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Acute heart failure with improved ejection fraction in a middle-aged patient with myocarditis and COVID-19 infection

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ABSTRACT

This clinical image describes the management of a 48-year-old male with a history of asthma, anxiety-depressive disorder, and prior COVID-19 infection, who was diagnosed with NYHA Class IV heart failure with reduced ejection fraction due to myocarditis.

The patient received an intensive heart failure treatment regimen, including ramipril, eplerenone, bisoprolol, dapagliflozin, ivabradine, and loop diuretics, resulting in a notable clinical improvement and a subsequent reduction in NT-proBNP levels. Upon follow-up, the initiation of sacubitril/valsartan over four months further enhanced functional capacity, leading to an increase in left ventricular ejection fraction to 41% and a decrease in NT-proBNP to 315 pg/ml. Due to these substantial improvements, the need for implantable cardioverter-defibrillator implantation was deemed unnecessary. This case underscores the necessity for early and intensive management of acute heart failure in patients with reduced ejection fraction to facilitate significant recovery. It highlights the rising incidence of myocarditis-related heart failure and emphasises the need for further investigations to optimise therapeutic strategies for patients with restored left ventricular function.

Literature review

The overall global incidence of myocarditis was estimated at approximately 1.5 million cases in 2021, with about 0.5% to 4% of these cases progressing to heart failure (HF). According to the latest ESC guidelines for managing HF, and in the absence of sufficient evidence, a standard HF with reduced ejection fraction (HFrEF) treatment approach is recommended for patients with left ventricular dysfunction. This includes the use of angiotensin-converting enzyme inhibitors (ACE-I) or an angiotensin receptor-neprilysin inhibitor (ARNI), beta-blockers, mineralocorticoid receptor antagonists (MRA), and sodium-glucose cotransporter 2 (SGLT2) inhibitors. Immunosuppression is considered only in select cases of chronic inflammation with inactive viral infection. Once the indicators of HF have resolved, therapy should be continued for at least six months [1,2].

Since the emergence of COVID-19, numerous studies have investigated its cardiovascular complications, including myocarditis and subsequent heart failure (HF). However, the literature addressing post-COVID-19 myocarditis leading to HF remains limited and heterogeneous. Current research focuses predominantly on epidemiological trends and recovery statistics rather than optimal treatment strategies, and guidelines have not been established.

Sawalha et al. conducted one of the first systematic reviews of published case reports on COVID-19-associated myocarditis, highlighting the wide variability in clinical presentations and outcomes, with reported recovery rates ranging from approximately 60% to as high as 91% based on small patient cohorts with short-term follow-up. The review considered therapeutic interventions for severe cases, suggesting a potential benefit from glucocorticoid therapy, IL-6 inhibitors, intravenous immunoglobulins, and colchicine. However, these were primarily applied to critically ill patients, many of whom required intubation, indicating that these recommendations might be more applicable to individuals with concomitant acute respiratory distress syndrome [4]. Also, Mrudula et al. assessed the recovery rate from post-COVID myocarditis as 92.1% (35/38 cases) [5].

Castiello et al. concluded that acute myocarditis related to COVID-19 may present even after the resolution of an upper respiratory tract infection and that, if promptly treated, it is associated with a moderate prognosis. Furthermore, his comprehensive review of the challenges in diagnosing and managing COVID-19-related myocarditis emphasised that current evidence cannot offer definitive treatment guidance. Castiello underscored the urgent need for further research to elucidate optimal therapeutic approaches for patients who develop HF secondary to post-COVID-19 myocarditis, highlighting the necessity for prospective, large-scale studies to identify prognostic markers and refine management strategies [6].

In 2024, Semenzato et al., in a nationwide population-based study, examined the long-term prognosis of patients with myocarditis attributed to various etiologies, including post-COVID-19. In the context of treatment for post-COVID-19 myocarditis (298 individuals), their findings indicate myocarditis secondary to SARS-CoV-2 infection displays a more heterogeneous prognosis, with a higher incidence of persistent cardiac dysfunction than myocarditis following COVID-19 mRNA vaccination. HF occurred in 7/298 (2.3%), compared to 3/558 (0.5%) patients for postvaccine myocarditis and 45/3779 (1.2%) for conventional myocarditis. These data suggest that patients with post-COVID-19 myocarditis may benefit from more aggressive therapeutic strategies, such as prolonged pharmacotherapy and tailored device therapy. Moreover, Semenzato et al. emphasise the importance of individualised management and close follow-up to optimise outcomes for this patient population [7].

