AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

POST–COVID-19 DILATED CARDIOMYOPATHY: CURRENT EVIDENCE AND CLINICAL CHALLENGES

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ABSTRACT: Background: The long-term cardiovascular sequelae of SARS-CoV-2 infection have emerged as a significant global health concern. Dilated cardiomyopathy (DCM) is a serious potential complication of the post-acute phase of COVID-19, often resulting from persistent myocardial inflammation or autoimmune dysregulation. Objective: This article reviews current evidence regarding the pathophysiology, diagnosis, and management of post-COVID-19 DCM and highlights the clinical challenges in distinguishing it from idiopathic forms. Methods: A comprehensive review of literature published between 2020 and 2024 was conducted using PubMed, Scopus, and Google Scholar databases. Studies focusing on cardiac magnetic resonance (CMR) findings, biopsy-proven myocarditis, and heart failure outcomes in post-COVID patients were analyzed. Results: Evidence suggests that post-COVID-19 DCM develops through direct viral toxicity, cytokine-mediated injury, and endothelial dysfunction. CMR studies reveal late gadolinium enhancement (LGE) in a subset of recovered patients, indicating myocardial fibrosis. Conclusion: Post-COVID-19 DCM requires a high index of suspicion. Early initiation of guideline-directed medical therapy (GDMT) is crucial. Long-term longitudinal studies are needed to determine the reversibility of this condition.

Keywords: COVID-19, dilated cardiomyopathy, heart failure, myocarditis, cardiac MRI, SARS-CoV-2.

POST–COVID-19 DILATATSION KARDIOMIOPATIYASI: JORIY DALILLAR VA KLINIK MUAMMOLAR.

ANNOTATSIYA: Kirish: SARS-CoV-2 infeksiyasining uzoq muddatli yurak-qon tomir asoratlari global sog‘liqni saqlashning dolzarb muammosiga aylandi. Dilatatsion kardiomiopatiya (DKMP) COVID-19 ning o‘tkir davridan keyingi jiddiy asorat bo‘lib, ko‘pincha davomli miokard yallig‘lanishi yoki autoimmun buzilishlar natijasida yuzaga keladi. Maqsad: Ushbu maqola post-COVID-19 DKMP patofiziologiyasi, diagnostikasi va davolash bo‘yicha mavjud dalillarni ko‘rib chiqadi hamda uni idiopatik shakllardan ajratishdagi klinik muammolarni yoritadi. Usullar: 2020–2024 yillar oralig‘ida PubMed, Scopus va Google Scholar bazalarida chop etilgan adabiyotlar tahlil qilindi. Tadqiqotda asosan yurak magnit-rezonans tomografiyasi (MRT) natijalari va yurak yetishmovchiligi oqibatlariga e’tibor qaratildi. Natijalar: Post-COVID-19 DKMP virusning to‘g‘ridan-to‘g‘ri toksik ta’siri, sitokinlar bo‘roni va endotelial disfunksiya orqali rivojlanishi aniqlandi. MRT tekshiruvlari tuzalgan bemorlarning bir qismida kech gadoliniy to‘planishini (LGE) ko‘rsatdi, bu miokard fibrozidan dalolat beradi. Xulosa: Post-COVID-19 DKMP yuqori darajadagi klinik hushyorlikni talab qiladi. Qo‘llanmalarga asoslangan dori terapiyasini (GDMT) erta boshlash juda muhimdir. Ushbu holatning qaytuvchanligini aniqlash uchun uzoq muddatli kuzatuvlar talab etiladi.

AMERICAN ACADEMIC PUBLISHER

INTERNATIONAL JOURNAL OF MEDICAL SCIENCES

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Kalit so‘zlar: COVID-19, dilatatsion kardiomiopatiya, yurak yetishmovchiligi, miokardit, yurak MRT, SARS-CoV-2.

ДИЛАТАЦИОННАЯ КАРДИОМИОПАТИЯ ПОСЛЕ COVID-19: ТЕКУЩИЕ ДАННЫЕ И КЛИНИЧЕСКИЕ ПРОБЛЕМЫ.

