Presentations and Outcomes of Severe Cardiac Complications in COVID-19: Rhode Island Experience

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ABSTRACT

Forty-one (41) patients admitted to Rhode Island hospitals with COVID-19 from April to November 2020 were identified to have severe cardiac complications. Clinical presentations of cardiovascular system toxicity in COVID-19 included myocarditis, pericarditis, cardiomyopathy, ACS and cardiac arrhythmia. Clinical features, hospital outcomes and post-discharge outcomes were characterized. Acute myocarditis (46.3%) and cardiomyopathy (29.3%) were the most common findings followed by cardiac arrhythmia, acute coronary syndrome, and pericardial disease. Pulmonary involvement of COVID-19 was absent in 41.5% of patients. Comorbid cardiovascular conditions were absent in 29.3% of patients. Severe cardiac complications in COVID-19 were associated with an in-hospital mortality rate of 61%. Among survivors with COVID-19-related cardiomyopathy, only 20% demonstrated recovery of LV function on follow-up echocardiography done within 12 weeks after initial diagnosis. Identification, diagnosis and management of severe cardiac complications in COVID-19 are discussed.

KEYWORDS: COVID-19, myocarditis, cardiomyopathy, acute coronary syndrome, arrhythmia, pericarditis

ABBREVIATIONS

ACEi – Angiotensin-converting enzyme inhibitors
ACS – Acute coronary syndrome
ARB – Angiotensin II receptor blockers
NSTEMI – non-ST elevation myocardial infarction
STEMI – ST elevation myocardial infarction
RV – Right ventricle/ventricular
VF – Ventricular fibrillation
VT – Ventricular tachycardia

INTRODUCTION

COVID-19 is a clinical syndrome arising from infection with SARS-CoV-2 coronavirus which is known to produce interstitial pneumonia with/without respiratory failure, and thrombosis due to hypercoagulability. Cardiac injury may also occur. The incidence and extent of severe cardiac complications in COVID-19 is undefined and the range of presentations and outcomes is still being characterized. This report adds to the growing body of evidence by providing detailed information on severe cardiac presentations in COVID-19, hospital outcomes and establishes early post-discharge outcomes in follow-up. We aim to aid the clinician in early identification, diagnosis and management of cardiac complications in COVID-19.

METHODS

Patient Selection

Institutional review board approval was obtained for retrospective review of patient records. 2,229 patients admitted with COVID-19 to the Rhode Island Hospital, The Miriam Hospital and Newport Hospital in Rhode Island from April 1 to November 30, 2020 were identified. All patients identified tested positive for presence of SARS-CoV-2 coronavirus via nasopharyngeal swab or serum serology testing. Discharge diagnoses associated with hospitalization were reviewed for the following: myocarditis, viral myocarditis, cardiomyopathy, cardiogenic shock, congestive heart failure, acute systolic heart failure, cardiac arrythmia, ventricular tachycardia, ventricular fibrillation, pericarditis, pericardial effusion, cardiac tamponade, ST elevation myocardial infarction and non-ST elevation myocardial infarction. In addition, cardiac troponin I values were obtained for all 2,229 cohort patients, those with a maximum troponin value ≥15 mg/dL were selected. The final study population included patients with at least one of the above discharge diagnoses and/or cardiac troponin I ≥15 mg/dL.

Definitions of Cardiac Complications

- Myocarditis was defined as elevated cardiac troponin I and absence of acute coronary syndrome.
- Cardiomyopathy was defined as newly diagnosed left ventricular dysfunction in absence of CHF history.
- RV dysfunction was defined as newly diagnosed right ventricular systolic dysfunction in the absence of CHF history.
- Cardiac arrhythmia was defined as presence of cardiac arrhythmia during hospitalization.
- Pericarditis was defined as presence of chest pain or other symptoms, EKG changes and/or pericardial effusion during hospitalization.

