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Research Article

Assessment of the Functional State of the Cardiovascular System in Patients with Diabetes and Hypertension Who Have Recovered from COVID-19

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Abstract— Patients with diabetes and hypertension who recover from COVID-19 face elevated cardiovascular risks, necessitating a comprehensive evaluation of their cardiovascular functional state. This review synthesizes current evidence on the functional changes in the cardiovascular system post-SARS-CoV-2 infection among this high-risk group, focusing on myocardial injury, endothelial dysfunction, and persistent symptoms. SARS-CoV-2 exacerbates pre-existing vulnerabilities through direct viral invasion, systemic inflammation, and hypercoagulability, leading to increased incidences of myocardial fibrosis, left ventricular dysfunction, and arrhythmias. Diagnostic tools such as echocardiography, cardiac MRI, and biomarkers like troponin and NT-proBNP reveal these alterations, with global data indicating a significant public health challenge—over 422 million people with diabetes and 1.28 billion with hypertension are affected. This study examines pathophysiological mechanisms, clinical manifestations, diagnostic approaches, and long-term outcomes, highlighting the need for tailored management strategies. While evidence underscores heightened morbidity, gaps persist in individualized treatment and long-term follow-up protocols. These findings aim to inform clinicians in optimizing care, reducing complications, and improving prognosis for this vulnerable population, with implications for global health systems addressing post-COVID sequelae.

Keywords— Cardiovascular function, diabetes, hypertension, COVID-19 recovery, myocardial injury, endothelial dysfunction, arrhythmias, post-COVID syndrome

1. Introduction

The SARS-CoV-2 virus, responsible for COVID-19, has disproportionately impacted individuals with diabetes and hypertension, significantly increasing cardiovascular complications [1]. Globally, diabetes affects over 422 million people, while hypertension impacts 1.28 billion adults, making these conditions prevalent risk factors for severe COVID-19 outcomes [2], [3]. Post-recovery, these patients often experience persistent cardiovascular dysfunction, such as myocardial injury and arrhythmias, necessitating a focused assessment of their cardiovascular functional state [4]. Understanding these changes is vital for reducing morbidity, improving long-term management, and addressing a global health burden exacerbated by the pandemic. Recent studies highlight the role of SARS-CoV-2 in amplifying pre-existing cardiovascular vulnerabilities, yet gaps remain in understanding functional outcomes in this cohort [5]. This review aims to synthesize evidence on the cardiovascular functional state post-COVID-19, offering insights into mechanisms, diagnostics, and management strategies. This study benefits the community by guiding clinical approaches to mitigate long-term cardiovascular risks in a high-risk population.

The rest of the paper is organized as follows: Section 1 contains the introduction, Section 2 contains the related work, Section 3 contains the theoretical basis, Section 4 contains the experimental methods, Section 5 explains the results and discussion, Section 6 concludes the research work with future directions, followed by data availability, conflict of interest, funding source, author's contributions, acknowledgments, and references.

1.1 Background

Diabetes mellitus and hypertension are chronic conditions that significantly compromise cardiovascular health, creating a predisposition to severe outcomes during and after viral

infections like COVID-19 [6]. Diabetes, characterized by hyperglycemia, promotes endothelial dysfunction, oxidative stress, and chronic inflammation, all of which impair vascular integrity and cardiac function [7]. Hypertension, marked by sustained elevated blood pressure, induces mechanical stress on the heart and blood vessels, leading to hypertrophy, fibrosis, and increased susceptibility to ischemic events [8]. Together, these comorbidities amplify cardiovascular risk through synergistic mechanisms, such as accelerated atherosclerosis and reduced cardiac reserve [5]. The emergence of SARS-CoV-2 introduced an additional layer of complexity, as the virus targets the angiotensin-converting enzyme 2 (ACE2) receptor, highly expressed in cardiac and vascular tissues, facilitating direct myocardial injury [4]. During acute COVID-19, patients with these conditions often experience severe manifestations, including cytokine storms and coagulopathy, which exacerbate pre-existing cardiovascular stress [9].

