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POST-ACUTE-COVID-19 CARDIOVASCULAR SYNDROME FROM PATHOPHYSIOLOGY TO CLINICAL CARE

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1. ABSTRACT

COVID-19 symptoms for weeks or even months, is expected to affect the lives of millions of individuals Long COVID or post-acute Coronavirus disease 2019 (COVID-19), a malady defined by the persistence of worldwide significantly. Cardiopulmonary symptoms such as chest discomfort, shortness of breath, fatigue, and autonomic manifestations such as postural orthostatic tachycardia syndrome, and arrhythmias are prevalent and widely recognized a variety of cardiovascular problems, including myocardial inflammation, myocardial infarction, ventricular dysfunction, and endothelial dysfunction, have been described in individuals following the initial acute phase. With over 10,000 published publications on COVID-19 and the cardiovascular system, presenting an unbiased thorough analysis of how SARSCoV-2 affects the system is essentially challenging. This review will provide an overview of frequent cardiovascular manifestations, emphasizing consequences, proposed pathophysiology, and clinical diagnostic manifestation strategy.

KEYWORDS: COVID-19; long COVID-19; SARS-CoV-2; cardiovascular system; post-acute COVID-19; PACS.

2. INTRODUCTION

The year 2020 was a momentous occasion in both history and global health. The Coronavirus disease 2019.

(COVID-19) pandemic has emphasized the dangers of fatal epidemic-prone illnesses wreaking havoc on the globalized world.

In Wuhan, China, pneumonia with an unknown origin became common in December 2019. RNA was isolated and sequenced from bronchoalveolar lavage fluid samples from these individuals.

The culprit responsible for COVID-19 was discovered to be a new beta coronavirus, SARSCoV-2, which has caused morbidity and mortality on an unparalleled worldwide scale.[1-2]

A significant number of organ dysfunctions have been discovered because of considerable and on-going studies on COVID-19.

While the pharmaceutical armamentarium for COVID-19 is still being developed in order to minimize morbidity and death in COVID-19 patients, health communities must contend with a unique condition experienced by some COVID-19 survivors.

This syndrome is associated with persistent symptoms and/or delayed or long-term complications beyond four weeks from the onset of symptoms, known as long haulers, long COVID, or post-acute COVID-19 syndrome (PACS).[3]

3. Proposed Patho-mechanisms of Long COVID in the Cardiovascular System

SARS-CoV-2 is already known to be responsible for the global COVID-19 pandemic on March 11, 2020.[4]

This entity resembles SARS-CoV-1 in many ways since both are positive-stranded RNA viruses with four structural proteins that anchor on the viral envelope.[5]

Among these structural proteins, the spike (S) glycoprotein is the utmost important structure that is responsible for the host cell entrance mechanism.[6]

The SARS-CoV-2 entrance pathway occurs when the S glycoprotein binds to the host cell's

angiotensin- converting enzyme-2 (ACE2) receptor, primarily in the type 2 pneumocytes, which results in viral membrane and host cell fusion.[7]

The process is facilitated by the type II trans-membrane serine protease (TMPRSS2) by activating the S protein. ACE2 receptors are ubiquitously expressed in various organs such as the lungs, intestines, kidneys, and importantly, the heart and endothelium.

Although both SARS-CoV-1 and 2 attach to the same receptors (ACE), enhanced infectivity has been observed in SARS-CoV-2. The reasons are twofold. To begin, SARS-CoV-2 has two-unit S glycoprotein, S1 and S2.[8]

Then, changes in the virus's receptor binding region dramatically boosted SARS-CoV-2 affinity to ACE- 2 by 10 to 20-fold over SARS-CoV-19.[9]

The heightened virulency of SARS-CoV-2 also translates to causing more harm as we highlighted later in the review.

