

COVID-19 and acute myocarditis: current literature review and diagnostic challenges

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<http://dx.doi.org/10.1590/1806-9282.66.S2.48>

SUMMARY

INTRODUCTION: In the current literature, there has been an upsurge of cases of COVID-19-induced acute myocarditis. In this case-based review, we aimed to describe the clinical characteristics, imaging findings, and in-hospital course of acute myocarditis. In addition, the limitations of the myocarditis diagnosis were discussed since only fulminant myocarditis cases have been mentioned in the current literature.

METHODS: We performed a review of the literature of all patients who were diagnosed with COVID-19-induced acute myocarditis using the databases of PubMed, Embase, and the Cochrane.

RESULTS: 16 case reports were found to be related to COVID-19-induced acute myocarditis. We observed that the ECG findings in most of the COVID-19 patients were non-specific, including diffuse ST-segment elevation, non-specific intraventricular conduction delay, sinus tachycardia, and inverted T-waves in anterior leads. Echocardiographic findings of COVID-19-induced acute myocarditis patients ranged from preserved left ventricular ejection fraction (LVEF) without segmental abnormalities to reduced LVEF with global hypokinesia. Interestingly, a few patients with COVID-19-induced acute fulminant myocarditis were steroid-responsive and had an amelioration with glucocorticoid and immunoglobulin therapy.

CONCLUSION: Despite the COVID-19 pandemic worldwide, a limited number of cases has been shared in the current literature. There are a lot of difficulties in the differential diagnosis of acute myocarditis in the context of COVID-19.

KEYWORDS: Coronavirus Infections. Betacoronavirus. Myocarditis. Cardiomyopathies.

INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a new variant form of coronavirus that is responsible for the coronavirus disease 2019 (COVID-19). On March 2020, the World Health Organization (WHO) declared COVID-19 a global pandemic after the confirmation of cases in five continents¹. Although the virus mainly infects the lung epithelial

cells causing respiratory signs and symptoms, there has been an upsurge of cases that presented COVID-19-induced acute myocarditis. Currently, several mechanisms have been proposed to explain the underlying pathophysiology of COVID-19-related acute myocarditis². It has been suggested that direct viral contact through angiotensin-converting enzyme 2 (ACE-2)

DATE OF SUBMISSION: 23-Jun-2020

DATE OF ACCEPTANCE: 11-Jul-2020

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signaling pathways might have a role in the myocardial injury³. In addition, cytokine release syndrome has been proposed to be the main pathophysiology of COVID-19-induced acute fulminant myocarditis³. In this case-based review, we aimed to describe the clinical characteristics, imaging findings, and in-hospital course of acute myocarditis as well as the limitations in regard to myocarditis diagnosis. In addition, we tried to identify the possible underlying mechanisms of COVID-19-related acute myocarditis, whether it is caused by direct viral damage or an inadequate host immune response.

METHODS

We performed a review of the literature of all patients who were reported to have COVID-19-induced acute myocarditis using the databases of PubMed, Embase, and the Cochrane. All databases were searched in June 2020 using the following keywords: ‘COVID-19, acute myocarditis’ and ‘COVID-19, acute myopericarditis’. In total, 16 case reports were found to be related to COVID-19-induced acute myocarditis. Despite the fact that neither endomyocardial biopsy (EMB) nor cardiac magnetic rezones imaging (CMR) have been performed in several reports, the cases reported under the title of acute myocarditis were included in order to provide the current circumstance in terms of acute myocarditis in the COVID-19 era.

Baseline clinical characteristics

Tables 1 and 2 summarize the clinical characteristics of COVID-19-induced acute myocarditis patients⁴⁻¹⁷. The majority of the cases were male, and hypertension is the most commonly observed risk factor. In all patients, chest pain and dyspnea were the most common symptoms even though one case presented with atypical symptoms such as neck pain and diarrhea⁴⁻⁸. Fever is the most commonly observed sign in COVID-19 patients with acute myocarditis. Patients with lower systolic blood pressure developed cardiogenic shock following admission⁴.

