Impact of COVID-19 on cardiovascular diseases: considerations for patients and healthcare systems

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Abstract

Coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). It mostly presents with fevers and respiratory symptoms that can progress from simple pneumonia to severe acute respiratory distress syndrome (ARDS). It also has significant implications for the cardiovascular care of patients. Troponin, which is a sign of cardiac injury, usually goes up in patients who are hospitalized, and it requires careful consideration. Microvascular activation by cytokines might cause myocardial damage, and it can harm other organs usually involved in COVID-19, such as lungs and kidneys. COVID-19 infection is also associated with venous thromboembolism, arrhythmias, and myocarditis. On the other hand, the cardiovascular care provision might position workers in the healthcare system in vulnerability once they become hosts or vectors for the transmission of coronavirus. In this study, we have investigated the articles on cardiovascular considerations concerning COVID-19 and then introduced the gaps which require more investigation pertinent to patients and healthcare systems.

Keywords: COVID-19; SARS-CoV-2; cardiovascular disease; atherosclerosis; healthcare

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a new strain of coronavirus, causes coronavirus disease 2019 (COVID-19) [1, 2]. It was first reported in China in late 2019 and spread rapidly worldwide [3-5]. The symptoms of COVID-19 are dry cough, high temperature, headache, tiredness, shortness of breath, loss of taste or smell, and gastrointestinal symptoms such as abdominal pain, anorexia, nausea, diarrhea. Increased liver enzyme and low counts of lymphocytes (lymphocytopenia) along with increased C-reactive protein (CRP) levels existed in patients with COVID-19 [5-7]. This disease could eventually lead to acute respiratory distress syndrome (ARDS) [6].

It was originally thought novel coronavirus was mainly a respiratory disorder, but as a great number of patients received the virus, it became obvious that it has many other physiological manifestations. The influence of COVID-19 goes well beyond the lungs to impact the cardiovascular system and cause complications in the brain, kidneys, and other organs. Based on reports that showed a strong relationship between increased D-dimer levels and weak prognosis, concerns have appeared about thrombotic complications in COVID-19 patients [8-10]. The National Institute for Public Health of the Netherlands suggests a respiratory failure in COVID-19 is not caused only by the development of acute respiratory distress syndrome, but that microvascular thrombotic processes might have an impact [11].

Treatment of COVID-19 infections consisted of new, standard, and experimental therapies. Particular antiviral, anti-inflammatory, immunosuppressive, and cell-based therapies carry on to change [12]. Nonetheless, it is suggested that a considerable number of patients who survive COVID-19 and have verification for myocardial issues at the time of the first presentation might have lasting overt or subclinical myocardial or electrophysiological disorders. It is not known whether the intensity of the cardiopulmonary injury, treatment type, or other factors impact lasting sequelae. Heightened observation is, accordingly, validated in these patients with consideration of foremost prevention approaches. Although the COVID-19 infection is novel, there have been many case series and review articles on cardiovascular complications during the COVID-19 infection. In this paper, the potential cardiovascular complications that might happen during the

recovering phase in the COVID-19 survivors, as well as considerations for the health system, are discussed.

Severe Acute Respiratory Syndrome Coronavirus

The Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) outbreak started in the Guangdong Province, southern China in 2002, and was most probably connected to a zoonotic event in the market of wild animals [13-15]. The SARS-CoV is one of the β -CoVs group viruses and ties to the zinc peptidase angiotensin-converting enzyme 2 (ACE2), a molecule that is put on the endothelial cells of artery and veins surfaces, epithelia of the small intestine, respiratory tract epithelium, respiratory tract epithelium, arterial smooth muscle, and immune cells, to enter the host cell [16]. Subduing of ACE2 expression during the infection of SARS-CoV has been suggested to have an impact on the pathologic variations in the lung and cause severe pneumonia and acute lung failure seen with this virus [17-20].