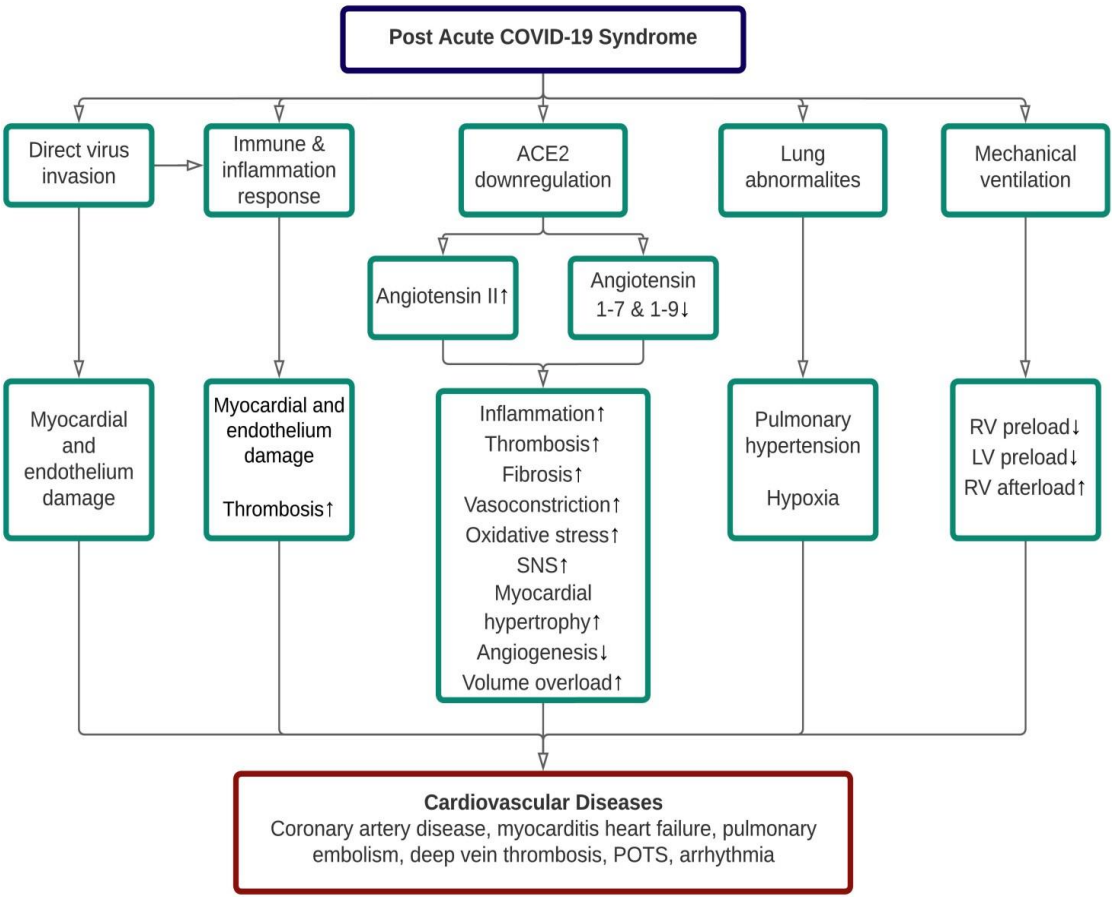

Fig. 1: Pathophysiology of cardiovascular diseases in post-acute COVID-19 syndrome.[3]

- ACE-, angiotensin-converting enzyme; syndrome; RV, right ventricle; SNS, sympathetic nervous system. LV-, left ventricle; POTS, postural orthostatic tachycardia.

4. ABERRANT IMMUNE AND INFLAMMATORY RESPONSE

Types of immunities

- ***Innate Immune Response***

Once within the human body, any pathogen, include in SARS-CoV-2, will elicit innate and adaptive immunity responses. Activation of toll-like receptors 7 and 8 (TLR7 & TLR8), as well as NOD-like receptors (NLR) on the surface of infected lung epithelial cells and alveolar macrophages, increases the production of type I and type III antiviral interferon (IFNs) and several distinct chemokines in the early phase of infection. These IFNs boost the expression of major histocompatibility complex (MHC) class in additional infected cells, allowing CD8+ cytotoxic cells and natural killer cells to block virus replication and restrict viral spread. Concurrently, other chemokine attract additional antigen-presenting cells (APCs) to the site of damage, such as dendritic cells, macrophages, and neutrophils, which then create more chemokine to recruit more CD4+ and CD8+ T cells.[10]

