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Review Article

Cardiovascular Complications of COVID-19: Acute Injury, Post-Acute Sequelae, and Long-Term Cardiovascular Risk

Riya Mehra*, Abhijeet Ojha, Arun Kumar, Vikas Bhatt, Divya Negi, Mamta Goswami, Navin Chandra Pant, Amit Budhori

Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India

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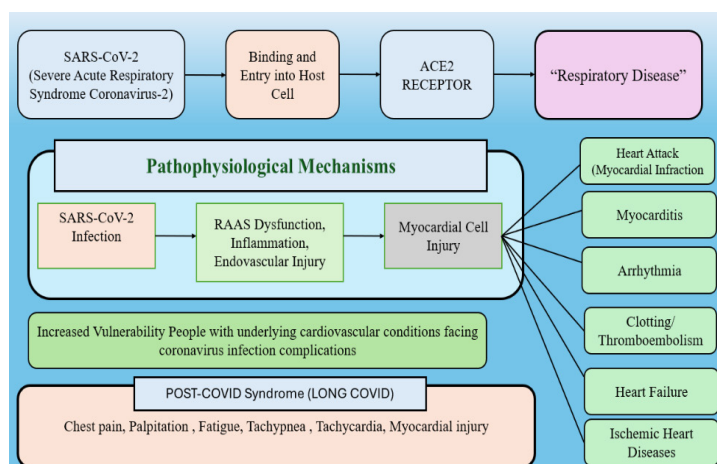
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ABSTRACT

The virus responsible for the disease COVID-19, which has greatly influenced health care worldwide, is referred to as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Although it is clear that this infection impacts the respiratory system, there have been many incidences where coronavirus affects the circulatory system as well. Various studies have identified correlations between coronavirus and different cardiovascular complications. These viruses infect the host cells via ACE2 receptors, which interfere with the renin angiotensin aldosterone systems, causing inflammation and damaging the blood vessels, resulting in damage to cardiac muscle cells. Consequently, heart attack, myocarditis, arrhythmia, thrombosis, heart failure, and ischemic heart disease develop in patients with SARS-CoV-2 infection. People who have cardiac disease before the infection are at increased risk of the impact of the coronavirus infections. Finally, post-COVID syndrome (long COVID) is also associated with cardiovascular complications including chest pain, palpitation, dyspnea, fatigue, tachypnea, tachycardia, and myocardial damage. Numerous studies have found that there are patterns in the relationship between coronavirus and different complications related to heart health.



Graphical abstract representing COVID-19 connected with cardiovascular complications and mechanisms

*Corresponding Author: Riya Mehra

Address: Faculty of Pharmaceutical Sciences, Amrapali University, Haldwani, Uttarakhand, India

Email ✉: rm841275@gmail.com

Tel.: +91-8279418792

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INTRODUCTION

Coronavirus disease 2019 (COVID-19), which is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has been related to high morbidity and mortality around the world. The effect of SARS-CoV-2 infection on cardiovascular systems has been extensively studied. [1] The effect of COVID-19 on the cardiovascular (CV) system has been seen since the initial phase of the disease although it is mainly a respiratory illness. Mechanistically, SARS-CoV-2 uses angiotensin-converting enzyme 2 (ACE2) (Fig. 1). Cardiac troponin raised due to the infection of COVID-19, as a marker for heart muscle damage, could be seen as a pathological feature of COVID-19 because of an imbalance in the renin-angiotensin-aldosterone system (RAAS).⁷ Without a doubt, the presence of hyper-inflammation in those with severe COVID-19 can be caused by the RAAS imbalance and in severe cases of COVID-19, multiorgan injury is brought about through pro-oxidative and fibrotic actions. [2] Progressive heart muscle damage in the process of the development of COVID-19 may lead to heart failure among other adverse outcomes after recovery. [3] Cardiovascular disease that comprises several conditions such as cerebrovascular disease, rhythm problems, ischemic or non-ischemic heart disease, pericarditis, heart failure, and thromboembolic disease is among diseases that are associated with COVID-19.