Lab findings

It is recommended to screen for cardiac injury in patients hospitalized for COVID-19 infection because an early diagnosis has an ultimate role in changing management. Therefore, the measurement of cardiac biomarkers, including troponin and brain natriuretic peptide (BNP), should be performed on admission.

We noted that most cases had elevated troponin and BNP levels on admission. Lymphopenia was present in patients with COVID-19-related acute myocarditis, as expected in a usual viral infection.

Imaging features

In patients who are suspected of having COVID-19-related acute myocarditis, baseline electrocardiography (ECG) should be carried out on admission. In this review, we observed that ECG findings in most of the COVID-19 patients were non-specific, including diffuse ST-segment elevation, non-specific intraventricular conduction delay, sinus tachycardia, and inverted T-waves in anterior leads^{4-8,10,14,17}. Despite frequent changes in ECG, no specific changes were detected in some cases^{9,16}. Because there is the possibility of using a point-of-care ultrasound, it is recommended that echocardiography (ECHO) should be performed if there is a suspicion of COVID-19-related acute myocarditis. In this case-based review, the ECHO findings of patients with COVID-19-induced acute myocarditis ranged from preserved left ventricular ejection fraction (LVEF) without segmental abnormalities to reduced LVEF with global hypokinesia^{4,10,14-17}. Those with a severe and depressed LV function developed cardiogenic shock, thereby having a worse prognosis^{4,6}. Although CMR was not performed in all cases because of prolonged acquisition time and the fact that COVID-19 infection is highly contagious, it showed myocardial edema and sub-epicardial late gadolinium enhancement in some parts of the LV^{7-12,16}. Interestingly, no EMB was performed to confirm the diagnosis in all patients even though it is considered a gold standard. Hemodynamic instability, a contagious risk due to prolonged acquisition time, and coagulopathy were the main reasons for not performing EMB.

In-hospital treatment

There is no consensus for the prescription of the standard heart failure treatment in addition to the antiviral therapy to patients hospitalized with COVID-19-induced acute fulminant myocarditis. Interestingly, intravenous (IV) inotrope treatment and circulatory support have been used in selected cases. Moreover, IV glucocorticoid and immunoglobulin therapy have been implemented in patients with complicated acute myocarditis^{4,6,10,15,17}. Most patients received heart failure therapy, antiviral, and antibiotic treatment according to the current literature. A few patients with COVID-19-induced acute fulminant myocarditis showed an