- **Adaptive Immune Response**

APCs and infected host cells initiated the adaptive immune response by presenting the antigen to naive CD4+ helper T cells and CD8+ cytotoxic T cells via MHC class I and II, respectively. This entire process eventually resulted in cytotoxic factors lysis of the infected cells; activation of B cells, which produce specific antibodies to kill SARSCoV-2; and secretion of numerous pro-inflammatory cytokines. such as IFN-, IL-4, IL-5, and IL-13, which activate macrophages and create a vicious cycle resulting in the pathological inflammatory process.[11]

5. PACS

In contrast to acute COVID-19, PACS is defined by the persistence of COVID-19 symptoms for >3–4 weeks. The pathophysiological mechanisms for PACS are not well defined but appear to be different from acute COVID-19. In a follow-up study of 384 patients hospitalized for COVID-19, elevations of D- dimer, ferritin, and C-reactive protein (CRP) normalized within 2 months following discharge. Chest radiographs remained abnormal or worsened in just over 10% of patients. However, in contrast to improving laboratory and imaging findings, fatigue and breathlessness were present in nearly two-thirds of patients.[12]

Chest ra-diagraphs remained abnormal or worsened in just over 10% of patients. However, in contrast to improving laboratory and imaging findings, fatigue and breathlessness were present in nearly two-thirds of patients. The course of PACS tends to be significantly prolonged. Huang et al. found 76% of patients had at least 1 persistent symptom of COVID-19 at a mean of 186 days from hospital discharge.[13]

Generalized decon-ditioning was most common with 63% reporting fatigue and muscle weakness. One quarter had 6-minute walk times below the age- predicted lower limit of normal months after hospitalization. These findings of persistent symptoms are not limited to hospitalized patients. In a cohort of 98 non-hospitalized patients, fatigue was present in 29%, loss of smell and/or taste in 23%, and dyspnea in 9% several months after their initial COVID-19 illness.[14]

Other studies in cohorts of asymptomatic or mild COVID-19 report lower incidences of PACS, but persistent symptoms as far as 8 months are still reported in over 10% of these patients.[15]

Cardiovascular manifestations of PACS

The most common cardiovascular (CV) symptoms present in PACS are chest pain or tightness, palpitations, dizziness, and an increase in resting heart rate (Table 1). These symptoms appear in both hospitalized and non-hospitalized groups. There is no clear relationship with CV symptoms and pre- existing CV disease in PACS.

The underlying pathophysiological link between PACS and the CV system has not definitively been established, but several CV syndromes may be implicated. The evidence for each is summarized in the following sections.[16]

Table 1
Longitudinal studies of Post-Acute COVID-19 Syndrome.