The mean age of the female patients was 46.3 years; the patients were young and showed multiple circulatory system symptoms including chest pain (66%). All the patients had greater than 5% proportion of circulatory system symptoms mainly during episodes of sinus tachycardia. Out of three patients two showed <40% LV ejection fraction, the mean left ventricular (LV) ejection fraction was 62.7%, out of the total 23 patients showed the symptom of Diastolic dysfunction. 70 Patients where data is available Global Longitudinal Strain (GLS) was low in 2020. One Patient showed the results that were consistent with active myocarditis and endangered by cardiac MRI to evaluate chest pain out of 37 cases, 19% cases showed T1/T2 Abnormalities. Amongst 41 patients who were

preliminarily diagnosed as a case of COVID-19 in Wuhan, five patients showed the symptom of Increased Levels of Cardiac Troponin I (Tn-I) (>28 pg/mL). Four of which have shown signs of Myocardial Injury and admitted in ICU. SARS-CoV-2 infection demonstrates an aggressive course in patients with pre-existing CV circulatory conditions with a concurrent increase in mortality rates. Acute complications of the cardiovascular system including thrombosis, myocarditis, cardiac arrhythmia have all been linked with the infection. These patients are particularly aware of strict personal hygiene and physical distancing protocols due to their CV disease. [1]

Cardiovascular Complications Associated With COVID-19

Acute Cardiac Injury and COVID-19

Acute cardiovascular complications mean the development of cardiovascular problems while one is infected with SARS-CoV-2 in the course of its acute clinical manifestation. Such conditions include myocardial injury, myocarditis, myocardial infarction/acute coronary syndrome, cardiac arrhythmias, thrombotic problems, and acute or exacerbated heart failure. Depending on how severe this disease and cardiovascular problems in the patients are, as well as some other factors, such complications will appear and their severity will differ from case to case. Infection can have an impact on the heart, damaging this important and vital organ. Morbidity and mortality rates depend on heart problems. [2] The effect of chronic damage/injury and acute damage of the cardiovascular system caused by the COVID-19 infection. [4-6] By use of myocardial injury biomarker it is possible to estimate the in-hospital mortality of the patients who have been infected by COVID-19. Such kind of deaths have been associated with the old age of the patient, inflammation response and heart-related condition. But as per the mechanism found out by the experts of this field with regard to myocardial injuries some of the important causes include the following: inflammation damage, fibrosis in the ventricle, cardiomyocyte cells, overreactions by type one and two helper T-cell, cardiac plaque destabilization, hypoxia and last but not least interferon-mediated immunological responses. It is clear that increased levels of high-sensitivity cardiac troponin I (cTnI) causes Myocardial injuries caused by COVID-19 infections for some people. Ten out of 138 COVID-19 patients developed myocardial injury while the ones admitted to the intensive care unit ICU were prone to the development of heart diseases, with high level of sensitivity to the TnI. Conclusion. cTnI concentration was found to be significantly higher among severe conditions of COVID-19 infection. [3]

Myocardial Injury and Myocarditis

Myocardial injury and myocarditis accompanied by elevations in serum level of Troponin have been attributed

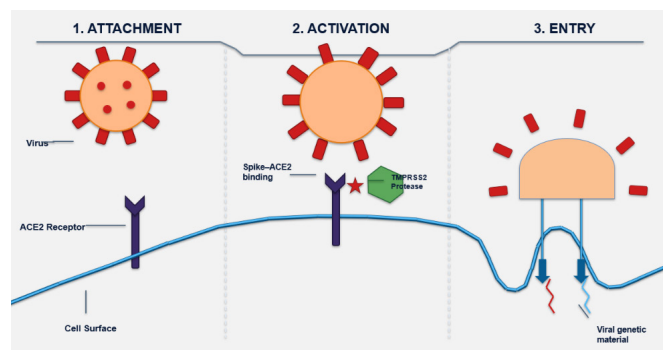


Fig. 1: The virus surface glycoprotein binds to the target cell ACE2 binding site, facilitating the initial attachment

to MERS-CoV due to increased physiological stress on heart, hypoxemia, and myocardial injury. [7-8] Cases of myocarditis have also been reported in patients suffering from SARS-CoV-2 infection where high viral load and mononuclear infiltrate were observed on autopsies performed on patients who succumbed to the infection. Acute myocarditis is very hard to diagnose because of its diversity of forms. [9] Acute myocarditis presents itself with multiple forms, making it very challenging to diagnose in cases of coronavirus disease 2019. Patients with coronavirus disease 2019 infection present themselves with symptoms such as chest pain, dyspnea, arrhythmia, and left ventricular failure, acute myocarditis/myocardial damage with troponin elevations observed in patients with COVID-19, which have been proven to be poor prognostic factor, particularly in severe cases of the infection (Fig. 2). [4]