TABLE 1. BASELINE CHARACTERISTICS OF ALL COVID-19 ACUTE MYOCARDITIS PATIENTS

Case, author	Age, sex	Risk factors	Presenting symptoms	Admission findings	ECG findings	Lab findings
Case 1, Inciardi et al. ⁴	53, F	-	Fatigue, chest pain and dyspnea	Fever: 36.6 °C HR:100 beats SBP: 90 mm Hg DBP:50 mm Hg Spo ₂ :98	Minimal diffuse ST-segment elevation (more prominent in the inferior and lateral leads), and an ST-segment depression with T-wave inversion in lead V1 and aVR	WBC: 8.0×10 ³ /μL Lymphocyte: 1.4×10 ³ /μL CRP: 25 mg/dl D-dimer: 500 U/F Troponin: 300 ng/L BNP: -
Case 2, Kim et al. ⁵	21, F	-	Coughing, sputum, diarrhea, and shortness of breath	-	Nonspecific intraventricular conduction delay and multiple premature ventricular complexes	WBC: - Lymphocyte: - CRP: - D-dimer: Troponin: 1.26 ng/L BNP: 1929 pg/mL
Case 3, Zeng et al. ⁶	63, M	-	Shortness of breath and chest tightness after activity	Fever: 39.3 °C HR: - SBP: - DBP: - Spo ₂ : 91	Sinus tachycardia and no ST-segment elevation	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: 11.37 g/L BNP: 22.600 pg/ mL
Case 4, Doyen et al. ⁷	69, M	Hypertension	Cough, fever, dyspnea	Fever: 39.1°C HR: - SBP: - DBP: - Spo ₂ : 91	Inverted T waves in anterior leads	WBC: 15.400 g/L Lymphocyte: 141.9×10 ⁹ per L CRP: - D-dimer: - Troponin: 9002 ng/L BNP: 22.600 pg/ mL
Case 5, Trogen et al. ⁸	17, M	Obesity, asthma, spondylolysis	fever and neck pain, diarrhea	Fever 103 °F HR: 150 bpm SBP: 79 mm Hg DBP: 66 mm Hg Spo ₂ : 91	Sinus tachycardia and T-wave inversion particularly in the inferior leads	WBC: 15.4 g/dL Lymphocyte: 0.9×10 ³ /μL CRP: 167 mg/L D-dimer: 1218 ng/mL Troponin: 2.97 ng/ml BNP: 2124 pg/mL
Case 6, Sardari et al. ⁹	31, M	-	Dyspnea on exertion and low-grade fever	Fever: 37.8 °C HR: 70 bpm SBP: 110 mm Hg DBP: 70 mm Hg Spo ₂ : 98	Normal findings	WBC: - Lymphocyte: - CRP: 105 mg/L D-dimer: - Troponin: 2.97 ng/ml BNP: -
Case 7, Coyle et al. ¹⁰	57, M	Hypertension	Shortness of breath, fevers, cough, myalgia, decreased appetite, nausea and diarrhea	Fever: - HR: - SBP: - DBP: - Spo ₂ : -	Sinus tachycardia without ST/T wave changes	WBC: - Lymphocyte: Lymphopenia CRP: Elevated D-dimer: - Troponin: Rapid rise BNP: Rapid rise
Case 8, Beşler and Arslan ¹¹	20, M	-	Febrile sensation and chest pain	Fever: 39 °C HR: 111bpm SBP: 149 mm Hg DBP: 63 mm Hg Spo ₂ : 97	-	WBC: 6.74 × 10 ⁹ per L Lymphocyte: Lymphopenia CRP: 0.0812 g/L D-dimer: - Troponin: 0.572 ng/mL BNP: 127 ng/L
Case 9, Yuan et al. ¹²	33, M	Hypertension	Chest pain, fever, and muscle ache.	Fever: 37.3 °C HR: 121bpm SBP: 115mm Hg DBP: 79 mm Hg Spo ₂ : -	Ventricular tachycardia.	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: - BNP: -
Case 10, Cizgici et al. ¹³	78, M	-	Chest pain and shortness of breath.	Fever: 37.3 °C HR: 150 bpm SBP: 115 mm Hg DBP: 79 mm Hg Spo ₂ : -	Atrial fibrillation with 150 beats/minute and concave ST elevation except for aVR lead	WBC: Leukocytosis Lymphocyte: Lymphopenia CRP: 94.6 mg/L D-dimer: - Troponin: 998.1 ng/L BNP: 127 ng/L
Case 11, Asif and Ali ¹⁴	64, M	Hypertension, hyperlipidemia	Dyspnea	Fever: - HR: 80 bpm SBP: 85 mm Hg DBP: 50 mm Hg Spo ₂ : 70	Non-specific T wave changes	WBC: Leukocytosis Lymphocyte: Lymphopenia CRP: 94.6 mg/L D-dimer: - Troponin: 0.17 ng/ml. BNP: -

TABLE 1.

(CONCLUSION)

