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Features of the myocardial infarction course in convalescents of the new coronavirus infection COVID-19

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Abstract

Acute myocardial injury is one of the possible complications of the new coronavirus infection COVID-19 and can be diagnosed both in the acute phase of the infection, and also after stabilization or clinical improvement of the patient's condition. This review is devoted to the actual problem of the acute myocardial infarction development during the period of COVID-19 convalescence. The pathophysiological mechanisms of acute myocardial infarction during recovery from COVID-19 are diverse. The key role belongs to the state of hypercoagulation and the systemic inflammatory response, which can provoke destabilization and rupture of unstable atherosclerotic cardiac plaques. Type 2 acute myocardial infarction is most often diagnosed in COVID-19 convalescents against the background of intact coronary arteries. In this case, acute myocardial infarction develops due to an imbalance between increased myocardial oxygen demand (increased levels of cytokines in the blood, hypercatecholaminemia, hyperthermia

and tachycardia) and a decrease in oxygen supply to cardiomyocytes due to hypoxemia and hypotension. Subclinical myocardial injury may be present despite normal coronary arteries on angiography and intact left ventricular ejection fraction. There is no clear relationship between the development of acute myocardial infarction during the period of COVID-19 convalescence and the severity of the infection, the time from the initial diagnosis, and the presence of traditional risk factors for cardiovascular diseases in the patient. Cardiac complaints in COVID-19 convalescents are often interpreted as a post-COVID syndrome, especially given the absence of a history of coronary heart disease in many patients, which can lead to late diagnosis. Further study of the features of acute myocardial infarction during the period of COVID-19 convalescence is needed.

Keywords: COVID-19, convalescence, acute myocardial infarction

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AMI, acute myocardial infarction

CA, coronary artery

Introduction

The novel coronavirus infection COVID-19 caused by the SARS-CoV-2 virus is known to primarily affect the respiratory system and has clinical manifestations ranging from asymptomatic and signs of mild respiratory illness to the development of severe viral pneumonia,

respiratory distress syndrome, respiratory failure, and death [1]. Meanwhile, clinical and pathologist anatomical studies indicate frequent heart damage in patients with COVID-19, which significantly increases the risk of complications and poor outcome of new coronavirus infection [2, 3].

We should note that cardiovascular diseases can develop in any period of COVID-19 course; however, their risk increases from the 15th day from the onset of SARS-CoV-2 infection; in addition, they can be diagnosed after the stabilization or clinical improvement and recovery of the patient [3]. One possible complication in COVID-19 survivors is acute myocardial infarction (AMI) requiring emergency care. According to a large Swedish study of 86,742 patients with COVID-19 and 348,481 controls, the risk of MI (odds ratio, OR) makes 2.89 (95% CI [1.51;5.56]) within 1 week after infection, 2.53 (95% CI [1.29;4.94]) during week 2, and 1.60 (95% CI [0.84;3.04]) during weeks 3 and 4 [4]. However, to date there are no published results of any original studies related to investigating AMI peculiarities in COVID-19 convalescents. The presented literature review is devoted to this issue.

The aim was to identify the features of the acute myocardial infarction course in COVID-19 convalescents, based on a sample from Google Scholar search databases, PubMed, and Cyberleninka for the period from 2020–2022.

Patients who suffered COVID-19 had not been vaccinated. In the course of the study, the risk factors and timing of acute myocardial infarction in COVID-19 convalescents within 6 months were reviewed.

Pathophysiological mechanisms of acute myocardial infarction in COVID-19

A large number of pathophysiological mechanisms for the AMI development have been identified both in the acute period of coronavirus infection caused by SARS-CoV-2 and in the convalescence period. They were as follows: direct viral cardiomyocyte damage mediated by angiotensin-converting enzyme 2 receptor; destabilization of atherosclerotic coronary plaque; endothelial dysfunction; hypercoagulation state; systemic inflammatory response; vasculitis; hypoxia [2, 5, 6]. The infectious diseases, including viral pneumonia, have been established to provoke a disagreement between the heart's need for oxygen and its supply [7], and also be prothrombotic risk factors that contribute to the instability of atherosclerotic plaques [8]. According to autopsy, fibrin-rich microthrombi and microangiopathic pathology are the main causes of COVID-19-associated myocardial necrosis [9]. At the same time, myocardial pathology can be caused by acute ischemic injury due to thrombosis of the epicardial coronary arteries (CAs), or by the microvascular obstruction due to microthrombi, and in one way or another be associated with an increased risk of mortality [10].

D. Pellegrini et al. (2021) in a study of 40 hearts of patients who died from COVID-19 found the signs of heart damage that were present in 35% of cases in the form of small areas of infarction and cardiomyocyte necrosis foci, which arose primarily due to multiple thromboses in small vessels [11]. Further analysis of these thrombi showed that they were composed of a large amount of fibrin and C5b-9 terminal complement, suggesting an immune-mediated reaction. The thrombogenic nature of AMI during recovery from COVID-19 infection was confirmed by increased rates of hypercoagulation in patients without previous risk factors for cardiovascular disease [12].

