WALKOWSKI, Radoslaw, WASINIEWSKA, Weronika, KOSEK, Szymon, KLONOWSKA, Justyna, BARAŃSKI, Marcin, KANDEFER, Tomasz and SROKA, Maria Izabela. Myocarditis in COVID-19 – Epidemiology, Pathophysiology, Diagnosis, Treatment and Clinical Outcomes. Quality in Sport. 2025;40:59784. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.40.59784
https://apcz.umk.pl/QS/article/view/59784

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 26.03.2025. Revised: 02.04.2025. Accepted: 04.04.2025 Published: 14.04.2025.

Myocarditis in COVID-19 – Epidemiology, Pathophysiology, Diagnosis, Treatment and Clinical Outcomes

Authors:
Radosław Jan Walkowski
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0006-9933-6437
radwalk.med@gmail.com

Weronika Wasiniewska
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0001-9263-5710
wasiwer@gmail.com

Szymon Kosek
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0001-7350-2306
kosek.med@gmail.com

Justyna Klonowska
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0002-8170-9187
justynajklonowska@gmail.com

Marcin Barański
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0003-7792-5837
baranskimarcin23@gmail.com

Tomasz Kandefer
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0004-2714-8344
tomekkandefer92@gmail.com

Maria Izabela Sroka
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0002-8743-8963
marsrokaa@gmail.com

ABSTRACT
Myocarditis is a rare inflammatory disease affecting the myocardium and is caused by a variety of factors – medications, toxins, viruses and other processes that can include the heart muscle. It presents with multiple symptoms and can lead to heart failure and in some cases even sudden death. The diagnosis of myocarditis especially in COVID-19 poses many challenges. In regards to SARS-CoV-2 the post-infection myocarditis is more common, however there were some cases of post-vaccination myocarditis present. Treatment strategies are primarily supportive and focused on battling the symptoms. In this article we focused on research about pathophysiology, clinical presentation, diagnosis, treatment and long-term effects of COVID-19 on the heart.

Keywords: COVID-19, myocarditis, diagnosis, treatment, epidemiologic factors

Introduction

Myocarditis is a disease affecting the myocardium that presents a broad clinical spectrum, typically ranging from fatigue and mild chest discomfort to dangerous arrythmias and cardiac shock. The diagnosis may prove difficult, as the symptoms presented by the patient might be unspecific and usually resemble the coronary artery disease.[1] Epidemiological studies suggest that SARS-CoV-2 has increased the prevalence of myocarditis approximately 15-fold, but the condition still remains rare. Pre-COVID cases were reported at 1-9/100 000 individuals and increasing to150-4000/100 000 with COVID-19. Before the COVID era some vaccines reported rare side effects of myocarditis, however the new platform of mRNA vaccines introduced with the SARS-CoV-2 vaccine led to a higher number of reported myocarditis. [2]
SARS-CoV-2 infection affects not only the frequency of myocarditis in infected individuals but also may affect the incidence of some cardiovascular diseases, such as myocardial infarction, heart failure, Takotsubo cardiomyopathy and arrythmias[3]. In this article we will focus on myocarditis.

Epidemiology

Myocarditis typically follows viral infections caused by enteroviruses, specifically Coxsackie group B, adenoviruses, parvovirus B19, hepatitis C, cytomegalovirus, and human immunodeficiency virus. Research suggests that the prevalence of myocarditis before COVID-19 was at a level of 1-10/100 000 patients and after COVID-19 was significantly bigger, at a level of 150-4000/100 000 [2,4]. Between March 2020 and January 2021, patients that contracted COVID-19 had a 15,7 times higher risk of developing myocarditis after adjusting for patient and hospital characteristics. Taking age into consideration the risk ratios ranged from approximately 7.0 for patients aged 16–39 years to >30.0 for patients aged <16 years or ≥75 years[5]. Two large studies of 3 000 000 and 200 000 patients, respectively, detected no sex difference in whether patients tested PCR positive for SARS-CoV-2, although men had higher rates of hospitalization, intensive care unit admission, and mortality. However, this was not the case for all research. A study of ≈100 000 patients found that men were more often PCR positive for SARS-CoV-2 and had greater mortality than women[2]. The reason behind it isn't yet known. Overall, myocarditis still remains uncommon, however COVID-19 is associated with a significantly increased risk of developing it. The risk difference for myocarditis between persons with and without COVID-19 was higher among males than among females, consistent with some earlier studies [5].