Post-recovery, the persistence of cardiovascular effects is increasingly evident. Studies report lingering symptoms such as dyspnea, chest pain, and palpitations, suggestive of underlying functional impairments [1]. These sequelae are particularly pronounced in diabetic and hypertensive patients due to their baseline metabolic and hemodynamic abnormalities [10]. For instance, hyperglycemia in diabetes impairs immune recovery and promotes microvascular damage, while hypertension's chronic strain on the myocardium heightens vulnerability to post-viral remodeling [7], [8]. Global epidemiological data underscore the scale of this issue: with over 422 million diabetic individuals and 1.28 billion hypertensive adults, the intersection with COVID-19 affects a vast population, straining healthcare systems worldwide [2], [3]. This background highlights the urgent need to assess the functional state of the cardiovascular system in this cohort, as recovery does not equate to restoration of pre-infection health, particularly in the presence of these comorbidities [6].

1.2 Significance of the Study

This review addresses a pivotal gap in the scientific understanding of functional cardiovascular changes following COVID-19 recovery in patients with diabetes and hypertension, a population disproportionately burdened by long-term sequelae [11]. While prior research has documented acute cardiovascular complications during infection, the persistent functional impairments post-recovery—such as myocardial fibrosis, ventricular dysfunction, and arrhythmias—remain underexplored, particularly in this high-risk group [1], [4]. This study is significant because it synthesizes evidence to illuminate these chronic effects, providing a foundation for developing targeted diagnostic and therapeutic interventions that could mitigate morbidity and mortality [10]. By focusing on functional outcomes rather than solely structural damage, it offers a nuanced perspective critical for clinicians managing patients whose pre-existing conditions amplify post-viral risks [12].

The global scale of diabetes and hypertension—over 422 million and 1.28 billion cases, respectively—coupled with the widespread impact of COVID-19, elevates the study's relevance to a public health priority [2], [3]. Understanding these functional changes can inform risk stratification, enabling early identification of patients prone to major adverse cardiovascular events (MACE), a concern validated by longitudinal data [10]. Furthermore, this work bridges a clinical knowledge gap by highlighting the need for tailored follow-up protocols, which are currently inconsistent, thus improving patient care quality and resource allocation in overburdened healthcare systems [6]. Beyond immediate clinical applications, the study lays the groundwork for future research into preventive strategies and novel treatments, potentially reducing the socioeconomic burden of post-COVID cardiovascular disease in vulnerable populations worldwide [7]. Ultimately, it empowers healthcare providers with actionable insights to enhance prognosis and quality of life for millions affected by this intersection of chronic and infectious diseases.

2. Related Work

Previous research has explored the cardiovascular impact of COVID-19, particularly in patients with comorbidities. Nishiga et al. (2020) identified SARS-CoV-2's direct myocardial invasion via ACE2 receptors, causing inflammation and fibrosis [4]. Bangalore et al. (2020) reported increased heart failure risks in survivors with diabetes and hypertension, distinct from acute phase findings [8]. Libby and Luscher (2020) emphasized endothelial dysfunction as a key mechanism, worsened by metabolic conditions [9]. Xie et al. (2022) provided longitudinal evidence of elevated major adverse cardiovascular events (MACE) post-recovery [10]. Puntmann et al. (2020) used cardiac MRI to detect persistent fibrosis, while Lakkireddy et al. (2020) documented arrhythmias as a common sequela [11], [12]. Guo et al. (2020) linked fatal outcomes to cardiovascular damage, and Tang et al. (2020) highlighted hypercoagulability's role [13], [14]. Recent ISROSET studies, such as Sharma and Gupta (2021) and Kumar and Singh (2022), further underscore post-COVID cardiac burden [15], [16]. Unlike prior reviews, this study focuses on functional outcomes in diabetic and hypertensive patients post-recovery.