Myocardial Infarction and Acute Coronary Syndrome

Epidemiologic data show a raised risk of developing myocardial infarction in subjects who have contracted coronavirus infection. A systematic literature review and meta-analysis, which covered data from 13 studies, showed that there was a more than two-fold raised risk of myocardial infarction after developing COVID-19. The calculated combined relative risk (RR) of myocardial infarction development was 2.2 (95% CI 1.4-3.5). The pooled RR for myocardial infarction developing, when the date of the beginning of the COVID-19 course was counted within the period of observation, was 2.4 (95% CI: 1.1-5.3), whereas the RR was also more than doubled in hospitalized patients (RR: 2.7; 95% CI: 1.3-5.3). It can be concluded that SARS-CoV-2 infection is likely to have a clinically important role in the development of myocardial infarction. [5]

Cardiac Arrhythmias

Tachycardia was found to be the most frequently occurring abnormality of the ECG, albeit typically isolated, with an

incidence from 72% among those infected with SARS. The frequency of bradycardia was moderately lower, varying from 2% to 15%, and was recorded among those with COVID-19 infection, according to one survey we found. In addition, ST-T abnormalities and cardiac dysrhythmias like branch block, atrial fibrillation (AF), premature contractions, QT interval prolongation, or even SCD have been found in SARS. [10-11] In patients with COVID-19 infection, according to one review, detect that AF was the most common abnormal rhythm of heart. [12] As mentioned before because of the systemic infection which can lead to atrial fibrillation among these patients, cardiomyocytes damage, direct viral effects, hypoxemia, and high risk of the general population because of their old age, in conclusion, overactivity of the sympathetic nervous system. [13] More commonly found among those with raised troponin T levels caused malignant dysrhythmias, including ventricular tachycardia/ventricular fibrillation compared with patients with normal troponin T levels (6 patients [11.5%] vs 7 patients [5.2%]). [14]

Thrombotic Events

It can disrupt coagulation system as well. High frequency of cases such as venous thrombosis and pulmonary embolism associated with the occurrence of COVID-19 has been noticed in patients. The average incidence rate of venous thromboembolism in 1988 patients with COVID-19 from 20 clinical trials was 31.3%. [15]

Apart from venous thrombosis and pulmonary embolism, other kinds of thrombotic disorders have also been reported among patients having COVID-19 such as the appearance of thrombosis in arteries and myocardial infarction. High mortality risk is associated with such events. Moreover, in a number of studies elevated D-dimer levels in COVID-19 patients have also been linked with higher risk of mortality. In addition, thrombotic events in patients having COVID-19 have been associated with mortality risks. It is recommended that all patients of hospitalized COVID-19 receive prophylaxis dose of low-molecular-weight heparin. [16]

Heart Failure

Heart failure is an essential cardiovascular disorder that can develop during the course of or after SARS-CoV-2 infection. Myocardial damage, myocarditis, inflammation, hypoxia, endothelial dysfunction, and regulation problems of renin-angiotensin-aldosterone system can affect the functioning of the heart in patients who have or develop heart failure due to SARS-CoV-2 infection. Moreover, myocardial inflammation and fibrosis after acute infections may cause prolonged dysfunction of ventricles. Patients who already suffer from heart failure may be more vulnerable to developing complications because of excessive hemodynamic load and systemic inflammatory response.

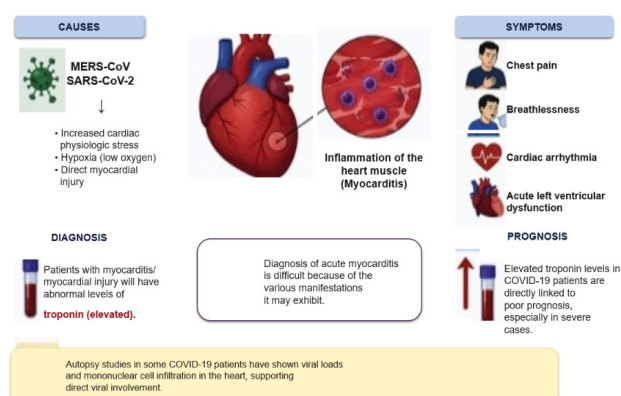


Fig. 2: Myocardial injury and myocarditis in coronavirus infections.