More investigations of wild animals show robust evidence that SARS-CoV may have come from bats when a SARS-like CoV was found in Chinese horseshoe bats with a sequence resemblance of 87% to 92% with human SARS-CoV [21]. It has also been alleged that raccoon dogs and palm civets prepared the intermediate amplification host for SARS-CoV before spreading it to animal handlers in the animal market [22, 23]. The spread of SARS-CoV is mainly from people to people who are in contact, through respiratory droplets, with an incubation period of 2 to 14 days after exposure [5]. The SARS-CoV might be spread into the atmosphere and transmitted from surfaces to the hands of people who are patient and health care system clinicians. In this regard, the synergic effects of anionic surfactants on coronavirus have been studied to improve the virucidal efficiency of commonly used sanitizers [24]. Transmission of infection can be performed via contact with the eyes, nose, and mouth. The ability of a patient who is infected to transmit the virus to other people is assessed by R_0 (i.e., R naught: basic reproduction number). The approximate R_0 for SARS-CoV is about 3, that means each person with SARS-CoV is anticipated to be able to infect three other individuals in a vulnerable population [25].

Atherosclerotic lesion

Arteriosclerosis happens when the blood vessels that transfer oxygen and nutrients from heart to the remainder of the body (arteries) become stiff and thick. It often limits blood flow to the tissues and organs. Healthy arteries are pliant and elastic, but when time passes, the walls in the arteries could stiffen, a situation usually called hardening of the arteries. Atherosclerosis refers to the growth of cholesterol, fats, and other substances in and on the artery walls (plaque), that can limit the blood flow [26]. Atherosclerosis mostly forms in left main coronary bifurcation to left anterior descending (LAD) and left circumflex (LXC) branches [10]. The plaque in the arteries, can rupture, prompting a blood clot. Although atherosclerosis is mostly considered a heart issue, it could impact the arteries anywhere in the body. Clinically, given the expansion of the arteries, symptomatic atherosclerosis is usually related to males in their 40s and females in their 50s to 60s [27]. Sub-clinically, the illness starts to appear in childhood and seldom is already present at birth. Perceptible signs could start developing at puberty. Though symptoms are seldom shown in children, early screening of children for cardiovascular illnesses can be useful to the children and also their relatives [28].

The demographic mostly affected by lethal COVID-19 has a high prior likelihood of formed atherosclerotic lesions: they are old people, mainly male, and have underlying lung disease, including that related to cigarette smoking, a risk factor for atherosclerosis. Distant infections such as the severe pneumonitis that too frequently complicates COVID-19 could obtain an acute irritation of the chronic smoldering inflammation that marks coronary atherosclerotic lesions [29]. The inflammatory cells at a location of zonal infection such as the lungs in COVID-19 pneumonitis could cause cytokines such as interleukin-1 and -6 and tumor necrosis factor to be produced, mediators that not only disseminate regional inflammation but could enter the systemic circulation [30]. These circulating cytokines could prompt macrophages in the plaque to increase the production of regional cytokine and arouse a growth in tissue factor expression that makes lesions more thrombogenic. We have referred to this local response to systemic stimuli as an “echo” event. These same systemic cytokines could trigger leukocyte adhesion molecule expression on the endothelial cells overspreading established atheroma, increasing regional recruitment of these inflammatory cells. These changes in underlying plaques could intensify their tendency to disturb,

be it by fibrous cap fissure or by external erosion, and stimulate an acute coronary syndrome [31, 32].