First author	Patients	Mean follow up time	Population	General symptoms	CV symptoms	Notes
Chopra [83]	488	~60 days	Hospitalized	Persistent symptoms of illness, 32.5%; DOE, 22.9%	"Chest problem" on exertion, 16.6%	15.1% readmitted 24.1% with CV disease
Huang [17]	1733	186 days	Hospitalized	Fatigue and muscle weakness, 63%; 6MWT below age predicted normal, ~25%	Palpitations, 9%; dizziness, 6%; chest pain, 5%	7% with CV disease
Mandal [16]	384	54 days	Hospitalized	Fatigue, 67.3–76.9%[a]; Breathlessness, 54.8–63.3%[a]	Not reported	9.7% with CV disease
Petersen [18]	98	125 days	96% non-hospitalized	Fatigue, 29%; loss of smell/taste, 23%; dyspnea, 9%	Chest tightness, 5%	No differences found between those with hypertension, hypercholesterolemia, or diabetes
Carfi [84]	143	60 days	Hospitalized	Fatigue 53.1%; dyspnea 43.4%	Chest pain, 21.7%	35% with hypertension; 7% with diabetes; 4.9% with CV disease
Xiong [85]	538	97 days	Hospitalized	Fatigue, 28.3%	Dizziness, 2.6%; chest pain, 12.3%; "CV related symptoms," 13%; resting HR increase, 11.2%	All symptoms were significantly more common compared to risk factor matched controls
Kamal [86]	287	>20 days	80.2% mild severity of disease	Fatigue, 72.8%; dyspnea, 28.2%	Chest pain, 28.9%	Self-reported: Myocarditis, 1.4%; arrhythmia, 0.3%
Carvalho-Schneider [87]	150	60 days	65% non-hospitalized	At day 60: respiratory symptoms, 91%; flu-like symptoms, 87%; dyspnea, 45%	Chest pain, 16%	49% with no comorbid conditions; 34% drop out rate
Garrigues [88]	120	110 days	Hospitalized	Fatigue, 55%; dyspnea, 42%; cough, 25%	Did not resume sports, 28%; chest pain, 11%	47% with hypertension; 22% with diabetes
Halpin [89]	100	48 days	Hospitalized	Fatigue, 64%; breathlessness, 50%; difficulty with usual activities, 44%	Not reported	10% with CAD, 5% with HF, 41% with hypertension
Moreno-Perez [90]	277	77 days	66% Hospitalized	Dyspnea, 34.4%; abnormal spirometry, 9%	Not reported	36.5% with hypertension, 11.6% with diabetes, 6.9% with CV disease
Logue [19]	177	169 days	91% with mild or asymptomatic COVID-19	Fatigue, 13.6%; shortness of breath, ~5%; myalgia ~5%	Not reported	13.0% with hypertension, 5.1% with diabetes
Morin [91]	478	113 days	All hospitalized, 30% in ICU	Fatigue, 31.1%; dyspnea, 16.3%	Chest pain 8.1%	25.3% with RV dilation, 12.0% with LVEF ≤ 50% (all ICU, no baseline reported), 19.4% with fibrotic lung lesions
Havervall [20]	323	8 months	Healthcare workers without severe COVID-19	Fatigue, 4.0%; dyspnea, 1.9%	Palpitations, 0.6%	Average age 43 years, 83% were women, 0.7% reported palpitations in COVID-19 negative control
Shang [92]	1174	6 months	All hospitalized	Fatigue, 25.3%; dyspnea, 20.4%; myalgia, 13.8%	Chest pain, 9.9%;	No difference in rate of symptoms found between age < 65 and age > 65, 52.1% with abnormal CT chest
Meije [93]	294	7 months	All hospitalized	Fatigue, 26.5%; myalgia, 13.3%; dyspnea, 9.5%	Chest pain, 2.7%	Patients with severe hypoxia had worse respiratory status at 7 months but similar incidence of other symptoms
Armange [94]	214	6 weeks	All non-hospitalized	Dyspnea, 40.2%; cough 19.2%	Chest pain, 10.7%	Only 55% of patients were able to resume sports at 6 weeks
Darley [95]	65	69 Days	86% non-hospitalized	Fatigue, 26.2%; dyspnea, 23.1%	Chest pain, ~5%	Average age 47 years
Jacobson [96]	118	3–4 months	81.4% non-hospitalized	Fatigue, 30.8%; dyspnea, 26.5%; myalgias, 17.9%	Chest pain, 13.7%; palpitations, 6.0%	Symptom burden did not differ between hospitalized and non-hospitalized patients except dyspnea was more common in hospitalized patients
de Graaf [97]	81	6 weeks	All hospitalized, 41% admitted to ICU	Dyspnea, 62%	Any chest pain, 14%; anginal chest pain, 1%; atypical chest pain, 4%; palpitations, 15%	Mean troponin 11 ng/L, mean NT-ProBNP 190 ng/L
Venturelli [98]	767	68 days	88.4% hospitalized, 8.6% admitted to ICU	Fatigue, 36.5%; dyspnea 32.7%; myalgia 5.7%	Chest pain, 4.7%; palpitations, 5.9%	8.2% received cardiology consultation in ambulatory setting
Liang [99]	76	3 months	Hospitalized	Fatigue, 59%; dyspnea, 61%	Chest pain, 62%; palpitations, 62%	86% healthcare worker

Abbreviations: 6MWT, 6-minute walk time; CT, computed tomography; CV, cardiovascular; DOE, dyspnea on exertion; ICU, intensive care unit; LVEF, left ventricula ejection fraction; NT-ProBNP, n-terminal-pro brain natriuretic peptide; RV, right ventricle.
[a] Depending on level of care.

