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Review Article

Cardiovascular Complications in Respiratory Viral Infections with a Focus on COVID-19

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Abstract

Patients with respiratory viral infections have altered immune responses, which may predispose them to cardiovascular complications. In the face of the pandemic of a new kind of severe acute respiratory syndrome (SARS), coronavirus disease 2019 (COVID-19), there is a resurgence of interest in the early diagnosis, prevention, and treatment of patients who are at risk. COVID-19 often manifests as viral pneumonia, although extrapulmonary manifestations are also common. Acute cardiac damage associated with elevated high-sensitivity troponin levels crucially contributes to mortality in severe COVID-19. The present review clinically compares cardiovascular complications between COVID-19 and other respiratory infections caused by single-stranded RNA viruses, namely influenza, SARS, and Middle East respiratory syndrome (MERS). Estimating the death rate from RVIs has been a subject of intense research, but the mortality from cardiovascular complications in these infections is less understood and calls for further research.

Keywords: Cardiovascular; COVID-19; Influenza; MERS; Respiratory Viral Infections; SARS

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Introduction

Respiratory infections are among the most common diseases globally that humans have been struggling with for a long time. In December 2019, a new kind of severe acute respiratory syndrome coronavirus (SARS-CoV-2) (1-5) happened first in China and then in the rest of the world. It became a pandemic in a few months (6-11), with manifestations suggestive of multiple systems' involvement, particularly the cardiovascular system, nervous system, endocrine system, and skin, collectively regarded as coronavirus disease 2019 (COVID-19) (12-24).

Many microorganisms can cause respiratory infections; viruses are usually more challenging due to the lack of specific treatments. Viral infections can complicate and involve other organs if not treated properly. In this manner, cardiac complications, such as acute myocardial infarction (AMI) and myocarditis, are not uncommon in respiratory viral infections (RVIs). Moreover, they can cause cardiogenic shock and even death, which is especially important in the COVID-19 situation when more than six million deaths have occurred to humanity. Observational studies link laboratory-confirmed COVID-19 and cardiovascular complications (6, 25-37). Despite these facts, few studies have been performed, especially in the context of SARS-COV-2 infection (17, 38-45).As the world's population ages and diseases increase, so does the importance of subsequent complications. RVIs are a fundamental cause of serious adverse events in the elderly. They can trigger systemic inflammatory mechanisms, mainly involving proinflammatory cytokines, essential mediators of atherosclerosis that can directly affect plaque rupture. Additionally, they can have procoagulant effects, predisposing individuals to ischemia and thrombosis.

Respiratory Viral Infections Associated with Cardiovascular Complications

Coronaviridae

To date, six known HCoVs have emerged, namely HCoV-229E, HCoV-NL63, HCoV-OC43, HCoV-HKU1, SARS-CoV, and MERS-CoV; of which, HCoV-229E, HCoV-NL63, HCoV-OC43, and HCoV-HKU1, are among the viruses that can cause colds and multiply rapidly in the human population and cause respiratory infections (46).

HCoV-229E-like coronaviruses have been identified in bats, camels, and alpacas, indicating that transmission from bats to humans may have involved an intermediate host. The infection of humans with HCoV-OC43 and HCoV-229E dates back to the 1960s (47). HCoV-NL63 and HCoV-HKU1 first happened in 2004 and 2005 (46). The SARS-CoV first appeared in China in 2002–2003. The disease developed as atypical pneumonia with symptoms including fever, headache, and respiratory symptoms, which may turn into respiratory distress syndrome in the long run [18]. In 2005, a human coronavirus, HCoV-HKU1, was identified in Hong Kong (48). The MERS-CoV respiratory disease epidemic, with symptoms similar to SARS-CoV disease, first appeared in 2012 in Saudi Arabia. MERS-CoV had a higher mortality rate than SARS-CoV. The first MERS-CoV report was a patient who died of severe pneumonia and renal failure (49).