Given the high potential for myocardial recovery in myocarditis-related HF, current guidelines for non-ischemic HFrEF offer a Class IIa recommendation for implantable cardioverter-defibrillator (ICD) implantation in selected patients. Additionally, the role of non-invasive alternatives, such as wearable cardioverter defibrillators (WCD), is discussed, particularly in patients with reversible HF etiologies [1]. Despite these insights, the optimal management strategy, including the timing and escalation of pharmacotherapy and the use of device therapy, remains to be clearly defined.

In summary, while preliminary data suggest that patients with post-COVID-19 myocarditis HF may experience significant recovery, the current literature is limited. This underscores an urgent

need for further research to establish optimal treatment protocols, identify reliable prognostic markers, and refine management strategies for patients with HF secondary to post-COVID-19 myocarditis.

Case report

A 48-year-old male with asthma and anxiety-depressive disorder, with no history of cardiovascular diseases, experiencing his first COVID-19 infection, confirmed by PCR testing fourteen days before hospitalisation, was referred to cardiology for reduced exercise tolerance and severe dyspnoea (NYHA IV), which began three weeks prior. Hospital imaging and tests led to a diagnosis of HF due to myocarditis. His NT-proBNP level was 10,846 pg/ml (normal: 0–125 pg/ml). Echocardiography showed severe left ventricular dysfunction with 10% left ventricular ejection fraction (LVEF), and cardiac magnetic resonance (CMR) confirmed myocarditis [**Figure 1A-C**]. Coronary angiography was negative for significant stenosis, and toxic and other viral etiologies were excluded. Given the patient's favourable clinical status and refusal to undergo the procedure, a myocardial biopsy was not performed.

The patient received intensive HF therapy, including ramipril, eplerenone, bisoprolol, dapagliflozin, ivabradine, and loop diuretics, resulting in NT-proBNP reduction to 7,255 pg/ml. After eight days, he was discharged with symptoms improvement to NYHA II. Sacubitril/valsartan was introduced and titrated at a follow-up over four months. This led to improved functional capacity, LVEF increase to 41%, and NT-proBNP reduction to 315 pg/ml after nine months [1–3]. Due to clinical improvement, CMR-confirmed LVEF recovery [**Figure 1D-F**] and absence of ventricular arrhythmia, using WCD or ICD implantation was deemed unnecessary.

This case highlights the rising incidence of post-COVID-19 HF and underscores the need for early, intensive HF treatment in patients with reduced LVEF, which can lead to significant recovery. Guidelines recommend sustained pharmacotherapy to maintain clinical stability and functional improvements in such patients [1–3,8]. Currently, there are no established guidelines for the follow-up of patients with myocarditis; however, frequent monitoring in the early phase after diagnosis is essential, combined with continuous escalation of medication dosages. In the initial period, follow-up visits are recommended every 2 to 4 weeks, followed by evaluations every 3 months, and subsequently annually. It is suggested that serial echocardiographic assessments, such as improvements in LVEF and reductions in left ventricular dimensions combined with serial laboratory assessments indicating declining levels of natriuretic peptides, may collectively serve as favourable prognostic indicators. These observations require further validation in more extensive, prospective studies to establish their predictive value in this patient population. Additional studies are needed to refine management strategies for restoring left ventricular function.

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Conflict of interest statement
The authors declare no conflict of interest.