Pathophysiology

Viral-induced myocarditis has historically been defined by three phases: acute viral exposure with an innate immune response (<1 week); activation of an acquired immune response with cytokine and chemokine release (1-4) weeks; disease progression with clearance of the virus and development of fibrosis, remodeling and cardiomyopathy (>4 weeks). Cases of myocarditis following SARS-CoV-2 infections largely follow this pattern, with a delay of onset after viral infection of days to weeks. Data indicate that a maladaptive host immune response occurs, with excessive activation of innate immune pathways, a surge of proinflammatory cytokines, deregulated thrombo-inflammation, thrombotic microangiopathy, endothelial dysfunction, and even molecular mimicry; other hypotheses include demand ischemia, stress cardiomyopathy, and hypoxia-induced myocardial injury[6,7].

The damage caused by SARS-CoV-2 infection can be divided into two stages: viral infection and uncontrolled inflammation. The viral infection stage is characterized by viral replication, resulting in direct virus-mediated tissue damage. After SARS-CoV-2 combines with Angiotensin-converting enzyme 2 (ACE2) on the surface of the target cell membrane, the type 2 transmembrane serine protease (TMPRSS2) existing in the target cell promotes the uptake of the virus by splitting ACE2 and activating the SARS-CoV-2 S protein. After the virus enters the cell, it releases its RNA, replicates, releases it, and further infects adjacent cells, causing tissue damage.

The second stage is a local inflammation and cytokine storm caused by tissue damage, an uncontrolled systemic inflammatory reaction related to the release of a large number of pro-inflammatory cytokines [including interleukin-6 (IL-6), IL-1, IL-2, IL-10, tumor necrosis factor alpha (TNF-α), and interferon-γ] and coagulation dysfunction. IL-6 is a primary mediator of cytokine storm, a life-threatening condition observed in some patients who developed COVID-19, which is characterized by extreme increases in pro-inflammatory cytokines and an uncontrolled immune response. This systemic inflammation may further increase the risk of thrombus formation within coronary vessels due to platelet activation and high levels of coagulation factors (including factors V and VIII). Excessive inflammatory reactions may be related to changes in the myeloid reaction and the production of pathogenic autoantibodies. Research suggests that this uncontrolled inflammatory reaction is the core of developing myocarditis in patients infected with SARS-CoV-2 [8,9].In addition, interleukin IL-1β and IL-17 cause cardiac remodeling and fibrosis, which eventually lead to dilated cardiomyopathy and heart failure. Myocardial fibrosis leads to disruption of the conduction system, resulting in an increased risk of developing arrhythmias. [9]

Clinical manifestations and diagnosis

The clinical presentation of myocarditis associated with acute COVID-19 infection is typically a viral prodrome beginning with symptoms of fever, cough and shortness of breath, followed by cardiac symptoms like chest tightness on exertion or characteristic chest pain or lethargy[10]. The European Study of the Epidemiology and Treatment of Inflammatory Heart Disease tested 3055 individuals with suspected myocarditis and found out that 72% of them experienced dyspnea, 32% had chest discomfort, and 18% had arrhythmias[11]. Clinical course can vary significantly, from mild symptoms not requiring hospitalization to severe cases. Many patients deteriorate, showing signs or symptoms of heart failure, and/or cardiac arrhythmias. Early circulatory failure symptoms such as a narrow arterial pulse pressure, sinus tachycardia, cool or mottled extremities, or elevated lactate, patient being febrile more often suggest sepsis, but may also be signs of a severe myocarditis in rarer cases[12]. Some cases lead to fulminant myocarditis – a severe diffuse cardiac inflammation that typically develops within three weeks of contracting the virus and manifests as clinically significant ventricular arrhythmia, cardiogenic shock, and acute onset of heart failure[10,13,14]. Patients may also present with signs of right-sided heart failure, including raised jugular venous pressure, peripheral edema, and right upper quadrant pain[15].