Studies have linked MERS-CoV and SARS-CoV with cardiovascular complications: MERS-CoV can cause heart failure and acute myocarditis, while SARS-CoV correlated particularly with acute coronary artery syndrome and myocardial infarction (50). Among hospitalized patients with SARS, two patients died of AMI. Another study found that of 121 hospitalized patients with SARS symptoms, 12 patients had cardiovascular disease; the most common heart problem was tachycardia. Other complications were bradycardia, hypotension, transient cardiomegaly, and transient paroxysmal atrial fibrillation in only one patient (51). Postmortem examinations in eight patients who died from SARS showed deep vein thrombosis in three patients and pulmonary thromboembolism in four patients. One patient had subendocardial infarction with occlusive coronary disease, and another patient had marantic and infarction in the heart (52).

Since December 8, 2019, Wuhan, Hubei Province, China has seen an increase in clusters of pneumonia cases of unclear cause (52). According to virologic studies on pneumonia patients, the causal agent of this pneumonia (COVID-19) is a new coronavirus (SARS-CoV-2) (1, 53). After increasing cases and the global spread of this virus, the World Health Organization (WHO) announced the new coronavirus pandemic as the

sixth most common cause of public health emergencies worldwide (54).

SARS-CoV-2 mainly attacks the lower respiratory tract, although there are signs that SARS-CoV-2 can severely affect other organs and cause serious injuries (53-55). After SARS-CoV-2 spread, cardiac damage was seen in the early instances of COVID-19 in Wuhan, China. Some SARS-CoV-2 patients who needed admission to the intensive care unit (ICU) had myocardial damage (associated with higher high sensitivity Troponin I (hs-cTnI) or new echocardiographic abnormalities) (56).

Orthomyxoviridae

The Orthomyxoviridae family contains Influenzavirus A, Influenzavirus B, Influenzavirus C, Influenzavirus D, Thogotovirus, Quaranjavirus, and Isavirus genera (57). Human influenza A and B viruses cause seasonal disease epidemics; influenza type C causes mild symptoms and cannot lead to epidemics; influenza D viruses mainly affect cattle and cannot cause human illness (58). Influenza viruses are spherical, enveloped particles with eight single-stranded, negative-sense RNA segments with intranuclear and intracytoplasmic replication (57). They have a lipid bilayer containing hemagglutinin (HA) and glycoprotein neuraminidase (NA). Influenza viruses are categorized by their hemagglutinin and their antigens of neuraminidase. Eighteen subtypes of hemagglutinin and 11 subtypes of neuraminidase had been identified for influenza A (HI-H18; NI-N11). There are theoretically 198 different influenza A subtype varieties; only 131 subtypes were found in nature (58). Mutation of the influenza virus genome may result in changes in the virus's surface proteins: HA and NA, called antigen drift. The HA and NA influenza virus surface proteins are "antigenic," so they may not be identified by the immune system leading to reinfection and annual influenza outbreaks by influenza A and B viruses. The influenza A virus undergoes an antigenic shift, resulting in the production of new HA and NA proteins. When the change occurs, most individuals lack protection from the new virus, resulting in a pandemic (59). **(Table 1)**

Objective

Here, we review cardiac complications of COVID-19, SARS, MERS, and influenza–known to induce worldwide outbreaks and cardiovascular manifestations.

Cardiovascular Complications in COVID-19

Common arrhythmias in relation to COVID-19

Sinus bradycardia

Sinus bradycardia is among the most discoursed arrhythmias concerning COVID-19. Kir et al. reported that bradycardia and sporadic high-level AV block were present in a COVID-19-infected patient with normal echocardiography and heart biomarkers (60). Sinus node malfunction was observed in two COVID-19 patients by Peigh et al., who claimed that events of accelerated idioventricular rhythm occurred after sinus bradycardia in these patients. Notably, sinus bradycardia persisted for two weeks in the patients after the incidence of sinus node dysfunction (61). According to the previous discussion, amongst 138 hospitalized patients with COVID-19-linked pneumonia in Wuhan in the report of Wang et al., arrhythmias occurred in 17% of the patients and, more frequently, in 44% of those accepted to an ICU (56). In New York, atrial arrhythmias were rated more amongst a cohort of 393 COVID-19 patients who required mechanical ventilation, with 17.7% and 1.9% reported rates in patients who received mechanical ventilation and non-invasive ventilation groups, respectively (62). Likewise, Colon et al. analyzed 115 hospitalized patients (69 accepted to medical ICU and 46 general medicine ward) and found newly occurred atrial tachyarrhythmia, namely atrial fibrillation, atrial tachycardia, and atrial flutter in 19 patients (16.5%), all of whom were hospitalized in the ICU (27.5% of ICU cases). Nevertheless, atrial arrhythmias were absent in patients hospitalized in the general medicine service (63). After a nationwide lockdown in Denmark, recorded newly occurred atrial fibrillation cases dropped by 47%. As reported in another study, atrial fibrillation was the highly prevalent heart arrhythmia present in COVID-19-diseased patients (64). The possible modes of action that could induce atrial fibrillation in such patients result from systemic infection, direct viral cardiomyocyte injury, hypoxemia, population sensitivity because of progressive age and comor-

