Annals of Case Reports and Clinical Studies
Case Report (ISSN: 2834-5673)

COVID-19 Associated Cardiomyopathy with Recovery Demonstrated at 1-Year Follow Up: A Case Report

Okonkwo MC[1], Aguwa1UT[2], Racherla M[3], Kashif M[4], Ali S[4], Naroo M[3], Aziz MA[3*]

[1]Johns Hopkins University School of Medicine, USA

[2]Wills Eye Hospital, Philadelphia, USA

[3]Johns Hopkins Howard County Medical Center, Johns Hopkins Medicine, USA

[4]Allama Iqbal Medical College, Pakistan

Citation: *Okonkwo MC, Aguwa1UT, Racherla M, Kashif M, Ali S, Naroo M, Aziz MA. COVID-19 Associated Cardiomyopathy with Recovery Demonstrated at 1-Year Follow Up: A Case Report. Ann Case Rep Clin Stud. 2024;3(4):1-7.*

Received Date: 05 June, 2024; **Accepted Date:** 10 June, 2024; **Published Date:** 17 June, 2024

[*]**Corresponding author:** Muhammad Asif Aziz, Johns Hopkins Howard County Medical Center, Johns Hopkins Medicine, USA

ABSTRACT

Cardiomyopathy is now well-established as a potential manifestation of COVID-19 infection. Although many studies have focused on the acute presentation of COVID-19 associated cardiomyopathy, there have been fewer cases demonstrating recovery after significant derangements in cardiac function. Data on the recovery of cardiac function in the setting of COVID-19 associated cardiomyopathy is mixed, with some studies suggesting that cardiac function may remain impaired months after acute hospitalization. Cases demonstrating recovery of cardiac function may provide greater insight into the natural history and long-term outcomes of COVID-19 infection.

In this case, a woman in her 30s with past medical history of recent COVID-19 infection presented to the emergency department with new respiratory distress. Echocardiogram during acute hospitalization demonstrated systolic heart failure with reduced ejection fraction (25-30)% and grade 3 diastolic dysfunction. At an 11-month follow-up visit, echocardiogram demonstrated significant recovery of left ventricular ejection fraction (60-65)% with resolution of previously diagnosed cardiomyopathy. Here, management of COVID-19 associated cardiomyopathy included supportive care and medical management for left ventricular dysfunction with resolution demonstrated in the outpatient setting. Further studies are necessary to describe long-term outcomes of COVID-19 associated cardiomyopathy in larger cohorts.

Keywords: Cardiomyopathy; COVID-19; Echocardiogram; Case report

INTRODUCTION

The COVID-19 pandemic imposed devastating impacts worldwide, with significant acute and post-acute complications of illness. Cardiac dysfunction is a potential manifestation of COVID-19 infection, with studies

identifying cardiovascular complications in 18% to 60% of cases [1]. Acute ischemic heart disease and acute heart failure were found to be the most prevalent cardiac events in adults during a COVID-19 associated hospitalization [2]. Although some studies investigate associations between incident cardiac dysfunction and recent COVID-19 infection [3-5], less robust data exists to describe the long-term outcomes of COVID-19 associated cardiomyopathy that was diagnosed during an acute hospitalization. Therefore, data on the recovery of cardiac function in the setting of COVID-19 associated cardiomyopathy is mixed. Some studies suggest that cardiac function remains reduced at 2 and 6 months after diagnosis of COVID-19 associated cardiomyopathy [6,7], while some data suggests that there are trends toward normalization of cardiac function [8].

We describe COVID-19 associated cardiomyopathy in a patient with few previous risk factors for cardiac disease. This case report outlines management of the acute illness and recovery of cardiac function demonstrated on echocardiogram at follow-up visit 11 months post-infection.

CASE PRESENTATION

Written informed consent was obtained from the patient for publication of this case report and accompanying images and videos. A copy of the written informed consent is available for review by the editorial office of this journal (Table 1).

Table 1: Timeline of case events.

Time	Events
Two weeks prior to admission	Symptom onset, positive COVID-19 test
5 days prior to admission	Worsening of symptoms with new cough, shortness of breath, nausea, vomiting, paroxysmal nocturnal dyspnea
2 days prior to admission	Diagnosis of bronchitis at urgent care, initiation of oral doxycycline
Presentation to emergency department, Hospital Admission Day 1	Right perihilar infiltrate on chest x-ray, initiation of intravenous azithromycin and ceftriaxone
Hospital Admission Day 2	Diagnosis of new cardiomyopathy, echocardiogram showing ejection fraction 25 to 30%
Hospital Admission Day 3	Discharged with recommendations for follow-up studies with outpatient cardiologist (ANA serology, iron studies, HIV, ischemic evaluation, cardiac magnetic resonance imaging)
11-month follow-up	Recovery of cardiac function demonstrated on echocardiogram

IV: Intravenous; ANA: Antinuclear Antibody.