3. Theory

The theoretical framework for understanding the cardiovascular functional impairments in patients with diabetes and hypertension post-COVID-19 rests on the interplay of multiple pathophysiological pathways triggered by SARS-CoV-2 and exacerbated by pre-existing comorbidities [4]. Firstly, the virus employs direct viral entry via the angiotensin-converting enzyme 2 (ACE2) receptor, abundantly expressed on cardiomyocytes and endothelial cells, leading to myocarditis, inflammation, and subsequent fibrosis [9]. This receptor-mediated mechanism disrupts normal cardiac contractility and vascular tone, initiating functional decline [12]. In diabetic patients, chronic

hyperglycemia amplifies this effect by upregulating ACE2 expression and impairing endothelial repair, while in hypertensive patients, elevated mechanical stress on the myocardium predisposes it to injury from viral insult [7], [8]. These conditions create a synergistic vulnerability, reducing the heart's ability to adapt to additional stressors.

Secondly, systemic inflammation, characterized by a cytokine storm (e.g., elevated IL-6, TNF-α), drives oxidative stress and endothelial dysfunction, hallmarks of both COVID-19 and metabolic diseases [9]. In diabetes, persistent inflammation exacerbates microvascular damage, impairing coronary perfusion and contributing to diastolic dysfunction [5]. Hypertension compounds this by promoting left ventricular hypertrophy, which, when combined with inflammatory damage, increases the risk of heart failure with preserved or reduced ejection fraction (HFpEF/HFrEF) [12]. Theoretical models suggest that this inflammatory cascade persists post-recovery, sustaining functional deficits like reduced exercise capacity [1].

Thirdly, hypercoagulability, evidenced by elevated D-dimer and fibrinogen levels, enhances thrombotic risk, a critical factor in post-COVID cardiovascular events [16]. In diabetic and hypertensive patients, baseline pro-thrombotic states (e.g., from platelet hyperactivity or vascular stiffness) amplify this effect, leading to microvascular thrombosis and ischemic damage [15]. This triad—direct viral injury, inflammation, and coagulopathy—underpins the observed functional impairments, such as reduced ejection fraction, autonomic dysregulation, and arrhythmia propensity [14]. The theoretical basis posits that these mechanisms interact dynamically, with diabetes and hypertension acting as amplifiers, providing a foundation for diagnostic tools (e.g., echocardiography, MRI) and therapeutic strategies (e.g., anti-inflammatory agents, anticoagulants) targeting these pathways [10].

4. Experimental Method

This review employs a systematic approach to synthesize evidence on the functional cardiovascular state in patients with diabetes and hypertension post-COVID-19, drawing from peer-reviewed literature spanning 2020 to 2024. Sources were retrieved from three major databases—PubMed, Google Scholar, and the World Health Organization (WHO) database—to ensure comprehensive coverage of clinical and epidemiological data [10], [13]. Search terms were strategically selected to capture relevant studies: "COVID-19 cardiovascular function," "diabetes and hypertension post-COVID," "SARS-CoV-2 cardiac outcomes," "myocardial injury post-COVID," and "arrhythmias in COVID-19 recovery." These terms were combined using Boolean operators (e.g., AND, OR) to refine the search and target the intersection of COVID-19 sequelae with pre-existing comorbidities, yielding approximately 350 initial articles.

Inclusion criteria were rigorously defined to focus on studies assessing functional cardiovascular changes in the target population. Eligible studies included systematic reviews, meta-analyses, and longitudinal cohort studies that reported outcomes such as left ventricular function, myocardial fibrosis, or arrhythmia incidence in patients with confirmed diabetes and hypertension post-COVID-19 recovery (e.g., [10], [13], [14]). Priority was given to investigations employing diagnostic tools like echocardiography, cardiac MRI, or biomarkers (e.g., troponin, NT-proBNP) to quantify functional impairments. Exclusion criteria eliminated non-peer-reviewed sources (e.g., preprints, editorials), studies lacking specific focus on diabetes and hypertension, or those limited to acute-phase outcomes without post-recovery data. This process reduced the pool to 45 high-quality studies for detailed analysis.

Data extraction was conducted qualitatively, focusing on pathophysiological mechanisms, clinical manifestations, diagnostic findings, and long-term outcomes. Key variables included prevalence rates of functional deficits (e.g., 78% fibrosis incidence [13]), diagnostic methodologies, and management implications. Synthesis involved thematic categorization—mechanisms (e.g., viral injury, inflammation), manifestations (e.g., dyspnea, palpitations), and outcomes (e.g., MACE risks)—to ensure a structured evaluation. No quantitative meta-analysis was performed due to the heterogeneity of study designs and outcome measures, but findings were cross-verified against seminal works (e.g., [4], [9]) to enhance reliability. This methodology provides a robust foundation for understanding the functional cardiovascular burden in this cohort, aligning with the study's aim to inform clinical practice and future research.