Fig. No. 1: Longitudinal Studies of Post-Acute COVID-19 Syndrome.[16]

- **Management Of Cardiovascular Manifestations Of PACS**

The true risk of an underlying cardiovascular pathology for those with PACS is difficult to assess. While more cases of myocarditis, POTS, and pericarditis will undoubtedly be identified, indiscriminate testing will incur a substantial burden on the healthcare system. On the other hand, future studies are needed to prospectively follow and thoroughly evaluate those with PACS to determine the most appropriate workup.

Basic science research is needed to identify the underlying pathophysiology of PACS and any links to similar syndromes such as MCAS and CFS. CMR techniques and reporting need to be more standardized to detect true myocarditis and these findings need to be correlated with symptoms and objective findings on ECG, ambulatory cardiac monitoring, and echocardiogram.

- **Table 2: Summarizes Priority Areas for Additional Study.[17-20]**

Table 2
Priority areas for further study of cardiovascular involvement in Post-Acute COVID-19 Syndrome.

Syndrome	Area of need	Potential impact
Myocarditis/ pericarditis	Long term significance of myocardial edema, LGE, and pericardial abnormalities seen in patients recovered from severe COVID-19	Understanding of incidental CMR findings, long-term functional consequence, and natural history Reduction in unnecessary diagnostic testing
POTS	Prevalence of patients with POTS following COVID-19	Aggregation of large cohorts of patients to trial potential treatments
Arrhythmia	Ambulatory cardiac monitoring following hospitalization with COVID-19 or in patients with persistent palpitations	Determination of the frequency of long-term arrhythmia in patients recovered from COVID-19
CFS, MCAS, and deconditioning	Link and overlap of these syndromes with PACS	Identification of a plausible biological mechanism for PACS symptoms
"Unmasked" coronary artery disease	Long-term outcomes of patients with troponin elevations during hospitalization for COVID-19	Identification of a population with sub-clinical CAD that will benefit from medical optimization

Abbreviations: CAD, coronary artery disease; CMR, cardiac magnetic resonance imaging; CFS, Chronic Fatigue Syndrome; LGE, late gadolinium enhancement; MCAS, Mast Cell Activation Syndrome; PACS, Post-Acute COVID-19 Syndrome; POTS, Postural Orthostatic Tachycardia Syndrome.

Fig. No. 2: Priority areas of further study of cardiovascular involvement in post-Aute COVID-19 Syndrome.[21]

6. Pathophysiology Of Angiotensin-converting enzyme 2 (ACE2) receptor

ACE2 receptors are the binding sites for SARS-CoV-2 which uses its S-spikes to bind ACE2 as the point of entry into the host cell. The pneumocyte like type 1 and type 2 are expressed and other cell types, like endothelial cells too. The renin-angiotensin-aldosterone system is inversely regulated by ACE2.[3] ACE2 not only functions as a SARS-CoV-2 receptor.[22]