The diagnosis of myocarditis is made through a combination of laboratory, electrocardiogram (ECG), and imaging studies. Laboratory diagnosis typically involves elevated cardiac biomarkers like troponin T and B-type natriuretic peptide, however troponin T is not universally present in myocarditis, and its sensitivity for diagnosing myocarditis is limited, around 34%[16,17].

In contrast, a study conducted on children found that cardiac enzymes were elevated in 89% of the cases, especially troponin-I levels were positive in most of the patients, confirming myocardial damage[18] and another study conducted on adults reported similar findings[19]. Patients with a reduced ejection fraction (EF) had higher maximum troponins during their admission, but similar rates of requiring intubation, intensive care-unit length of stay and inpatient mortality. [20]

Other inflammatory markers, such as lactate, C-reactive protein , erythrocyte sedimentation rate , and procalcitonin, are elevated in myocarditis, but these markers were not followed as they are often raised in the presence of infection[16].

ECG changes were found to be vague but included sinus tachycardia and nonspecific ST-segment (such as ST elevation), T-wave abnormalities and PR depression, and may be used in conjunction with other studies; however, these findings are not sensitive in detecting the disease and their absence is not exclusionary[15,16]

Because of its availability and portability, transthoracic echocardiography (TTE) is used as the initial imaging modality in patients with suspected COVID-19 myocarditis. Left or biventricular dysfunction, abnormal strain, ventricular hypertrophy, and peri cardial effusion can all be seen on TTE in COVID-19 myocarditis, but the findings are nonspecific[21]. Cardiac magnetic resonance imaging (cMRI) is the most precise tool for patients with myocarditis, due to its ability to accurately detect inflammation, hyperemia, and the extent of reversible or irreversible cardiac injury, however cMRI is not generally indicated unless there is diagnostic uncertainty[16], probably due to its reduced availability. The cMRI significantly enhances the detection of myocarditis, particularly through the identification of myocardial edema on T2-weighted imaging and nonischemic scar on late gadolinium enhancement (LGE) imaging[21,22]. Injury in consequence of a viral infection is usually located in inferolateral and, less frequently, anteroseptal segments[23]. It is important to note that the cMRI although being probably the best non-invasive method of imaging an extent of myocarditis in COVID-19 might be rarily available due to it requiring highly trained personnel and highly specialist equipment; it might also be impossible to obtain an image from patients in critical condition due to the specific conditions needed to be met for this imaging technique.

Studies have shown that myocarditis can occur even in patients with mild COVID-19 infections, with some cases identified through cMRI imaging in asymptomatic or mildly symptomatic individuals. In one study 43% of the consecutive patients shortly after COVID-19 pneumonia requiring hospitalization presented with radiological signs of myocarditis. However, at the time of the CMR examination, they had no evident acute clinical symptoms suggesting myocarditis[12,24]. This could indicate that myocarditis may be present even without or with very mild clinical symptoms after COVID-19 pneumonia.