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References

1. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, Burri H, Butler J, Čelutkienė J, Chioncel O, Cleland JGF, Coats AJ, Crespo-Leiro MG, Farmakis D, Gilard M, Heymans S, Hoes AW, Jaarsma T, Jankowska EA, Lainscak M, Lam CSP, Lyon AR, McMurray JJ, Mebazaa A, Mindham R, Muneretto C, Piepoli MF, Price S, Rosano GMC, Ruschitzka F, Skibelund AK; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021 Dec 21;42(48): 4901. doi: 10.1093/eurheartj/ehab368. PMID: 34447992.
2. Kasprzak JD, Gorczyca-Głowacka I, Sobczak-Kaleta M, Barylski M, Drożdż J, Filipiak KJ, Kapłon-Cieślicka A, Lelonek M, Mamcarz A, Ochijewicz D, Ryś-Czaporowska A, Starzyk K, Szymański FM, Wełnicki M, Wożakowska-Kapłon B. Pharmacotherapy of heart failure A.D. 2023. Expert opinion of Working Group on Cardiovascular Pharmacotherapy, Polish Cardiac Society. Kardiol Pol. 2023; 81: 537-556. doi: 10.33963/KP.a2023.0110. PMID: 37179465.
3. Heidenreich PA, Bozkurt B, Aguilar D, Allen LA, Byun JJ, Colvin MM, Deswal A, Drazner MH, Dunlay SM, Evers LR, Fang JC, Fedson SE, Fonarow GC, Hayek SS, Hernandez AF, Khazanie P, Kittleson MM, Lee CS, Link MS, Milano CA, Nnacheta LC, Sandhu AT, Stevenson LW, Vardeny O, Vest AR, Yancy CW. 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

