

Influence of COVID-19 Pandemic on Early-Onset Myocardial Infarction in Adults

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ABSTRACT

Myocardial infarction (MI) is one of the leading causes of morbidity and mortality worldwide and has shown a significant increase following the COVID-19 pandemic. Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) infection is strongly associated with cardiovascular complications such as myocarditis, arrhythmia, thromboembolism, stroke, and heart failure. The present review revealed the relationship between COVID-19 and cardiovascular injury, focusing on the role of cardiac troponin as a rapid and reliable biomarker for myocardial infarction diagnosis. Elevated troponin levels were found to be closely associated with disease severity and mortality risk, particularly among hospitalized and ICU-admitted patients. Comparative analysis demonstrated that troponin I/T possesses higher sensitivity and specificity than CK-MB and ECG for diagnosing myocardial injury. The review further discusses the major mechanisms responsible for cardiovascular damage that is cytokine storm, endothelial dysfunction, oxidative stress, hypoxia, and hypercoagulability. Graphical and tabular analyses also revealed increased long-term cardiovascular risks following COVID-19 infection, especially among elderly individuals and critically ill patients. Also, myocarditis risk after COVID-19 infection was significantly higher than after vaccination.

Keywords- Myocardial Infarction; COVID-19; Cardiac Troponin; Cardiovascular Complications; SARS-CoV-2.

I. INTRODUCTION

Myocardial infarction (MI), commonly known as a heart attack, is one of the leading causes of global morbidity and mortality. It occurs due to obstruction of coronary blood flow, resulting in ischemic injury to the cardiac muscle (Thygesen et al., 2018; Reed et al., 2020). Following the COVID-19 pandemic, the incidence of cardiovascular diseases, including MI, has increased considerably worldwide (Madjid et al., 2020; Bavishi et al., 2021). Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2) infection has been strongly associated with cardiovascular complications such as myocarditis, arrhythmias, thromboembolism, and heart failure (Clerkin et al., 2020; Guo et al., 2020; Nishiga et al., 2020). These complications are mainly linked to systemic inflammation, cytokine storm, endothelial dysfunction,

and a hypercoagulable state, all of which contribute to myocardial injury (Libby et al., 2020; Zheng et al., 2020).

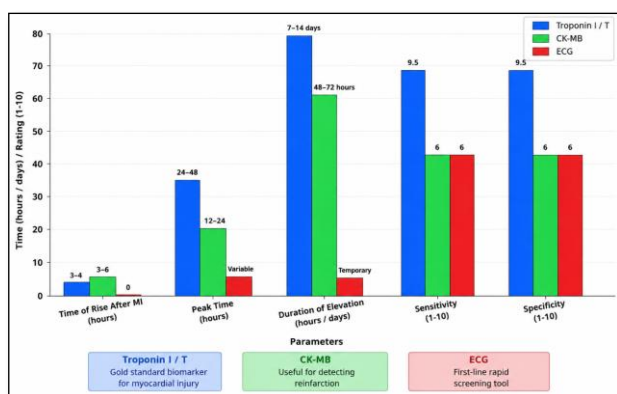
Clinical investigations have shown that myocardial injury is common among hospitalized COVID-19 patients and is frequently indicated by elevated cardiac biomarkers such as troponin I (Guo et al., 2020; Shi et al., 2020). Troponin is a highly sensitive and specific biomarker of cardiac muscle injury and plays an important role in the early diagnosis of myocardial infarction (Apple & Sandoval, 2020; Thygesen et al., 2018). Normal serum troponin levels are generally below 0.04 ng/mL, and elevations beyond this threshold are considered clinically significant for myocardial damage (Chapman et al., 2017; Keller et al., 2020). Increased troponin levels in COVID-19 patients are also associated with greater disease severity and mortality risk (Guo et al., 2020; Shi et al., 2020).