Patient information

A woman in her 30s with past medical history of hypertension and recent COVID-19 infection 3 weeks prior presented to the Emergency Department (ED) with a chief complaint of shortness of breath. Associated symptoms included cough, sore throat, nausea and vomiting. The patient had been prescribed doxycycline for bronchitis two days prior to presentation with persistence of symptoms. The patient had obtained COVID-19 booster vaccination 6 months prior to presentation. Patient social and family history was non-contributory.

Clinical findings

In the emergency department, the patient was febrile, tachycardic, and hypertensive with increased work of breathing. Notably, the patient was not hypoxic. Physical exam was notable for decreased breath sounds. Labs demonstrated no leukocytosis or derangements in electrolytes or renal function. However, D-dimer was elevated to 1.26. Lactate was within normal limits (Table 2).

Table 2: Relevant laboratory values from patient's initial presentation in the emergency department. Reference ranges are provided within the table. Abnormal lab values have been highlighted in red. "H" represents the particular value is elevated compared to reference range.

	Reference Range	Labs – Initial Presentation
White Blood Cell Count	4.50 – 11.00	5.9
Platelet Count	150 – 305 K/ cu mm	376 (H)
Lactate	0.5 – 2.0 mmol/L	1.1
D-Dimer	0.19 – 0.79 mg/L FEU	1.26 (H)

Diagnostic assessment

Electrocardiogram (EKG) demonstrated sinus tachycardia with non-specific ST and T changes and were not suggestive of an acute coronary syndrome (Figure 1A). Chest x-ray featured right perihilar infiltrates. Subsequent Computed Tomography (CT) of the chest did not show pulmonary embolism but was notable for alveolar consolidation with surrounding ground glass infiltrates in the right lower lobe, cardiomegaly, and small pericardial effusion (Figure 1B). Trans-Thoracic Echocardiogram (TTE) showed mildly dilated left ventricle with reduced Left Ventricular (LV) ejection fraction (25-30)% and grade 3 diastolic dysfunction (Supplemental Digital Content, Video S1). Results were also notable for moderately dilated left atrium, moderate mitral regurgitation, and mild tricuspid regurgitation.

Together, presentation with dyspnea, tachycardia, along with evidence of new cardiac dysfunction on echocardiogram in the setting of recent COVID-19 infection in a young, healthy patient was suggestive of a COVID-19 associated cardiomyopathy.

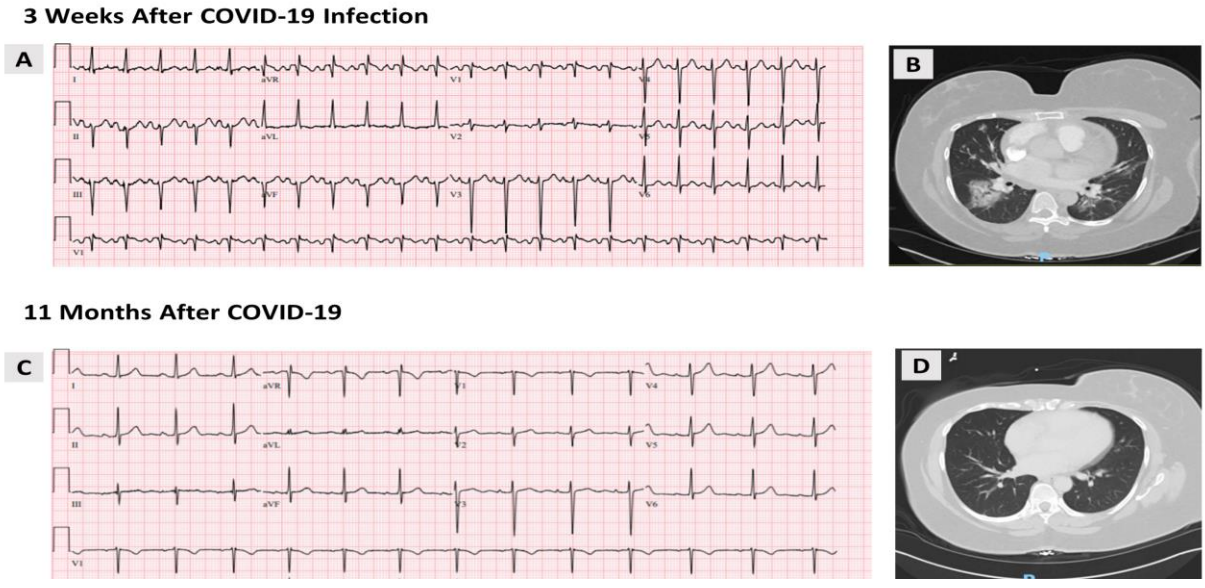

Figure 1: EKG and CT imaging from original presentation (A and B) and follow-up appointment after 11 months (C and D). Initial EKG shows sinus tachycardia and left axis deviation with non-specific ST and T wave changes. B): CT scan from initial presentation demonstrates lower lobar consolutation and ground glass infiltrates in the right lung, with nodular infiltrates scattered between both lungs. C): EKG from 11 months after initial presentation shows normal sinus rhythm and normal axis. D): CT scan from 11 month follow-up shows resolution of consolidations in lung.

