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Research Article

# Cardiac Manifestations of COVID-19 - Post COVID Syndromes and Prevalence of Acute Coronary Disease and Heart Failures

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## Abstract

**Background**: The onset of coronavirus has brought an aggressive impact on the cardiology departments of different hospitals. There has been an upsurge of patients and a terrifying increase of mortality. We have witnessed various strains of the SARS-COV-2 virus causing havoc in different parts of the world and affecting all the countries. The virus which more dominantly causes respiratory problems and lung fibrosis now is also seen to have cardiac presentations. The purpose of this study is to find out a relation between COVID 19 and cardiovascular disease and to define the trends observed in the Armenian population since no such data/literature exists in this sphere. This article also aims to find potential pathophysiology associated with the subject.

**Methods:** The medical records of 30 adult and geriatric patients were collected from the cardiology departments of different hospitals in Yerevan, Armenia and were reviewed. 100% percent of patients were identified to have a current or history of coronavirus infection. The age, gender, date of infection, hospital admission, severity of COVID, past medical history, diagnosis and the name of the medications prescribed were noted and analysis was done and trends were observed.

**Results**: Ninety-four percent of patients were identified to have exacerbations plus new manifestations of cardiac problems due to COVID. A direct correlation between severity of the disease course of COVID and the severity of cardiac manifestation was seen. The proposed pathophysiology for cardiac damage appears to be direct myocardial damage caused by viral involvement of cardiomyocytes and the influence of systemic inflammation.

**Conclusion:** The cardiovascular symptoms of the patients are thought to have exacerbated/originated after acute COVID infection which is described in most recent studies and therefore our study aligns itself with other large-scale studies done in this sphere. More information and studies are needed to understand the incidence, aetiology, clinical symptoms, and outcomes of different cardiovascular manifestations in individuals with COVID19, given the disease's enormous burden and the considerable negative prognostic impact of cardiac involvement.

***Keywords:*** *Cardiac Manifestations; Syndromes and Prevalence; Coronary Disease and Heart Failures*

## Introduction

The pathogen responsible for coronavirus disease 2019 (COVID-19), severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has caused unprecedented worldwide morbidity and mortality [1]. The subacute and long-term effects of COVID-19, which can influence many organ systems, are still being researched. Early studies have shown that SARSCoV2 infection has left residual symptoms of fatigue, shortness of breath, chest discomfort, cognitive impairment, arthralgia, and poor quality of life. The procoagulant state produced by SARSCoV2 infection, cell damage and the innate immune response that produces inflammatory cytokines can all be involved in these complications. Initial reports of the post-acute infection effects of COVID19 have been published, and studies from the United States, Europe, and China report results for patients who survived acute COVID19 hospitalization. The majority of patients with COVID19 recover from poor health within a few weeks, but some develop post-COVID symptoms. More than four weeks after being infected with COVID 19, various new, recurrent, or chronic health problems known as 'post-COVID disorders' can occur [2]. Even people who do not show signs of COVID 19 days or weeks after infection can develop post-COVID complications. Over time, these illnesses can manifest themselves as health problems of different types and combinations [3]. Long COVID, long-haul COVID, post-acute COVID-19, long-term COVID effects, and chronic COVID are all terms used to describe these post-COVID problems. This study is the first of its kind for the Armenian population and attempts to show and explain the discrepancies observed.

## Materials And Methods

### Study design and participants

Using medical record abstraction, questionnaires, and physician review -an observational cohort study from two hospitals in Yerevan, Armenia was done. We analysed the outcomes of 30 patients discharged live at 60 days from Heratsi Hospital Complex N1 and Mikaelyan Institute of Surgery which are designated hospitals to treat and admit COVID patients and also specialized cardiology centres in the country. This study was designed to quantify patient infection date with COVID19 and proportionate it with exacerbation of existing heart problems or development of new symptoms. The exacerbation was defined as any event leading to Myocardial infarction, Acute Heart failure, and progression to decompensated state. The use of defibrillator and the number of times resuscitation was performed on the patient additionally qualifies them to be included as exacerbation. Patients were included in the study if they had a discussed cardiac manifestation with COVID19. Inclusion criteria was not limited to one variable like serum markers or ECG findings (Figure 4).

### Data collection/patient selection

All 30 patients who are a part of this study were selected by a trained team of physicians working at Heratsi hospital N1 and Mikaelyan Hospital who analysed the handed the patients' electronic medical records to the researchers. Patient data was collected and evaluated, including demographics, medical history, laboratory exams, comorbidities, complications, and the medical treatments administered to them at the hospital and a detailed history of their routine medications (included antiviral, antibiotic, corticosteroid treatments, immunological glucocorticoid therapy, anti-aggregants, anticoagulants, anti-lipid agents, diabetic medications, blood pressure medications and respiratory support), and outcomes. The EKGs of all patients involved in this study were taken and scanned for further references and diagnosis on acuteness or exacerbation of already existing cardiovascular disease (also including acute on chronic HF) was made based on serum biomarkers – Troponin I and Troponin T, Chest-X-ray and Echocardiography. Lung function tests were also taken in order to have a complete picture. Routine examination like CBC, Hepatitis Panel, lipid and coagulation screening was done on all patients newly admitted to the hospital (Figure 5).

### Statistical analysis

In accordance with PRISMA guidelines, a comprehensive literature search was conducted on PubMed, Cochrane database, Embase, Google Scholar, and Ovid to identify articles that discussed cardiac manifestations in COVID19 patients and cardiac biomarkers with their

clinical implications on COVID19. An extensive literature search was done and relevant data and information is cited and referenced in the discussion. Due to a lack of research data among the papers on this topic, it was not able to conduct an adequate meta-analysis.