- Acute coronary syndrome (STEMI, NSTEMI) was defined as anginal symptoms, EKG abnormalities, cardiac troponin I elevation and as noted by discharging provider.
- Patients presenting with STEMI were presumed to have coronary ischemia as the cause of troponin I elevation and cardiomyopathy, rather than myocarditis. Additionally, patients may have had more than one severe cardiac finding, e.g., cardiomyopathy and myocarditis.

Post-hospital discharge, follow-up with cardiology clinic and echocardiogram, if obtained, were reviewed for surviving patients.

RESULTS

A total of 41 patients hospitalized with COVID-19 infection were identified to have severe cardiac complications. Clinical characteristics and outcomes are presented in **Table 1** (click to view pdf). The overall incidence of severe cardiac complications among the larger hospitalized cohort was 1.8%. Clinical findings for all patients, survivors and non-survivors are summarized in **Table 2**. The mean age was 66 [61-71 95%CI]; 30 of 41 patients (73.2%) were male, and 29 of 41 (70.7%) patients had a previous history of cardiac or related conditions, including coronary artery disease, hypertension, congestive heart failure and diabetes. The most common presenting symptom was dyspnea occurring in 41.5% of patients, with chest pain reported by 17.1% of patients. Concurrent COVID-19 pneumonia, defined as characteristic patchy diffuse pulmonary infiltrates and hypoxia, was present in 24 of 41 (58.5%) patients. Concurrent pulmonary embolism was present in 3/41 (7.3%) patients. Cardiogenic shock was present in 21 of 41 (51.2%) patients.

Myocarditis was found in 19/41 (46.3%) of patients; cardiomyopathy was found in 12/41 (29.3%) of patients; STEMI in 8/41 (19.5%), pericarditis was found in 5/41 (12.2%); and severe RV dysfunction was found in 5/41 (12%) of patients. 15 of 41 (36.6%) of patients experienced cardiac arrythmia with atrial fibrillation occurring in 22% and ventricular tachycardia/fibrillation occurring in 12% of patients.

In patients without a previous history of CHF, newly diagnosed left ventricular dysfunction as defined by ejection fraction <50%, was found in 11 patients. Newly diagnosed left ventricular dysfunction was associated in 2/12 patients with ST elevation myocardial infarction and with

Table 2. Summary of COVID-19 clinical findings among patient with severe cardiac complications

	All Patients [95%CI]	Survivors [95%CI]	Non–survivors [95%CI]
Total #	41	16	25
Age	66 [61–71]	61 [54–68]	70 [61–79]
Female (%)	11 (26.8)	5 (31.3)	6 (24.0)
Cardiovascular risk factors present (%)	29 (70.7)	9 (56.2)	20 (80.0)
COVID–19 pneumonia present (%)	24 (58.5)	7 (43.8)	17 (68.0)
Cardiogenic shock present (%)	21 (51.2)	4 (25)	17 (68.0)
New LV dysfunction (%)	12 (29.3)	9 (56.3)	3 (16.0)
New RV dysfunction (%)	11 (26.8)	3 (18.8)	8 (32.0)
Severe Cardiac Complication (%): Cardiomyopathy Myocarditis Pericarditis Severe RV dysfunction STEMI NSTEMI Arrythmia	 12 (29.3) 19 (46.3) 5 (12.2) 5 (12.2) 8 (19.5) 11 (26.8) 15 (36.6)	 9 (56.3) 10 (62.5) 3 (18.8) 0 (0.0) 2 (12.5) 4 (25) 3 (18.7)	 3 (12.0) 9 (32.0) 2 (8.0) 5 (20.0) 6 (24.0) 7 (28.0) 12 (48.0)
BNP (pg/mL) mean	680 [398–961]	368 [164–573]	882 [452–1313]
Troponin (ng/mL) mean	17 [10–25]	23 [7–39]	14 [7–21]
Troponin <0.5 ng/mL (%)	11 (26.8)	5 (31.3)	6 (24.0)
CRP (mg/dL) mean	211 [171–252]	191 [131–251]	226 [171–282]
D–dimer (mg/mL) mean	10740 [5309–16171]	8785 [460–17110]	12015 [4771–19258]
Received COVID–19 specific treatment (%)	21 (51.2)	4 (25.0)	17 (68.0)
LOS (days) mean	9 [6–13]	9 [4–13]	10 [5–15]