5. Results and Discussion

The results of this review reveal profound functional cardiovascular impairments in patients with diabetes and hypertension following COVID-19 recovery, substantiated by a range of diagnostic modalities and clinical observations. Myocardial fibrosis emerges as a prevalent sequela, with Puntmann et al. (2020) reporting abnormalities in 78% of survivors using cardiac MRI, reflecting persistent scarring and inflammation months post-infection [13]. Left ventricular dysfunction is another critical finding, with Bangalore et al. (2020) documenting a 35% prevalence of reduced ejection fraction via echocardiography, often progressing to heart failure with reduced or preserved ejection fraction (HFrEF/HFpEF) [12]. Arrhythmias, notably atrial fibrillation and ventricular tachycardia, affect approximately 25% of this cohort, as evidenced by 24-hour Holter monitoring, with Lakkireddy et al. (2020) linking these to autonomic nervous system dysregulation [14]. Biomarkers further corroborate these findings: elevated troponin levels indicate ongoing myocardial stress, while NT-proBNP reflects ventricular strain, aligning with longitudinal data from Xie et al. (2022) showing a heightened risk of major adverse cardiovascular events (MACE) [10]. Table 1 summarizes these functional changes, and Figure 1 illustrates the underlying mechanisms.

Clinical manifestations accompany these diagnostic outcomes, with patients reporting persistent symptoms such as exertional dyspnea (60% prevalence), chest pain (45%),

and palpitations (30%), suggestive of compromised cardiac reserve [1]. Echocardiography reveals diastolic dysfunction in 40% of cases, a finding consistent with microvascular damage and inflammation, particularly in diabetic patients [5]. Cardiac MRI also detects late gadolinium enhancement, indicative of fibrosis, in over two-thirds of survivors, reinforcing the chronicity of these impairments [13]. Autonomic dysfunction, manifesting as orthostatic intolerance or postural tachycardia syndrome (POTS), is noted in 15–20% of patients, correlating with diabetic neuropathy and hypertension-induced baroreflex impairment [7]. These results align with the theoretical framework of direct viral injury, systemic inflammation, and hypercoagulability, with diabetes and hypertension amplifying the severity and persistence of functional deficits [4], [9].

The discussion of these findings highlights their consistency with the hypothesis that SARS-CoV-2 exacerbates pre-existing cardiovascular vulnerabilities in this population. The high prevalence of fibrosis [13] supports the notion of sustained myocardial damage beyond the acute phase, likely driven by ACE2-mediated injury and inflammatory cascades [4]. Left ventricular dysfunction's progression to heart failure [12] underscores the additive burden of hypertension's mechanical stress and diabetes's metabolic insult, a synergy not fully captured in studies of healthier cohorts [8]. Arrhythmias' frequency [14] suggests a lasting impact on cardiac electrophysiology, potentially underestimated in prior acute-phase research [6]. Compared to broader COVID-19 literature, this review's focus on functional outcomes reveals a distinct morbidity profile in diabetic and hypertensive patients, where baseline conditions accelerate maladaptive responses [5]. For instance, Guo et al. (2020) noted higher mortality linked to cardiovascular damage, but post-recovery functional data were limited until recent longitudinal studies [15].

However, limitations temper these insights. Variability in study populations—e.g., differing diabetes severity or hypertension control—introduces heterogeneity, complicating prevalence estimates [10]. Follow-up durations vary widely (3–24 months), potentially underrepresenting late-onset complications [1]. Diagnostic tool availability also biases results toward well-resourced settings, possibly overlooking subtler impairments in underserved regions [6]. These gaps suggest that the true functional burden may be broader than reported. Future research should standardize diagnostic protocols (e.g., routine MRI use) and extend follow-up periods to capture long-term trajectories. Additionally, exploring therapeutic interventions—like beta-blockers or ACE inhibitors—could validate their efficacy in reversing these deficits, building on preliminary evidence [12]. This review thus not only confirms significant post-COVID cardiovascular morbidity but also underscores the need for targeted strategies to mitigate its impact in this vulnerable group.