Post-COVID cardiovascular complications have increasingly been observed among younger adults aged 25–45 years (Knight et al., 2021; Patone et al., 2021). Studies suggest that even mild COVID-19 infection may result in long-term cardiovascular consequences, including acute coronary syndrome and ischemic stroke (Taquet et al., 2021; Xie et al., 2022). Although the precise mechanisms are not fully understood, both direct viral invasion of cardiac tissue and indirect effects such as hypoxia, immune-mediated injury, oxidative stress, and systemic inflammation are believed to contribute to cardiovascular damage (Bavishi et al., 2021; Nishiga et al., 2020).

1.1 Rapid Diagnostic Test for Myocardial Infarction

One of the most reliable and rapid diagnostic methods for myocardial infarction is the measurement of cardiac troponins through blood testing (Apple & Sandoval, 2020; Thygesen et al., 2018). Cardiac troponins are regulatory proteins involved in cardiac muscle contraction and are highly specific to myocardial tissue (Jaffe et al., 2018). Under normal physiological conditions, troponin remains within cardiac cells; however, during ischemia or coronary artery obstruction, troponin proteins are released into the bloodstream (Katus et al., 2017).

Elevated troponin levels serve as a highly sensitive and accurate indicator of myocardial injury and are essential in diagnosing acute coronary syndromes, including MI (Chapman et al., 2017; Thygesen et al., 2018). Clinically, troponin testing is recommended in patients presenting with chest pain, angina, or suspected cardiac ischemia for risk stratification and therapeutic decision-making (Apple & Sandoval, 2020).



Graph: 1. Comparison of Diagnostic Methods for Myocardial Infarction

Graph 1 ITroponin I/T showed the highest sensitivity and specificity for myocardial infarction diagnosis, making it the gold standard biomarker. CK-MB was moderately effective and useful for detecting reinfarction, while ECG served as the fastest initial screening tool despite moderate accuracy.

Compared with other diagnostic approaches, troponin assays are considered the gold standard

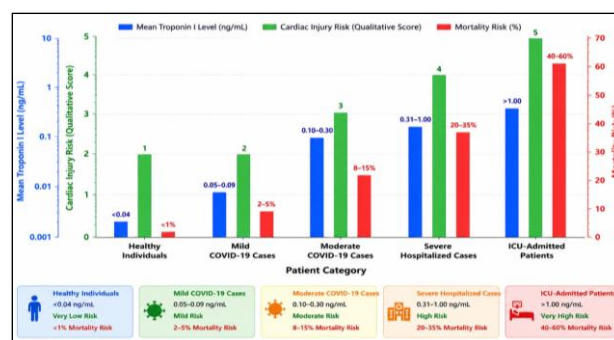
biomarkers for myocardial injury (Reichlin et al., 2012; Thygesen et al., 2018). Normal serum troponin concentrations are generally below 0.04 ng/mL, while elevated levels indicate cardiac muscle damage (Keller et al., 2020). High-sensitivity troponin assays further enhance early diagnosis and prognostic assessment in acute myocardial infarction (Chapman et al., 2017; Mueller et al., 2019).

1.2 Comparison of Troponin with ECG and CK-MB in MI Diagnosis

The diagnosis of myocardial infarction depends on clinical evaluation, biomarker assessment, and electrocardiographic findings (Thygesen et al., 2018). Among the available biomarkers, cardiac troponins (cTnI and cTnT) are considered the most sensitive and specific indicators of myocardial injury and have largely replaced older biomarkers such as creatine kinase-MB (CK-MB) (Apple & Sandoval, 2020; Jaffe et al., 2018).

Electrocardiography (ECG) remains the first-line, rapid, and non-invasive diagnostic tool for suspected MI (Ibanez et al., 2018). ECG detects ischemic abnormalities including ST-segment elevation, ST-segment depression, and T-wave inversion. However, ECG findings may appear normal during early-stage myocardial infarction or in non-ST elevation myocardial infarction (NSTEMI), thereby reducing its diagnostic sensitivity (Thygesen et al., 2018).