Although not commonly performed due to its invasive nature, endomyocardial biopsy (EMB) still remains the gold standard for diagnosing myocarditis. It allows for direct histopathological examination and identification of viral genomes[13,25]. In histopathology CD68+ macrophages with fewer T cells are a characteristic finding of immunohistochemistry performed on endomyocardial biopsy of patients with COVID-19 myocarditis/pericarditis, and macrophages

are the primary infiltrate with fewer T cells in autoimmune models of myocarditis. Thus, the characteristics of myocardial inflammation are similar between COVID-19 myocarditis and Coxsackie B3 virus and autoimmune myocarditis animal models.[2]

Management and treatment strategies

Treatment needs to be tailored to the variable phenotype of the disease and reflect the need to manage arrhythmia and/or decompensated ventricular function[26,27]. Selected treatments for COVID-19-associated myocarditis were variable, but the most common approach was supportive treatment alone (43.1%). In the acute state of the disease, patients with severe disease are generally hospitalized. Supportive therapy included intravenous/oral hydration, beta-blockers, or diuretics. Additional interventions were vasopressor or inotropic support (31.3%), steroids (19.6%), and antivirals (7.8%)[28]. In viral myocarditis, beta-blockers may be helpful in patients with arrhythmias but can precipitate cardiogenic shock, so the clinical picture must be carefully examined[26]. Use of intravenous corticosteroids may be considered in those with suspected or confirmed COVID-19 myocarditis with hemodynamic compromise or multisystem inflammatory syndrome in adults, a hyperinflammatory state with acute heart failure and/or cardiogenic shock in the absence of sepsis, as this approach was associated with a favorable prognosis in a small series. Empiric use of immunosuppressive therapy (eg, corticosteroids) may also be considered in those with biopsy evidence of severe myocardial inflammatory infiltrates or fulminant myocarditis, balanced against infection risk. Patients with myocarditis and COVID-19 pneumonia (with an ongoing need for supplemental oxygen) should be treated with corticosteroids. For patients with suspected pericardial involvement, treatment with NSAIDs, colchicine, and/or prednisone is reasonable[6].

In some reported cases of SARS-CoV-2-related fulminant myocarditis, mechanical circulation support therapy and immunoglobulin combined with steroid shock therapy also showed good therapeutic effects. In addition, some immune-specific drugs have been proven to be beneficial, especially those against the IL-6 pathway, including tocilizumab and sarilumab. Janus kinase-signal transducer and activator of transcription protein (JAK-STAT) signaling pathway inhibitors such as axitinib and tofacitinib show potential[8].

Clinical outcomes

Myocarditis in COVID-19 patients was associated with worse outcomes than non-myocarditis COVID-19 patients both in terms of morbidity and mortality, including increased acute kidney injury needing hemodialysis, cardiogenic shock with vasopressor support, mechanical ventilation and sudden cardiac death[29]. The total duration of the treatment was revealed in 29 cases (76.31%) and is highly variable, ranging from 3 days to 1 year[30]. Vaccination may play a pivotal role in reducing the incidence of COVID-19-related myocarditis[29].

Post-discharge management

Assessment of a cardiac function is essential in monitoring patients with COVID-19 myocarditis. It may be achieved in several ways including imaging methods, biomarkers and clinical evaluation.

A valuable insight in the function of the heart may be visible in echocardiography, especially in relation to the left and right ventricle. Numerous studies have shown that the most common cardiac pathology among hospitalized patients with acute COVID-19 infection is RV dilatation and/or dysfunction. Most RV parameters improved (although did not normalize) 3 months after hospitalization.

In marked contrast to the impressive recovery of RV function, LV dysfunction observed during acute COVID-19 rarely improved 3 months post-COVID-19. In fact, LVEF even decreased compared to exams performed during the acute phase. Nevertheless, it is important to note that the reduction in LV functional parameters rarely reached the abnormal range[31].

The cMRI may show a lesser extent of LGE, better left ventricular systolic function, lower left ventricular end-diastolic volume but a higher rate of pericarditis and septal predilection of LGE in comparison to control group, some patients show signs of myocardial edema. In patients without clinical evidence of myocarditis, areas with abnormal LGE may just reflect scars but not acute inflammation. Septal fibrosis, as reported, is not specific for inflammation and may be present in diseases such as dilated and hypertrophic cardiomyopathy and sarcoidosis and can even be sometimes seen in healthy individuals[32].

