

Acceleration of Coronary Artery Disease in Patients with Previous COVID-19 Infections

Original
Article

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ABSTRACT

Background: The pandemic COVID-19 has raised concerns about its impact on cardiovascular (CAV) health.

Aim: To evaluate the acceleration of coronary artery disease (CAD) in individuals with previous COVID-19 infections.

Methods: This retrospective cohort study examined 100 CAD patients at AL-Azhar University Hospitals from July 2023-2024. Participants underwent comprehensive evaluations including history, clinical examination, ECG, echocardiography, and coronary angiography. Primary outcome: incidence of CAD in COVID-19 patients. Secondary outcomes: CAD progression, inflammatory markers, endothelial function, and CAV events.

Results: The prevalence of CAD was much greater in individuals with previous COVID-19 (57.14%) than those without (30.56%, $p=0.014$), with increased arrhythmias (17.86% vs. 2.78%, $p=0.017$), and coronary artery calcium scores were significantly high in the COVID-19 group ($p=0.030$). There were no significant differences in hematological and biochemical parameters between both groups. Elevated inflammatory markers were noted but not statistically significant.

Conclusions: This research suggests an acceleration of CAD in patients with previous COVID-19 infections, characterized by increased prevalence, greater plaque presence, and elevated coronary artery calcium scores. These results underscore the importance of CAV monitoring in COVID-19 survivors.

Key Words: Arrhythmia, COVID-19, Coronary Artery Disease, Inflammation, Plaque.

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INTRODUCTION

The pandemic COVID-19, resulting from the SARS-CoV-2 virus, has become a substantial issue in public health, with its ability to impact multiple organ systems, including the cardiovascular (CAV) system^[1]. Emerging evidence suggests that COVID-19 may exacerbate existing CAV conditions, particularly coronary artery disease (CAD), due to various mechanisms such as inflammation, endothelial dysfunction, and thrombosis^[2, 3].

People who already have heart problems, like CAD, are more likely to develop serious complications from COVID-19, primarily due to the burden of comorbidities and the physiological stress induced by the illness^[4]. Autopsy studies and clinical investigations have revealed widespread myocardial injury, endothelial dysfunction, and thromboembolic outcomes in individuals infected with COVID-19, which may contribute to the accelerated progression of CAD^[5, 6].

Furthermore, long-term CAV complications, known as "prolonged COVID-19," have been observed, with patients reporting persistent symptoms such as exertional dyspnea, fatigue, and chest pain well after the acute phase of the infection has subsided^[7]. This phenomenon has prompted increased research efforts to understand the underlying pathophysiological mechanisms and their impact on CAV health.

The intersection of COVID-19 and CAV disease has become a focal point for researchers, with studies exploring the specific risk factors, potential mechanisms of acceleration, and strategies for management and treatment^[2, 8]. Factors such as immune system dysregulation, cytokine storms, and hypercoagulability have been identified as playing crucial roles in worsening CAV outcomes and accelerating CAD in those recovering from COVID-19^[9, 10].

AIM OF THE STUDY

To evaluate the acceleration of CAD in individuals with previous COVID-19 infections.

METHODS

Subjects in this retrospective cohort study included 100 individuals who were aged 18 years or older of both sexes, diagnosed with CAD based on clinical, imaging, or biochemical criteria at AL-Azhar University Hospitals, Egypt, from July 2023 to July 2024.

ETHICAL CONSIDERATIONS

The research received institutional ethical committee approval (RESEARCH/AZ.AST./CAR032/4/243/6/2023). All individuals who took part in the study provided their written informed consent.

CAD^[11] was defined by the presence of at least one epicardial coronary vessel with >75% stenosis on coronary angiography, a history of acute coronary syndrome, or previous coronary revascularization (either percutaneous transluminal coronary angioplasty or coronary artery bypass grafting). Individuals with other significant comorbidities that could independently accelerate CAD and pregnant women were excluded from the study.

The PCR was considered the gold standard for COVID-19 diagnosis because of its accuracy and reliability^[12].

A comprehensive methodology was employed, beginning with a thorough history taking. This included demographic information, risk factors for CAD (such as diabetes mellitus, hypertension, smoking history, and family history of CAD), prior CAD history, and COVID-19 history, including severity, treatment, and recovery within the past 6-12 months. A complete clinical examination was performed, and routine laboratory investigations were conducted.

