World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-16, November 2022
ISSN: 2749-3644

POSSIBLE EFFECTS OF CORONAVURUS INFECTION (COVID-19) ON THE CARDIOVASCULAR SYSTEM

Begijonova Dilfuza Tokhirovna, teacher
Ferghana Medical Institute of Public Health
Uzbekistan, Fergana

Article history:

Received: September 20th 2022
Accepted: October 24th 2022
Published: November 30th 2022

Abstract:

Acute viral respiratory infections can increase the risk of progression of a pre-existing condition, including a cardiovascular pathology. Life-threatening complications of Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) necessitate research into the cardiovascular effects of COVID-19 crucial for developing adequate treatment strategy for infected patients, especially those of advanced age. This article reviews the literature on the clinical and functional characteristics of patients with COVID-19, including those with poor outcomes.

Keywords: coronavirus, cardiovascular diseases, infection, severe acute respiratory syndrome, coronavirus infection 2019, angiotensin-converting enzyme, SARS-CoV-2, COVID-19

Coronaviruses are a family of viruses that includes, as of 2020, 40 types of RNA-containing viruses united into two subfamilies that affect humans and animals. The genome of coronaviruses (SOUS) is represented by single-stranded (+)RNAs with the ability to rapidly mutate and recombine. The name of the family is associated with the structure of the virus, the spiny processes of which resemble the solar corona. The purpose of the "crown" in coronaviruses is associated with their specific mechanism of penetration through the cell membrane by imitating "fake molecules" of molecules to which the transmembrane receptors of cells react. After the receptor captures a fake molecule from the "crown", it is pushed by the virus into the cell, and the RNA of the virus follows it.

Acute respiratory tract infections, including influenza, respiratory syncytial infection, bacterial pneumonia, are generally recognized triggers of cardiovascular diseases (CD), and the initial CD, in turn, are associated with other concomitant pathology and may increase the likelihood of the development and progression of the infectious process.

The appearance of severe acute respiratory syndrome against the background of coronavirus 2 (severe acute respiratory syndrome coronavirus 2, or SARS-CoV-2), causing coronavirus disease 2019 (Coronavirus disease 2019, or COVID-19), quickly turned into a pandemic, and, as reported, a significant proportion of patients affected by the virus have CD.

Prospective cohort observational study ARIC (atherosclerosis risk in communities study) showed that patients have a high probability of developing coronary heart disease (ISA) and stroke, especially 90 days after the infection process.

In this regard, pathophysiological changes occurring in the cardiovascular system as a result of the potential effects of coronavirus are of undoubted interest. Since information about the mechanisms of action of COVID-19 is still limited, the analysis of data from previous studies of outbreaks of viral pneumonia and acute respiratory syndrome in the Middle East (middle east respiratory syndrome Coronavirus, or MERS-CoV), as well as seasonal influenza, will help to get a more complete understanding of the mechanism of action of coronaviruses on the cardiovascular system. Understanding the cardiovascular effects of COVID-19 is essential for the development and provision of timely comprehensive medical care for patients, especially older people with CD.

Coronaviruses, which got their name because of the characteristic features of the structure (crown-like spikes on the surface of the virus), belong to the subfamily Coronaviridae, which has four groups: a, b, y and 5 CoVs by phylogenetic clustering, of which a and b cause infection in humans. Coronaviruses contain four main structural proteins: spike protein (S) (provides attachment to the host cell receptor and subsequent fusion of the virus with the cell membrane), nucleocapsid protein (N), membrane protein (M) and envelope protein (E).

Coronavirus was first identified in humans (HCoV) in 1965 in cultured tracheal tissues of a human embryo and until 2003 only two types of HCoV were recognized: HCoV-229E and HCoV-OC43.

Currently, seven different CoV strains have been found to infect humans, including HCoV-229E, HCoV-NL63, HCoV-OC43 and HCoV-HKU1, which usually cause self-resolving symptoms. In addition, coronavirus can cause severe acute respiratory syndrome (SARS),

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Middle Eastern respiratory syndrome (MERS-CoV) and lethal acute respiratory syndrome in humans, which is caused by the recently identified SARS-CoV-2.

Four types of HCoV, including HCoV-229E (a-CoV), HCoV-NL63 (a-CoV), HCoV-OC43 (β-CoV) and HCoV-HKU1 (β-CoV), are endemic to humans and usually cause mild respiratory infection with self-resolving symptoms, which accounts for 15-30% acute respiratory diseases (ARI). As a rule, this type of infection occurs in young people, but in older age, especially in patients with cardiovascular and bronchopulmonary pathology, it can cause hospitalization, including emergency.