There is also a method of echocardiography that showed promising results in monitoring - two-dimensional speckle tracking echocardiography (2D-STE). A study showed that global myocardial strain, including longitudinal strain and circumferential strain, are sensitive, non-invasive, and reproducible indices that are clinically relevant to the subtle changes in the left ventricular myocardium that may occur as a consequence of the cardiac impact of COVID-19 vaccination. It was found that the potential capability of tissue speckle tracking analysis can evaluate and indicate early myocardial dysfunction and adverse cardiac events after immunization with COVID-19 vaccines[33].

Research gaps and future directions.

Despite the growing number of research on COVID-19 related myocarditis, many gaps need more research. The long-term outcomes of COVID-19 related myocarditis are not yet fully understood. The reason behind higher prevalence and mortality in COVID-19 related myocarditis in male population is not yet known. There is a need for studies conducted on the development of chronic heart failure or the risk of arrhythmias over years post-infection. Diagnosis for myocarditis still proves a challenge, with the only definitive technique being an invasive biopsy; research could focus on finding a biomarker specific to COVID-19 myocarditis. Because of being a rare manifestation of SARS-CoV-2 treatment options of COVID-19 related myocarditis should be explored in further detail. There is a need to evaluate the efficacy of new and possibly more effective therapies, for example JAK-STAT signaling pathway inhibitors.

Conclusions

COVID-19 related myocarditis might be a significant and dangerous complication of COVID-19 infection associated with relatively higher risk of mortality. The pathophysiology is a complex mechanism of viral infection, inflammation, cytokine storm and immune dysregulation. Diagnosis requires a multitude of different approaches, including clinical, laboratory and imaging findings with cardiac MRI proving to be the best of the non-invasive detection tools. Treatment strategies are primarily supportive and focused on battling the symptoms, although there is a promise of new targeted therapies. Further research is needed to develop more effective diagnostic and therapeutic interventions.

Disclosure

Author's contribution

Conceptualization: *Radosław Jan Walkowski*; Methodology: *Marcin Barański*; Check: *Radosław Jan Walkowski*; Formal analysis: *Justyna Klonowska*; Investigation: *Maria Izabela Sroka*; Resources: *Szymon Kosek*; Data curation: *Tomasz Kandefer*; Writing - rough preparation: *Marcin Barański*; Writing - review and editing: *Weronika Wasiniewska*; Visualization: *Tomasz Kandefer*; Supervision: *Radosław Jan Walkowski*; Project administration: *Justyna Klonowska*; Receiving funding - no specific funding.

All authors have read and agreed with the published version of the manuscript.

Financing statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflict of interest

The authors deny any conflict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process

In preparing this work, the author(s) used *Perplexity AI* for the purpose of improving language and readability. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References:

1. Sagar S, Liu PP, Cooper LT, Jr. Myocarditis. *Lancet*. Feb 25 2012;379(9817):738-47. doi:10.1016/S0140-6736(11)60648-X
2. Fairweather D, Beetler DJ, Di Florio DN, Musigk N, Heidecker B, Cooper LT, Jr. COVID-19, Myocarditis and Pericarditis. *Circ Res*. May 12 2023;132(10):1302-1319. doi:10.1161/CIRCRESAHA.123.321878
3. Krishna BA, Metaxaki M, Sithole N, Landin P, Martin P, Salinas-Botran A. Cardiovascular disease and covid-19: A systematic review. *Int J Cardiol Heart Vasc*. Oct 2024;54:101482. doi:10.1016/j.ijcha.2024.101482
4. Thaker R, Faraci J, Derti S, Schiavone JF. Myocarditis in SARS-CoV-2: A Meta-Analysis. *Cureus*. Oct 2023;15(10):e48059. doi:10.7759/cureus.48059
5. Boehmer TK, Kompaniyets L, Lavery AM, et al. Association Between COVID-19 and Myocarditis Using Hospital-Based Administrative Data - United States, March 2020-January 2021. *MMWR Morb Mortal Wkly Rep*. Sep 3 2021;70(35):1228-1232. doi:10.15585/mmwr.mm7035e5
6. Writing C, Gluckman TJ, Bhave NM, et al. 2022 ACC Expert Consensus Decision Pathway on Cardiovascular Sequelae of COVID-19 in Adults: Myocarditis and Other Myocardial Involvement, Post-Acute Sequelae of SARS-CoV-2 Infection, and Return to Play: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. May 3 2022;79(17):1717-1756. doi:10.1016/j.jacc.2022.02.003
7. Spychalska M, Spychalski J. COVID-19 Sequelae: Long-Term Impairments in Physical Performance. *Quality in Sport*. 03/08 2025;39:58900. doi:10.12775/QS.2025.39.58900
8. Shu H, Zhao C, Wang DW. Understanding COVID-19-related myocarditis: pathophysiology, diagnosis, and treatment strategies. *Cardiol Plus*. Apr-Jun 2023;8(2):72-81. doi:10.1097/CP9.0000000000000046
9. Cersosimo A, Di Pasquale M, Arabia G, Metra M, Vizzardi E. COVID myocarditis: a review of the literature. *Monaldi Arch Chest Dis*. Nov 3 2023;doi:10.4081/monaldi.2023.2784
10. Yendrapalli U, Hassoun A, Zalamea RM, Hritani A. Myocarditis presentation following COVID-19 virus infection versus COVID-19 vaccination. *Am J Cardiovasc Dis*. 2022;12(5):272-277.
11. Hufnagel G, Pankuweit S, Richter A, Schonian U, Maisch B. The European Study of Epidemiology and Treatment of Cardiac Inflammatory Diseases (ESETCID). First epidemiological results. *Herz*. May 2000;25(3):279-85. doi:10.1007/s000590050021
12. Kociol RD, Cooper LT, Fang JC, et al. Recognition and Initial Management of Fulminant Myocarditis: A Scientific Statement From the American Heart Association. *Circulation*. Feb 11 2020;141(6):e69-e92. doi:10.1161/CIR.0000000000000745