АННОТАЦИЯ: Введение: Долгосрочные сердечно-сосудистые последствия инфекции SARS-CoV-2 стали значительной глобальной проблемой здравоохранения. Дилатационная кардиомиопатия (ДКМП) является серьезным потенциальным осложнением подострой фазы COVID-19, часто возникающим в результате персистирующего воспаления миокарда или аутоиммунной дисрегуляции. Цель: В данной статье рассматриваются текущие данные о патофизиологии, диагностике и лечении постковидной ДКМП, а также освещаются клинические проблемы дифференциальной диагностики. Методы: Был проведен всесторонний обзор литературы, опубликованной в период с 2020 по 2024 год, с использованием баз данных PubMed, Scopus и Google Scholar. Анализировались исследования, посвященные данным МРТ сердца и исходам сердечной недостаточности. Результаты: Данные свидетельствуют о том, что постковидная ДКМП развивается вследствие прямой вирусной токсичности, цитокинового повреждения и эндотелиальной дисфункции. Исследования МРТ выявляют позднее накопление гадолиния (LGE) у части выздоровевших пациентов, что указывает на фиброз миокарда. Заключение: Постковидная ДКМП требует высокой клинической настороженности. Раннее начало медикаментозной терапии согласно рекомендациям (GDMT) имеет решающее значение. Необходимы долгосрочные продольные исследования для определения обратимости этого состояния.

Ключевые слова: COVID-19, дилатационная кардиомиопатия, сердечная недостаточность, миокардит, МРТ сердца, SARS-CoV-2.

INTRODUCTION

The outbreak of Coronavirus Disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), initially manifested as a respiratory syndrome. However, it rapidly became evident that the cardiovascular system is a primary target of the virus. While acute cardiac injury—manifested by elevated troponin levels, arrhythmias, and thromboembolism—is well-documented during the active infection phase, the long-term sequelae known as "Long COVID" or Post-Acute Sequelae of SARS-CoV-2 (PASC) present a growing challenge for cardiologists.

Among these sequelae, the development of Dilated Cardiomyopathy (DCM) represents a particularly severe trajectory. DCM is characterized by left ventricular (LV) dilation and systolic dysfunction in the absence of abnormal loading conditions or coronary artery disease sufficient to explain the global systolic impairment. In the context of COVID-19, DCM may arise as a consequence of fulminant myocarditis that transitions into a chronic inflammatory state or as a *de novo* process driven by autoimmune mechanisms post-recovery. Understanding the transition from acute viral injury to chronic cardiomyopathy is essential for developing effective screening and management strategies.

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LITERATURE REVIEW

Pathophysiological mechanisms - Current literature posits three primary mechanisms for post-COVID-19 myocardial injury leading to DCM:

Direct Viral Cytotoxicity - Studies utilizing endomyocardial biopsy (EMB) have demonstrated the presence of SARS-CoV-2 viral particles within cardiomyocytes and endothelial cells, facilitated by the ACE2 receptor binding (Basso et al., 2021).

Hyperinflammation (Cytokine storm) - The excessive release of pro-inflammatory cytokines, particularly IL-6 and TNF-alpha, can lead to myocardial edema, necrosis, and subsequent fibrosis. This "bystander" damage is believed to be a major contributor to remodeling (Tschöpe et al., 2021).

Autoimmunity - Molecular mimicry between viral antigens and cardiac proteins (e.g., myosin heavy chain) can trigger autoimmune myocarditis, which may progress to DCM weeks or months after the initial infection.

Prevalence and imaging findings - A landmark study by Puntmann et al. (2020) using Cardiac Magnetic Resonance (CMR) imaging revealed that 78% of patients recovered from COVID-19 had cardiac involvement, and 60% showed ongoing myocardial inflammation independent of preexisting conditions. Although later studies suggested lower prevalence rates, the persistent risk of myocardial fibrosis (scarring) remains a predictor for arrhythmogenic DCM.

Clinical presentation - Patients typically present with symptoms of heart failure (dyspnea, fatigue, edema) 1 to 6 months post-infection. Unlike classic viral myocarditis (e.g., Coxsackievirus), which often presents acutely, post-COVID DCM can have an insidious onset, often masked by the generalized fatigue associated with Long COVID.

METHODS

This article employs a narrative review methodology. A systematic search was conducted on major medical databases including PubMed, MEDLINE, and Google Scholar for articles published between January 2020 and March 2024.

Search terms included - "COVID-19 cardiomyopathy", "SARS-CoV-2 dilated cardiomyopathy", "Post-COVID heart failure", and "viral myocarditis mechanisms".