Standard 12-lead electrocardiograms (ECGs) were obtained for all patients. The ECGs, captured during breathing exercises, were used to autonomously fine-tune the system's settings. Multiple distinct reconstructions were conducted using various cardiac stages, where automatic provision was not possible, by selecting

the optimal reconstruction stage. To reduce radiation exposure, we migrated the ECG and synchronized data reconstruction with the ECG signal using a backward pass technique. Subsequent investigation was conducted on the reconstruction period that exhibited the lowest amount of motion artifact.

Transthoracic echocardiography and coronary angiography were performed as part of the diagnostic workup.

The primary outcome of the study was the prevalence of COVID-19 patients who suffered from CAD. Secondary outcomes included the progression of CAD, defined by an increase in plaque volume or stenosis severity on imaging, elevation of biomarkers indicating worsening CAD, and new CAV events. Additionally, changes in inflammatory markers and endothelial function, as well as the frequency of negative CAV incidents, were monitored.

Sample size calculation:

To determine the appropriate sample size, researchers used the statistical program EpI-Info 2002, which was developed by the WHO and the CDC. Factors such as these were used to determine the sample size: The prevalence of COVID-19 individuals with CAD was 50.9% with a 95% confidence level and $\pm 10\%$ confidence limit, as reported in a prior study^[3]. Dropout was mitigated by adding four scenarios. This is why we gathered 100 cases.

Statistical analysis

Statistical analysis was done by SPSS v27 (IBM®, Armonk, NY, USA). The normality of the data distribution was assessed using the Shapiro-Wilks test and histograms. The quantitative parametric data was examined using an unpaired student t-test and was provided as mean and standard deviation (SD). The Chi-square test was used to assess the qualitative variables, which were reported as percentages and frequencies. It was deemed statistically significant if the two-tailed *P value* was less than 0.05.

RESULTS

Age, sex, DM, hypertension, smoking, and family history of CAD were insignificantly different between individuals with previous COVID-19 and those without COVID-19 (Table 1).

Table 1: Demographic data and history of risk factors of the studied groups.

		With previous COVID-19 (n=28)	Without COVID-19 (n=72)	P
Age (years)		47.04 ± 14.73	51.63 ± 15.72	0.185
Sex	Male	16 (57.14%)	44 (61.11%)	0.716
	Female	12 (42.86%)	28 (38.89%)	
History of risk factors	DM	8 (28.57%)	19 (26.39%)	0.825
	Hypertension	12 (42.86%)	27 (37.5%)	0.622
	Smoking	6 (21.43%)	17 (23.61%)	0.816
	Family history of CAD	6 (21.43%)	9 (12.5%)	0.262

Data are presented as mean ± SD or frequency (%); DM: Diabetes mellitus; CAD: Coronary artery disease.

Hemoglobin, platelets, white blood cells, neutrophil, creatinine, urea, alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, CRP, Interleukin-6

and procalcitonin were insignificantly different between individuals with previous COVID-19 and those without COVID-19 (Table 2).

Table 2: Laboratory investigation of the studied groups.

	With previous COVID-19 (n=28)	Without COVID-19 (n=72)	P
Hemoglobin (mg/dl)	10.36±0.95	10.81±1.16	0.070
Platelet (10 ⁹ /L)	248.36±31.52	269.61±69.29	0.122
White blood cells (10 ⁹ /L)	6.32±0.72	6.4±1.11	0.721
Neutrophil (10 ⁹ /L)	5.18±0.9	5.26±0.99	0.690
Creatinine (mg/dl)	0.83±0.23	0.92±0.24	0.079
Urea (mg/dL)	14.71±4.02	15.58±4.55	0.378
Alanine aminotransferase (U/L)	33.75±9.88	30.63±9.82	0.157
Aspartate aminotransferase (U/L)	31.14±7.64	29.26±6.95	0.241
Alkaline phosphatase (U/L)	73.04±9.88	77.78±12.72	0.079
CRP (mg/dl)	3.78±1.08	3.6±0.98	0.408
Interleukin-6 (pg/mL)	8.39±3.22	7.58±2.83	0.220
Procalcitonin (ng/mL)	0.16±0.1	0.14±0.04	0.109

Data are presented as mean ± SD; *Significant as *P* value ≤0.05; CRP: C reactive protein.