Case, author	Age, sex	Risk factors	Presenting symptoms	Admission findings	ECG findings	Lab findings
Case 12, Asif and Ali ¹⁴	71, F	Multiple myeloma	Fever, cough, and dyspnea	Fever: - HR: 125 bpm SBP: 70 mm Hg DBP: 41 mm Hg SpO ₂ : 70	1 mm ST elevation in leads V2–V6 with associated Q waves in leads V4–V6	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: 1.6 ng/ml BNP: -
Case 13, Juusela et al. ¹⁵	45, F Pregnant (39 weeks)	Obesity, gestational diabetic	Contractions and emesis	Fever: 99.6 °F HR: 120 bpm SBP: 183 mm Hg DBP: 114 mm Hg SpO ₂ : 96	Nonspecific T-wave abnormalities	WBC: - Lymphocyte: - CRP: - D-dimer: - Troponin: 0.046 ng/mL BNP: 114 pg/mL
Case 14, Juusela et al. ¹⁵	26, F Pregnant (33 weeks)	Obesity, polycystic ovary syndrome	Shortness of breath, dyspnea,	Fever: 99.6 °F HR: 130 bpm SBP: 110 mm Hg DBP: 70 mm Hg SpO ₂ : 95	Supraventricular tachycardia	WBC: - Lymphocyte: - CRP: 7.68 mg/dL D-dimer: - Troponin: 0.046 ng/mL, BNP: <10 pg/mL,
Case 15, Pavon et al. ¹⁶	64, M	Pulmonary sarcoidosis and epilepsy	Chest pain and dyspnea.	Fever: 39.3°C HR: - SBP: - DBP: - SpO ₂ : -	Unremarkable	WBC: 18.7 gr/L Lymphocyte: - CRP: - D-dimer: 1210 ng/ml Troponin: 1843 ng/L BNP: -
Case 16, Irbien-Ortiz et al. ¹⁷	59, F	Hypertension, cervical degenerative arthropathy, chronic lumbar radiculopathy, erythema nodosum, migraine	Anginal chest pain in the absence of respiratory symptoms.	Fever: 39.3°C HR: - SBP: 75 mm Hg DBP: 53 mm Hg SpO ₂ : 96	Concave ST-segment elevation and PR-segment depression, as well as low voltages	WBC: 14.17x 10 ⁹ /L Lymphocyte: 2.59x10 ⁹ /L CRP: - D-dimer: 23.242 ng/mL Troponin: Elevated BNP: 4421 ng/L

Abbreviations: F; female, M; male, HR; heart rate, SBP; systolic blood pressure, DBP; diastolic blood pressure, WBC; white blood cell, CRP; c-reactive protein, BNP; brain natriuretic peptide.

TABLE 2. IMAGING FINDINGS, IN-HOSPITAL TREATMENT, AND COURSE OF ALL COVID-19 ACUTE MYOCARDITIS PATIENTS

Case, author	Echocardiographic findings	CMR imaging findings	EMB findings	In-hospital treatment	In-hospital course
Case 1, Inciardi et al. ⁴	Diffuse hypokinesia, with an estimated left ventricle ejection fraction of 40%	Diffuse biventricular hypokinesia, especially in the apical segments, and severe LV dysfunction	-	Hydroxychloroquine (200 mg twice daily), lopinavir/ritonavir (2 tablets of 200/50 mg twice daily), and intravenous methylprednisolone (1 mg/kg daily for), 50 mg of kanrenone, 25 to 50 mg of furosemide, and 2.5 mg of bisoprolol	Cardiogenic shock, clinical follow-up
Case 2, Kim et al. ⁵	Severe left ventricular systolic dysfunction	Diffuse high signal intensity in the left ventricle myocardium on T2 short inversion recovery image	-	-	Discharged
Case 3, Zeng et al. ⁶	An enlarged left ventricle (61 mm), diffuse myocardial dyskinesia along with a low left ventricular ejection fraction of 32%	-	-	High-flow oxygen, lopinavir-ritonavir, interferon α -1b, methylprednisolone, immunoglobulin, piperacillin-tazobactam, and continuous renal replacement therapy	Cardiogenic shock, exitus
Case 4, Doyen et al. ⁷	Mild left ventricle hypertrophy, the left ventricular ejection fraction and wall motion were within normal limits	Subepicardial late gadolinium enhancement of the apex and inferolateral wall—suggestive of myocarditis	-	Hydrocortisone, aspirin, fondaparinux	Acute respiratory distress syndrome, discharged

TABLE 2.