Both new onset heart failure and heart failure aggravation are possible in case of COVID-19. Dyspnea, fatigue, swelling, chest pain, and poor exercise tolerance are common manifestations of heart failure but may overlap with the symptoms of post-COVID disorders, and thus, adequate clinical examination is necessary. Diagnostic examination may include electrocardiography, cardiological biological markers like cardiac troponin, natriuretic peptides, echocardiography for evaluation of ventricular dysfunction, and cardiac magnetic resonance imaging in cases of myocarditis or myocardial injury. The management approach should be tailored based on the specific cardiac disease and severity of heart failure. Patients with established heart failure should receive guideline-based treatment, and the management of myocarditis, arrhythmias, hypoxia, among others, should be tackled if present. Future research is needed to define the incidence, mechanisms, prognosis, and optimal management of heart failure after SARS-CoV-2 infection.^[16-22]

Mechanism

Cardiac ailments in the wake of the disease could occur because of myocardial damage, which was due to a lack of blood supply or the loss of oxygen supply. This is not all, as heart complications might develop due to microvascular damage, as well as myocarditis caused by the virus. The hypothesis relies on the assumption that factors involved in cardiac dysfunction are systemic inflammation and the infection of the heart cells by the virus via ACE2 receptors (Fig. 3).

SARS-CoV-2-induced cardiovascular damage may be a result of a variety of interrelated pathways leading to myocardial and vascular injury. Viral effects either directly or indirectly in conjunction with ACE2/RAAS system dysregulation might lead to endothelial dysfunction and injury of blood vessels. The endothelial damage would then increase inflammation and immune system abnormalities that activate platelets and disrupt the clotting system. Such phenomena can affect microcirculation and predispose patients to developing microthrombi. During a severe infection, hypoxia and increased oxidative stress levels might further worsen endothelial and myocardial tissue damage. It appears that all of those factors combined together can cause ischemic injury and myocardial damage contributing to an increased likelihood of developing acute coronary syndrome, myocarditis, arrhythmias, and heart failure.

Integrated Pathophysiological Mechanisms of Cardiovascular Injury Following SARS-CoV-2 Infection

ACE2 is a Type I membrane-bound protein, which has the capability of performing enzymatically for converting Ang II into Angiotensin^[23] the ACE2 enzyme is a protein that is found on the surface of cells. ACE2 may be regarded as

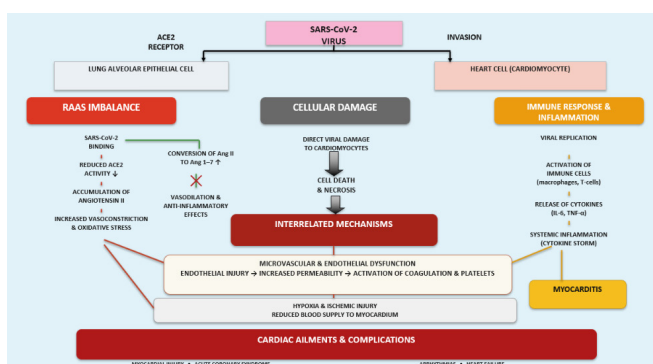


Fig. 3: Mechanisms of SARS-CoV-2-induced cardiovascular damage

antagonizing the actions of RAAS even though it has the similarity of ACE. ACE2 exists in different organs like the lung, heart, and kidney.^[24]

The cardiovascular effects of SARS-CoV-2 infection can also not be explained by dysregulation of ACE2 alone. Endothelial damage, systemic inflammation, immune system derangement, activation of platelets, and coagulation disorders could possibly have an interplay with dysregulation of ACE2/RAAS system and cause micro-vascular dysfunction. Reduced oxygen supply to the myocardium caused by microcirculatory insufficiency can also be followed by injury through hypoxia and oxidative stress. Thus, these mechanisms of pathophysiology create an indirect relationship between SARS-CoV-2 infection and myocardial damage, myocarditis, etc.^[25-26]

Through binding with the extracellular domains of the transmembrane Angiotensin converting enzyme 2 protein, and through viral spike (S)-protein, which has been reported to reduce the production of angiotensin-converting enzyme 2, the coronavirus gains access into ss cells. The production of the oligopeptide angiotensin II is normally done through the production of angiotensin-converting enzyme 2.^[27] However, recently some research has indicated that the production of Ang II was much higher in coronavirus patients than healthy individuals. This indicates that there might be low production of angiotensin-converting enzyme 2, implying a systemically imbalanced RAAS. In general, multivariate damage caused by SARS-CoV-2 infection is as a result of an imbalance in the RAAS.^[28] The different angiotensin-converting enzyme 2 polymorphisms have been associated with CVD in multiple studies.^[29]