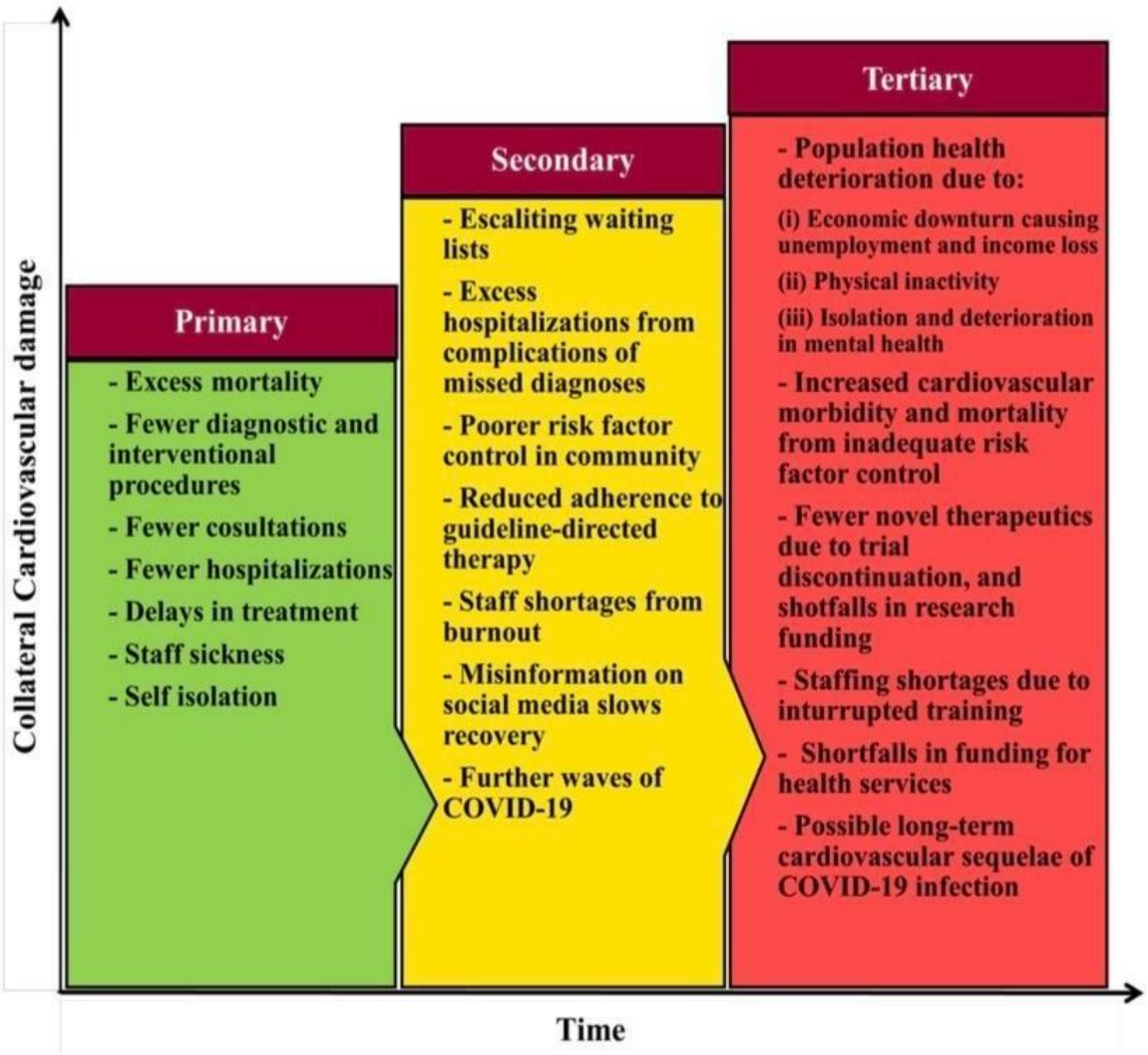

Fig. No. 3: Predicted potential collateral damage of the COVID-19 pandemic to cardiovascular services.[23]