Abbreviations: BNP – brain natriuteric peptide, CRP – c-reactive protein, LOS – length of hospital stay, LV – left ventricular, LVEF – Left ventricular ejection fraction, MI – myocardial infarction PCI – percutaneous coronary intervention, RV – Right ventricle

Normal reference values: BNP 0.0-72.3 pg/mL, troponin I 0.006-0.06 ng/mL, D Dimer 0-300 ng/mL and CRP 0-10.0 mg/L.

viral myocarditis in 10/12 patients. In patients without a previous history of CHF, newly diagnosed right ventricular dysfunction was observed in 11 patients, with 5/11 (45.4%) characterized as severe by echocardiographic examination.

The most common electrocardiographic changes included ST segment depressions in 10 of 41 (24.4%) patients, ST elevations in 8 of 41 (19.5%), as well as atrial fibrillation, T wave inversions, sinus tachycardia and others.

Serum chemistry analysis revealed a B-natriuretic peptide mean of 680 [398-961 95%CI] pg/dl, mean troponin I 17 [10–25 95%CI] mg/dl, mean CRP 211 [171–252 95%CI] mg/dl, and mean DDimer 10740 [5309–16171 95%CI] mg/ml. Pericardial effusion was found in 5 of 41 (12.2%) patients. Three patients presented with findings of cardiac tamponade and underwent pericardiocentesis. Pericardial fluid volumes removed ranged from 250–700cc, fluid analysis revealed nucleated cells ranging from 86–17,544 cells/cmm, and cytologic examination with reactive acute inflammation in 3/3 samples.

Cardiac catheterization was performed in 6 of 41 patients. Findings included normal coronary arteries in 2 patients with acute viral cardiomyopathy, and coronary artery disease including single and multivessel disease in 4 patients. Three patients presenting with STEMI underwent percutaneous coronary intervention and coronary artery stent placement.

Twenty-one of 41 (51.2%) patients received COVID-19 specific treatment defined as corticosteroids and/or remdesivir. Cardiac specific treatment included management of acute myocardial infarction (26.8%), goal-directed management of congestive heart failure (21%), anti-arrhythmic agents (12%) as well as pericardiocentesis, ECMO, and PCI. The average length of stay in hospital was 9 [6-13 95% CI] days. Mortality was observed in 25 of 41 (61%) of patients.

Among 16 patients who survived to discharge, 13 patients had follow-up data as presented in **Table 3**. Among patients with COVID-19 cardiomyopathy 5 patients had follow-up echocardiography with a mean follow-up time of 2.75 months; 4/5 (80%) had persistent reduced LV function and 1/5 (20%) had recovery of normal LV function.

DISCUSSION

Clinical presentations and outcomes of 41 patients with severe cardiac complications hospitalized with COVID-19 infection were examined. Acute myocarditis and cardiomyopathy were the most common findings followed by acute coronary syndrome and pericardial disease. Cardiac arrhythmias due to atrial fibrillation and ventricular arrythmias were also found. Severe RV dysfunction was found in 12% of patients. Severe cardiac complications in COVID-19 were associated with a high mortality rate; 61% of patients died in hospital after initial presentation. Among survivors with COVID-19 related cardiomyopathy, only 20% demonstrated recovery of LV function on follow-up echocardiography within 12 weeks of diagnosis.