Inclusion criteria - Peer-reviewed original research, meta-analyses, and major case series involving adult patients.

Exclusion criteria - Studies focusing solely on acute phase shock without follow-up, and single case reports with insufficient diagnostic data (e.g., lack of echocardiography or MRI).

Data was synthesized to categorize findings into pathophysiology, diagnostic challenges, and therapeutic outcomes.

RESULTS

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Structural and functional changes - The analysis of aggregated data indicates that post-COVID DCM is distinct from ischemic cardiomyopathy. The primary structural change is global biventricular hypokinesis, often with a predisposition for right ventricular involvement due to concurrent pulmonary vascular damage (pulmonary embolism or microthrombi).

Table 1: Comparison of classic viral myocarditis vs. post-COVID-19 DCM

Feature	**Classic viral myocarditis (e.g., Coxsackie)**	**Post-COVID-19 dilated cardiomyopathy**
Onset	Often acute, fulminant	Can be acute, but frequently subacute/insidious
Primary mechanism	Direct viral lysis prominent	Mixed: Viral persistence + Microvascular thrombosis + Autoimmunity
Endothelial involvement	Less common	Prominent (Endotheliitis and microthrombi)
Fibrosis pattern (CMR)	Subepicardial (Inferolateral wall)	Patchy, diffuse, or non-ischemic patterns
Systemic features	Usually isolated cardiac	Part of multi-organ inflammatory syndrome

Diagnostic yield - Echocardiography remains the first-line tool, showing reduced Left Ventricular Ejection Fraction (LVEF) < 50% and increased Left Ventricular End-Diastolic Diameter (LVEDD). However, CMR is superior in the post-COVID setting for detecting myocardial edema (T2 mapping) and fibrosis (Late Gadolinium Enhancement), which correlates with poor long-term prognosis.

Biomarkers Persistently elevated NT-proBNP and high-sensitivity Troponin T (hs-TnT) are observed in the subacute phase, signaling ongoing remodeling before overt DCM symptoms manifest.

DISCUSSION

Clinical challenges One of the primary challenges identified is the overlap with "Long COVID" symptoms. Dyspnea and fatigue are common in post-COVID patients due to pulmonary sequelae or deconditioning. Consequently, cardiac dysfunction may be overlooked until advanced DCM develops. Clinicians must differentiate between "subjective" Long COVID and objective myocardial impairment.

Another challenge is differentiating *de novo* DCM from exacerbation of subclinical preexisting heart disease. Without pre-COVID imaging, it is often difficult to attribute DCM solely to the viral infection, complicating epidemiological data.

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Management strategies - There is no specific antiviral therapy for post-COVID DCM. Management aligns with standard Heart Failure (HF) guidelines: 1) Guideline-Directed Medical Therapy (GDMT): Beta-blockers, ACE inhibitors/ARBs/ARNIs, MRAs, and SGLT2 inhibitors form the cornerstone of therapy. 2) Immunomodulation: In cases with biopsy-proven active inflammation, corticosteroids or intravenous immunoglobulin (IVIG) have been used, though high-quality randomized trial data for the chronic phase is lacking. 3) Anticoagulation: Given the thrombogenic nature of COVID-19, prophylactic anticoagulation is often considered in patients with severe LV dysfunction (LVEF < 30%) or intracardiac thrombus.

CONCLUSION

Post-COVID-19 dilated cardiomyopathy represents a distinct and evolving clinical entity that extends the burden of the pandemic well beyond the acute infection. The evidence suggests that a combination of persistent inflammation, endothelial damage, and autoimmune reactions drives myocardial remodeling.

1. Surveillance - Patients with moderate-to-severe COVID-19, particularly those with troponin elevation during the acute phase, warrant cardiovascular follow-up 3-6 months post-recovery.

2. Early intervention - Early detection via Echocardiography and CMR allows for the timely initiation of GDMT, which may reverse remodeling (reverse remodeling) in this specific cohort more effectively than in chronic ischemic DCM.

3. Future directions - Longitudinal registries are urgently needed to determine the 5-year and 10-year outcomes of these patients and to establish whether post-COVID DCM is a progressive or self-limiting condition.

For the practicing clinician, a low threshold for cardiac imaging is recommended in any post-COVID patient presenting with disproportionate dyspnea or new-onset arrhythmia.

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