulating exosomal biomarkers in patients with coronary artery disease and metabolic syndrome // European journal of clinical investigation. – 111 RIVER ST, HOBOKEN 07030-5774, NJ USA : WILEY, 2022. – T. 52.

[4] Novel CP. The epidemiological characteristics of an outbreak of 2019 novel coronavirus diseases (COVID-19) in China. Zhonghua Liu Xing Bing Xue Za Zhi. 2020;41(2):145.

[5] Orziev D. et al. Features of platelet induces in patients with atherosclerotic coronary artery disease // European journal of clinical investigation. – 111 RIVER ST, HOBOKEN 07030-5774, NJ USA : WILEY, 2022. – T. 52.

[6] Payziev D. D. et al. Influence of diet with low glycemic index on anthropometric parameters in patients with atherosclerotic coronary artery disease // European Journal of Cardiovascular Nursing. – 2022. – T. 21. – №. Supplement_1. – C. zvac060. 023.

[7] Uzokov, J., Mukhamedova, M. G., Narzullaeva, D. S., Nosirova, G. A., Orziev, D. Z., & Majidov, I. (2022). Influence of sacubitril/valsartan on diastolic parameters in patients with chronic heart failure with reduced ejection fraction. European Journal of Cardiovascular Nursing, 21(Supplement_1), zvac060-017.

[8] Ng TM, Toews ML. Impaired norepinephrine regulation of monocyte inflammatory cytokine balance in heart failure. World J Cardiol. 2016;8(10):584–589.

[9] Klok FA, Kruip MJ, Van der Meer NJ, Arbous MS, Gommers DA, Kant KM, Kaptein FH, van Paassen J, Stals MA, Huisman MV, Endeman H (2020) Confirmation of the high cumulative incidence of thrombotic complications in critically ill ICU patients with COVID-19: an updated analysis. Thromb Res 191:148–150.

[10] Paranjpe I, Fuster V, Lala A, Russak A, Glicksberg BS, Levin MA, Charney AW, Narula J, Fayad ZA, Bagiella E, Zhao S (2020) Association of treatment dose anticoagulation with in-hospital survival among hospitalized patients with COVID-19. J Am Coll Cardiol 76(1):122–124.

[11] Ruan Q, Yang K, Wang W, Jiang L, Song J (2020) Clinical predictors of mortality due to COVID-19 based on an analysis of data of 150 patients from Wuhan, China. Intensive Care Med 46(5):846–848.

[12] Mukhitdinova O. et al. Changes of blood D-dimer level after COVID-19 in patients with coronary heart disease //European Heart Journal: Acute Cardiovascular Care. – 2022. – T. 11. – №. Supplement_1. – C. zuac041. 136.

[13] Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, Zhang L, Fan G, Xu J, Gu X, Cheng Z. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497–506.

[14] Uzokov, J., Alyavi, B., Alyavi, A., Orziev, D., Sobirova, G., & Karimov, M. (2022). Features of characteristics of metabolic syndrome components in COVID-19 patients associated with nonalcoholic fatty liver disease. Hepatology International, S287-S287.

[15] Salah, H.M., Fudim, M., O'Neil, S.T. et al. Post-recovery COVID-19 and incident heart failure in the National COVID Cohort Collaborative (N3C) study. Nat Commun 13, 4117 (2022).

[16] Abbasi J. The COVID Heart—One Year After SARS-CoV-2 Infection, Patients Have an Array of Increased Cardiovascular Risks. JAMA. 2022;327(12):1113–1114.

[17] Grigoricheva E., Gessen G.R, Features of chronic heart failure in COVID-19 pandemic, European Heart Journal, Volume 42, Issue Supplement_1, October 2021, ehab724.0758,

[18] Araiza-Garaygordobil D, Montalto C, Martinez-Amezcua P, et al. Impact of the COVID-19 pandemic on hospitalizations for acute coronary syndromes: a multinational study. QJM. 2021;114(9):642-647.

[19] Inciardi RM, Lupi L, Zaccone G, Italia L, Raffo M, Tomasoni D, et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). JAMA Cardiol. (2020) 5:819–24.

[20] Basso C, Leone O, Rizzo S, De Gaspari M, van der Wal AC, Aubry MC, et al. Pathological features of COVID-19-associated myocardial injury: a multicentre cardiovascular pathology study. Eur Heart J. (2020) 41:3827–35.

[21] Bradley BT, Maioli H, Johnston R, Chaudhry I, Fink SL, Xu H, et al. Histopathology and ultrastructural findings of fatal COVID-19 infections in Washington State: a case series. Lancet. (2020) 396:320–32.

[22] Oprinca GC, Muja LA. Postmortem examination of three SARS-CoV-2-positive autopsies including histopathologic and immunohistochemical analysis. Int J Legal Med. (2021) 135:329–39.

[23] Dolhnikoff M, Ferreira Ferranti J, de Almeida Monteiro RA, Duarte-Neto AN, Soares Gomes-Gouvêa M, Viu Degaspare N, et al. SARS-CoV-2 in cardiac tissue of a child with COVID-19-related multisystem inflammatory syndrome. Lancet Child Adolesc Health. (2020) 4:790–4.

[24] Zhou R. Does SARS-CoV-2 cause viral myocarditis in COVID-19 patients? Eur Heart J. (2020) 41:2123.