## Discussion

Similar results have been observed in European studies, specifically from the post-acute outpatient service studies in Italy. The most commonly reported symptoms were fatigue, dyspnoea, arthralgia, chest pain, with more than half of patients showing 3 or more symptoms. In their study, approximately 45% of patients had poor quality of life when evaluated on the EuroQol visual analog scale [4,17]. The long-term effects of acute COVID-19 were also evaluated in a prospective cohort study from Wuhan, China, based on a complete face-to-face examination of 1,733 patients 6 months after the onset of symptoms. To assess post-acute COVID-19 end organ damage, researchers used questionnaires, physical examinations, a 6-minute walk test (6MWT), blood tests, and possibly lung tests. Pulmonary functional examination/tests (PFT), high-resolution chest computed tomography (HRCT), and ultrasound [18,19]. Fatigue/weakness was the most commonly reported sign, followed by sleep disorders and anxiety [4,20]. COVID19 infections are associated with a variety of comorbid cardiovascular diseases such as hypertension, obesity, diabetes, coronary artery disease, and congestive heart failure [8]. It is unclear whether these findings are age-related, but it is clear that the presence of these comorbidities makes individuals who develop serious illness more sensitive [9]. Reported case fatality rates vary from country to country, probably due to different reporting methods. Nonetheless, COVID19 appears to be disproportionately affecting ethnic groups of men, the elderly and minorities, with a more serious illness course and higher mortality rates. Based on a study by Oronski., *et al.* during hospitalization, up to quarter percent of patients developed serious illness, out of which 5% required intensive care and 2.3% required intubation. The case fatality rate for COVID19 infection was expected to be 1% [5].

### Cardiac complications in covid-19

Chest pain, elevated cardiac biomarkers, arrhythmias, heart damage (acute coronary syndrome, stress ECG, myocarditis) left ventricular (LV) and right ventricular (RV) disorders, pulmonary hypertension, thromboembolic events, arrhythmia, hemodynamic instability, and sudden death are all acute cardiovascular complications in active COVID 19 diseases [10]. Studies show that COVID19 has a high prevalence of cardiac involvement with a variety of clinical symptoms. These include elevated myocardial enzyme levels in 54% of patients, cardiac dysfunction in 41%, and acute cardiac injury in 9% of patients. [5] Acute cardiac injury is associated with poor outcome in patients with COVID 19 [11,12]. The aetiology here may differ from the aetiology of traditional acute coronary artery occlusion / plaque rupture followed by STEMI and Non-STEMI. SARSCoV2 can damage the myocardium (both RV and LV), and coronary circulation directly or indirectly increasing the risk of thrombus formation and acute coronary artery occlusion. Stress cardiomyopathy, atypical myocardial infarction due to tachycardia / stress-induced myocardial demand discrepancies, microvascular embolization, endothelial dysfunction, and cytokine storms due to altered immune and inflammatory responses are all examples of acute cardiac injuries in these patients. Acute pericarditis may be followed by cardiac tamponade, which requires immediate pericardial drainage [5,48].

### Diagnostic approach

Patients with suspected cardiac involvement, such as heart failure, cardiac arrhythmias, or other unexplained ECG abnormalities symptoms should be included in a targeted cardiac assessment. For patients with suspected heart failure, this should include physical examination, serum troponin levels, and serum natriuretic peptide (BNP) measurements. Serum troponin is seen to be elevated in up to 36% of patients with COVID 19, and higher levels have been reported to indicate worse clinical outcomes. Patients with a slight increase in troponin and then a decrease in the next few days had a better prognosis than those patients with a moderate increase in troponin followed by an increase in other biomarkers (interleukin 6, ferritin, lactate dehydrogenase and D-dimer). BNP levels are often high in patients with COVID 19 and are associated with a five-fold increased risk of death. In up to 29% of patients, EKG findings include atrial

tachyarrhythmia, right bundle branch block (RBBB), and abnormal repolarization. RBBB, interventricular conduction retardation, premature atrial contractions, and nonspecific repolarization abnormalities are all ECG functions associated with poor prognosis [40]. Bedside transthoracic echocardiography has been proposed by both European and American Cardiology Society to assess acute cardiac injury or cardiovascular problems in patients with COVID 19. TAPSE (tricuspid valve annulus systolic range of motion) may be maintained until late inCOVID19 patients, but RV radial function may be impaired. Other factors such as RV systolic function markers (such as RV systolic pressure and partial area change divided by RV longitudinal strain) have also been found to be important markers of disease severity and mortality in COVID19. More generally, heart abnormalities are found in50% of all people undergoing echocardiography. Patients with bloodindicators of myocardial damage and abnormal echocardiography showed higher mortality than patients with elevated troponin and BNPlevels [32,33]. Thromboembolic complications in covid-19: Due to its high coagulable state, COVID19 causes thromboembolism problems in both the venous and arterial systems. Intracardiac thrombosis, stroke, deep vein thrombosis, and pulmonary embolism are all the result of common thromboembolism in COVID 19 [32,33].

Post-COVID-19 Cardio Vascular response: ACE2 is a cardio protective transmembrane protein whose expression is downregulated with the aid of using SARS-CoV-2 infection according to the literature. A poorer prognosis, a larger demand for mechanical breathing, and an increased fatality rate are all symptoms and signs of acute cardiac damage [43]. Some people may acquire prolonged sinus tachycardia and high blood pressure after recovering from COVID-19 illness. Although the specific aetiology of this phenomena is unknown lengthy durations of mechanical breathing, the use of inotropic medications, fluid overload, elevated adrenergic tone, and inflammation (interleukins) have all been suggested as possible contributors.

Moreover, cytokine storms may be responsible for short- and long-term CV repercussions such as chronically raised blood pressure and a high resting heart rate, comparable to their acute influence on cardiac damage. Finally, findings link COVID-19 to diabetic complications (such as new-onset diabetes with diabetic ketoacidosis or poor diabetic management) and acute kidney damage.