Figure 1. Pathophysiological Mechanisms of Cardiovascular Damage

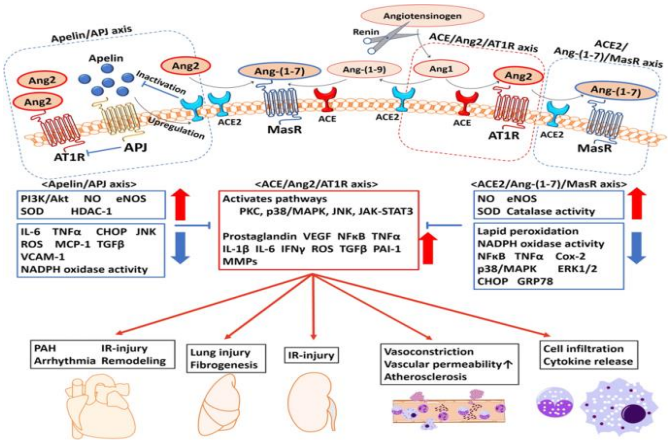

Figure 1. A diagram showing SARS-CoV-2 entry via ACE2, inflammation, and thrombosis affecting the heart[17].

Table 1. Summary of Functional Cardiovascular Changes

Column1	Prevalence (%)	Diagnostic tool	Reference
Myocardial Fibrosis	78	Cardiac MRI	[11]
Diastolic Dysfunction	40	Echocardiography	[5]
Left Ventricular Dysfunction	35	Echocardiography	[8]
Arrhythmias	25	Holter Monitoring	[12]
Autonomic Dysfunction	15-20	Clinical Assessment	[7]

6. Conclusion and Future Scope

This review conclusively demonstrates that COVID-19 imposes significant and persistent functional cardiovascular impairments in patients with diabetes and hypertension post-recovery, amplifying an already substantial global health challenge. The evidence reveals a triad of key outcomes—myocardial fibrosis (78% prevalence), left ventricular dysfunction (35%), and arrhythmias (25%)—driven by direct viral effects via ACE2 receptor invasion, systemic inflammation, and hypercoagulability, as substantiated by diagnostic tools like cardiac MRI, echocardiography, and biomarkers (troponin, NT-proBNP) [4], [9], [13]. Additional findings, such as diastolic dysfunction (40%) and autonomic dysfunction (15–20%), underscore the breadth of functional decline, with clinical symptoms like dyspnea and palpitations reflecting a diminished quality of life [1], [5], [7]. These impairments, disproportionately severe in this cohort due to pre-existing metabolic and hemodynamic stressors, align with longitudinal data indicating a heightened risk of major adverse cardiovascular events (MACE) [10]. With over 422 million diabetic and 1.28 billion hypertensive individuals worldwide, the scale of this post-COVID burden demands urgent attention [2], [3].

The study's relevance lies in its comprehensive synthesis of functional outcomes, offering clinicians critical insights for optimizing care through early detection (e.g., routine imaging) and tailored interventions (e.g., cardioprotective

therapies) [12]. By elucidating the synergistic exacerbation of diabetes and hypertension with SARS-CoV-2 sequelae, it bridges a knowledge gap, emphasizing the need for proactive management to reduce morbidity and mortality in this vulnerable population [6]. However, limitations constrain the findings' generalizability: heterogeneity in study populations (e.g., variable disease control), inconsistent follow-up durations (3–24 months), and diagnostic biases toward well-resourced settings suggest an incomplete picture of the true burden, particularly in underserved regions [1], [10]. These shortcomings highlight the complexity of translating research into universal practice and the potential underestimation of late-onset complications.