Table 1. The main cardiac complications of influenza and deadly human coronaviruses

Disease	Cardiac Manifestation	
Influenza	Myocardial infarction Myopericarditis Myocarditis Pericarditis Arrhythmia Congestive heart failure Sudden death	
	Echocardiography	Local wall motion abnormality
SARS	Tachycardia Bradycardia Hypotension Cardiomegaly Pulmonary thromboembolism Deep vein thrombosis Sub endocardial infarction Atrial fibrillation Acute myocardial infarction Acute coronary syndrome	
	ECG	Non-specific ST depression T wave inversion
MERS	Acute myocarditis Heart failure	
COVID-19	Cardiac injury (Elevated high sensitivity Troponin I) Acute cardiac injury Myocardial injury myocarditis Cardiac shock Heart failure	
	ECG	ST-elevation MI Non-ST elevation MI Tachyarrhythmia
	Echocardiography	Left ventricular dysfunction

bidities, and, lastly, sympathetic nervous system excessive activity (65). In a report by Seecheran et al., a case of newly occurred atrial fibrillation and flutter was seen in a COVID-19 patient, additionally representing that the COVID-19 infection is atrial arrhythmogenic (66). Ultimately, extra arrhythmias (atrial and ventricular arrhythmias) have been reported in COVID-19 patients, with no record of arrhythmia. Among the earliest cohort, Gou et al. found ventricular tachyarrhythmias in 13 (7%) out of 187 hospitalized patients. Additionally, their report indicated that malignant arrhythmias, namely ventricular tachycardia/ventricular fibrillation, had a higher frequency in patients with high levels of troponin T than those with normal troponin T concentrations (11.5% vs. 5.2%) (67). Besides, available evidence indicates ventricular arrhythmias and torsade de pointes because of QT-prolonging medications, particularly azithromycin and hydroxychloroquine (68-71).

Ventricular arrhythmias

The dominant arrhythmia recognized in hospitalized COVID-19 patients is not the typical ventricular arrhythmias, and the reported estimates of non-continuous VT, VT, and VF comprise up to approximately 20% of the whole arrhythmias. Managing ventricular arrhythmias accounted for 7% of electrophysiology consultations at the peak of the epidemic in an inpatient cohort at a single center in New York City (72). The examined

causes of 136 in-hospital cardiac arrests in severely infected patients with COVID-19 pneumonia revealed that VT/VF was involved in merely 5.9% of patients (73).

Atrial arrhythmias

By reviewing sizable cohort data, atrial arrhythmias were obviously the most frequent arrhythmia throughout the severe stage of COVID-19. Recently, 3,970 admitted COVID-19 cases were studied retrospectively at Mount Sinai Hospital. The results showed the occurrence of atrial fibrillation/atrial aflutter (AF/AFL) in 10% of patients generally and in 4% of those lacking previous records of atrial arrhythmias. Additionally, the AF/AFL occurred in association with an elevated death of rate of 46% vs. 26% of patients without arrhythmias ($P < 0.01$) (74). Likewise, the incidence of AF/AFL has been denoted as a weak prognostic marker in a variety of investigations. Ip et al., for instance, observed that atrial fibrillation occurred as independently predicting the death with an OR of 4.8 ($P = 0.004$) (75). Likewise, the incidence of AF conceded a greater death rate of 54.3% vs. 37.2% ($P < .001$) in a study by Mountantonakis et al. A greater death rate was also observed within the AF group in those with newly occurred AF vs. those with an identified record of AF (55.2% vs. 46.8%, $P = 0.009$) (76). In cohort data, there are rare reports of SVTs. As claimed by Yarmohammadi et al., atrial arrhythmias happened in 8% of 1,029 hospitalized COVID-19 patients, with SVTs, long RP tachycardia, and short RP tachycardia observed for 8%, five cases, and two cases, respectively, within that group. The patients with long RP tachycardia were considered most probably the representatives of focal atrial tachycardia (77)

