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Original Research Article

Prevalence of Myocarditis in Adults with Prior COVID-19 Infection: A Cross-Sectional Study

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Abstract:

Background: COVID-19 has been linked to a wide spectrum of cardiovascular complications, with myocarditis drawing particular attention. Although vaccination is known to lessen the severity of illness, uncertainty remains about the persistence of myocardial inflammation in individuals who contract the infection after immunization.

Methods: A cross-sectional study was carried out over a one-year period in the Department of Medicine, Assam Medical College and Hospital, Dibrugarh. The study recruited 100 adults aged 18–59 years with a documented history of COVID-19 infection and complete vaccination. Participants with pre-existing cardiac, autoimmune, or chronic systemic diseases were excluded. Each subject underwent detailed clinical assessment, electrocardiography (ECG), and contrast-enhanced cardiac magnetic resonance imaging (MRI) to evaluate myocardial changes.

Results: Abnormal ECG patterns were common, with sinus tachycardia detected in 48% and non-specific ST–T wave alterations in 38% of cases. Cardiac MRI demonstrated late gadolinium enhancement (LGE)-a characteristic marker of myocarditis—in 8% of participants. The relationship between previous COVID-19 infection and myocardial involvement was statistically significant ($p = 0.01$), with the majority of cases seen among men aged 30–39 years. Although no major structural heart disease was evident, subtle inflammatory changes were observed.

Conclusion: This study suggests that even fully vaccinated individuals who recover from COVID-19 may experience silent myocardial injury. Incorporating ECG and cardiac MRI into follow-up protocols can aid in early detection. Younger male patients with lingering symptoms may benefit most from targeted cardiac surveillance.

Keywords: COVID-19, Myocarditis, Vaccination, Cardiac MRI, Post-Infectious Sequelae.

Introduction

Myocarditis refers to inflammation of the myocardium, the muscular middle layer of the heart. It may occur secondary to infections, autoimmune processes, or toxic exposures, with a global incidence estimated at 22 per 100,000 individuals.[1] Clinically, it can present as acute, fulminant, subacute, or chronic inflammatory cardiomyopathy. [2,3] Histopathology typically reveals inflammatory cell infiltrates with or without myocyte necrosis. The 2013 ESC criteria highlight diagnostic challenges, particularly since the disease often affects young to middle-aged adults (20–50 years).[4] Common symptoms include chest pain and dyspnoea, reported in nearly half of patients, usually following flu-like prodromes.[4,5] Severe forms occur in ~26%, frequently with left ventricular dysfunction or arrhythmias.[5] Evidence suggests that SARS-CoV-2 infection confers a greater risk of myocarditis than vaccination, although the underlying mechanisms remain incompletely understood.[6]

SARS-CoV-2, the causative virus of COVID-19, contains structural proteins such as spike (S), envelope (E), membrane (M), and nucleocapsid (N), in addition to its RNA genome within a lipid bilayer.[7] The clinical spectrum of COVID-19 ranges from mild symptoms such as fever, cough, sore throat, or anosmia, to severe respiratory failure requiring hospitalization. Diagnosis is confirmed via RT-PCR, rapid antigen tests, NAATs, or antibody assays (IgM/IgG).[8]

During the pandemic, myocarditis became a key concern. Its global burden is underestimated, though

reported rates range between 10.2 and 105.6 per 100,000, translating to roughly 1.8 million new cases annually. In children, the incidence is much lower; a Finnish study reported 1.95/100,000 in those aged 0–15.[9,10] Post-mortem studies show myocarditis in up to 42% of sudden cardiac deaths, contributing to a quarter of such deaths in people younger than 21.[9] It is also a known precursor to dilated cardiomyopathy (DCM). Male predominance is marked, with peaks at 16–20 years in men and 56–60 years in women.[11]