Therapeutic intervention

Medications at the time of presentation include doxycycline 100 mg twice daily and benzonatate 100 mg three times daily. Supplemental oxygen was administered via nasal cannula in the setting of respiratory distress. Given the consolidation seen on lung exam, the patient was started on intravenous ceftriaxone. Beta blockers were prescribed for blood pressure control, and fluids were avoided.

To address her new cardiomyopathy, the patient was started on sacubitril/valsartan, spironolactone, and low dose torsemide. She was discharged on day 3 of hospitalization following symptom improvement with follow-up with an outpatient cardiologist. To address the new diagnosis of HFrEF, she received recommendations at discharge for initiation of a sodium-glucose transport protein 2 (SGLT-2) inhibitor.

Follow-Up and outcome

The patient presented for a follow-up appointment nearly 1-year post-hospitalization as part of peri-surgical evaluation for an unrelated elective procedure. At time of follow-up, the patient had maintained adherence to treatment with carvedilol, losartan, and torsemide with corresponding prescription fill history and regular follow-up with outpatient cardiologist. EKG and CT scan at time of follow-up demonstrated resolution of ST changes and infiltrates, respectively (Figure 1C,1D). Repeat TTE nearly 1-year post-hospitalization demonstrated markedly improved ejection fraction of 60% with mild concentric LV hypertrophy (Supplemental Digital Content, Video S1). TTE results also showed lack of valvular abnormalities and resolution of previous cardiomyopathy. Patient continues follow-up with an outpatient cardiologist.

DISCUSSION

This case describes a patient with scant medical history and recent COVID-19 infection. Initial work-up revealed cardiomyopathy with significant left ventricular dysfunction with EF to 25%-30%. These findings are suggestive of acute cardiomyopathy as sequelae of COVID-19 infection. Prompt medical management was initiated with a diuretic to minimize complications from volume overload and goal-directed medical therapy following standard treatment guidelines for HFrEF [9]. Echocardiogram at 11-month follow-up appointment demonstrated resolution of the cardiomyopathy.

It is now well-established that COVID-19 disease is caused by the SARS-CoV-2 virus, which enters human cells and causes cytokine release and inflammatory response [1]. The damage produced by these inflammatory processes

are thought to be precursors of cardiac dysfunction and have been associated with arrhythmias, acute coronary syndromes, acute myocardial damage, and heart failure [2].

Our patient's presentation is generally consistent with clinical findings reported in studies of COVID-19 associated cardiomyopathies. According to Omidi et al's comprehensive analysis, dyspnea, elevated troponin and D-dimer, and LV dysfunction are commonly reported findings of cardiomyopathies in patients with confirmed COVID-19 infection and are consistent findings in this patient's presentation [10]. Although ischemic disease is a cause of cardiac dysfunction, global cardiomyopathy would be expected to demonstrate regional wall motion abnormalities on echocardiogram or be associated with severe multivessel Coronary Artery Disease (CAD) [11]. This patient's lack of risk factors for coronary artery disease and subsequent recovery without coronary intervention make diagnoses of ischemic cardiomyopathy or new underlying CAD unlikely [12]. Overall, the patient's history, presentation, and findings of new global cardiac dysfunction with non-specific EKG findings are suggestive of a COVID-19 associated cardiomyopathy.

Of note, the pathogenesis of cardiomyopathy in COVID-19 is not well understood. Infection with the SARS-COV-2 virus induces widespread cytokine release and dysregulated immune activity, processes that are implicated in proposed mechanisms of COVID-19 associated cardiomyopathy [1,13]. Myocarditis has been identified on cardiac MRI and endomyocardial biopsy in cases of cardiac dysfunction in COVID-19 [13,14]. Similarly, stress or Takotsubo cardiomyopathy, classically triggered by physical, psychological, or metabolic stress, has been identified in several cases reports of COVID-19 associated cardiomyopathies [15]. It is possible that these or other processes may represent a primary mechanism through which COVID-19 associated myopathies evolve. Further studies of cardiac pathology through tissue biopsy may better elucidate the contributing pathophysiology in cases of COVID-19 associated cardiomyopathy.