Subsequent studies, including those conducted on wild animals, proved that SARS-CoV could develop in bats, after SARS-like CoV was identified in Chinese horseshoe bats with high similarity of nucleotide sequences with those of SARS-CoV isolated from humans (87-92%). Presumably palm civets and raccoon dogs acted as an intermediate host for the amplification of SARS-CoV before transferring it to other animals during contact with them on the market. Transmission of SARS-CoV occurs mainly from person to person in close contact, the mechanism is airborne (aerosol-aerogenic).

There is an opinion about the existence of a fecal-oral mechanism of transmission of SARS-CoV-2, since patients infected with SARS and MERS during their outbreak often had gastrointestinal symptoms in the form of diarrhea and abdominal pain, and SARS-CoV RNA was detected in the feces of 14.6% of patients with SARS and MERS. In some patients, the disease began with fever and diarrhea before the development of pronounced respiratory symptoms. In vitro studies have shown that MERS-CoV can infect and replicate in human intestinal epithelium by acting through the dipeptidyl peptidase-4 receptor. In vivo studies revealed the development of inflammation and epithelial degeneration in the small intestine, followed by the development of pneumonia, confirming that MERS-CoV lung infection was secondary to intestinal infection.

The ability of an infected patient to transmit the virus to other people is determined by a basic reproduction estimate of R0. R0 for SARS-CoV is about 3, i.e. a person with SARS-CoV is likely to infect three other people in a susceptible population, for comparison, the average R0 for seasonal influenza (swine flu, H1N1) is about 1.3.

To date, there is no vaccine or effective drug against SARS-CoV. Treatment of SARS includes supportive symptomatic therapy and the administration of broad-spectrum antimicrobials for the treatment of secondary bacterial infection. Older age (especially 60 years and older), multimorbidity (diabetes mellitus, CD, oncological diseases, COPD), high levels of lactate dehydrogenase serve as predictors of mortality in the presence of SARS-CoV. A number of authors note the absence of significant morbidity and mortality among children and adolescents during previous outbreaks of SARS-CoV.

Among the main symptoms of SDS-19 are fever, cough, a feeling of lack of air (shortness of breath, rapid breathing). Myalgia, anorexia, nausea, weakness, sore throat, nasal congestion, headache are less common. Symptoms may appear after 2 days or by the 14th day after contact with the patient. The detectable viral load is the same in patients with symptoms and without symptoms of SBU-19, which suggests the potential possibility of transmission of the virus from an asymptomatic or low-symptomatic patient to another person. The greatest viral load was noted in the nasal region compared to the pharynx. The diagnosis is confirmed by a polymerase chain reaction, the material for which is scraping from the mucosa of the upper and lower respiratory tract. The case of SOW-19 is considered confirmed with a positive result of laboratory testing for the presence of RNA 8A8-SoW-2, regardless of clinical manifestations. New serological tests that are convenient and possible to use at home are likely to appear in the near future.

The danger of ARVI is associated with the fact that during epidemics, mortality from chronic somatic diseases increases significantly, especially in the group of patients with cardiovascular diseases.

The hypothesis that influenza can act as a provoking factor of acute cardiovascular events and death was proposed in the 1930s. At that time, for the first time, a link was noted between seasonal influenza virus activity and higher mortality from all causes, including bronchopulmonary pathology, pulmonary tuberculosis, diabetes mellitus, organic heart pathology and hemorrhagic stroke [18].

The lessons of previous epidemics caused by coronaviruses suggest that viral infections can provoke the development of acute coronary syndrome, arrhythmias, decompensation of heart failure, thromboembolic complications mainly due to a combination of a significant systemic inflammatory response and localized inflammation of the vascular wall.

The severity and degree of clinical manifestations, short-term and long-term cardiovascular changes against the background of SSR-19, along with the effects of specific treatment, are not currently known and are subject to careful study. It should be noted that during influenza epidemics, most patients die more often from cardiovascular problems, and not from pneumonia caused by the virus. Taking into account the most powerful inflammatory load caused by COIU-19 and the previously presented clinical data on other coronavirus infections, significant cardiovascular complications can be expected against

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the background of COIU-19, the prevalence and severity of which is likely to be lower in outpatient, non-hospitalized patients.