Myocardial injury develops either through direct viral replication and cytotoxicity or an autoimmune-mediated process persisting beyond viral clearance.[12] Outcomes vary, ranging from full recovery to chronic inflammation leading to DCM. Cytokine release syndrome (CRS), a hyperinflammatory state often linked with COVID-19, may further aggravate myocardial injury, leading to arrhythmias, hypoxia, and hemodynamic collapse. [13,14]

mRNA vaccines (BNT162b2, mRNA-1273) have been associated with rare myocarditis cases, primarily in adolescent males after the second dose, with reported incidences of 162.2/million in the U.S. and 97/million in Denmark. [15,16] Risks are lower in younger children (4.8/million, ages 5–11) compared with adolescents (57.4/million) (17). CDC monitoring noted 147 cases among 2.2 million adolescents. [15,17,18] Most were mild and self-limiting, and overall risk remained lower than after SARS-CoV-2 infection. [16,19,20]

Classification of myocarditis relies on histology (Dallas criteria), underlying cause, and clinical course-acute, subacute, chronic, or chronic inflammatory cardiomyopathy.[21,22] Presentations include chest pain (up to 95%), arrhythmias, syncope, and fulminant myocarditis with high mortality.[21,23,24] Diagnostic evaluation encompasses ECG (abnormal in ~90%), inflammatory and cardiac biomarkers, echocardiography, cardiac MRI, PET imaging, and confirmatory endomyocardial biopsy.[24,25]

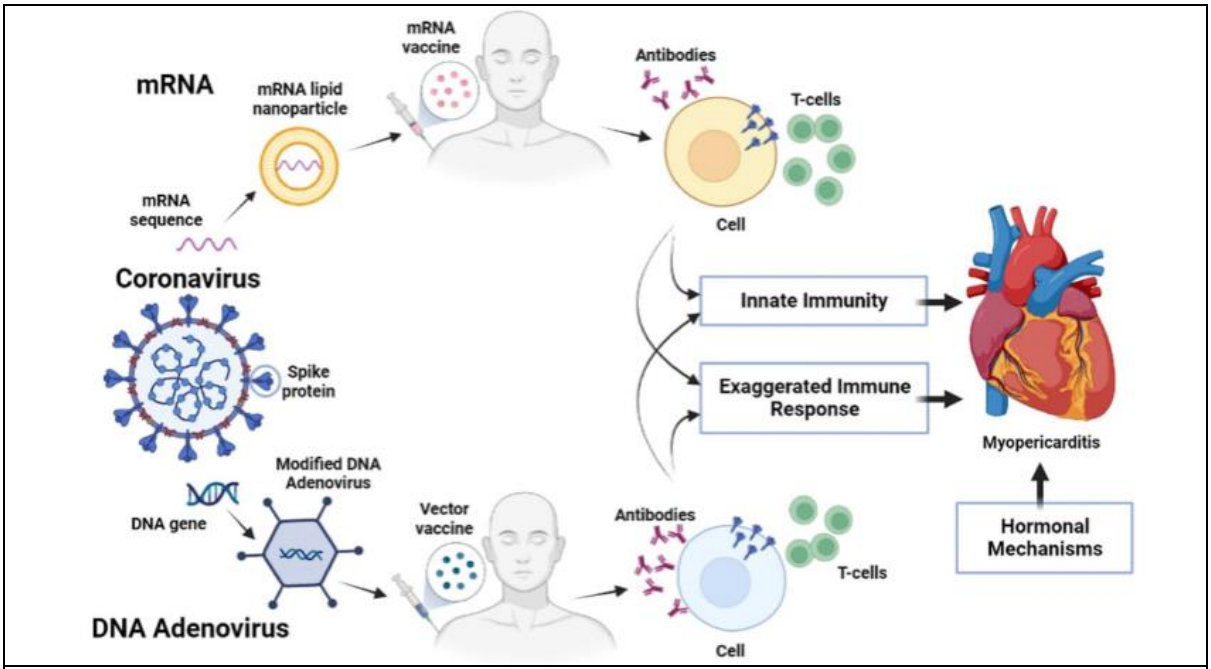

Figure 1: Possible Molecular Mechanisms of COVID-19 Vaccine-Induced Myocarditis [26]

Materials and Methods

Place of Study: This was a hospital-based, cross-sectional study conducted in the Department of General Medicine at Assam Medical College and Hospital, Dibrugarh over a duration of one year on 100 patients.