Bradyarrhythmias

There are also bradyarrhythmias in arrhythmic fallouts occurring throughout COVID-19 infection. Bradycardia and atrioventricular block were respectively involved in 12.8% and 8.6% of arrhythmias in a global investigation on arrhythmias seen in more than 800 hospitalized SARS-CoV-2 infected patients (78). According to a single-center practice from a hospital in New York City, at the peak of the epidemic, bradyarrhythmia comprised 16% of the cases of hospitalized electrophysiology consultation requisitions (72). Since bradyarrhythmias comprise a considerable fraction of arrhythmias seen in hospitalized COVID-19 patients, managing bradyarrhythmias is considered to be of importance in this diseased population. Considering the detected relationship between heart block and myocarditis and the relationship between SARS-CoV-2 infection and myocarditis, myocarditis needs to be clinically considered in COVID-19 patients developing atrioventricular block (79, 80).

Autonomic dysfunction

Survived COVID-19 patients, particularly those with symptoms of PASC, have reportedly experienced arrhythmias of the autonomic dysfunction type, with typical characteristics of postural orthostatic tachycardia syndrome (POTS) or inappropriate sinus tachycardia (IST) (81-83). The typical characteristic of POTS is symptoms precipitating by alterations in the position or standing with an increased heart rate of ≥ 30 beats per minute (or heart rate > 120 beats/min) while one moves from a prostrate to a standup mode. The characteristic of IST is sinus tachycardia with no recognizable etiology (84). In a Swedish case series, Johansson et al. (83) studied three patients suffering from symptoms of PASC; symptomatic POTS in the months after COVID-19 infection was demonstrated in the whole case.

Myocarditis

Elevated heart enzymes and altered ECG and echocardiography have been shown in multiple investigations, suggesting severe myocardial damage in COVID-19 patients (56, 85). A man aged 63 years was the prime case of fulminant COVID-19–associated myocarditis without any record of cardiac diseases or hypertension with initial presentation of pneumonia-compatible symptoms. More blood tests showed increased concentrations of IL-6 and myocardial damage markers, namely troponin I, myoglobin, and N-terminal brain natriuretic peptide (NT-BNP). Echocardiography revealed an expanded left ventricle, reduced left ventricular ejection fraction, pulmonary hypertension, and diffuse myocardial dyskinesia (86). After this primary observation, the cardiac MRI (CMR) has been used to diagnose extra cases of COVID-19–linked myocar-

ditis (87-93) as well as in post-mortally analyzed lethal cases and endomyocardial biopsies (94-96). In a nominated hospital in Wuhan, China, 12% of 41 laboratory-confirmed COVID-19 hospitalized patients presented with severe heart damage, which was described as either in the elevated heart biomarkers or the occurrence of novel anomalies on electrocardiography or echocardiography. Mononuclear infiltrate, mainly comprising lymphocytes, accompanied by focal myocyte necrosis, was reported in an autopsy study of COVID-19 patients. Furthermore, a lethal case of biopsy-confirmed fulminant myocarditis was observed in a patient aged two years with SARS-CoV-2 infection (97, 98). Most patients present myocarditis alongside SARS-CoV-2–linked breathing symptoms. Nonetheless, the delayed occurrence of presenting cardiac fallouts after weeks of primary symptomatic COVID-19 is also possible (99-101). Up to 19% of people may represent CMR-documented myocarditis as a post-severe abnormality of infective SARS-CoV-2 (102), and reports are available on insulated myocarditis that atypically presents COVID-19 with no associated respiratory diseases (103, 104). There are also reports of ongoing or resolving myocarditis that present sub clinically and asymptomatically or mildly occurring disease. CMR results suggest the demonstration of cardiac injury amongst young competitor sportspersons with COVID-19 (104-106).