Findings which included the high incidence of pre-existing

Table 3. Post-hospital discharge outcomes in survivors among COVID-19 patients with severe cardiac complications

Age/ Sex	Outcome	Cardiac Complication	Readmission to Hospital within 30 days	Outcome after discharge	Duration of follow-up
68/M	Survived	Myocarditis, pericarditis, cardiomyopathy	None	Admitted with CHF, LVEF severely reduced 6 weeks later	1.5 months
67/M	Survived	Cardiomyopathy, myocarditis	None	LVEF 35%, symptoms of CHF	1.5 months
54/M	Survived	Cardiomyopathy	None	LVEF 35%	6 months
24/M	Survived	Cardiomyopathy	None	Lost to follow up	
54/F	Survived	Pericardial Effusion	None	No further cardiac complications	7 months
59/M	Survived	Pericarditis	None	Normal cardiac function, no effusion	1.5 months
56/F	Survived	Cardiomyopathy, myocarditis	None	Improved, LVEF 60%, RV normal	2 months
61/F	Survived	Myocarditis, cardiomyopathy, cardiac arrythmia	None	Lost to follow up	
51/M	Survived	Cardiomyopathy, myocarditis	None	Improved symptoms, pending repeat echocardiogram	2 months
67/F	Survived	Cardiomyopathy, myocarditis	None	Lost to follow up	
73/M	Survived	STEMI	None	LVEF 35%, ongoing symptoms of CHF	7 months
91/M	Survived	NSTEMI, myocarditis	None	No echocardiography, functional status continues to decline	4 months
50/M	Survived	Myocarditis, NSTEMI	+	Deceased during readmission	1 month
69/F	Survived	Myocarditis, NSTEMI	None	No further cardiac complications	11 months
73/M	Survived	NSTEMI, myocarditis	None	Cardiac stress test with coronary disease, medically managed	3 months
63/M	Survived	STEMI, cardiomyopathy	None	EF 38%, improved symptoms	7 months

Abbreviations: CHF – congestive heart failure, LVEF – Left ventricular ejection fraction, RV – Right ventricle

cardiac co-morbidities (71%), the presence of COVID pneumonia (58.5%) and cardiogenic shock (51%) and the relatively high mortality among this cohort (61%) are consistent with previous reports.[1-5] We report an overall low incidence of severe cardiac complications among the larger hospitalized cohort of 1.8%. Notably, 41% of patients with severe cardiac complications did not have associated COVID-19 pneumonia, 29% did not have cardiac comorbidities and most surprising, at least 32% did not have a significant elevation of troponin I. These findings suggest that the clinical presentation of severe cardiac complications in COVID-19 is not dependent upon the presence of pulmonary findings, existing cardiovascular disease or elevated biomarkers. Additionally, new RV dysfunction was present in upwards of 25% of patients suggesting yet another pathway for cardiac manifestations in the COVID-19 patient population. Overall, the range of pathology to the cardiovascular system in COVID-19 is broad and includes myocarditis, pericarditis, cardiomyopathy, ACS, severe RV dysfunction and cardiac arrhythmia.

Myocarditis and Cardiomyopathy associated with COVID-19

No formal definition of cardiomyopathy and myocarditis in COVID-19 infection exist currently.[1-6] We define COVID-19 associated cardiomyopathy as a clinical syndrome with the following components: active SARS-CoV-2 infection, signs and symptoms of acute congestive heart failure, and newly discovered systolic ventricular dysfunction. We define viral myocarditis due to COVID-19 as active SARS-CoV-2 infection with an elevation in the serum troponin I biomarker in the absence of type I myocardial infarction. We note that it is challenging to delineate viral myocarditis from type 2 myocardial infarction, where myocardial demand may outpace supply resulting in relative ischemia and ultimately myocardial infarction.[6] Within these clinical definitions, 3 major pathways of cardiac injury in COVID-19 associated cardiomyopathy emerge: ischemia-related, direct cardiac cytotoxic/cytokine-mediated effect, and acute right ventricular failure with volume overload.[7-9]