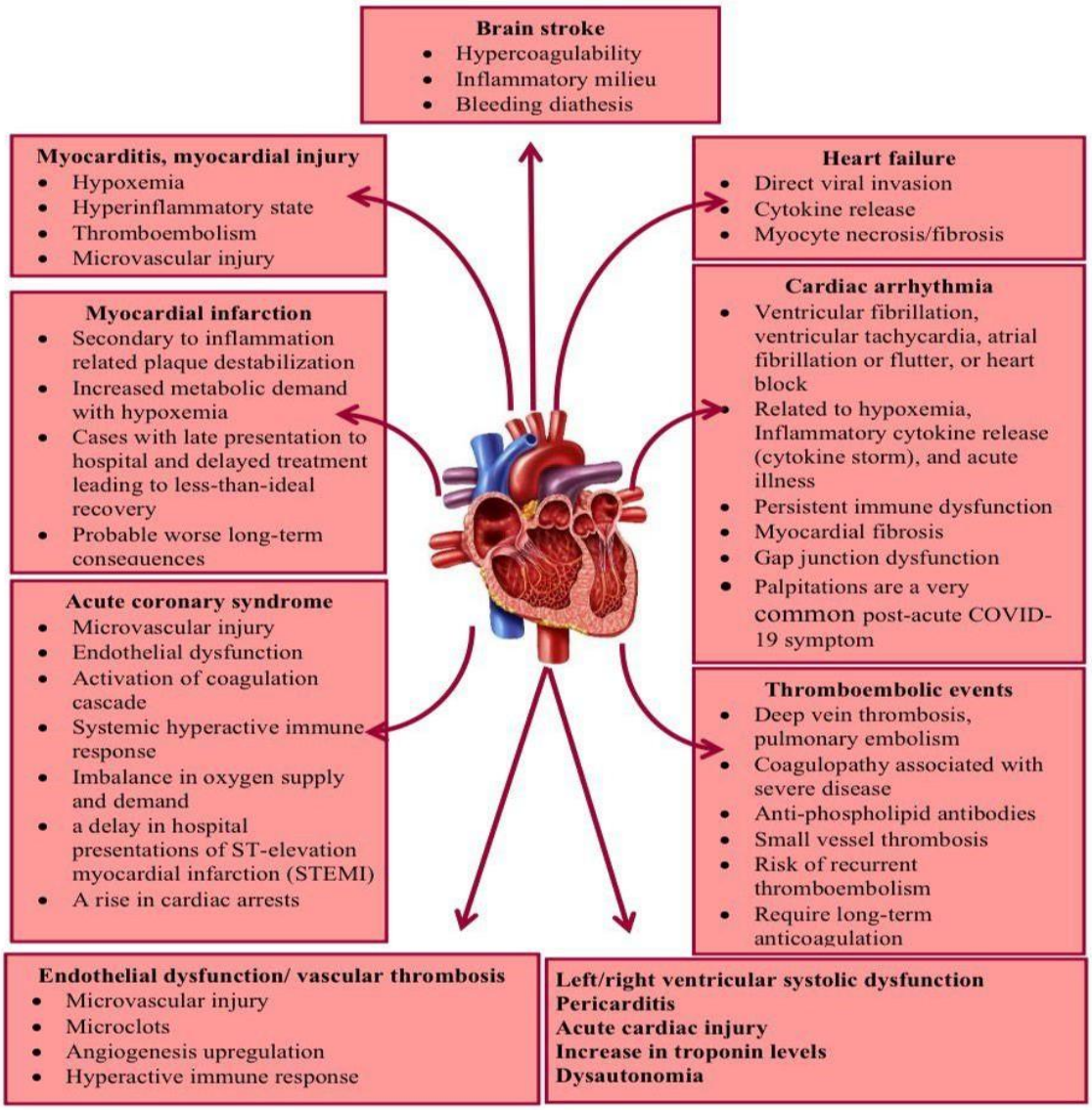

Fig. No. 4: Post-COVID-19 associated cardiovascular complications.[24]

7. *Acute COVID-19 Treatment*

- *Invasive Mechanical Ventilation*

A substantial number of severe and critical COVID-19 patients need mechanical ventilation to support ventilation and gas exchange in the alveoli.[25]

Nonetheless, there are cardiac complications associated with mechanical ventilation use, primarily to the right ventricle (RV) and the left ventricle (LV). Generally, mechanical ventilation could decrease the RV preload and concurrently increase the RV afterload.[26]

Proposed algorithm for management of COVID-19 recovered patients with persistent cardiovascular symptoms or a previous COVID-19 hospitalization with cardiac complications.

a-POTS can generally be managed by primary care physicians, but in atypical or refractory cases, referral to a cardiologist or neurologist is advised.

Abbreviations: BNP, brain natriuretic-peptide; CAD, coronary artery disease; CMR, cardiac magnetic resonance imaging; CRP, C-reactive protein; ECG, electrocardiogram.