(CONCLUSION)

Case, author	Echocardiographic findings	CMR imaging findings	EMB findings	In-hospital treatment	In-hospital course
Case 5, Trogen et al. ⁸	Left ventricular ejection fraction qualitatively noted to be mildly depressed without obvious intracardiac clots or pericardial effusion	Normal size left ventricle (LV) with mildly decreased systolic function (40%) and normal right ventricular (RV) size with mildly diminished systolic function (RVEF of 39%). There was an area of mid-wall late gadolinium enhancement at the inferior LV–RV junction corresponding to an area of increased T2 signal, as well as an area of hypokinesia	-	Hydroxychloroquine, piperacillin/tazobactam, enoxaparin	Discharged
Case 6, Sardari et al. ⁹	Mild left ventricular dysfunction	Normal left ventricular size with a mildly reduced ejection fraction of 50%. T2-weighted sequence with its post-analysis showed edema/inflammation in the mid inferoseptal and inferior wall. Late gadolinium enhancement showed subepicardial fibrosis in the mid inferior wall	-	Bisoprolol and lisinopril	Discharged
Case 7, Coyle et al. ¹⁰	Moderate diffuse hypokinesia with relative apical sparing and a left ventricular ejection fraction of 35–40%	Diffuse bi-ventricular and bi-atrial edema with a small area of late gadolinium enhancement	-	Hydroxychloroquine, azithromycin, ceftriaxone, methylprednisolone, colchicine	Discharged
Case 8, Beşler and Arslan ¹¹	-	Short tau inversion recovery (STIR) sequence revealed a subepicardial high signal intensity in the mid posterolateral wall of the left ventricle which suggests myocardial wall edema	-	Hydroxychloroquine, azithromycin, ceftriaxone, tigecycline, favipiravir, colchicine	Discharged
Case 9, Yuan et al. ¹²	-	The signal of T2 weighted image in the apical region of the left ventricle was increased, which indicated the possibility of myocardial cell edema. Left ventricular systolic function was slightly decreased	-	-	-
Case 10, Cizgici et al. ¹³	Not being able to perform echocardiography	Not being able to perform magnetic resonance imaging	-	Furosemide, beta-blocker, and angiotensin-converting enzyme inhibitor was added to his Covid-19 specific therapy.	-
Case 11, Asif and Ali ¹⁴	Normal left ventricle ejection fraction of 70–75% with no regional wall motion abnormalities	-	-	Aspirin 81 mg, clopidogrel 75 mg, heparin, azithromycin, hydroxychloroquine, meropenem, tocilizumab, norepinephrine, phenylephrine, vasopressin, atracurium, propofol, fentanyl	Intubated
Case 12, Asif and Ali ¹⁴	Normal left ventricle ejection fraction of 65–70% with no regional wall motion abnormalities	-	-	Aspirin 81 mg, clopidogrel 75 mg, heparin, azithromycin, cefepime, vancomycin, tocilizumab, norepinephrine, phenylephrine, midazolam, and fentanyl	Intubated
Case 13, Juusela et al. ¹⁵	Moderately reduced left ventricular ejection fraction of 40% with global hypokinesia	-	-	IV methylprednisolone, hydroxychloroquine, tocilizumab	Intubated, (a primary cesarean)
Case 14, Juusela et al. ¹⁵	Moderately reduced left ventricular ejection fraction of 40–45% with global hypokinesia	-	-	Metoprolol	Cesarean
Case 15, Pavon et al. ¹⁶	Moderately reduced left ventricular ejection fraction of 47%.	T2-mapping sequences showed myocardial edema and sub-epicardial late gadolinium enhancement in the anterior interventricular septum, in the inferior and inferolateral walls	-	Piperacillin/tazobactam	Discharged
Case 16, Irabien-Ortiz et al. ¹⁷	Preserved left ventricular ejection fraction without segmental abnormalities, and moderate pericardial effusion with no clear signs of hemodynamic deterioration	-	-	Immunoglobulins (80 mg/d) methylprednisolone (500 mg/d), antiviral treatment consisting of interferon-B (0.25 mg/48 h) and ritonavir/lopinavir (400 mg/100 mg/12 h)	Cardiogenic shock

Abbreviations: CMR; cardiac magnetic resonance, EMB; endomyocardial biopsy

amelioration using IV glucocorticoid and immunoglobulin therapy. This finding appears as a clue highlighting the cytokine storm due to inadequate host immune response, which might be the main pathophysiology of COVID-19-induced acute fulminant myocarditis.