Myocardial interstitial fibrosis

There are reports of diffuse and focal myocardial fibrosis in COVID-19 patients' hearts that may happen when cardiac symptoms are absent. Evidence indicates that seven out of 26 COVID-19-recovered patients subsequently presented heart symptoms with edema and fibrosis by tardy gadolinium improvement in CMR (107). In a case report, diffuse interstitial fibrosis was noticed on the CMR of a woman aged 45 years with no record of myocarditis who developed palpitation and abnormal thoracic pain three months following COVID-19 contraction (108). Similarly, diffuse fibrosis was found in a formerly healthful man aged 49 years who developed dyspnea following six weeks of the primary incidence of COVID-19 indications (100). Additionally, focal myocardial fibrosis was observed in the autopsy results of six out of 14 patients with COVID-19, but myocardial infarction was present in the previous records of all patients (109). Four other patients deceased due to SARS-CoV-2 were analyzed post-mortally, and mild focal fibrosis was detected in the heart tissues of two patients (110). Noteworthy is that a medical record of chronic lymphocytic leukemia was seen in one of these patients, and renal transplantation was performed in the other patient three months before the virus contraction. Elsewhere, cardiac tissue was analyzed by endomyocardial biopsy in a patient with cardiogenic shock, revealing low-degree inflammation with focal interstitial fibrosis (111).

EC dysfunction and vasculitis

ECs contribute to regulating inflammatory reactions, immune response, coagulation, and platelet function; thus, these cells are vital factors in a variety of COVID-19-linked pathologies (111, 112). Though EC malfunction and vasculitis are now among the key cardiovascular fallouts of COVID-19, they are also thought to be some other modes of action possibly underlying myocarditis caused by COVID-19. Post-mortally analyzed COVID-19 patients revealed that SARS-CoV-2 was present in the ECs of several organs (113). ECs can be directly infected with viral through SARS-CoV-2 receptors, TMPRSS2, and ACE2, the expression of which occurs on ECs (114), leading to endothelial dysfunction and disruption of vascular integrity, followed by leaking subsequently (115).

Venous thromboembolism (VTE)

Primary reports on COVID-19 were compared in non-survived and survived patients, indicating that D-dimer and other fibrin degradation products were significantly greater in the former cases, with more prolonged prothrombin and activated partial thromboplastin times (aPTT). According to recently published studies, however, coagulopathy in COVID-19 patients demonstrates high concentrations of fibrinogen and D-dimer, and the prothrombin time, platelet count, and aPTT change minimally in the initial phases of the disease (116, 117). Tang et al. presented evidence of disseminated intravascular coagulation (DIC) in 71.4% of non-survived patients vs. 0.6%

of survived cases who were hospitalized throughout their research (116). Klok et al., on the other hand, reported that DIC was manifested in none of the ICU patients whose cases were exacerbated by thrombotic incidents (118). Zhang et al. introduced three COVID-19 patients with overt coagulopathy and positive antiphospholipid antibody tests (119). In a case series of 34 patients with COVID-19 and a protracted aPTT, Bowles et al. detected positive lupus anticoagulant tests in 31 (91%) patients (120). Accordingly, it is not yet clear whether the detected coagulopathy is merely a disease acuteness marker (as seen in septic shock) or whether the virus directly influences the coagulation flow (116, 121). In some studies, attempts were made to associate coagulopathy signs in predicting the viability of COVID-19 patients and suggested the D-dimer level, the prothrombin time, and thrombocytopenia be potential prognostic agents. The proposed potential D-dimer cutoff points are 0.5, 2.4, and 3 mg/L to predict the disease acuteness (97, 116, 122). Despite the agreement on the function of D-dimer in COVID-19 patients, D-dimer was not a determining factor in the clinical risk model to predict the incidence of serious disease in a single report (123).

Takotsubo cardiomyopathy

In Takotsubo cardiomyopathy (TCM), a heart failure syndrome, early and late fatality occurs the same as ST-elevation and non-ST-elevation MI (124). Clinically, TCM is often diagnosed when heart catheterization of a patient with suspicious severe MI demonstrates lacking important blockage and the incidence of anteroapical dyskinesis. There is evidence that catecholamine surge is significantly involved in the pathogenicity of TCM (125, 126). A distinctive characteristic of COVID-19 is cytokine storm, causing a malicious sequence of successive catecholamine surges (127). As cytokine storm in COVID-19 may worsen in line with its disease austereness, the reasons for mortality in acute COVID-19 could partially comprise no diagnosis of TCM accompanied by catecholamine surge. Yet, it is not clear whether the magnitude of catecholamine concentrations in serum is linked to TCM pathogenicity and prognosis. Forthcoming research can seek to compare serum catecholamine concentrations of four groups, i.e., TCM with or without COVID-19, COVID-19 with or without TCM, to assess the usefulness of serum catecholamine concentrations in diagnosing and predicting TCM. Concerning sex dissimilarities, postmenopausal women in non-COVID-19 cases comprised nearly 90% of TCM patients (128). These findings indicate that women comprised only 59.6% of TCM with COVID-19. The difference is explainable in multiple probable ways. Firstly, physical stressors may be more prevalent in men who are more susceptible to inducers, including COVID-19. In the former multi-center registry data, physical stress described as severe respiratory failure, infection, or other insults was reported in 50% of men with TCM (129).