CK-MB was historically used for MI diagnosis because it rises within 3–6 hours after myocardial injury, peaks at 12–24 hours, and returns to normal within 48–72 hours (Collinson et al., 2017). Nevertheless, CK-MB lacks specificity because it can also be elevated in skeletal muscle injury (Apple et al., 2012).



Graph: 2. Troponin Levels and Severity of COVID-19-Associated Cardiac Injury

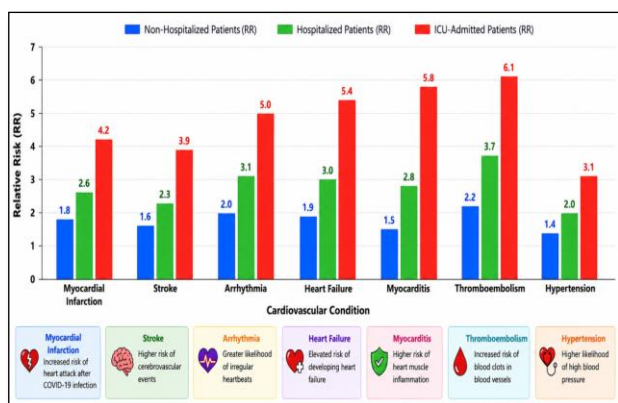
The graph 2 shows that higher troponin levels in COVID-19 patients are associated with increased cardiac injury severity and mortality risk. ICU-admitted patients had the highest troponin levels and greatest risk compared to mild and moderate cases.

In contrast, cardiac troponins rise within 3-4 hours after myocardial injury, peak at approximately 24-48 hours, and may remain elevated for 7-14 days (Reichlin et al., 2012). Due to their superior sensitivity and specificity, troponins are currently regarded as the

gold standard for MI diagnosis (Chapman et al., 2017; Thygesen et al., 2018).

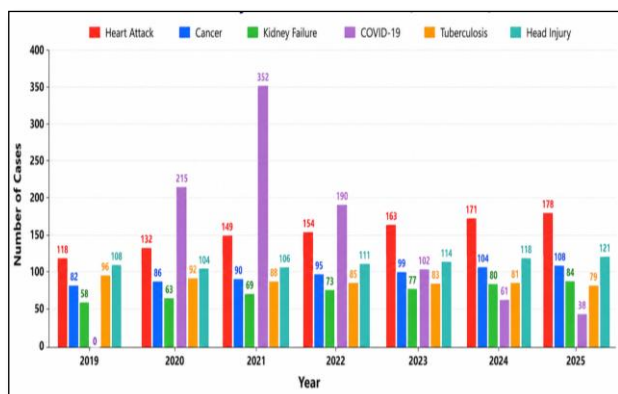
1.3 Dramatic Rise in Heart Attack after COVID-19

Heart attacks remain a major cause of mortality globally; however, the COVID-19 pandemic has been associated with a marked increase in myocardial infarction, stroke, cardiac arrest, and heart failure (Madjid et al., 2020; Wadhera et al., 2021).



Graph 3. Increased Risk of Cardiovascular Conditions Following COVID-19 Infection

The graph 3 indicates that COVID-19 infection significantly increases the risk of cardiovascular diseases, with the highest risk observed in ICU-admitted patients compared to hospitalized and non-hospitalized individuals.

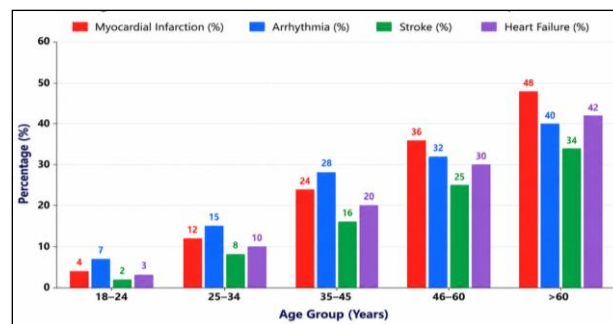


Graph 4. Trend of Major Diseases Over Time (2019-2025)

The graph 4 shows that heart attack, cancer, kidney failure, and head injury cases gradually increased from 2019 to 2025, while COVID-19 cases sharply rose in 2021 and then declined afterward.