Study Population: The study population included all adult individuals aged between 18 to 59 years who had a history of COVID-19 vaccination, with a prior history of COVID-19 infection, as per the inclusion criteria.

Inclusion Criteria: Patients with 18 to 59 years age who were hospitalized with COVID 19 infection and history of COVID vaccination who has received at least two vaccine doses.

Exclusion Criteria: Patient with hypertension, diabetes mellitus, known cardiac disease, COPD, history of smoking, Chronic Kidney Disease, known autoimmune diseases, any other known chronic illness, did not take two doses of COVID vaccine, did not give consent.

Data Collection Procedure: Data were collected using a pre-designed and pre-tested structured proforma tailored to capture both clinical and investigational parameters related to the cardiovascular health of the participants. The study process included:

1. **Informed Consent**

Participants were informed about the purpose, procedures, and potential risks and benefits of the study. Written informed consent was obtained before enrolment.

2. **History and Clinical Examination**

A comprehensive medical history, including previous COVID-19 infection, vaccination status, comorbidities, and medications, was recorded. General and systemic clinical examinations were carried out with emphasis on cardiovascular signs and symptoms.

3. **Recruitment**

Participants from the Post-COVID group were identified from hospital records and contacted for follow-up through the Post-COVID Clinic, held weekly at the institution.

Investigations Conducted

1. **Electrocardiography (ECG)**

- **Equipment Used:** EDAN SE- 1201 GmbH (Europe)
- **Purpose and Parameters Assessed**
- ECG was performed to evaluate basic cardiac electrical activity and identify rhythm abnormalities. Key findings included non-specific ST segment changes, which may suggest myocardial stress or inflammation, and sinus tachycardia.

Cardiac Magnetic Resonance Imaging (Cardiac MRI)

- **Equipment Used:** MAGNETOM Avanto Fit 1.5 Tesla MRI Scanner Siemens Healthineers Germany.

- **Technique and Findings:**

Cardiac MRI was performed with gadolinium contrast to detect Late Gadolinium Enhancement (LGE)—a key imaging marker of myocardial inflammation, fibrosis, or scarring, typically seen in myocarditis. LGE allows precise tissue characterization, helping to differentiate active inflammation from irreversible damage. This modality was particularly valuable in confirming suspected myocarditis in patients with abnormal ECG or echocardiographic findings.

Observation and Result

Table 1: Comparison of Age in Post-Covid group

Age Group (Years)	Number of Patients	Percentage (%)
< 20	1	1%
20–29	19	18%
30–39	38	38%
40–49	27	27%
50–59	15	15%
Total	**100**	**100%**
Mean Age ± SD: 38.2 ± 11.3, **P-value:** 0.06		

Table 2: Comparison of Gender in Post-COVID Group

Sex	Post-COVID (n = 100)	Percentage (%)
Male	63	63%
Female	37	37%
Total	**100**	**100%**

Table 3: ECG Findings in Post-COVID Group (n = 100)

ECG Finding	Yes (n)	Yes (%)	No (n)	No (%)
ST-T Changes (Non-specific)	38	38%	62	62%
Sinus Tachycardia	48	48%	52	52%

Table 4: ECG Finding: Non-Specific ST-T Changes by Age in Post-COVID Group (n = 100)

Age Group	ST-T Changes: Yes	ST-T Changes: No	Total
<20 Years	9	1	10
20–29 Years	0	10	10
30–39 Years	13	25	38
40–49 Years	10	17	27
50–59 Years	6	9	15
Total	**38**	**62**	**100**
P-value (Chi-square test) = **0.0014** (Significant)			

Table 5: ECG Finding – Sinus Tachycardia by Age in Post-COVID Group (n = 100)

Age Group	Sinus Tachycardia: Yes	Sinus Tachycardia: No	Total
<20 Years	0	1	1
20–29 Years	9	10	19
30–39 Years	19	19	38
40–49 Years	12	15	27
50–59 Years	8	7	15
Total	**48**	**52**	**100**
P value (Chi- square test) – 0.206			