## Future directions

The confusion caused by the COVID 19 pandemic has led to postponement, cancellation, or modification of many clinical trials, and in some circumstances has led to the adoption of adaptive study designs to address the unique condition of the pandemic. Remote consent, telemedicine monitoring, and the use of proprietary research procedures have all improved the way clinical trials are conducted as a result of these new study designs [45]. The main focus of the ongoing epidemic was the detection and treatment of acute heart damage. Long-term data on COVID-19 survivors with documented cardiac involvement may reveal the true extent of cardiovascular problems and their long-term effects. The results of current and future studies are needed to fully understand the many unanswered concerns about the involvement of COVID-19 in causing heart abnormalities.

The severity of RV and LV dysfunction and its link to increased cardiac biomarkers, as well as pulmonary hypertension and their reversibility after months of infection may say something about the long-term effects of COVID on heart. Arrhythmias were found in 17 percent, 22 percent, and 29 percent of people in various studies. As a result, bigger COVID-19 causes of arrhythmias and conduction anomalies [36]. Speckle tracking echocardiography and advanced multimodality imaging are used to assess subclinical LV dysfunction and coronary flow reserve. Some studies show that endocarditis and subclinical valvular thrombosis may also be caused by COVID. Hereby, these problems give rise to these main questions as to 'What could be the best clinical follow-up plan for COVID-19 survivors who have had a heart attack?' and 'How often do we need a follow-up transthoracic echocardiography?' along with 'Is it required to perform a cardiopulmonary function test with maximal oxygen uptake and clinical and portable blood pressure measurements after the clinical evaluation?' Patients who recovered from COVID-19 had a significant prevalence of myocardial tissue alterations, according to cardiac MRI. It was seen as necessary to assess the impact of these modifications on prognosis.

Although several helpful prescriptions for the general public have been published in the literature during the COVID-19 epidemic, a well-structured cardiac rehabilitation program for COVID-survivors should be devised [46]. ACI was found in up to 50% and 21% of seriously ill and hospitalized COVID19 patients, at a higher rate than other serious illnesses caused by respiratory viruses. Patients with COVID 19 of ACI were older (mean age 70 years), had higher mortality, and had more evidence of systemic inflammation, malignant arrhythmias, shock, and the need for intensive care unit (ICU) treatment. Myocardial injury was the second leading cause of death after respiratory failure [6].

## Pathogenesis

Studies have revealed that direct viral toxicity, including endothelial and microvascular damage, irritation of immune system dysregulation and hyper inflammatory conditions, hyper coagulation with in situ thrombosis and macro-thrombosis, and angiotensin converting enzyme-2 (ACE2) signalling pathway discrepancies are the main pathophysiological mechanism of acute COVID19. Evolutionary similarities between the causative pathogenic coronaviruses may explain the overlap between post-acute COVID 19 episodes and SARS and MERS episodes. SARSCoV2 shares 79% genomic sequence similarity with SARSCoV1 and 50% with MERS-CoV. In addition, SARSCoV1 and SARSCoV2 share the same ACE2 host cell receptor [25]. There are some notable changes, such as the higher affinity of SARSCoV2 for ACE2 compared to SARSCoV1. These may be due to changes in the receptor binding that promotes interaction with ACE2. In contrast to other structural genes, the SARSCoV2 spike gene is branched, with only 73% amino acid similarity to SARSCoV1 in the receptor binding region of the spike protein [22]. After a serious illness, post-intensive care syndrome includes new or increasing disabilities in the physical, cognitive, and psychological areas. The pathophysiology of post-critical care syndrome is multifaceted and, according to which it is assumed to be associated with ischemia and damage of micro vessels, immobilization, and metabolic changes between critical illnesses [31]. Based on the literature, another proposed pathogenesis highlights other pathological pathways that underlie heart damage by COVID19. First, myocardial oxygen supply-demand imbalances in severe hypoxia, hypo perfusion, shock, and stress-induced cardiomyopathy can lead to ACI with elevated troponin and heart failure. Second, high post ventricular load, high prevalence of right ventricular insufficiency and cor pulmonale are associated with low lung compliance in acute respiratory distress syndrome (ARDS), pulmonary vascular dysfunction and positive pressure artificial respiration. [44] Nonspecific systemic inflammation, endothelial dysfunction, and platelet activation are the mechanisms of pulmonary vascular dysfunction in ARDS, along with hypoxemia, vasoconstriction due to inflammation, extrinsic vascular compression, and fibrosis due to pulmonary vascular remodelling. It is one of the failure mechanisms. Third, the interaction between SARSCoV2 and heart cells can cause cardiac dysfunction. Upon binding to the angiotensin converting enzyme 2 (ACE2) receptor, the virus causes significant activation of the innate immune system, causing a violent systemic inflammatory response with the production of pro-inflammatory cytokines [23]. Direct viral damage of endothelial and/or myocardial cells can potentially cause cardiac harm, albeit SARS-CoV-2 detection in these cells is uncommon. Various viral entry receptors, including the ACE2 transmembrane protein, have been found in myocardial cells, endothelial cells, smooth muscle cells, and fibroblasts, and the virus is directly or indirectly involved in the cytotoxic effects of the heart. It suggests that there is a possibility even in individuals with a healthy heart. These cytotoxic effects exacerbate inflammatory endothelial dysfunction and thrombosis-promoting phenotypes and can cause micro thrombosis in the heart tissue. Loss of ACE2 and over activation of the renin-angiotensin-aldosterone pathway can cause a variety of organ damage, including endothelial dysfunction and heart failure. In addition to the loss of immunological competence, patients after COVID are at risk of developing pulmonary fibrosis. This is different from interstitial pulmonary fibrosis (IPF) and is usually seen during follow-up of patients who have recovered. However, post-ARDS fibrosis is a real concern, as symptoms associated with dyspnoea, malaise, and weakness do not appear to be associated with the extent of ongoing lung injury or gas exchange disorders [7,20,21,24].

Direct viral entry, ACE2 downregulation, inflammation, and immune response all affect the structural integrity of the myocardium, pericardium, and conduction system, prolonging cardiovascular sequelae in post-acute COVID-19. In 39 patients with COVID 19, an autopsy study revealed the presence of the virus in 62.5 percent of patients' heart tissue. Subsequent inflammatory reactions can result in cardiomyocyte death and fibro-fatty replacement of desmosome proteins required for intercellular adhesions [26,27].