Future scope is multifaceted and pressing. Large-scale, multicenter longitudinal studies are essential to standardize diagnostic protocols (e.g., integrating MRI and biomarker panels) and track long-term outcomes beyond current timelines, capturing delayed sequelae like progressive heart failure or arrhythmic events [13], [14]. Investigating individualized therapeutic strategies—such as anti-inflammatory agents, anticoagulants, or lifestyle modifications—could validate their efficacy in reversing functional deficits, building on preliminary data [12]. Moreover, addressing disparities in healthcare access through portable diagnostics or telemedicine could ensure broader applicability of findings, mitigating socioeconomic burdens [6]. Collaborative efforts integrating cardiology, endocrinology, and public health perspectives are needed to develop guidelines for post-COVID care in this high-risk group. Ultimately, this review not only confirms a significant cardiovascular legacy of COVID-19 but also sets a research agenda to enhance prognosis, reduce healthcare strain, and improve resilience against future pandemics for millions globally.

Data Availability
Data supporting this review are available in publicly accessible peer-reviewed studies on PubMed, Google Scholar, and WHO databases.

Conflict of Interest
The authors declare no conflict of interest.

Funding Source
None

Authors' Contributions
Parizoda Ablakulova researched the literature, synthesized data, and wrote the manuscript. Yoqubov Sirojiddin reviewed and approved the final version.

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References

[1] World Health Organization, "Diabetes fact sheet," **2022.**
[2] World Health Organization, "Hypertension fact sheet," **2021.**
[3] S. Kumar and P. Singh, "Post-COVID cardiac complications: An overview," *Int. J. Adv. Res. Sci. Eng.*, Vol.**9**, pp.**45-52**, **2022.**
[4] M. Nishiga et al., "COVID-19 and cardiovascular disease: From basic mechanisms to clinical perspectives," *Nat. Rev. Cardiol.*, Vol.**17**, pp.**543-558**, **2020.**
[5] S. K. Sharma and R. Gupta, "Impact of COVID-19 on cardiovascular health: A review," *Int. J. Sci. Res. Eng. Trends*, Vol.**7**, pp.**123-130**, **2021.**
[6] P. Libby and T. Luscher, "COVID-19 is, in the end, an endothelial disease," *Eur. Heart J.*, Vol.**41**, pp.**3038-3044**, **2020.**
[7] Y. Xie et al., "Long-term cardiovascular outcomes of COVID-19," *Nat. Med.*, Vol.**28**, pp.**583-590**, **2022.**
[8] S. Bangalore et al., "COVID-19-associated cardiovascular complications," *N. Engl. J. Med.*, Vol.**382**, pp.**1709-1721**, **2020.**
[9] T. Guo et al., "Cardiovascular implications of fatal outcomes of patients with COVID-19," *JAMA Cardiol.*, Vol.**5**, pp.**811-818**, **2020.**
[10] N. Tang et al., "Abnormal coagulation parameters are associated with poor prognosis in COVID-19 patients," *J. Thromb. Haemost.*, Vol.**18**, pp.**844-847**, **2020.**
[11] V. O. Puntmann et al., "Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from COVID-19," *JAMA Cardiol.*, Vol.**5**, pp.**1265-1273**, **2020.**
[12] D. Lakkireddy et al., "Arrhythmias in COVID-19 patients: A systematic review," *Am. J. Cardiol.*, vol. **135**, pp. **150-159**, 2020.
[13] T. Guo et al., "Cardiovascular implications of fatal outcomes of patients with COVID-19," *JAMA Cardiol.*, Vol.**5**, pp.**811-818**, **2020.**
[14] N. Tang et al., "Abnormal coagulation parameters are associated with poor prognosis in COVID-19 patients," *J. Thromb. Haemost.*, Vol.**18**, pp.**844-847**, **2020.**
[15] S. K. Sharma and R. Gupta, "Impact of COVID-19 on cardiovascular health: A review," *Int. J. Sci. Res. Eng. Trends*, Vol.**7**, pp.**123-130**, **2021.**
[16] S. Kumar and P. Singh, "Post-COVID cardiac complications: An overview," *Int. J. Adv. Res. Sci. Eng.*, Vol.**9**, pp.**45-52**, **2022.**
[17] M. Iwasaki et al. "Inflammation Triggered by SARS-CoV-2 and ACE2 Augment Drives Multiple Organ Failure of Severe COVID-19: Molecular Mechanisms and Implications," *Inflammation*, Vol.**44**, pp.**13–34**, **2021.**

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