Sars-CoV-2 impacts on Cardiovascular System

A schematic of the impact of SARS-CoV-2 on the cardiovascular system is shown in Fig. 1. It has been observed that patients with coronary artery disease (CAD) are susceptible to develop adverse outcomes and death because of COVID-19 [33-35]. As shown in previous studies, for other acute infections, the hyperdynamic circulation resulted from COVID-19 in patients with liable factors for CAD might aggravate the dangerous balance by increasing the demand for myocardial oxygen, bringing about acute coronary syndrome (ACS) [36, 37]. ACS in patients who are infected might be caused by immoderate cytokines causing atherosclerotic plaque instability and rupture [33]. A case report on a man who was 70-year-old was published. He was cured three years before by percutaneous coronary intervention (PCI) and infected by COVID-19, who showed up with ACS and non-ST segment elevation myocardial infarction (NSTEMI). The angiogram of coronary depicted atherosclerotic spontaneous coronary artery dissection (A-SCAD) on the coronary artery and proximal left anterior descending (LAD) and in-stent restenosis of a marginal branch (OM) [38].

The impact of coronavirus on lipid metabolism and development of the atherosclerotic procedure is verified by an observational study carried out to examine long-term effects of infection of acute Sars-CoV [39]. Twenty-five patients were enlisted 13 years after healing from Sars-CoV in 2003. The metabolomic examination was conducted, depicting a changed lipid metabolism with remarkably elevated amounts of free fatty acids, lysophosphatidylethanolamine, lysophosphatidylcholine and phosphatidylglycerol. In addition, 44% of the patients were affected by CVD. Sars-CoV pathogenesis depicts a significant resemblance to COVID-19, being marked by an unusual hyperactivation of the immune system, causing extra amounts of cytokines [40, 41]. Hyperactivation of pro-inflammatory patterns, primarily identified by cytokine storm, might augment the possibility of restenosis in patients who experienced PCI with stent implantation because of CHD. Pre-operative elevated extents of IL-6, TNF- α , and IL-23 might efficiently

anticipate the risk of restenosis after PCI related to the implantation of drug-eluting stent (DES) [42].

In addition to ACS, the cardiovascular system might be complicated in patients affected by heart failure because of hemodynamic decompensation and, in a little percentage of patients, acute myocarditis might happen without earlier confirmation of CVD because of the presence of ACE2 on cardiac myocytes, potentially causing chronic dilated cardiomyopathy [43-46].

Figure 1. Schematic of the impact of SARS-CoV-2 on the cardiovascular system

Potentially, the long-term effects of cardiovascular system involvement, primarily because of hemodynamic changes, atherosclerotic development, and causing an increased risk of thrombosis because of COVID-19, might directly influence the function of left ventricular systolic and elevate retrograde pressure on the right cardiac chambers resulting in decompensation. Increased occurrence of deep vein thrombosis (DVT) events because of abnormal blood clotting might result in more pulmonary hypertension and pulmonary embolism events [47-49].

Conclusions and future directions

The COVID-19 disease has impacted thousands of patients who are suffering from cardiovascular diseases such as atherosclerosis and causes a significant health warning on an international scale. The management and handling of patients who are impacted by this disease are crucial in addition to providing continuousness of care to patients who are not infected with pre-existing CVD. In the upcoming months, attempts for evaluating new therapies would be important to the healing of coronavirus, and as this procedure progresses, more recognition of the complex interplay among COVID-19, CVD, and the different stakeholders who are involved including patients, and health care systems, would be vital to developing results in at-risk and patients who are infected [50-52]. Potential unarranged clinical trials and group studies are ongoing and would be vital to helping heal patients who are impacted by this coronavirus.

A number of theories exist regarding the elevated risk of adverse events for patients with CVD who develop COVID-19. Particularly, good apprehension of the association engaging the ACE2 protein, antihypertensive agent use, and COVID-19 prognosis would have crucial implications for patients with both CVD and COVID-19. Outside of the area of individual trials, concerted attempts by all workers in the health care system and insightful control are needed to assist in alleviating the health risk to the at large, in addition to workers in CV health care, as shown by the hard decision to cancel the Scientific Sessions of 2020 American College of Cardiology. Organized use of assets, such as using the telehealth capacities and appropriate adhesion to preventative population-wide and measures of health care worker-level, would enable the move from this critical time till the disease outbreak is contained.

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