## Statistics

A strong predictor during severe outcome is also considered by the marker hsTnT (High Sensitivity Troponin T). [28,29,49] For individuals with cardiovascular sequelae between acute infections or persistent cardiac symptoms, continuous clinical and imaging evaluation by ECG and echocardiography over 4-12 weeks may be recommended [35,36]. Low-dose beta-blockers may help patients with postural orthostatic tachycardia syndrome and inappropriate sinus tachycardia regulate heart rate and reduce adrenergic activity [28,29]. Antiarrhythmic drugs (such as amiodarone) should be used with caution in people with fibrotic lung changes after COVID 19 [34]. Acute myocarditis has been reported in patients with COVID 19 with elevated cardiac troponin, abnormal echocardiography (most commonly changes in left ventricular function), and/or electrocardiogram with various intracardiac findings [47]. Few cases have been validated by endo- myocardial biopsy and/or cardiac magnetic resonance. Inflammatory and clot-promoting features, congestive cardiomyopathy, and damage from underlying diseases such as atherosclerosis, chronic ischemic cardiomyopathy, and myocardial hypertrophy are the most common cardiac tissue diseases in deceased COVID 19 patients [41]. Although it being a physical finding, few cases reported localized lymphocytic cardiomyopathy [50]. Some heart complications were also seen to have a relation with the vaccination for COVID19 - as in Myocarditis/pericarditis especially in young adults and adolescent boys, at a rate of approximately 12.6 cases per million doses of the double-dose mRNA vaccine aged 12 - 39 years [9-12].

COVID19 usually reports supraventricular and ventricular arrhythmias and conduction abnormalities. In a recent global study of 4,526 people, 827 had arrhythmias (70% atrial, 20% ventricle), which was associated with increased morbidity and mortality [50]. Due to the coexistence of hypoxia, electrolyte problems, comorbidities, and the use of arrhythmia-inducing agents (hydroxychloroquine, azithromycin, etc.), it is difficult to determine the direct and indirect effects of COVID 19 on cardiac arrhythmias. According to data from the Swedish National Cardiopulmonary Resuscitation Registry, COVID19 is also involved in at least 10% and 16% of all out-of-hospital and in-hospital cardiac arrests and is associated with a significant increase in 30-day mortality rate.) [13-16]. Higher incidence of cardiac problems and cardiac arrest, especially during the first wave of the pandemic, is partly due to the burden on the medical system and delays in seeking medical assistance, probably due to fear of in-hospital COVID 19 or restricted access to medical care [6,30].

According to other retrospective cohort studies in China- 23% patients developed heart failure as complication of COVID-19, whereas in Spain out of a cohort of 7 patients, 4 COVID-19 patients developed cardiogenic shock, resulting in 3 deaths (75% mortality). Similarly, in Germany, according to Bemtgen., *et al.* COVID-19 patient presenting with acute respiratory distress syndrome degenerated into cardiogenic and vasoplegic shock. France and Switzerland also had similar results where, one-third of all the children in the study developed acute heart failure associated with COVID-19 and multisystem inflammatory syndrome.

## Results

3.33% of the patients died throughout the research period, and 74 percent of patients required re-admission. 68% of the 30 patients in this research had chronic symptoms, with 93.33 percent reporting new or exacerbated symptoms. Out of 30 patients, 28 patients were observed to have either exacerbation of existing cardiac problems or develop new CVD altogether. 16 out of the 28 patients had no history of CV surgeries like PCT, CABG, implanting Pacemaker, etc. Majority of the patients of the study had mild arterial hypertension from at least 4 years before the COVID infection who later developed very serious conditions leading to MI and ACS. A detailed history of the patients' sex, age, presenting complaints, their date of infection, date of admission to hospital, diagnosis and medications is given above in table 1. It was also seen in this study that patients having severe COVID had a direct correlation with the severity of their cardiac problems. Comparing results of this study to other populations, it is shown that cardiac manifestations have been observed to be more severe in the Armenian population.

| SN | Gender | Age | COVID Infection | Date of COVID infection | Severity of COVID | Date of Hospital Admission | History | Diagnosis | Medications | Exacerbation of cardiac problems after COVID infection |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F | 84 | Yes | 12.10.2020 | Mild | 25.06.2021 | Diabetes Mellitus; Arterial Hypertension; PCI done previously and 2 stents placed in LCX and Diagonal Artery; 3 years ago Atrial Fibrillation | Arterial Hypertension III; Stenocardia; Anaemia | Furosemide; Omeprazole; Heparin; Atoris; Uperex; Jarnias; Glucotas; Clopidogrel; Egilol | Yes |
| 2 | M | 69 | Yes | Not Specified | Mild | 14.05.2021 | Arterial Hypertension; 2 Stents placed previously; Diverticulosis; Has previously been transfused with blood | Ischemic Heart Disease; Non-Stable Angina; Cardiosclerosis; Arterial hypertension II; Heart Failure II; Anaemia | B- Blockers; Unfractional Heparin; Ciprofloxacin; Atorvastatin | Yes |
| 3 | M | 65 | Yes | 09.10.2020 | Mild | 16.05.2021 | Myocardial Infarction in 2003; CABG surgery done in 2004 | Stable Angina; Ischemic Heart Disease II; Arterial Hypertension; Cardiosclerosis; | Aspirin; Clopidogrel; Ramipril; Atorvastatin; B- Blocker; H2 blocker; Nitro-glycerine; Calcium channel blocker; Amlodipine; Heparin | No |
| 4 | M | 79 | Yes | 02.11.2020 | Severe – b/l polysegmental pneumonia | 12.11.2020 | Admitted with h/o dyspnoea, fever; chest pain for 10 days | Ischemic Heart Disease; Non- STEMI; Acute HF II-III; Pulmonary oedema; Chronic HF III- decompensation; Pulmonary Insufficiency III | A,B- blockers – Carvedilol; Aspirin; Amproxal; Nitro-glycerine; Clopidogrel; Spironolactone; Meropenem; Heparin; Dexamethasone; i/v Furosemide; H2 blocker | Yes |
| 5 | M | 72 | Yes | 15.01.2021 | Severe – Pneumonia- | 16.01.2021 | Arterial HTN; Myocardial Infarction; Stents placed previously; Was admitted unconscious and breathing with ventilator, Resuscitation and Defibrillation done | Chronic IHD; STEMI; Ventricular Fibrillation; Dilated Cardiomyopathy; Heart Failure III; Arterial Hypertension; Chronic Kidney Disease III | Aspirin; Clopidogrel; Nitro-glycerine; B- Blocker; Heparin; Furosemide; NatriBicarbonate; Amiodarone; Ciprofloxacin; Dexamethasone | Yes – The patient passed away due to extreme exacerbation of his cardiac problems by COVID. |