An insult in angiotensin converting enzyme 2 causes neutrophils influx in the area of necrosis, causing the immunity response involving the process of interferon gamma, interleukin-6 (IL-6), chemotactic chemokines, and monocyte chemoattractant protein-1 which appeared to be upregulated. Phosphorylation in extracellular signal-regulated kinase $\frac{1}{2}$ and c-Jun N-terminal kinase $\frac{1}{2}$ signalling pathways was also upregulated. Angiotensin converting enzyme 2 receptor blockage in mice increased

the range of the infarct zone, activated metalloproteinase like MMP2 and MMP9 and disrupted extracellular matrix, which eventually leads to myocardial infarction-related heart failure.^[30] The latest study conducted by Nicin *et al.*, was centered around the investigation of the expression pattern of the ACE2 receptor on various cell phenotypes of the human heart organ. On the basis of individual nuclei ribonucleic acids composition, they analyzed ACE and ACE2 levels in single cells of AS and HFrEF hearts, along with healthy donor hearts. The results revealed a small presence of ACE2 in fibroblasts, endothelial cells, and leukocytes, whereas ACE2 was more abundant in cardiomyocytes and pericytes. The cardiomyocytes of diseased hearts had an increased expression of ACE2 compared with that in healthy hearts, whereas no difference was observed in the level of ACE2. Endothelial lining cells and connective tissue cells were detected between the contaminated and vigorous hearts. Possible interventions that can be taken to reduce damage associated with COVID-19 include the inhibition of ACE2 binding by SARS-CoV-2 using of chemical inhibitors, upregulation of Angiotensin converting enzyme 2 expression, genetically Angiotensin converting enzyme 2, Angiotensin converting enzyme 2-attachment peptides, or antibodies.^[8]

Post-Acute Cardiovascular Complications and Long Covid

Long COVID is defined by the continuation of symptoms far beyond the typical recovery time from a SARS-CoV-2 infection. "Long haulers" is a common designation for those affected by long-term symptoms.^[9]

Three distinct symptom clusters were identified:

Cluster 1: Patients belonging to this cluster have fatigue and joint/muscle pains as well as other symptoms, although in general they tend not to suffer from specific symptoms.

Cluster 2: This is where patients experience fatigue, muscle/joint pains, cardiorespiratory problems, which include irregular heartbeat sensation, reduced berating capacity, thoracic pain, and coughing.

Cluster 3: In this cluster, ageusia, anosmia, fatigue, and cardiorespiratory problems are frequent.

From our findings, we infer that there indeed exists symptom clustering in long COVID cases, and the clusters might persist even two years after suffering from a coronavirus infection.^[10]

COVID-19 Cardiovascular Epidemiology

In January 2020, the first case of coronavirus pneumonia was reported in Wuhan, China, coronavirus was the causative agent was identified as SARS-CoV-2.^[11] Pyrexia, inflamed throat, coughing, and body aches are primary symptoms of coronavirus (COVID-19). Other clinical cases may involve cardiac-related problems, including acute coronary syndrome or congestive heart failure.^[12]

Approximately 12% of patients with coronavirus infection experience persistent acute cardiac injury. Furthermore, another study revealed that the incidence of cardiac troponin elevation, which indicates heart muscle injury, in hospitalized coronavirus cases was within the range of 5-25%. It has also been demonstrated that patients suffering from myocardial injury are older and are at high risk for heart disease.^[13] In a study conducted by a cardiac failure Tertiary unit in London on hospitalization patterns, number in hospitalizations due to HF decreased by a drastic 66% during the pandemic. This was compared not only to the same time before the onset of the pandemic but also to the corresponding periods in 2017-2019. Of those who had to seek medical care for HF during the pandemic, the severity of their disease had increased, as evidenced by a higher number of NYHA class III-IV and patients with severe peripheral edema, which are considered indicators of poor prognosis in acute HF patients.^[14]

The determination of the occurrence of coronavirus infection among cases with cardiac-related diseases is complicated by the disparity between nations in their reporting of data. According to one study from China on 1,527 cases of COVID-19, 9.7 percent suffered from diabetes mellitus, 16.4 percent suffered from either heart or brain disease, and 17 percent were hypertensive. The significance of the co-morbidities cannot be understated since their presence increases the likelihood of adverse outcomes.^[15] The present study advises there could be held through effect of coronavirus on health of the circulatory system.^[16]