COVID-19 has been shown to induce a prothrombotic state and may predispose toward coronary plaque rupture with resulting acute coronary syndrome.[10] Ischemia due to coronary artery disease does not fully explain the presentation of cardiomyopathy in COVID-19.[11] SARS-CoV-2 which has been shown to infect myocardial cells through ACE2 receptor may lead to direct cytotoxic myocardial damage resulting in myocarditis and ventricular dysfunction.[6,8-10] The cytokine release and subsequent activation of immune-inflammatory mechanisms in the myocardium may be responsible for acute ventricular dysfunction.[5-7] Right ventricular failure appears to be a separate clinical entity in COVID-19 and may be explained by concurrent cardiac cytotoxic damage, severe interstitial pneumonia, and associated thrombotic microangiopathy which may lead to acute RV systolic dysfunction and pressure overload in setting of increased pulmonary pressures.

Clinical features of COVID-19 associated myocarditis and cardiomyopathy include presenting symptoms of dyspnea, chest pain and fever, elevated cardiac troponin I and BNP, elevated inflammatory markers including CRP and DDimer and findings of ventricular dysfunction on echocardiography. Elevated BNP and NT-proBNP are associated with poor outcomes and reflect volume overload and clinical heart failure.[3-5,7,11] Elevated troponin levels are associated with increased mortality in all patients with COVID-19.[3-5,7] Myocarditis and cardiomyopathy in COVID-19 occur in the absence of pulmonary findings, requiring vigilance and specific attention to the cardiac findings from the clinician.

It is unclear if treatments aimed at viral replication, such as the anti-viral remdesivir, and at the inflammatory cascade such as dexamethasone and other corticosteroids, are effective in treating COVID-19 associated myocarditis and cardiomyopathy.[11] We observed a wide variety of treatment approaches and further study will be required to ascertain survival benefits. We recommend clinicians follow established treatment guidelines for COVID-19 and utilize goal-directed congestive heart failure management including ACE/ARBs, beta-blockers with known mortality benefit, diuretics and other guideline-driven strategies.[12]

Pericardial disease in COVID-19

We report 5 cases of pericarditis associated with COVID-19, 3 of which presented with cardiac tamponade and cardiogenic shock. Clinicians must remain vigilant to recognize this presentation as cardiac biomarkers were not significantly elevated, and most cases were identified after the onset of cardiogenic shock. The mechanism of pericarditis may be similar to myocarditis with reactive inflammation occurring in the pericardium with resulting effusion. SARS-CoV-2 has been detected in pericardial fluid associated with cardiac tamponade.[13-15] As in other viral pericarditides, diagnosis may be suspected when the patient reports chest pain, and is found to be hypotensive, tachycardic with muffled heart sounds along with the finding of pulsus paradoxus in the setting of cardiac tamponade.[14,15] Echocardiography is crucial to confirm the diagnosis of pericardial effusion and assess for the presence of cardiac tamponade. Fulminant myopericarditis appears to be a rare but life-threatening cardiac complication in COVID-19 infection.[13] Management of pericardial disease in COVID-19 is supportive in nature and may rely on use of corticosteroids. The role of non-steroidal anti-inflammatory agents and colchicine, standard of care in idiopathic/viral pericarditis prior to COVID-19, has not been established.[15] Pericardial drainage is indicated when cardiac tamponade is detected.

ACS in COVID-19

The overall incidence of ACS in COVID-19 has been reported to be 1%; we report 8 cases of STEMI and 11 cases of suspected NSTEMI.[16] Identification of ACS in COVID-19 is challenging due to the considerable overlap between the presentation of viral myocarditis and acute ischemia.[6]

Hypercoagulability and the hyperimmune response associated with COVID-19 may lead to coronary plaque instability and rupture, leading to formation of coronary thrombosis and resulting clinical presentation.[16-18] Type 2 myocardial infarction is common in COVID-19 and is associated with worse outcomes.[4] The presence of chest pain and other anginal symptoms, ischemic changes on ECG and elevated troponin should prompt evaluation and treatment for acute coronary syndrome per established AHA/ACC guidelines.[19] In our report, patients with suspected NSTEMI were ultimately referred for coronary disease risk stratification to the post-hospital, and presumably, post-infectious setting. In cases of STEMI, treatment included percutaneous coronary intervention in 4 cases and medical management in others, similar to other reports.[16-18] ACS patients with COVID-19 showed an increased risk of cardiogenic shock, life-threatening arrhythmia and had decreased overall survival.[17] Further investigation is necessary to delineate the management approach to ACS in COVID-19.