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|---|---|---|---|---|---|---|---|---|---|---|
| 6 | M | 58 | Yes | 14.11.2020 | Severe- b/l poly-segmental pneumo-nia | 14.11.2020 | Arterial Hyper-tension | Chronic Ischemic Heart Disease; Non STEMI; PCI – Stenting done on Proximal LAD and Diagonal; Acute Heart Failure II; Pulmonary Edema | Monosodium-nitrate; Aspirin; Clopidogrel; B- Blocker; Ramipril; Famotidine; Atorvastatin; Furosemide; Ceftriaxone; Dexamethasone; Heparin; Albidol; Zinc | Yes |
| 7 | F | 80 | Yes | 05.11.2020 | Mild | 05.11.2020 | Arterial HTN; Atrial Fibrilla-tion; Pacemaker added | Chronic IHD; Non stable Angina; Permanent Atrial Fibrillation; HF II-III: Arterial Hyperten-sion | Aspirin; B-Blocker; Omeprazole; Ramipril; Spironolactone; Heparin; Furosemide; Ceftriaxone | Yes |
| 8 | M | 59 | Yes | 21.10.2020 | Severe – b/l oneu-monia | 21.10.2020 | DM; Arterial HTN; Placed stent in September 2020 | Arterial HTN; IHD; CKD | Aspirin; A,B Blocker – Carvediol; Nitroglycerin; Fluvoxetine; Furosemide; Dexamethasone; Humulin; Dopamine | Yes |
| 9 | M | 40 | Yes | 20.10.2020 | Severe – b/l poly-segemntal pneumo-nia with 30 fibrosis | 20.10.2020 | Arterial HTN | STEMI; Acute HF; Arterial HTN II; PCI done and stents placed | Aspirin; Clopidogrel; B-Blocker; Atorvastatin; Fametidine; Nitroglycerin; Heparin; Kaliklor; Moxitek; Dexamethasone | Yes |
| 10 | M | 65 | Yes | 20.09.2020 | Severe- with Pneumo-nia | 25.09.2020 | COPD | COPD -Remission; Tricuspid Regurgita-tion III | Aspirin; Furosemide; Spironolactone; Famotidine; Dexamethasone; Ceftriaxone; Auphillin; $MgSO_4$; Heparin; Clopidogrel | Yes |
| 11 | F | 63 | Yes | 03.12.2020 | Severe – with b/l polyseg-mental Pneumo-nia | 17.12.2020 | Hemithyroidec-tomy CL; Arterial HTN | Chronic IHD; Non Stable Angina; Arterial HTN II; Hyperthyroisis | B-Blocker; Calcium Channel Blocker; Heparin; Omeprazole; Dexamethasone | Yes |