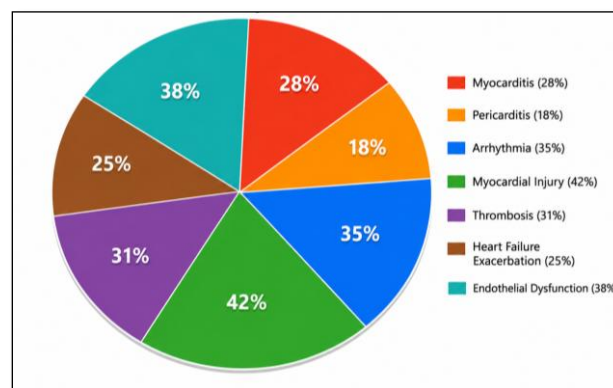
1.4 COVID-19 and Cardiovascular Disease

COVID-19 is now recognized as an independent risk factor for cardiovascular disease (Libby et al., 2020; Nishiga et al., 2020).



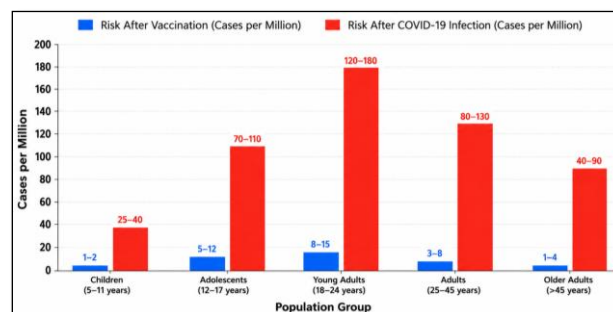
Graph 5. Age-Wise Distribution of Post-COVID Cardiovascular Complications

The graph 5 indicates that post-COVID cardiovascular complications increased with age, with the highest prevalence observed in individuals above 60 years.



Graph 6. Cardiovascular Complications Due to COVID-19 Infection

The graph 6 shows that myocardial injury and endothelial dysfunction were the most common cardiovascular complications associated with COVID-19 infection, while pericarditis had the lowest prevalence.



Graph 7. COVID-19 Vaccination and Myocarditis Risk

The graph 7 shows that the risk of myocarditis was much higher after COVID-19 infection than after vaccination across all age groups, especially among young adults.

1.5 Possible Causes of Heart Attack after COVID-19

Although COVID-19 primarily affects the respiratory system, it can also significantly impact the cardiovascular system (Clerkin et al., 2020; Libby et al., 2020).

Table: 1. Major Mechanisms of Cardiovascular Injury in COVID-19

Pathophysiological Mechanism	Cardiovascular Effect	Possible Outcome
Cytokine Storm (IL-6, TNF-α)	Systemic inflammation	Myocardial injury
Endothelial Dysfunction	Vascular damage	Thrombosis
Hypercoagulable State	Increased clot formation	Myocardial infarction
Hypoxia	Reduced oxygen supply	Ischemic injury
Direct Viral Invasion	Cardiomyocyte damage	Myocarditis
Oxidative Stress	Cellular injury	Heart failure
Immune-Mediated Injury	Autoimmune response	Pericarditis

The table 1 shows that multiple mechanisms such as cytokine storm, endothelial dysfunction, hypoxia, and hypercoagulability contribute to cardiovascular injury in COVID-19, leading to serious complications including myocardial infarction, myocarditis, thrombosis, and heart failure.