ETT, exercise treadmill test; HF, heart failure; MPI, myocardial perfusion imaging; POTS, Postural Orthostatic Tachycardia Syndrome; TTE, transthoracic echocardiogram.[27]

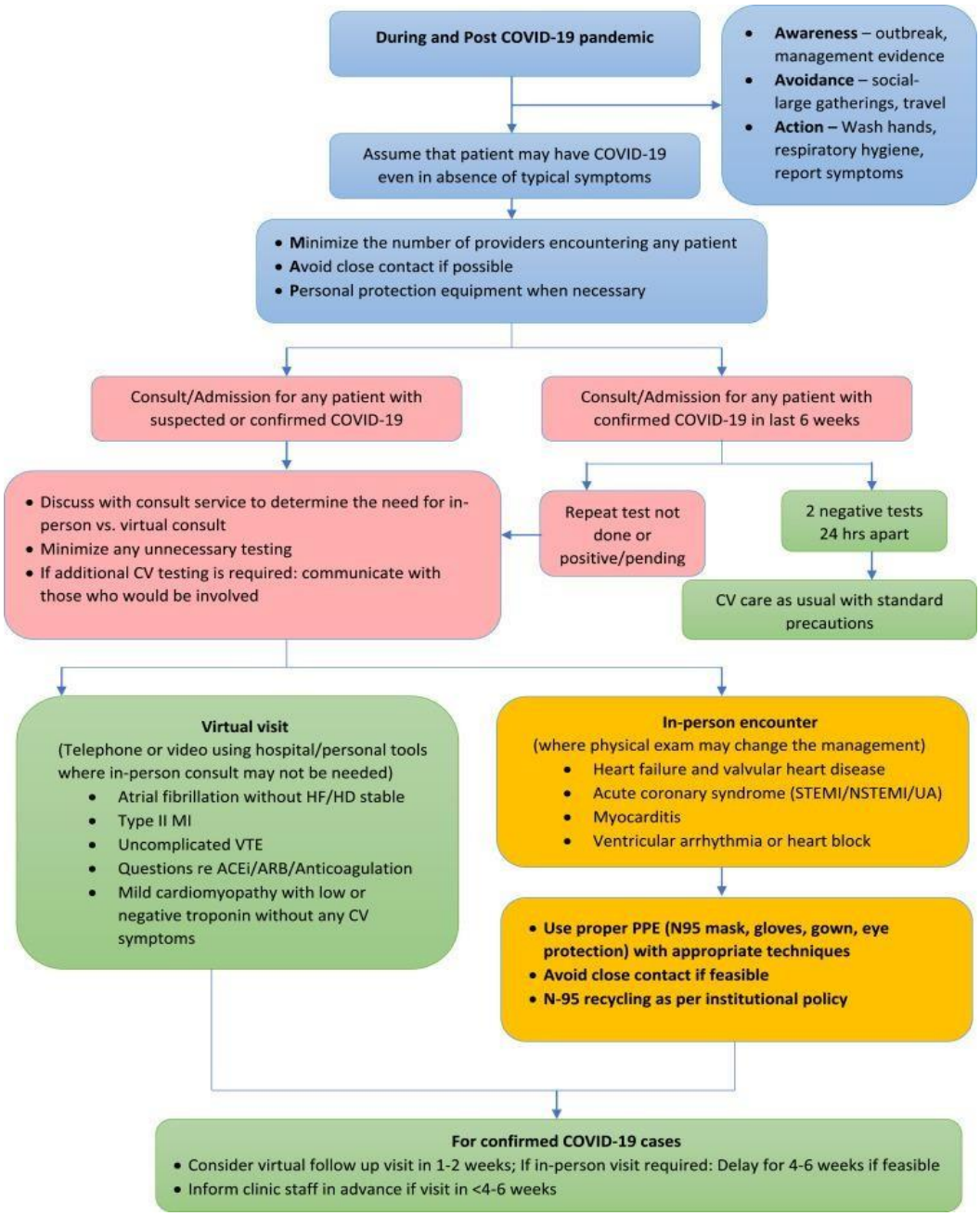

Fig. No. 5: Proposed management pathway for COVID-19-associated myocardial injury.[28]

8. CONCLUSIONS

With the emerging success of the COVID-19 vaccines, the burden of acute COVID-19 will wane, but we will likely be left with a significant number of patients with persistent symptoms even months after COVID-19 infection.

PACS has become a top priority for the healthcare system, and federal institutions such as the National Institutes of Health have dedicated significant funding for research into this new, likely chronic disease process. As the medical community gains a deeper understanding of PACS and its cardiovascular manifestations over the coming years, we will hopefully enhance our ability to identify those at increased risk of these complications and discover effective strategies to prevent and treat this syndrome.

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