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|---|---|---|---|---|---|---|---|---|---|---|
| 12 | M | 68 | Yes | 20.11.2020 | Severe with Pneumonia | 20.12.2020 | Arterial HTN since 2004; Myocardial Infarction; Coronary stenting done | Chronic IHD; Non Stable Angina; Cardiosclerosis; Arterial HTN | Aspirin; Clopidogrel; B-Blocker; Famotidine; Furosemide; Atorvastatin; Heparin; Nitroglycerin; Sulbactid; Dexamethasone | No |
| 13 | M | 55 | Yes | 20.05.2020 | Mild | 20.05.2020 | Arterial HTN I | Arterial HTN III; Ischemic Stroke with Hemiparesis; Ureteritis; Hyperplasia of prostate -Epiphysiotomy | Aspirin; B-Blocker; Adenophytin; Ramipril; Calcium Channel Blockers; Famotidin; Kaliklov; $MgSO_4$; Diclofenac; Ceftriaxone; Analgin dinstrot | No |
| 14 | F | 68 | Yes | 23.05.2020 | Severe with b/l polysegmental pneumonia | 26.05.2020 | Arterial HTN; Atrial Fibrillation; Infarction; | Ischemic Heart Disease; Non state Angina; Cardiosclerosis; Atrial Fibrillation; Heart Failure III; Respiratory insufficiency; Arterial HTNA | Aspirin; B-Blocker; Statins; Analapril; Spironolactone; Furosemide; Clopidogrel; Heparin; Nitroglycerin; Ceftriaxone | Yes |
| 15 | M | 73 | Yes | 15.03.2021 | Severe with pneumonia | 15.05.2021 | Diabetes Mellitus II; Stroke; Arterial HTN | IHD; Non stable Angina; Cardiosclerosis; Arterial HTN II; Ischemic Stroke; PCI with 2 stents placed ay Mid LAD and PCX | Aspirin; Clopidogrel; B-Blocker; Statins; Monosodium-Nitrate; Analapril; Diabloton; Heparin; Amiodarone | Yes |
| 16 | M | 72 | Yes | 28.05.2020 | Severe with b/l polysegmental pneumonia | 28.05.2020 | Myocardial Infarction; CHF with Decreased EF; Defibrillator placed; COPD; Chronic Kidney Disease; Adenoma of Prostate | Chronic IHD; Dilated cardiomyopathy; HF III; COPD; CKD | Aspirin; Famotidine; Furosemide; Statins; Clopidogrel; Heparin; Mostec; Sulbacansid; Dexamethasone; Auphillin | Yes |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 17 | F | 59 | Yes | 16.11.2020 | Severe with b/l Pneumonia | 16.11.2020 | Arterial HTN | Chronic IHD; NonSTEMI; Acute HF on Chronic HF; Decreased EF; Chronic Pancreatitis | Aspirin; Clopidogrel; Statin; Furosemide; B-Blocker; Heparin; Dopamine; Kaliklor; Ceftriaxone; Diltiazem; Auphillin; $MgSO_4$; Dexamethasone | Yes |
| 18 | M | 90 | Yes | 11.05.2020 | Mild | 11.05.2020 | Cholecystectomy | Chronic IHD; Non stable Angina; Chronic HF III | B-Blocker; Statins; Ramipril; Spironolactone; Furosemide; Clopidogrel; Aspirin; Ceftriaxone; Heparin; Nitro-glycerine; Cyneclor | Yes |
| 19 | M | 49 | Yes | 07.12.2020 | Severe with b/l polysegmental pneumonia | 07.12.2020 | Not given | IHD; NonSTEMI; Chronic HF III; Dilated Cardiomyopathy; Atrial Fibrillation | Aspirin; B-blocker; Porphyrine; Heparin; Furosemide; Ceftriaxone; Amliodarone; Dopamine; Digoxin; Spironolactone | Yes |
| 20 | M | 54 | Yes | 22.12.2020 | Severe with Right sided Pneumonia | 22.12.2020 | Arterial HTN; DM II | IHD; NonSTEMI; Dilated Cardiomyopathy; Chronic HF III; Arterial HTN III; | B-Blocker; Furosemide; Spironolactone; Ramipril; Fluvoxetine; Nitrate; Aspirin; Clopidogrel; A-B-Blocker; Insulin; Verapamil; Dexamethasone; Statins; Hypothiazid | Yes |
| 21 | M | 47 | Yes | 09.12.2020 | Mild | 09.12.2020 | Appendectomy | IHD; NonSTEMI; Arterial HTN II; PCI done on Proximal PRCA and MRCA | Aspirin; Clopidogrel; Statins; B-Blockers; Famitidine; Nitrates; Calcium Channel Blockers; Heparin; Nitroglycerine | Yes |

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|---|---|---|---|---|---|---|---|---|---|---|
| 22 | F | 67 | Yes | 07.10.2020 | Severe with b/l polysegmental pneumonia | 25.11.2020 | Arterial HTN; Chronic Lympholeukoxytosis | Arterial HTN III; Respiratory insufficiency II-III degree; Chronic Lympholeukocytosis | Aspirin; B-Blockers; Ramipril; Sulbactid; Heparin; Dexamethasone; Omeprazole; Amproxol; Moxitec | Yes |
| 23 | M | 70 | Yes | 13.11.2020 | Severe with Pneumonia | 13.11.2020 | Arterial HTN; CABG done in 2008 | IHD; Non STEMI; Respiratory insufficiency; Diabetes mellitus II; Heart Failure III | Aspirin; B-Blockers; Clopidogrel; Statins; Heparin; Furosemide; Fiperacillin; Fezobactam; Fluvoxetin; Kyliclov | Yes |
| 24 | F | 82 | Yes | 07.11.2020 | Severe with Pneumonia | 07.12.2020 | Not specified | Chronic IHD; NonStable Angina; Thrombo-embolism of pulmonary artery; b/l pneumo-fibrosis; Chronic HF III; Ascites; Chronic HCV | Clopidogrel; Famotidine; B-blocker; Furosemide; Heparin; Ceftriaxone; Dopamine; Kaliklor; Flumetin | Yes |
| 25 | M | 85 | Yes | 05.11.2020 | Severe with b/l polysegmental pneumonia | 05.12.2020 | HTN; Myocardial Infarction before 10 years | IHD; Non STEMI; Dilated Cardiomyopathy; Arterial HTN; Chronic HF | Aspirin; B-Blockers; Clopidogrel; Atorvastatin; Nitrate; Spironolactone; Furosemide; Heparin; Dexamethasone | Yes |
| 26 | F | 70 | Yes | 18.04.2021 | Severe with b/l polysegmental pneumonia | 18.04.2021 | Myocardial Infarction; Arterial HTN; DM II | IHD; NonSTEMI; Cardiosclerosis; Arterial HTN; Chronic HF II; DM II | Aspirin; Clopidogrel; B-Blockers; Statins; Lisinorpil; Calcium channel Blockers; Heparin | Yes |
| 27 | F | 79 | Yes | 25.11.2020 | Severe with Pneumonia | 02.12.2020 | Atrial Fibrillation; Breast cancer | Chronic IHD; NonSTEMI; Arterial HTN; Atrial fibrillation; Chronic HF; Respiratory Insufficiency | Ceftriaxone; Dexamethasone; Furosemide; Aspirin; Clopidogrel; Statins; Heparins; Omeprazole; B-Blockers; Amilodarone | Yes |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 28 | f | 67 | Yes | 22.12.2020 | Severe with Pneumo-nia | 22.12.2020 | Mitral valve commissuroto-my and replace-ment; Tricuspid valve annuloplasty | IHD; Non stable Anginal Valvular Heart disease; Arterial HTN II; Chronic HF III | B- blockers; Statins; Digoxin; Spironolactone; Vitamin B complex; Fluvoxetine; Heparin; Clopidogrel; Vitamin C; Analapril; Dexamethasone; Calcium channel blocker; Furosemide | Yes |
| 29 | F | 71 | Yes | 11.03.2021 | Severe with Pneumo-nia | 11.03.2021 | Paroxysmal Atrial Fibrilla-tion; Arterial HTN; Parkinson's disease | Respiratory insuf-ficiency I; Total thrombosis of vena Basilica; Non stable angina; Arterial HTN III; Chronic HF II | Aspirin; Clopidogrel; Cobafenon; Kylechloridr; Omeprazole; Heptral; Heparin; Ceftriaxone; $MgSO_4$; Furosemide | Yes |
| 30 | M | 56 | Yes | 01.05.2021 | Mild | 01.05.2021 | Myocardial infarction; Stents placed | IHD; Stable Angina II; Cardiosclerosis; Stenting done in CAD, OM I and OM II; Chronic HF I | Aspirin; Clopidogrel; Statins; Heparin; Pentaprazole; Kyleklor | Yes |

