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Cardiac Arrhythmias in COVID-19 Infection

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ABSTRACT

COVID-19 infection is a viral disease that affects the whole world with a markedly high risk of morbidity and mortality. This disease affects many systems. Cardiovascular system involvement has an important place among these systems. Arrhythmias have an important place in cardiovascular system involvement. Viral toxicity, secondary complications and drugs are involved in the occurrence of arrhythmias. Correction of the underlying mechanism plays an important role in treatment. Because of this importance, we present cardiac arrhythmia in COVID-19 infection.

KEYWORDS

COVID-19 infection; Arrhythmia; Morbidity; Mortality

INTRODUCTION

An epidemic of pneumonia, which is thought to have developed due to a new coronavirus, was detected in Wuhan, Hubei province of the People's Republic of China, in December 2019, and this epidemic could not be brought under control, and soon became a European continent that would cause a pandemic first in other provinces of China, spread around the world [1]. The causative virus has been named as "Serious Acute Respiratory Syndrome-Coronavirus-2" (SARS-CoV-2) and the disease it causes as COVID-19 by the World Health Organization [2,3].

This disease affects the lungs, causing pneumonia. However, it also has adverse effects on the heart. Adverse cardiovascular effects of this virus such as myocardial damage, thromboembolic events and fatal arrhythmias have been described [4]. In a study, arrhythmia was detected in 44% of individuals with severe COVID-19 disease [5]. Due to these reasons, we have mentioned, COVID-19 infection causes an increase in both morbidity and mortality risk. Because of this increased risk, we have presented this article.

ARRHYTHMIA MECHANISMS IN COVID 19 INFECTION

SARS-CoV-2 virus enters the cell by interacting with ACE-2 and TMPRSS2 receptors on the surface of type 2 pneumocytes. The main mode of entry into the cell is endocytosis. It replicates within the cell by the RNA-dependent RNA polymerase enzyme. It also enters the cells in the layers of the heart by the same mechanisms.

It can cause arrhythmia with hypoxia by causing acute respiratory distress syndrome in the lungs. Hypoxia leads to change in the depolarization threshold, tissue anisotropy and increment with reduced electrical coupling. Myocarditis may develop with virus entry into the myocardial cell and immune cell-mediated damage. The cytopathic effect and post-inflammatory myocardial scar play an important role in the physiopathology of myocarditis. Subsequently, arrhythmias may develop with electrical imbalance and re-entry. Thrombosis,

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disseminated intravascular coagulation and pulmonary embolism can cause pulmonary hypertension by increasing right heart pressures. Also, thrombosis can cause myocardial ischemia. Microvascular dysfunction, presence of atherosclerosis, increased heart rate and increased contractility can cause myocardial ischemia. Subsequently, changes in ion haemostasis, loss of intercellular connections, focal electrical activity, and conduction disturbances lead to arrhythmia. Activation of the cardiac sympathetic system, immune cell mediated damage, and IL-6 inhibition of cytochrome p450 can cause myocarditis and myocardial ischemia. Azithromycin and hydroxychloroquine may prolong OT. These drugs prolong OT by early after depolarization and hERG-K channel inhibition. Diarrhoea and acute renal injury may cause arrhythmias with intravascular volume imbalance and electrolyte imbalance [6-12].

ARRHYTHMIAS AND TREATMENT

Autonomic dysfunction in COVID-19 infection can cause sinus tachycardia, postural orthostatic tachycardia, and supraventricular tachycardia. Secondary causes such as pulmonary embolism, anaemia, hypotension, hyperthyroidism, and fever in unexplained inappropriate sinus tachycardia should be excluded. Holter electrocardiography and event recorders can be used in the diagnosis of inappropriate sinus tachycardia. While beta blockers have been found useful in the treatment, the benefit of ivabradine is not clear. Tilt table test can be used in the diagnosis of postural orthostatic tachycardia syndrome. These patients should take a break from competitive sports for 3-6 months after normalization of troponin, normalization of imaging methods, and resolution of infection. Secondary causes of supraventricular tachycardia should be ruled out. However, holter electrocardiography and event recorders can be used in diagnosis. Anticoagulant treatment should be initiated in the patient with Covid-19 infection. Radiofrequency catheter ablation can be performed. However, it is more appropriate to postpone the ablation for 3-6 months after infection in this process [13,14].

The most common arrhythmias in COVID-19 infection are atrial arrhythmias. Systemic inflammation can cause atrial fibrillation and atrial flutter. In addition, hypoxia, metabolic disorders, electrolyte disturbances, myocardial ischemia and proarrhythmic drug effects may cause atrial arrhythmias. Beta blockers should be used with caution in patients with atrial fibrillation and atrial flutter if restrictive and constrictive pulmonary lung disease is present. Attention should be paid to the use of Amiodarone in those who develop fibrotic lung disease. Protective equipment should be used if procedures such as intubation and transesophageal echocardiography will be performed to increase contamination. Cardiac computed tomography can be used as an alternative to rule out thrombus before cardioversion. We recommend the use of anticoagulants in relation to COVID in these patients [15].

Atrioventricular nodal injury may cause atrioventricular block. Bradycardia may occur due to the use of hydroxychloroquine, lopinavir/ritonavir and azithromycin. Bradycardia due to prone position or tracheal secretion can be seen, leading to temporary increase in vagal activity. Pacemaker replacement should be performed in patients with symptomatic bradycardia, patients with high-grade atrioventricular block, or in patients with complete atrioventricular block [16,17]. However, secondary causes such as drug use, hypothyroidism, and pulmonary embolism should be excluded in these patients.

Thrombosis and myocarditis can lead to ventricular tachycardia or ventricular fibrillation. Ventricular tachycardia or ventricular fibrillation may be due to secondary causes such as acute coronary syndrome, acute pulmonary embolism, cerebrovascular events, and drug use. Beta blockers should be used with caution in

terms of hypoxia due to bronchospasm. In addition, magnesium infusion can be used in the presence of Torsades de pointes tachycardia. Coronary reperfusion and intravenous beta blocker are very important, especially in polymorphic ventricular tachycardia, which is thought to be due to acute coronary syndrome [18]. There is insufficient evidence on implantable cardioverter defibrillators.

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