In addition to the hypercoagulation state, an influential factor predisposing to the AMI development is the inflammation that continues in the convalescence phase of COVID-19. In a series of studies conducted in Germany, V.O. Puntmann et al. (2020) used magnetic resonance imaging and found heart damage in 78% of 100 patients who recovered from COVID-19, and ongoing myocardial inflammation in 60% of cases [13]. It is noteworthy to note that the identified changes did not depend on the history of cardiovascular pathology, the severity and characteristics of the COVID-19 course, or the period from the initial diagnosis.

Systemic inflammation can provoke destabilization and rupture of unstable atherosclerotic cardiac plaques; and the hypercoagulation state contributes to coronary thrombosis, resulting in type 1 AMI [6]. However, the most common coronary artery disease during COVID-19 convalescence is manifested by type 2 AMI due to an imbalance between increased myocardial oxygen demand (increased cytokine levels, hypercatecholaminemia, hyperthermia, and tachycardia) and decreased oxygen supply to cardiomyocytes due to hypoxemia and hypotension [14]. Heart disease, which is regarded as type 2 AMI, is identified in 7-30% of patients with COVID-19 [15].

Features of coronary artery lesions and clinical characteristics of acute myocardial infarction

In a study by G. G. Stefanini et al. (2020), among 28 patients with COVID-19 who had a typical clinical pattern of AMI, 17 cases (60.7%) showed signs of coronary occlusion requiring revascularization, while in 11 patients (39, 3%), there was no lesion of the coronary artery in angiography results [5]. Echocardiography revealed local disorders of myocardial contractility in 23 patients (82.1%), diffuse hypokinesia in 3

patients (10.7%); and there were no contractility disorders in 2 patients (7.1%). In more than half of the cases (17 patients, 60.7%), the left ventricular ejection fraction was less than 50%.

Other authors also pay attention to the absence of CA obstruction in patients with AMI against COVID-19; meanwhile, the patients have typical retrosternal pain, abnormal findings in the electrocardiogram, and increased cardio specific enzymes. So, S. Bangalore et al. (2020) presented the clinical case series of 18 patients with COVID-19 and ST segment elevation AMI [16]. In 56% of patients, no hemodynamically significant stenoses of the coronary artery were detected in coronary angiography results.

In a number of cases, the classic clinical manifestations of AMI during the period of convalescence from new coronavirus infection are combined with persistent respiratory symptoms (cough, shortness of breath, chest pain, loss of smell, etc.) and asthenic syndrome, which are primarily interpreted as a post-Covid state, making it difficult to timely diagnose acute myocardial damage [17]. Blood levels of trooping are known to play an important role in establishing the diagnosis of AMI. However, in patients with COVID -19, an increase in this parameter may be due both to AMI, and also to the presence of viral myocarditis, microangiopathy, and subclinical course of coronary heart disease [18].

At the same time, many patients with AMI have no history of chronic coronary heart disease. A. Kini et al. (2021) found that individuals without a history of coronary heart disease, as well as patients under 65 years of age, have a worse prognosis in the development of AMI [19]. Thus, among patients under the age of 65, acute myocardial injury was associated with an almost two-fold increase in the risk of mortality compared to patients with chronic myocardial injury. As a possible explanation for this fact, the authors suggested a protective role of

antithrombotic drugs used as secondary prevention of acute coronary events in patients with an established diagnosis of coronary heart disease. It should be noted that the highest risk of death in acute myocardial injury occurs in the first 6 months after suffering COVID -19.

Currently, there is no data indicating a clear relationship between the development of AMI and the severity of COVID-19. So, according to T. Kotecha et al. (2021), among 148 patients with severe COVID-19 and elevated troponin levels, AMI was detected in 28 cases (19%) at average 68 days after discharge from the hospital [20]. Meantime, the function of the left ventricle was normal in 89% of patients (mean ejection fraction values were 67%±11%). At the same time, the cases of the AMI development were described in patients with a mild course of infection treated on an outpatient basis [21].

Considering the difficulties of differential diagnosis of chest pain in COVID-19, the frequent absence of coronary artery lesions at coronary angiography, and the absence of data on chronic coronary heart disease in patient's medical history, some authors suggest paying more attention to imaging techniques, in particular, magnetic resonance tomography of the heart for diagnosing AMI [21].

Conclusion

The risk of developing acute myocardial infarction does not disappear after the acute phase of the new coronavirus infection COVID-19, but persists in the following months, which requires cardiological vigilance at patient follow-up during the convalescence period. The development of acute myocardial infarction in COVID-19 convalescents is a multifactorial process, often not associated with traditional risk factors for cardiovascular disease or the severity of the infection and the time of initial diagnosis. The pathomorphological feature of acute

myocardial infarction in patients recovering from COVID-19 is the development of predominantly acute type 2 myocardial infarction against the background of intact coronary arteries. We should remember that subclinical myocardial injury may be present despite angiographic evidence of normal coronary arteries and a preserved left ventricular ejection fraction. It follows from this that the use of less invasive techniques for diagnosing coronary pathology may allow avoiding the use of invasive interventions, since the specificity of this patient category lies in the fact that they often look intact at coronary angiography. Thus, the peculiar features of the acute myocardial infarction course during convalescence of COVID-19 patients require further studying.

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