***Table 1***

The most prevalent general symptom was dyspnoea when going up the stairs, followed by cough and prolonged loss of taste and/or smell. The results from our study are aligned with studies from US, Europe, China and India thereby further increasing the credibility of this study (Figure 1 to 5).

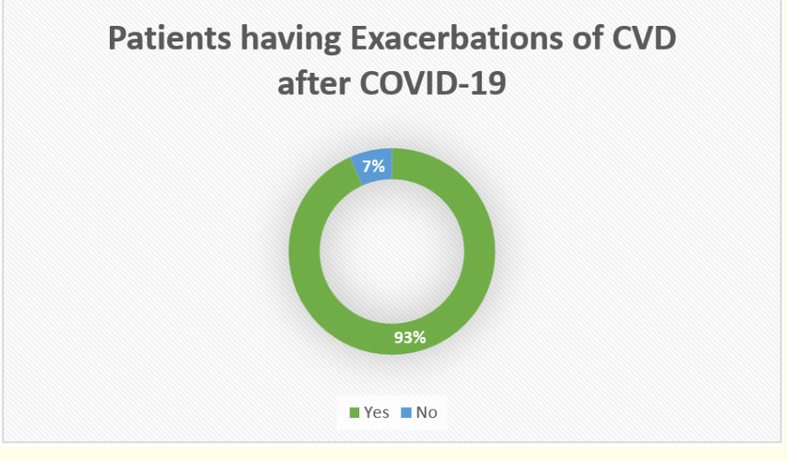


***Figure 1:*** *93% (28) patients having developed new cardiovascular symptoms/exacerbation of existing cardio-vascular disease and 7% (2) patients did not.*

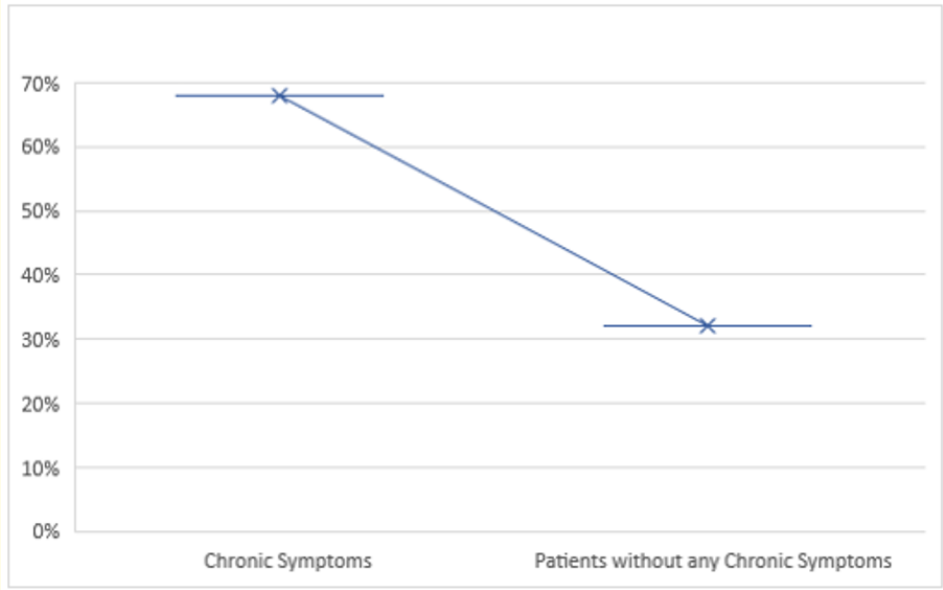


***Figure 2:*** *68% patients developed chronic symptoms and 32% patients presented without any chronic symptoms.*

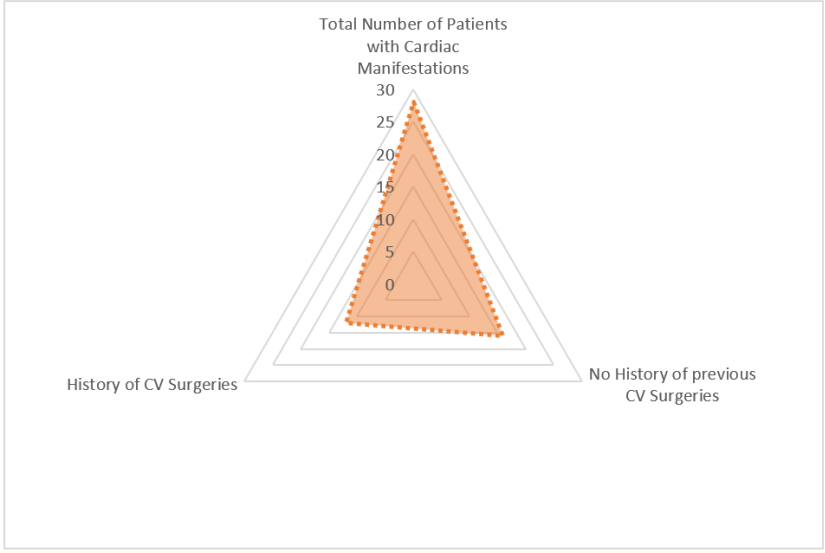


***Figure 3:*** *Out of 28 total number of patients with CM of COVID-19- 12 patients had a previous history of CV surgeries.*
**CM- Cardiac Manifestation.*