Diagnosis And Investigations of Post-COVID 19 Cardiovascular Complications

Assessment of post-COVID-19 heart conditions should start with the patient's full clinical evaluation. Close consideration needs to be given to any cardiovascular signs, including chest pain, dyspnoea, palpitations, fainting or near fainting, fast heartbeat, fatigue, and decreased exercise tolerance. Consideration must also be given to the extent of the prior coronavirus infection, hospital admission history, cardiovascular condition beforehand, cardiovascular risk factors, and presence or persistence of heart complaints following the recovery from the illness. Cardiovascular tests should be performed depending on the symptoms of the patient and suspected heart conditions but not as a standard test for everybody. Guidance recommends a symptom-based approach with ECG, lab tests, echocardiogram, ambulatory monitoring of heart rhythms, and other imaging tests based on suspected cardiovascular disorders.^[17-18]

Electrocardiogram (ECG)

The spectrum of arrhythmias in the clinical presentation was considered in patients with COVID-19 within two months of remission in relation to the age, comorbidity,



and mortality rate of the patient through the ECG profile. There were 256 (33.6%) males and 503 (66.4%) females with age $15-90$ (52.99 ± 11.68) years old studied using the ECG reports of 758 patients. There were 848 ECGs studied in acute duration and recovery period. Basal heart rates of all patients such as PR intervals, QRS intervals, and RR intervals were recorded. Mean values of three readings in each lead were determined and were included in the study. Records of more than eight leads available for assessment were included.

Electrocardiography serves as an ideal starting point for the detection of any rhythm disorders, any conduction abnormalities, and changes in the ST segment and T waves in patients with any cardiac complications associated with SARS-CoV-2 infection. Electrocardiographic findings may however be non-specific, and even a normal ECG does not absolutely rule out myocarditis or myocardium involvement.^[19]

Cardiac Biomarkers

Troponin

The concentrations of cardiac troponin 1 in the coronavirus disease patients were less than cardiac troponin T (ratio troponin 1/troponin T = 0.3; Interquartile range: 0.1–0.6). In the spasm, the cardiac troponin 1 concentration was 11 times higher compared to that of cardiac troponin T in 15 patients with the heart attack (troponin 1/troponin T coefficient = 11; Interquartile range: 7–14). The maximal ratio of cardiac troponin 1 to cardiac troponin T in heart attack patients who underwent successful percutaneous coronary intervention was 14 (troponin 1/troponin T ratio = 1) (Fig. 4).

But it is essential that myocardial injury should be distinguished from myocardial infarction and myocarditis when one is interpreting an increased level of cardiac troponins. The increase of cardiac troponins reflects myocardial injury but does not indicate myocardial infarction or myocarditis alone. For diagnosing myocardial infarction, a patient needs to have myocardial ischemia

besides a change of cardiac troponin values, and for diagnosing myocarditis, the patient must have evidence of inflammation of the myocardium. In patients having COVID-19, the increased troponin levels can be attributed to many factors like systemic inflammation, hypoxemia, myocarditis, micro vasculopathy, myocardial injury due to stress, or acute coronary syndromes.^[20]

D-DIMER

Co-activation of hemostasis and fibrinolysis can only synthesized D-dimer, it is a result of a persistent blood clot dissolution activity.^[21] 129 patients (median age 48.8 years; range 19–91 years) underwent D-Dimer assessment after a median (Interquartile Range) of 94 days (64–130) following COVID-19. significantly more common in patients who had observed a serious SARS-CoV2 infection result elevation of D-dimer found 15% (19/129).^[22]

Nonetheless, increased D-dimer levels are nonspecific findings that should not be taken to mean diagnosis in post-COVID cases of thrombotic disease without further testing. Increased D-dimer levels may occur in many diseases and may be due to factors such as infection, inflammation, malignancies, age, and more reasons related to coagulation activation. For this reason, elevated D-dimer level results must be interpreted according to a patient's clinical presentation and pre-test probability of suffering from thrombosis. In the case where thromboembolic diseases are suspected clinically, further tests to verify the condition need to be performed.^[23]

Natriuretic Peptides

However, natriuretic peptides such as BNP and NT-proBNP could also play a role in cases where there is the suspicion of heart failure or ventricular dysfunction after coronavirus infection. These have to be interpreted together with the findings from the clinical assessment and from cardiac imaging as natriuretic peptide elevation is non-specific for heart failure and can also occur in other states of cardiac stress or systemic disease.^[24]