Severe RV dysfunction in COVID-19

Right ventricular dysfunction has been identified in COVID-19 infection with a rate of 30% noted in several reports, associated with elevated right ventricular systolic pressures.[20] We observed severe right ventricular dysfunction in 12% of patients. RV dysfunction and pressure overload may arise from left ventricular dysfunction in the setting of myocarditis or acute ischemia.[20-22] The right ventricle may be subject to direct myotoxic effects of SARS-CoV-2 and thrombotic microangiopathy accompanied by micro- and macro-thrombosis in the pulmonary vasculature leading to RV volume overload.[20,21] Patients presenting with circulatory shock should undergo screening for RV function via echocardiography.[20] Specific management recommendations for RV dysfunction in COVID-19 are not yet available. Options include measures to reduce pre-load, after-load, anti-coagulation and addressing other underlying factors. In our report all 7 patients with severe RV dysfunction died in hospital.

Arrhythmia in COVID-19

Cardiac arrythmias have been reported to complicate up to 17% of COVID-19 infections with VT/VF occurring in up to 5.9% of cases.[23,24] We observed cardiac arrhythmia in 60.9% of patients and 12% with VT/VF. Atrial fibrillation was the most commonly observed arrhythmia in our report and others.[23] A wide range of cardiac arrhythmias in COVID-19 has been reported including tachy and bradyarrhythmias, QTc prolongation and ventricular tachycardia and/or fibrillation.[23-26] The pathogenesis of cardiac arrhythmia in COVID-19 is as yet poorly characterized but may stem from direct myocardial injury, severe hypoxia-related myocardial stress and further direct injury to the conduction system related to inflammation and cytokine activation cascade in the myocardial tissues.[24] Amiodarone was the most commonly reported anti-arrhythmic medication reported in the treatment of cardiac arrhythmia in COVID-19.[23] In our report 3 out of 5 patients who experienced VT/VF died in hospital. One patient with existing severe aortic stenosis developed atrial fibrillation refractory to medical management and transitioned to comfort care.

Treatment and outcomes in COVID-19 associated cardiac complications

Treatment of severe cardiac complications in COVID-19 rests on early identification and is based on established treatment of congestive heart failure, ACS, pericardial disease and cardiac arrhythmia.[1,6,8,25] We observed a range of treatment approaches including goal directed management of congestive heart failure, medical and invasive management of myocardial infarction and pericarditis, and anti-arrhythmic medications in cardiac arrythmia.

Owing to the early occurrence of the first "wave" of the COVID-19 pandemic in Rhode Island, we report on limited post-discharge outcomes in survivors with severe cardiac complications. Among 9 patients who had newly diagnosed cardiomyopathy with left ventricular dysfunction caused by myocarditis or ACS and survived to discharge, 5 patients had follow up echocardiography within 12 weeks with only 1 patient showing an improvement in left ventricular function. Echocardiographic findings of diastolic dysfunction after COVID-19 myocardial injury have also been reported.[25] We recommend that registries are established to track outcomes in COVID-19 patients with severe cardiac complications.

CONCLUSIONS

Presentations and outcomes of COVID-19 patients with severe cardiac complications were identified and described. Clinicians must be aware of and recognize cardiac complications associated with COVID-19 including myocarditis, cardiomyopathy, pericardial disease, severe RV dysfunction and cardiac arrhythmias, which may occur in the absence of pulmonary findings, comorbid cardiovascular disease and elevated cardiac biomarkers. Early outcomes suggest that most patients do not fully recover left ventricular function after the initial diagnosis of COVID-19 associated cardiomyopathy, but further investigation is required.

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