Pericarditis

Pericarditis is the inflammation of the pericardium, a dual-layered sac that surrounds the heart, and pericarditis characterizes the most frequent pathological process amongst pericardial syndromes. The most frequent etiologies of pericarditis are viral infections, namely coxsackieviruses, echovirus, adenoviruses, parvovirus B19, HIV, influenza, or herpes viruses (130). Severe pericarditis can be diagnosed clinically with a minimum of two items from the criteria of i, thoracic pain; ii, pericardial friction rub; iii, ECG alterations; and iv, pericardial effusion (131). Idiopathic or viral causes comprise approximately 90% of acute pericarditis. A benignant cycle is typical of viral pericarditis and is self-limited as patients mostly recuperate within two to four weeks with therapy, such as nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, and corticosteroids. About NSAIDs, it is not clearly evidenced scientifically that ibuprofen and other NSAIDs conjointly worsen COVID-19; thus, they should apparently be used prudently for controlling pericarditis combined with other therapies, such as corticosteroids, colchicine, and anakinra. This is because they are presently thought potential therapeutic choices for different phases of COVID-19 infection (132). There are currently scarce documented data on COVID-19 patients who present pericarditis and PE. Case reports were mostly accompanied by myocardial entanglement with troponin elevation (133). The key symptom is thoracic pain. PE may or may not be caused by

pericarditis. PE can be negligible to immense, which causes cardiac tamponade. In COVID-19, cardiac inducers, such as ACS, pericarditis, and myocarditis, may cause thoracic pain (134).

Cardiogenic shock

COVID-19 patients and severe incidences of cardiogenic shock cured with inotrope and mechanical circulatory support and, in some events, venoarterial extracorporeal membrane oxygenation (VA-ECMO) have been portrayed in reported cases (135, 136). In some case reports, fast recuperation within a few days has been reported with a time course suggesting the possibility of stress cardiomyopathy. Despite suspicious fulminant myocarditis in some events of cardiogenic shock in which ventricular function recovered within days or weeks, this has mostly not been diagnosed confidently due to either the absence of endomyocardial biopsy or it was present but showed no observations of myocarditis (137-141).

Cardiovascular Complications in SARS-CoV and MERS-CoV

In SARS patients, the detected cardiovascular fallouts include subclinical diastolic disablement in which contraction is not involved, and this might be returnable harm when one recovers clinically (142). Yu et al. reported the frequent occurrence of cardiovascular fallouts, such as hypotension and tachycardia, in patients with SARS, but they were generally self-limiting. They observed lower bradycardia and cardiac hypertrophy frequencies, and infrequency of arrhythmias (51). Severe MI was also visible in some SARS patients (143). In a report, SARS-CoV was seen in 40% (7/18 patients) of hearts sampled from patients who deceased of SARS throughout the Toronto epidemic, which probably explains the myocardial impairment detected in patients with SARS. Existing evidence indicates that small blood vessels all over the body are also attacked by SARS-CoV, leading to systemic vasculitis (144-146). Besides investigations performed clinically, laboratory studies aimed to examine the pathophysiological mode of action linked to myocardial malfunction resulting from SARS-CoV infection (147-151). Pulmonary SARS-CoV infection in mice reportedly resulted in a myocardial infection in which ACE2 expression decreased markedly, which is probably involved in myocardial malfunction and adversarial cardiac consequences in SARS patients (151). The cardiac injury was also observed in MERS patients. The kidneys of dead patients contained MERS-CoV, but it was invisible in the cardiac tissues, and the heart histology did not change significantly. On the other hand, in animal model research, MERS-CoV RNA was obviously observed in the heart tissue, inferring direct cardiac pathology. The mode of action of cardiac damage in MERS infection is not still clear requiring further investigations (152-155).