Viral infection and virus-induced immune responses in most cases underlie the inflammatory process in myocarditis. Invasion into the target cell of a viral particle with a tropicity to the myocardium, the direct cytopathogenic effect of the virus and the inclusion of non-specific mechanisms of antiviral protection (implemented by macrophages and YK cells) are the leading mechanisms of myocardial damage in the acute phase of the disease. Activated macrophages and other cells of the immune system, through the production of chemokines, attract T- and B-lymphocytes to the focus of inflammation. The latter implement the mechanisms of cell—mediated cytolysis and ensure the production of antiviral antibodies - the mechanism of cardiomyocyte apoptosis with further systolic myocardial dysfunction is triggered.

Myocarditis is a polyetiological disease. It can be caused by viral and bacterial agents, as well as non-infectious factors. In more than 50% of cases, the development of myocarditis is caused by viruses, among which parvoviruses B19, enteroviruses Coxsackie A and B, ESNO viruses, rubella virus, adenoviruses, human herpes virus type 6, Epstein-Barr virus, cytomegalovirus, influenza virus, etc. have a special cardiotropicity.

The clinical picture of myocarditis is diverse and non-specific, and their diagnosis is based on a triad of anamnestic data: acute onset of the disease, association of onset / exacerbations of clinical manifestations / arrhythmias with infection, less than a year old. Additional criteria include systemic immune manifestations, a combination of cardiac arrhythmias and conduction disorders, and the positive effect of steroid therapy.

Acute cardiovascular complications (myocardial infarction, arrhythmias), decompensation of heart failure with a confirmed diagnosis of COVID-19, 27.8% developed acute cardiovascular complications that led to cardiac dysfunction and rhythm disturbances, and the combination of cardiovascular complications with an increase in highly sensitive troponin was associated with high mortality.

Although the exact pathophysiological mechanisms underlying myocardial injury have not been sufficiently studied, existing data indicate the presence of the SARS-CoV genome in the myocardium in 35% of patients with SARS. These data increase the likelihood of possible direct damage to cardiomyocytes by viruses. SARS-CoV-2 may have the same mechanism of action as SARS-CoV, since these two types of viruses are very close, but not identical in genome. The presence of a close association of high troponin levels with the level of CRP indicates the inflammatory genesis of myocardial damage as the disease progresses. Viral particles, spreading through the mucous membrane of the respiratory tract and penetrating into the cells of the body, can cause a cytokine storm due to a violation of the balance of TM and T1t2 and a series of immune reactions leading to myocardial damage. The release of cytokines against the background of infection can cause a decrease in coronary blood flow, oxygen delivery, destabilization of atherosclerotic plaques and microthrombosis.

Myocarditis often manifests cardiac arrhythmias with the phenomena of progressive heart failure and sudden cardiac death, which can occur at any stage of the disease.

The first manifestations of myocarditis include weakness, fatigue, myalgia, occasionally subfebrility, which are caused not by the actual myocardial lesion, but by the manifestation of an infectious and inflammatory process. Sudden cardiac death due to ventricular tachycardia or ventricular fibrillation as a result of myocardial damage in the region of the cardiac conduction system, thromboembolic complications, syncopal conditions, cardiogenic shock and acute heart failure are also attributed to manifestations of myocarditis. The first clinical symptoms may occur on the background or a few days after the onset of ARVI.

Modern diagnosis of viral myocarditis has a number of difficulties. The main diagnostic criterion for myocarditis is the association of cardiac symptoms with a previous infection and the presence of signs of inflammation. In this case, a comprehensive clinical, laboratory and instrumental examination of the patient helps, as well as endomyocardial biopsy according to certain indications to exclude the inflammatory nature of heart damage.

Currently, according to the position of experts of the European Society of Cardiology, the American Heart Association and the Russian Society of Cardiology, patients with ESRD-19 who previously used ACE / ARA according to indications, it is necessary to continue taking them due to the lack of evidence for their ineffectiveness in this category of patients.

Existing data indicate a high incidence of concomitant pathology in patients with COVID-19 of middle and older age. Among cardiovascular diseases, hypertension (about 15%), diabetes mellitus (7.4-20%) and coronary heart disease (about 2.5%) prevail. Patients with COVID-19 and cardiovascular comorbidity have a high probability of developing SARS, septic shock and death. Acute cardiac dysfunction and SARS are considered as predictors of an unfavorable prognosis in patients with COV-19.

It is necessary to further study the features of screening, diagnosis, clinical manifestations, prevention and treatment in patients with COVID-19. As the disease spreads and new data become available, it is

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Volume-16, November 2022
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advisable to determine the risk factors for the development of cardiovascular complications in such patients.

It is possible that maintaining a register of patients with COVID-19 and systematic registration of clinical parameters, cardiovascular and other complications will allow us to determine the current characteristics of patients, approaches to treatment and prevention for the development of a model of the risk of complications.

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