13. Lovell JP, Cihakova D, Gilotra NA. COVID-19 and Myocarditis: Review of Clinical Presentations, Pathogenesis and Management. *Heart Int*. 2022;16(1):20-27. doi:10.17925/HI.2022.16.1.20
14. Okor I, Bob-Manuel T, Price J, et al. COVID-19 Myocarditis: An Emerging Clinical Conundrum. *Curr Probl Cardiol*. Sep 2022;47(9):101268. doi:10.1016/j.cpcardiol.2022.101268
15. Siripanthong B, Nazarian S, Muser D, et al. Recognizing COVID-19-related myocarditis: The possible pathophysiology and proposed guideline for diagnosis and management. *Heart Rhythm*. Sep 2020;17(9):1463-1471. doi:10.1016/j.hrthm.2020.05.001
16. Kamath S, Gomah MT, Stepman G, DiMartino P, Adetula I. COVID-19-Associated Acute Myocarditis: Risk Factors, Clinical Outcomes, and Implications for Early Detection and Management. *Cureus*. Sep 2023;15(9):e44617. doi:10.7759/cureus.44617
17. Ali M, Shiwani HA, Elfaki MY, et al. COVID-19 and myocarditis: a review of literature. *Egypt Heart J*. Apr 5 2022;74(1):23. doi:10.1186/s43044-022-00260-2
18. Ahmed A, Iqbal A, Abdullah A, Irshad M, Khan MQ. Clinical Presentation and Outcomes of Myocarditis Among the COVID-19 Pediatric Population: A Review of 100 Cases. *Cureus*. Sep 2024;16(9):e69885. doi:10.7759/cureus.69885
19. Ammirati E, Lupi L, Palazzini M, et al. Prevalence, Characteristics, and Outcomes of COVID-19-Associated Acute Myocarditis. *Circulation*. Apr 12 2022;145(15):1123-1139. doi:10.1161/CIRCULATIONAHA.121.056817
20. Medranda GA, Case BC, Fazlalizadeh H, et al. The Impact of Ejection Fraction on COVID-19 Patients With Myocardial Injury. *Cardiovascular Revascularization Medicine*. 2021/07/01/ 2021;28:S23. doi:https://doi.org/10.1016/j.carrev.2021.06.052
21. Crosier R, Kafil TS, Paterson DI. Imaging for Cardiovascular Complications of COVID-19: Cardiac Manifestations in Context. *Can J Cardiol*. Jun 2023;39(6):779-792. doi:10.1016/j.cjca.2023.01.022
22. Samimisedeh P, Jafari Afshar E, Tayebi A, Rastad H. Post-acute midterm follow-up cardiac MRI findings and clinical outcomes in patients with COVID-19 vaccine-associated myocarditis: a comprehensive systematic review and meta-analysis. *Infect Dis (Lond)*. Mar 2024;56(3):193-205. doi:10.1080/23744235.2023.2286289
23. Friedrich MG, Sechtem U, Schulz-Menger J, et al. Cardiovascular Magnetic Resonance in Myocarditis: A JACC White Paper. *Journal of the American College of Cardiology*. 2009/04/28/ 2009;53(17):1475-1487. doi:https://doi.org/10.1016/j.jacc.2009.02.007
24. Martusewicz-Boros MM, Boros PW, Piotrowska-Kownacka D, Paciorek M. Myocarditis after COVID-19 pneumonia: incidence and risk factors. *Pol Arch Intern Med*. Jun 23 2023;133(6)doi:10.20452/pamw.16510
25. Wiligórska N, Wiligórska D, Pawlak A. Zapalenie mięśnia sercowego. *Kardiologia Inwazyjna*. 2017-08-02 2017;12(3):47-58.
26. Sayegh MN, Goins AE, Hall MAK, Shin YM. Presentations, Diagnosis, and Treatment of Post-COVID Viral Myocarditis in the Inpatient Setting: A Narrative Review. *Cureus*. May 2023;15(5):e39338. doi:10.7759/cureus.39338

27. Zys D, Wojszczyk M, Ryniecka J, et al. Reviewing the current diagnostic and treatment approaches for myocarditis – analysis of literature. *Quality in Sport*. 01/07 2025;37:57145. doi:10.12775/QS.2025.37.57145
28. Haussner W, DeRosa AP, Haussner D, Tran J, Torres-Lavoro J, Kamler J, Shah K. COVID-19 associated myocarditis: A systematic review. *Am J Emerg Med*. Jan 2022;51:150-155. doi:10.1016/j.ajem.2021.10.001
29. Davis MG, Bobba A, Chourasia P, et al. COVID-19 Associated Myocarditis Clinical Outcomes among Hospitalized Patients in the United States: A Propensity Matched Analysis of National Inpatient Sample. *Viruses*. Dec 14 2022;14(12)doi:10.3390/v14122791
30. Sai Santhosha Mrudula A, Agarwal P, Vempati R, et al. Clinical outcome of established diagnostic and treatment modalities of COVID-19-associated myocarditis: a systematic review. *Ann Med Surg (Lond)*. Jul 2023;85(7):3583-3594. doi:10.1097/MS9.0000000000000964
31. Ghantous E, Topilsky Y. Recovery of cardiac function following COVID-19. *Eur J Heart Fail*. Nov 2021;23(11):1913-1915. doi:10.1002/ejhf.2364
32. Friedrich M, Garg R. COVID-19-induced myocarditis: A multicenter cardiovascular magnetic resonance study. *Kardiol Pol*. 2023;81(5):444-445. doi:10.33963/KP.a2023.0101
33. Chaichuum S, Tseng CL, Chang SC, et al. Assessment of cardiac adverse events following COVID-19 vaccination by speckle tracking echocardiography. *Sci Rep*. May 13 2024;14(1):10849. doi:10.1038/s41598-024-61641-y