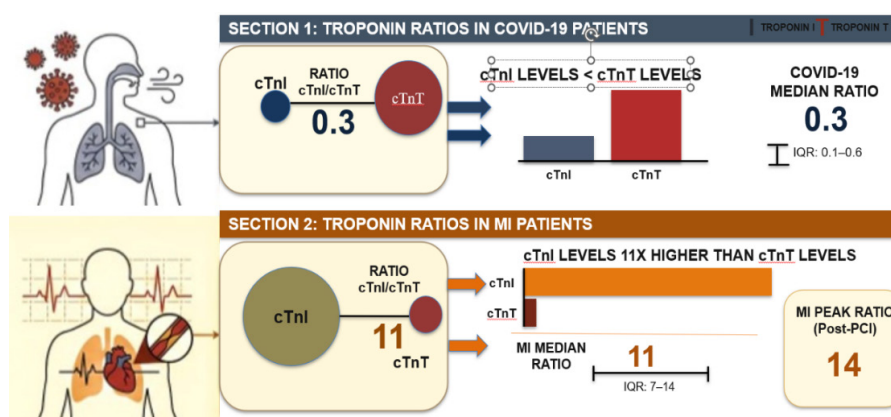


Fig. 4: Examination ratio of troponin 1 (cTnI) and troponin T (cTnT) ratio in COVID-19 and myocardial infarction.

Echocardiography

Transthoracic echocardiography is a very relevant non-invasive test that will allow us to assess the structure and function of the heart of the patient after COVID-19 infection, in which case we are suspicious about possible cardiovascular involvement. This procedure will allow us to evaluate left and right ventricle systolic function, left ventricle ejection fraction, regional wall movement abnormalities, ventricles dimensions, diastolic function, and pericardial effusion. Echocardiography thus might be helpful in diagnosing ventricular dysfunction or any other structural abnormalities, and it will help us to understand the necessity for additional evaluation of heart disease. [25-26]

Cardiac Magnetic Resonance Imaging (CMR)

Imaging using Cardiac Magnetic Resonance (CMR) enables an in-depth assessment of myocardial structure, heart pumping ability, and myocardial tissue properties. The diagnostic technique of Cardiac Magnetic Resonance imaging proves invaluable especially in cases where there is suspicion of myocarditis or persistent myocardial involvement. CMR can detect myocardial edema, myocarditis, necrosis, fibrosis, and pericardial involvement. It can be carried out in clinically suitable patients experiencing persistent signs or symptoms of heart disease, elevated heart biomarkers, changes in ECG, or ventricular dysfunction. [27]

Risk Factor for Cardiovascular Complications Following Sars-CoV-2 Infection

Certain characteristics can make it more likely for a person to develop cardiovascular complications following infection with SARS-CoV-2. Key risk factors include older age, underlying cardiovascular disease, hypertension, diabetes mellitus, obesity, chronic kidney disease, and other cardiometabolic conditions. Additionally, the seriousness of the acute COVID-19 illness can play a role, with those who get hospitalized and require intensive care showing a higher risk of cardiovascular complications when compared to those who have mild infection. Other risk factors that need to be considered in this context include persistent systemic inflammation, myocardial injury resulting from the acute illness, as well as thrombotic or embolic complications. There are suggestions from recently published research that patients with underlying cardiovascular disease have a higher chance of post-COVID syndrome. [28-29]

Pre-existing Cardiovascular and Metabolic Conditions

Previous cardiovascular diseases along with previous metabolic diseases are key risk factors for adverse cardiovascular events following SARS-CoV-2 infection. Diseases like hypertension, heart failure, coronary artery disease, diabetes mellitus, among others, might

contribute to the susceptibility of individuals towards developing cardiac complications due to the infection and even persistent post-COVID-19 symptoms. According to a 2024 systematic review, previous cardiovascular disease, which includes hypertension and heart failure, is significantly linked to the development of post-COVID-19 syndrome. [30-31]

Severity of Acute COVID-19

The severity of the primary SARS-CoV-2 infection is one of the factors linked with the increased future risk of heart problems. Hospitalized or critically ill patients may be exposed to a larger number of systemic inflammations, hypoxia, endothelial dysfunction, myocardial damage, and activation of coagulation pathways, thus possibly leading to the onset of heart complications following the infection. According to the results of a 2024 systematic review and meta-analysis, the patients who experienced SARS-CoV-2 infection were at a higher risk of heart disorders. [32]