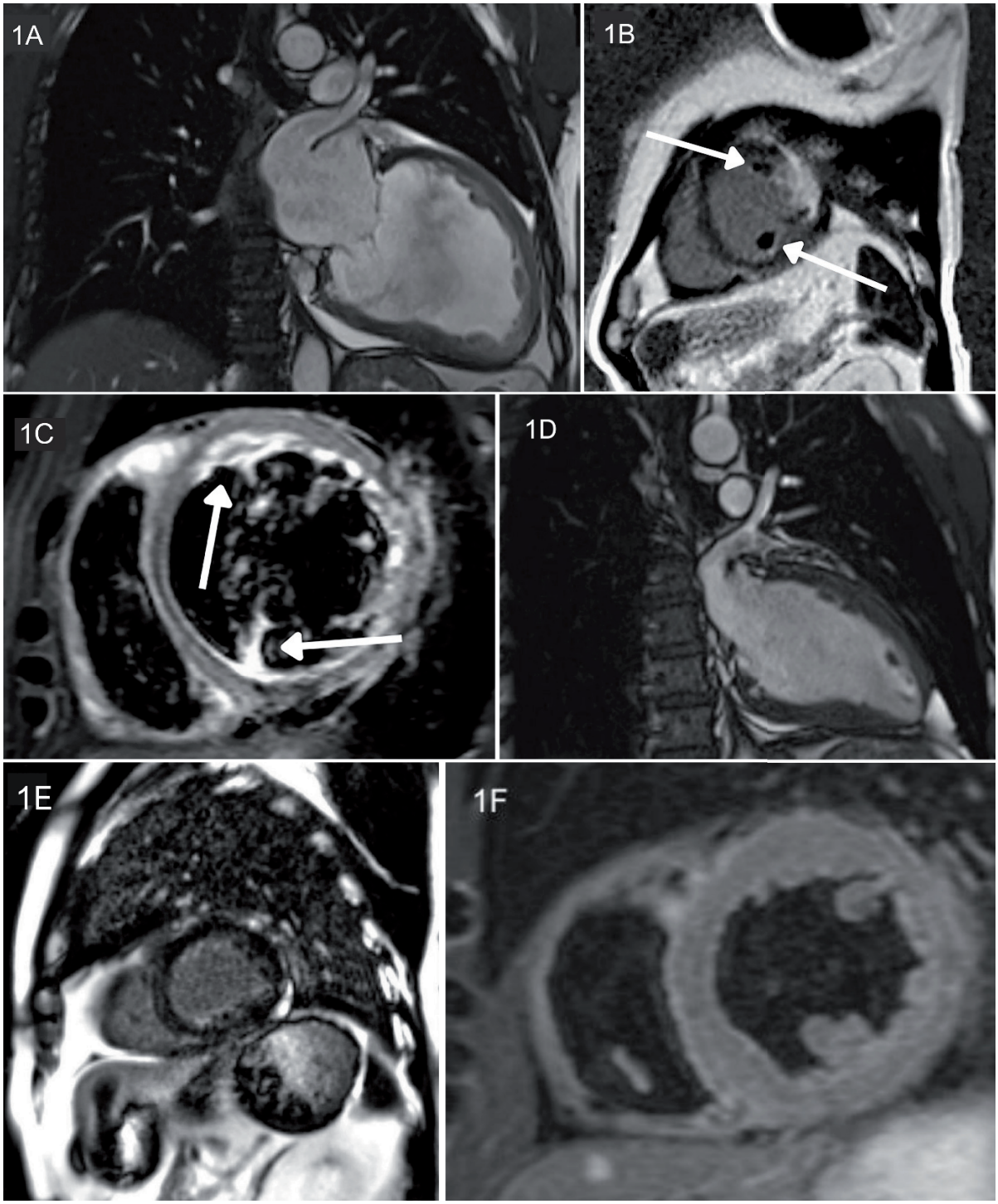

Figure 1. A – Cine-Magnetic Resonance Imaging 2-chamber view in end-diastole demonstrates left ventricular dilatation on admission. **B** – Magnetic Resonance Imaging 2-chamber short-axis, delayed enhancement images show intramural hyperenhancement (arrowheads) in the apical lateral wall and the adjacent mass (arrow) with no enhancement typical of thrombus formation in the left ventricle. **C** – Short-axis T2-weighted Magnetic Resonance Imaging shows myocardial oedema associated with acute myocarditis (arrows) predominantly involving the epicardial or transmural myocardium in the lateral wall on admission. **D –** Cine-Magnetic Resonance Imaging 2-chamber view in end-diastole demonstrates left ventricular reverse remodelling after 6 months of follow-up. **E –** agnetic Resonance Imaging 2-chamber short-axis delayed enhancement images show a reduction of the lesions shown in Figure 1B after 6 months. **F –** Short-axis T2-weighted magnetic resonance imaging shows the resolution of oedema, as shown in Figure 1C, after 6 months. Abbreviations: MRI – magnetic resonance imaging.

Clinical Practice Guidelines. Circulation. 2022; 145: e895–e1032. doi: 10.1161/CIR.0000000000001063. PMID: 35363499.
4. Sawalha K, Abozenah M, Kadado AJ, Battisha A, Al-Akchar M, Salerno C, Hernandez-Montfort J, Islam AM. Systematic Review of COVID-19 Related Myocarditis: Insights on Management and Outcome. Cardiovasc Revasc Med. 2021; 23: 107-113. doi: 0.1016/j.carrev.2020.08.028. PMID: 32847728; PMCID: PMC7434380.
5. Sai Santhosha Mrudula A, Agarwal P, Vempati R, Alla D, Balusu K, Tarannum S, Patel K, Devaragudi S, Patel D, Sultana Q, Paudel K. Clinical outcome of established diagnostic and treatment modalities of COVID-19-associated myocarditis: a systematic review. Ann Med Surg (Lond). 2023; 85(7): 3583-3594. doi: 10.1097/MS9.0000000000000964. PMID: 37427189.
6. Castiello T, Georgiopoulos G, Finocchiaro G, Claudia M, Gianatti A, Delialis D, Aimo A, Prasad S. COVID-19 and myocarditis: a systematic review and overview of current challenges. Heart Fail Rev. 2022; 27: 251-261. doi: 10.1007/s10741-021-10087-9. PMID: 33761041.
7. Semenzato L, Le Vu S, Botton J, Bertrand M, Jabagi M, Drouin J, Cuenot F, Zores F, Dray-Spira R, Weill A, Zureik M. Long-Term Prognosis of Patients With Myocarditis Attributed to COVID-19 mRNA Vaccination, SARS-CoV-2 Infection, or Conventional Etiologies. JAMA. 2024; 332(16): 1367–77. doi: 10.1001/jama.2024.16380. PMID: 39186694
8. Bielecka-Dabrowa A, Cichocka-Radwan A, Lewek J, Pawliczak F, Maciejewski M. Banach M. Cardiac manifestations of COVID-19. Rev Cardiovasc Med. 2021; 22: 365-371. doi: 10.31083/j.rcm2202043. PMID: 34258904.