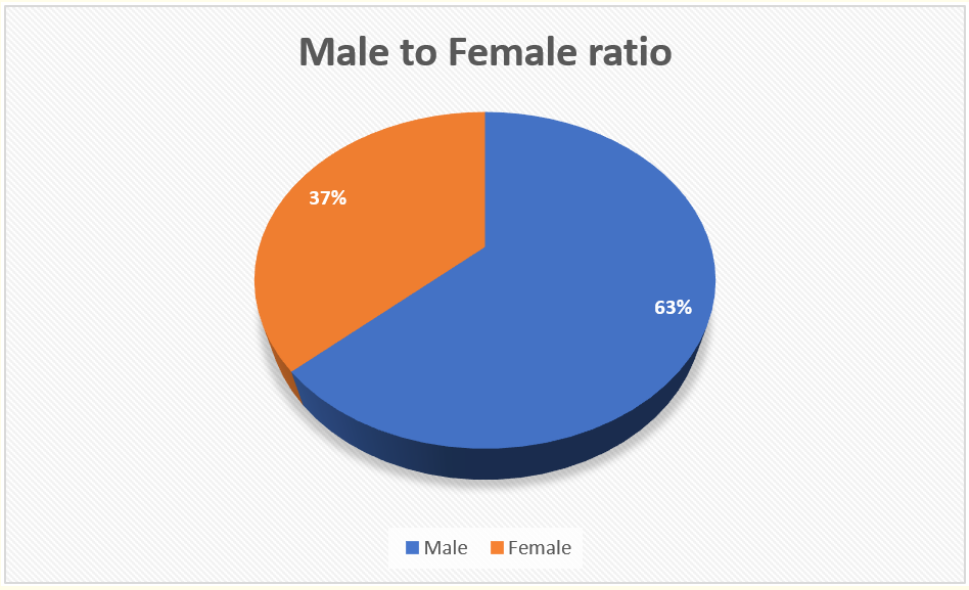


***Figure 4:*** *According to the male:female ratio 19 out of 30 patients were Male, and 11 Female.*

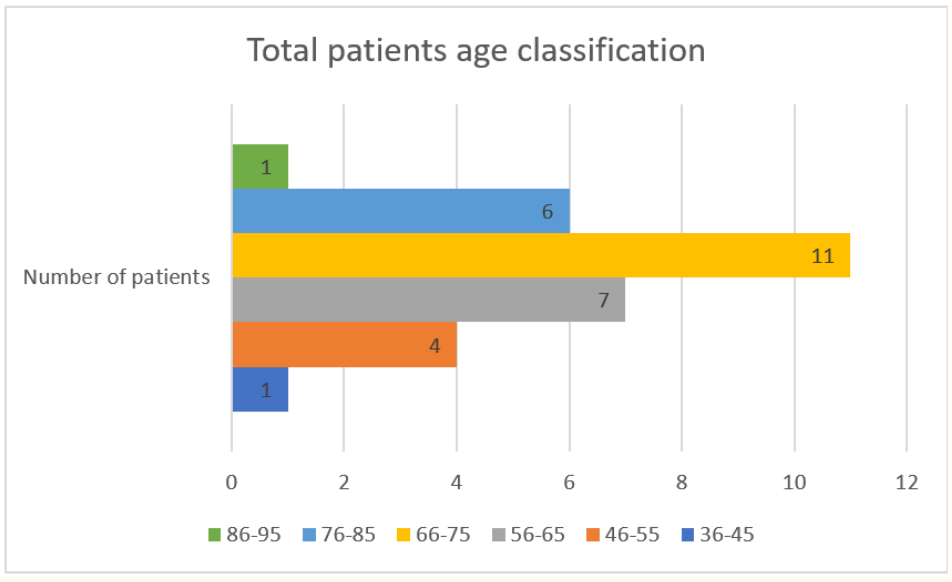


***Figure 5:*** *Maximum number of patients as a part of this study were placed in the age interval 66-75 years -11 patients followed by 56-65 years- 7 patients, followed by 76-85 years - 6 patients, 46-55 years - 4 patients, and 36-45 years and 86-95 years interval with 1 patient each.*

## Conclusion

As more data and clinical experience accumulate in this era, the multi-organic consequences and cardiac manifestations of COVID 19 beyond the acute phase of infection are becoming apparent [43]. Identifying and characterizing the major clinical, serological, imaging, and epidemiological features of COVID 19 in the acute, subacute, and chronic stages of the disease all provide a better understanding of the history and pathophysiology which are needed for future research projects that will further contribute to better understanding [38]. Active and future clinical studies such as prospective cohorts and clinical trials, as well as regular reviews of new discoveries by working groups and task forces, to build a comprehensive knowledge base and improve clinical practice in the field has become very important [39]. Health care providers currently caring for COVID 19 survivors need to identify existing or new symptoms, closely document them, investigate and treat them, and track organ-specific problems that occur during acute illness. Clinicians also need to share information in an easily accessible format, such as publicly available clinical studies and additional resources such as patient advocacy and support groups. In addition, it is clear that care for COVID19 patients does not end upon discharge, and complete outpatient treatment of these patients requires interdisciplinary cooperation [38,42]. As a result, medical institutions and hospitals need to understand the need to establish a specialized COVID 19 clinic where specialists from cardiac disciplines can provide integrated treatment [37]. For those at high risk of post-acute COVID-19 cardiac problems follow-up care should be considered prioritized as they are more susceptible to complications.

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## Authors Contribution

Dr. Rahul Sethi and Dr. Gayane F. Avetisyan: Providing critical revisions that are important for the intellectual content; Approving the final version of the manuscript.

Burhan Kantawala: Collecting the data and interpreting the data; Writing the manuscript.

Sanobar Shariff: Collecting the data and analysing the data, writing the manuscript.

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## Conflict of Interest

The authors of this study have no conflict of interest whatsoever and no affiliation to any of the patients involved in the study.

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