Table 6: MRI Findings – Myocarditis (LGE) in Post-COVID Vaccinated Group (n = 100)

Sex	LGE Present	LGE Absent	Total
Female	3	34	37
Male	5	58	63
Total	**8**	**92**	**100**
P value (Chi square test) - 0.798			

Discussion

This investigation offers valuable perspectives on the possible long-term cardiac effects of prior COVID-19 infection in vaccinated adults, with a particular emphasis on the occurrence of myocarditis. Conducted over a one-year period in the Department of Medicine at Assam Medical College & Hospital, the study adopted a cross-sectional comparative approach to assess differences in cardiac outcomes between individuals with and without a history of COVID-19 infection, all of whom had completed COVID-19 vaccination.

A key finding was the higher rate of myocarditis among those with a previous COVID-19 infection (8%), compared to just 1% in the non-COVID group, suggesting a lingering myocardial impact despite immunization. The use of late gadolinium enhancement (LGE) detected through cardiac MRI pointed to ongoing myocardial inflammation or fibrosis. The association between previous COVID-19 and myocardial involvement was statistically significant ($p = 0.01$), reinforcing findings from earlier studies that documented similar changes in cardiac MRI post-infection (Puntmann et al., 2020; Rajpal et al., 2021). [27,28]

On electrocardiography, non-specific ST-T changes and sinus tachycardia were observed more frequently among post-COVID individuals-38% and 48%, respectively-compared to much lower rates in the non-COVID group. Such changes can suggest underlying myocardial stress, inflammation, or ischemia, possibly driven by cytokine-related injury, autonomic dysregulation, or microvascular dysfunction. Previous studies by Mittal et al. and Rajpal et al. have documented similar post-infectious ECG abnormalities.[27]

The underlying mechanisms likely involve direct viral injury to cardiac myocytes or immune-mediated responses triggered by SARS-CoV-2, along with systemic inflammation characterized by cytokine overproduction (e.g., IL-6, TNF-α). Microvascular thrombosis may further aggravate myocardial ischemia, contributing to the observed decline in systolic function and subtle structural changes.

In line with global data, this study also resonates with existing literature on vaccine-associated myocarditis, particularly with mRNA-based platforms such as BNT162b2 and mRNA-1273. Reports from Israel, Europe, and the U.S. have noted higher myocarditis rates among younger males following vaccination, particularly after the second dose. However, our findings emphasize that prior COVID-19 infection may carry a greater risk for myocarditis than vaccination alone. [29,30]

In summary, the results of this study reinforce the need for routine cardiac monitoring in post-COVID individuals, especially in those presenting with non-specific symptoms like fatigue, palpitations, or decreased exercise capacity.

Conclusion

This study brings to light the important yet often overlooked cardiovascular complications in individuals who have recovered from COVID-19 but have already received full vaccination. The significantly higher incidence of myocarditis (8%) in the post-COVID group, as compared to the 1% incidence in those without prior infection, highlights the potential for residual myocardial involvement even after immunization.

Electrocardiographic signs such as non-specific ST-T abnormalities and sinus tachycardia, along with LGE findings on cardiac MRI, further support the presence of ongoing myocardial inflammation or injury. Despite the absence of gross structural changes like left ventricular dilatation, early indicators such as reduced strain and mild wall thickening point toward functionally relevant cardiac changes.

Importantly, our data suggest that the cardiac sequelae of COVID-19 may persist even after recovery and full vaccination, necessitating closer follow-up and cardiac evaluation. The use of imaging modali-

ties such as cardiac MRI proves essential in detecting early or subtle forms of cardiac dysfunction, which might otherwise go unnoticed.

Based on these observations, the study advocates for:

- Routine cardiac assessments in individuals with prior COVID-19 infection, particularly those with persistent post-viral symptoms;
- Implementation of post-COVID cardiac surveillance protocols, especially in populations at elevated risk;
- Continued public health efforts to refine vaccine safety monitoring and individualized risk assessment.

In conclusion, this research provides important evidence supporting the need for long-term cardiovascular follow-up in the post-COVID population and adds to the growing body of literature emphasizing the complex cardiac implications of SARS-CoV-2.

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