Table: 2. Post-COVID Long-Term Cardiovascular Outcomes

Cardiovascular Outcome	Relative Risk (RR) After COVID-19	Follow-Up Duration
Acute Myocardial Infarction	2.3	12 months
Ischemic Stroke	2.1	12 months
Arrhythmia	2.5	12 months
Heart Failure	2.7	12 months
Thromboembolic Events	3.1	12 months
Myocarditis	2.8	6–12 months
Pericarditis	1.9	6–12 months

The table 2 indicates that COVID-19 significantly increases the long-term risk of cardiovascular complications, with thromboembolic events and heart failure showing the highest relative risk during follow-up.

II. DISCUSSION AND CRITICAL ANALYSIS

The present review demonstrates a strong association between COVID-19 infection and cardiovascular complications, particularly myocardial infarction (MI), myocarditis, arrhythmia, and thromboembolic disorders (Clerkin et al., 2020; Libby et al., 2020; Nishiga et al., 2020). SARS-CoV-2 infection contributes to cardiovascular injury through systemic inflammation, endothelial dysfunction, cytokine storm, oxidative stress, and hypercoagulability (Zheng et al., 2020; Bavishi et al., 2021).

Graph 1 showed that cardiac troponin I/T has the highest sensitivity and specificity for MI diagnosis compared with CK-MB and ECG, confirming its role as the gold standard biomarker for myocardial injury (Thygesen et al., 2018; Apple & Sandoval, 2020; Chapman et al., 2017). Although ECG is useful for rapid screening, its moderate sensitivity limits early diagnosis in some patients (Ibanez et al., 2018).

Graph 2 demonstrated that elevated troponin levels were strongly associated with severe COVID-19 infection and increased mortality, especially among ICU-admitted patients (Guo et al., 2020; Shi et al., 2020). Similarly, Graph 3 indicated a substantial rise in cardiovascular complications following COVID-19 infection, particularly among critically ill individuals (Xie et al., 2022; Wadhwa et al., 2021).

The disease trend presented in Graph 4 revealed a progressive increase in heart attack cases after the COVID-19 pandemic, suggesting both direct and indirect cardiovascular impacts of SARS-CoV-2 infection (Madjid et al., 2020). Furthermore, Graph 5 showed that elderly individuals experienced the highest prevalence of post-COVID cardiovascular complications, likely due to pre-existing comorbidities and age-related endothelial dysfunction (Knight et al., 2021).

Graph 6 identified myocardial injury and endothelial dysfunction as major cardiovascular complications associated with COVID-19 infection, while Graph 7 demonstrated that myocarditis risk was significantly higher after COVID-19 infection than after vaccination, supporting the protective benefits of vaccination (Patone et al., 2021).

The mechanisms summarized in Table 1 indicated that cytokine storm, thrombosis, hypoxia, and immune-mediated injury play important roles in cardiovascular damage. In addition, Table 2 highlighted persistent long-term cardiovascular risks, particularly thromboembolic events and heart failure, even after recovery from COVID-19 infection (Taquet et al., 2021; Xie et al., 2022).

III. CONCLUSIONS

COVID-19 has emerged as a major risk factor for cardiovascular complications, including myocardial

infarction, myocarditis, arrhythmia, stroke, and heart failure. Elevated cardiac troponin levels remain the most reliable biomarker for early diagnosis and prognosis of myocardial injury. The results revealed that systemic inflammation, endothelial dysfunction, thrombosis, hypoxia, and immune-mediated injury play critical roles in COVID-19-associated cardiovascular damage. The study also highlights that severe COVID-19 infection and post-COVID conditions significantly increase long-term cardiovascular risk, particularly among elderly and critically ill patients. Furthermore, the risk of myocarditis following COVID-19 infection is substantially higher than after vaccination. Therefore, early diagnosis, continuous cardiovascular monitoring, and preventive strategies including vaccination are essential to reduce cardiovascular morbidity and mortality associated with COVID-19.

AUTHORS' CONTRIBUTIONS

Tanishq Gouniyal conducted the study and prepared the manuscript. Shreya Chaudhary assisted in data analysis and editing. Dr. Pradeep Kumar supervised the research work and approved the final manuscript.

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