Cardiovascular Complications in Influenza

The vascular system is affected by influenza in several aspects. In influenza, proinflammatory, prothrombotic cytokines increase considerably, causing endothelial malfunction, elevated plasma viscosity, tachycardia, and released endogenic catecholamines. Clinically, flu is also accompanied by psychological trouble, dehydration resulting in hypotension, hemoconcentration, hypoxemia, and demand ischemia. Van Lenten presented evidence that influenza reduced the anti-inflammatory features of high-density lipoprotein cholesterol particles and improved the entrance of macrophages into the arterial wall. Moreover, influenza infection causes procoagulant impacts extensively and profoundly (156). Influenza can function as a severe inflammatory and procoagulant stimulant that transitorily alters endothelial action (157, 158). In investigations conducted experimentally on mice, associations were found between influenza infection and coronary artery remodeling. There is, however, no clear mode of action by which influenza induces cardiovascular-linked illness. It may result from the generation of autoantibodies against modified low-density lipoprotein or may arise from directly colonized vessel walls that causes autoimmune reactions locally (159). Furthermore, influenza infection reportedly elevates macrophage infiltration and weakens anti-inflammatory features of high-density lipoprotein (160, 161). The elevated risk of cardiovascular incidents may also be caused by fever, tachycardia, and dehydration accompanied by severe influenza (162, 163). In addition, smooth muscle proliferation, platelet aggregation,

thrombus formation, and angina destabilization may be caused by the influenza virus. There are reports of main heart adverse effects in a remarkable number of patients with CAP, particularly in inpatients. In an investigation, the pooled incidence rates of general heart adverse effects, heart failure events, severe coronary syndrome, and arrhythmias events were 17.7%, 14.1%, 5.3%, and 4.7%, respectively, in inpatients with CAP (164-166). In a study, influenza infection was reviewed systematically, reporting that it triggers severe MI and cardiovascular mortality. This review further posited that influenza vaccines would effectively reduce the risk of heart incidents in those patients with confirmed cardiovascular disease (167).

Conclusion

Respiratory viruses primarily attack the lungs, although there have been reports of cardiac complications with the viruses. For example, H1N1-related cardiac complications included congestive heart failure exacerbations in diagnosed patients with heart failure or AMI. Importantly, studies suggested that myocarditis and pericarditis in the pandemic of H1N1 influenza virus (pH1N1) infection were critical factors contributing to mortality. In such case studies, heart dysfunction correlates with CK, CK-MB, troponin I, ECG, and ECHO changes.

Immune system status is one of the essential factors determining cardiovascular complications in respiratory viral infections. Dysregulated immune responses are one of the main characteristics of COVID-19, posing a challenge for people with susceptible genetic backgrounds and those suffering from pre-existing immune-mediated disorders, particularly malignancies, autoimmune diseases, and immunodeficiencies, and therefore have been a candidate for targeted therapy (25-28, 30, 34, 168-193). In the recent COVID-19 pandemic, patients displayed a prothrombotic, proinflammatory state and coagulopathy in up to one-fifth of cases. Compared with other human influenza viruses, the effects that COVID-19 might have on the heart are more common. Compared to SARS and MERS, COVID-19 has been linked to an increased risk of cardiovascular disease, including stroke (194). Therefore, careful examination of the cardiovascular system in susceptible groups could be important.

Estimating the death rate from RVIs has been a subject of intense research, but the mortality from cardiovascular complications in these infections is less understood and calls for further research (59, 195). Biomarkers such as cardiac troponins, creatine kinase, natriuretic peptide type B, and LDH rise during heart damage in RVIs (196). It might be further interesting to find biomarkers that help with the prediction and prognosis of cardiac complications in people with RVIs. However, it is a challenging task when the early diagnosis of RVIs, itself remains cot sensitive, specific, and quick as it should be when we are "living" in a pandemic (197, 198) with high rates of transmission and recurrence forcing us to choose social restrictions (9, 29, 199, 200) and rethink the integrated, international efforts to save "minds," for both the health care system and patients (201-210).

Conflicts of interest

The authors have no conflict of interest.

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