Limitations in the diagnosis of COVID-19-related acute myocarditis

Acute viral infections are one of the most common etiologic factors of acute myocarditis. Following the COVID-19 pandemic, several cases of acute myocarditis were reported worldwide, which were diagnosed with different modalities other than EMB. In our review, all of the published cases were mentioned in terms of clinical characteristics, imaging findings, and in-hospital course.

Troponin has been indicated as an independent predictor of mortality in hospitalized patients with COVID-19^{18,19}. Since troponin has been defined as a noteworthy prognostic factor, the etiology of the higher levels of troponin gains clinical importance to regulate adequate medications. In COVID-19 patients who present with elevated troponin levels, it might be difficult to reach a definitive diagnosis of acute coronary syndrome (ACS) or acute myocarditis because there are similarities among them in regard to the elevation of troponin levels and ECG changes. It is reasonable not to perform CMR and MRI in most patients due to the contiguousness of COVID-19. On the other hand, underusing the aforementioned modalities in the differential diagnosis reveals a gap in the definite etiology of the myocardial injury. Thus, we may be underestimating the prevalence and importance of

COVID-19-related acute myocarditis. Moreover, in severe patients, both ACS and acute myocarditis may present together because of the procoagulant and inflammatory nature of the COVID-19 infection.

CONCLUSION

Despite the COVID-19 pandemic worldwide, a limited number of cases has been shared in the current literature. There are a lot of difficulties for the differential diagnosis of acute myocarditis in the context of COVID-19, and information about COVID-19-related acute myocarditis remains unclear. Also, there is no consensus about the diagnostic and treatment algorithms in patients with COVID-19-induced acute myocarditis. Hence, further studies and case reports on COVID-19-associated acute myocarditis are needed to clarify an appropriate approach to these patients.

Conflict of interest

The authors have no conflicts of interest relevant for this article

Funding

The authors declare that this article has received no financial support.

Author's Contribution

Concept: T.Ç., M.İ.H.; Design: T.Ç., M.İ.H.; Supervision: M.U., A.L.O.; Funding: V.Ç.; Materials: T.Ç., M.İ.H., V.Ç.; Data collection: T.Ç., V.Ç.; Analysis: M.İ.H.; Literature review: T.Ç., M.İ.H., V.Ç.; Writing: T.Ç.; Critical review: M.U., A.L.O.

RESUMO

INTRODUÇÃO: Na literatura atual, houve um aumento dos casos apresentados com doença coronavírus de 2019 (COVID-19) induzida por miocardite aguda. Nesta revisão baseada em casos, buscamos descrever as características clínicas, achados de imagem e curso hospitalar de miocardite aguda. Além disso, as limitações em relação ao diagnóstico de miocardite foram discutidas, uma vez que apenas casos de miocardite fulminante foram mencionados na literatura atual.

MÉTODOS: Fizemos uma revisão da literatura de todos os pacientes diagnosticados com miocardite aguda induzida por COVID-19 com a utilização das bases de dados PubMed, Embase e Cochrane.

RESULTADO: Dezesesseis casos relatados estão relacionados com a miocardite aguda induzida pela COVID-19. Observamos que os achados de ECG na maioria dos pacientes com COVID-19 não eram específicos, incluindo elevação difusa do segmento ST, atraso não específico da condução intraventricular, taquicardia sinusal e ondas T invertidas em pistas anteriores. Os resultados ecocardiográficos de doentes com miocardite aguda COVID-19 variaram entre a fração de ejeção ventricular esquerda preservada (LVEF) sem anomalias segmentais e a LVEF reduzida com hipocinesia global. Curiosamente, alguns pacientes com COVID-19 induzidos à miocardite aguda fulminante eram sensíveis aos esteróides e tinham uma melhoria com glucocorticoides e terapia com imunoglobulina.

CONCLUSÃO: Apesar da pandemia de COVID-19 em todo o mundo, um número limitado de casos tem sido compartilhado na literatura atual. Há muitas dificuldades para o diagnóstico diferencial de miocardite aguda no contexto da COVID-19.

PALAVRAS-CHAVE: Infecções por coronavírus. Betacoronavírus. Miocardite. Cardiomiopatias.

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