With limited data on recovery from acute COVID-19 associated cardiomyopathy6-8, further study is required to better describe long-term natural history. For patients such as the one that we describe here, the lasting effects of COVID-19 infection may lead to prolonged follow-up with specialty care providers or chronic medical management of cardiac dysfunction. Studies therefore can also investigate the long-term outcomes and healthcare burden in patients with diagnosed COVID-19 cardiomyopathy.

CONCLUSION

This case highlights a presentation of COVID-19 associated cardiomyopathy with subsequent management and recovery at long-term follow-up. Future studies can investigate the long-term natural history, outcomes, and healthcare burden of COVID-19 associated cardiomyopathies. Together, these studies continue to build an understanding of COVID-19 related complications and options for management.

FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

AUTHOR CONTRIBUTIONS

MCO and UTA wrote the manuscript and compiled data. MR retrieved echocardiogram images and revised the manuscript. MK, SA, MN, and MAA wrote and revised the manuscript.

DATA SHARING STATEMENT

All data generated or analyzed during this study are included in this published article and its supplementary information files.

CONFLICT OF INTEREST STATEMENT

The authors declare no financial conflict of interest with regard to the content of this manuscript.

REFERENCES

1. Fairweather D, Beetler DJ, Di Florio DN, Musigk N, Heidecker B, Cooper LT. COVID-19, myocarditis and pericarditis. Circ Res. 2023;132(10):1302-19.
2. Patone M, Mei XW, Handunnetthi L, Dixon S, Zaccardi F, Shankar-Hari M, et al. Risks of myocarditis, pericarditis, and cardiac arrhythmias associated with COVID-19 vaccination or SARS-CoV-2 infection. Nature. 2022;28(2):410-22.
3. Xie Y, Xu E, Bowe B, Al-Aly Z. Long-term cardiovascular outcomes of COVID-19. Nat Med. 2022;28(3):583-90.
4. Wang H, Li R, Zhou Z, Jiang H, Yan Z, Tao X, et al. Cardiac involvement in COVID-19 patients: mid-term follow up by cardiovascular magnetic resonance. J Cardiovasc Magn Reson. 2021;23(1):14.
5. Mitrani RD, Dabas N, Alfadhli J, Lowery MH, Best TM, Hare JM, et al. Long-term cardiac surveillance and outcomes of COVID-19 patients. Trends Cardiovasc Med. 2022;32(8):465-75.
6. Lassen, MCH, Skaarup, KG, Lind, JN, Alhakak AS, Sengeløv M, Nielsen AB, et al. Recovery of cardiac function following COVID-19 – ECHOVID-19: a prospective longitudinal cohort study. Eur J Heart Fail. 2021;23(11):1903-12.
7. Fayol A, Livrozet M, Boutouyrie P, Khettab H, Betton M, Tea V, et al. Cardiac performance in patients hospitalized with COVID-19: a 6 month follow-up study. ESC Heart Fail. 2021;8(3):2232-9.
8. van den Heuvel FMA, Vos JL, van Bakel B, et al. Comparison between myocardial function assessed by echocardiography during hospitalization for COVID-19 and at 4 months follow-up. Int J Cardiovasc Imaging. 2021;37(12):3459-67.
9. Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2022;145(18):e895-1032.

10. Omidi F, Hajikhani B, Kazemi SN, Tajbakhsh A, Riazi S, Mirsaeidi M, et al. COVID-19 and cardiomyopathy: a systematic review. Front Cardiovas Med. 2021;8:695206.
11. Pellikka P, Arruda-Olson A, Chaudhry FA, Chen MH, Marshall JE, Porter TR, et al. Guidelines for performance, interpretation, and application of stress echocardiography in ischemic heart disease. J Am Soc Echocardiogr. 2020;33(1):1-41.
12. Alkhawam H, Sogomonian R, El-Hunjul M, Kabach M, Syed U, Vyas N, et al. Risk factors for coronary artery disease and acute coronary syndrome in patients ≤ 40 years old. Future Cardiol. 2016;8(12):728-34.
13. Fairweather D, Beetler DJ, Di Florio DN, Musigk N, Heidecker B, Cooper LT. COVID-19, myocarditis and pericarditis. Circ Res. 2023;132(10):1302-19.
14. Heidecker B, Dagan N, Balicer R, Eriksson U, Rosano G, Coats A, et al. Myocarditis following COVID-19 vaccine: incidence, presentation, diagnosis, pathophysiology, therapy, and outcomes put into perspective. A clinical consensus document supported by the Heart Failure Association of the European Society of Cardiology (ESC) and the ESC Working Group on Myocardial and Pericardial Diseases. Eur J Heart Fail. 2022;24(11):2000-18.
15. Shah RM, Shah M, Shah S, Li A, Jauhar S. Takotsubo syndrome and COVID-19: Associations and implications. Curr Probl Cardiol. 2023;46(3):100763.