Age and Other Patient-Related Factors

Advanced age, as well as multiple underlying conditions, may add more risks of cardiovascular diseases due to SARS-CoV-2 infection. Patients at high risk for cardiovascular problems are more likely to experience myocardial damage, thrombosis, arrhythmia, and heart failure when infected with the virus or after infection. On the other hand, some cases of cardiovascular problems may arise even among younger individuals who were not reported to have cardiovascular disorders before being infected by the novel coronavirus. [33]

Reinfection and Vaccination Status

The effect of vaccination status on the possibility of developing cardiovascular problems in connection with infection with SARS-CoV-2 is not ruled out. In recent studies on large cohorts, it was shown that vaccination significantly reduces the risk of cardiovascular and thromboembolic problems in patients with COVID-19 in comparison with unvaccinated infected persons. However, some cases of myocarditis and pericarditis in connection with vaccination were identified, especially after the use of mRNA vaccines. Consequently, there must be made a differentiation between the infection-induced cardiovascular problems and myocarditis/pericarditis due to vaccination. [34-35]

Management and Prevention

The approach to managing the cardiac complications caused by SARS-CoV-2 infection should be individual and depend on the extent and nature of cardiovascular involvement. Patients suffering from myocarditis, arrhythmias, clotting disorders, and heart failure will require evaluation by specialists along with condition-tailored treatments. The guideline-directed management of heart failure and other forms of known cardiovascular disease will have



to be considered, and thrombotic complications should be addressed as per standard recommendations for the use of anticoagulants or antithrombotic. Post-COVID cardiac complications that include palpitations, chest pain, shortness of breath, and intolerance to exercise will need to be ruled out to confirm heart disease.^[36]

The prevention needs to concentrate on minimizing the danger of serious infection by managing the cardiovascular risk factors which can be managed. This could include hypertension, diabetes mellitus, obesity, smoking, and lack of exercise. Additionally, through adequate vaccinations as well as following the cardiovascular management strategies, there would be an even lower possibility of serious effects of coronavirus disease. Patients suffering from serious cardiovascular effects following COVID-19 need to undergo appropriate clinical evaluation.^[37]

Limitation and Knowledge Gap

While there is an accumulating number of observations regarding cardiovascular complications after SARS-CoV-2 infection, a number of important weaknesses and lack of knowledge still exist. Different designs of the conducted studies, diverse patient groups, varying degrees of illness, differences in follow-up periods, diagnostic criteria, and definitions of post-COVID complications limit direct comparison across different research. The majority of studies are observational, which creates significant challenges in fully attributing the effects associated with SARS-CoV-2 infection to those associated with preexisting cardiovascular risk factors and other confounding factors. Long-term risk of incidence and prognosis of myocardial injury, myocarditis, arrhythmias, thromboembolism, heart failure, and other cardiovascular complications still need to be further studied, especially for non-hospitalized patients and young people. Furthermore, the impact of SARS-CoV-2 variants, re-infections, vaccine administration, and evolving treatment options on long-term cardiovascular risk needs to be better characterized as well.

CONCLUSION

SARS-CoV-2 infection is associated with a diverse set of cardiovascular complications which can arise both acutely or during the post-acute phase. Cardiovascular complications which have been reported due to SARS-CoV-2 infection include myocardial injury, myocardial infarction, myocarditis, arrhythmias, thrombosis, and heart failure. Multiple different overlapping pathways of injury leading to cardiovascular complications caused due to infection appear to involve viral effects, immunological effects, endothelial dysfunction, inflammation, disturbances in coagulation, microvascular dysfunction, hypoxia, and oxidative stress. Factors involved in determining the risk of developing cardiovascular complications might include the severity of the infection as well as the presence of underlying cardiovascular and metabolic disease.

Proper understanding is required since raised cardiac troponin levels reflect myocardial injury but not myocardial infarction and myocarditis, and similarly, D-dimer levels which are elevated nonspecifically require clinical correlation and confirmatory investigation. A symptom-based and risk-based cardiovascular workup using electrocardiogram (ECG), cardiac biomarkers, echocardiography, and advanced imaging techniques, if necessary, might be helpful in identifying those requiring further investigation.

However, while vaccination might alleviate the burden of cardiovascular complications secondary to SARS-CoV-2 infection through reduction of the severity of the illness, the cardiovascular complications secondary to infection should be distinguished from the rare myocarditis/pericarditis due to vaccination, particularly in predisposed patients. Long-term follow-up and well-designed clinical researches are required to elucidate the mechanisms, outcome, prevention, and treatment of the cardiovascular complications following COVID-19.

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