The prevalence of CAD was significantly higher in patients with previous COVID-19 16 (57.14%) than in those without COVID-19 22 (30.56%) (*P*=0.014). Plaque was significantly higher in individuals with previous COVID-19 than in those without COVID-19 (*P*=0.035).

Plaque type was insignificantly different between patients with previous COVID-19 and individuals without COVID-19. CAC score was significantly higher in individuals with previous COVID-19 than those without COVID-19 (*P*=0.030) (Table 3).

Table 3: Prevalence of CAD, plaque, plaque type, and CAC score of the studied groups.

	With previous COVID-19 (n=28)	Without COVID-19 (n=72)	P value	RR (95%CI)
Prevalence of CAD	16 (57.14%)	22 (30.56%)	0.014*	1.87(1.16:3)
Plaque	14 (50%)	20 (27.78%)	0.035*	1.8(1.06:3.04)
Plaque type	Soft Calcific Mix	7 (9.72%) 11 (15.28%) 2 (2.78%)	0.132	0.69(0.51:0.94)
CAC score	0(0 - 41.75)	0(0 - 9.25)	0.030*	---

Data are presented as frequency (%); *Significant as *P* value ≤0.05; CAC: Coronary artery calcium.

Arrhythmia was significantly higher in individuals with previous COVID-19 than in those without COVID-19 (*P*=0.017). Acute myocardial injury, heart failure, and

cardiac arrest were insignificantly different between patients with previous COVID-19 and patients without COVID-19 (Table 4).

Table 4: Adverse CAV events of the studied groups

	With previous COVID-19 (n=28)	Without COVID-19 (n=72)	P value	RR (95%CI)
Acute myocardial injury	2 (7.14%)	1 (1.39%)	0.188	5.14(0.49:54.49)
Arrhythmia	5 (17.86%)	2 (2.78%)	0.017*	6.43(1.32:31.23)
Heart failure	2 (7.14%)	0 (0%)	0.076	---
Cardiac arrest	1 (3.57%)	0 (0%)	0.280	----

Data are presented as frequency (%); *Significant as *P* value ≤0.05.

DISCUSSION

COVID-19 has had profound implications for CAV health, with emerging evidence suggesting a complex interplay between SARS-CoV-2 infection and the progression of CAD^[1-3].

Our analysis revealed no significant differences between groups across various hematological and biochemical parameters. Notably, hemoglobin levels were 10.36 ± 0.95 mg/dl in the COVID-19 group and 10.81 ± 1.16 mg/dl in the non-COVID-19 group (*p* = 0.070). Platelet counts were 248.36 ± 31.52 × 10⁹/L and 269.61 ± 69.29 × 10⁹/L in the COVID-19 and non-COVID-19 groups, respectively (*p* = 0.122). These findings contrast with some previous studies that reported significant alterations in blood cell parameters among COVID-19 patients.

For instance, *Wang et al.*^[13] observed a transient increase in platelet counts following COVID-19 infection, with peak levels occurring on Day 14. Our study, conducted at a later time point post-infection, did not detect such differences, suggesting that any acute changes in platelet counts may normalize over time. Similarly, Zinellu and Mangoni^[14] reported associations between altered blood cell parameters and COVID-19 progression and mortality. However, their meta-analysis focused on parameters within 48 hours of admission, whereas our study examined patients at a later stage post-infection.

The lack of significant differences in inflammatory markers (CRP, interleukin-6, and procalcitonin) between our COVID-19 and non-COVID-19 groups also diverges from some previous findings. *Elshazl et al.*^[15] reported that elevated levels of these markers were associated with higher odds of progression to severe COVID-19. This discrepancy may be attributed to the timing of our assessments, potentially after the acute inflammatory phase had subsided.

Our study found a significantly higher prevalence of CAD in the COVID-19 group (57.14%) than in the non-COVID-19 group (30.56%) (*p* = 0.014, RR = 1.87, 95% CI: 1.16-3). Similarly, the presence of plaque was more frequent in the COVID-19 group (50%) than in the non-COVID-19 group (27.78%) (*p* = 0.035, RR = 1.8, 95% CI: 1.06-3.04). These findings align with emerging evidence suggesting a potential link between COVID-19 and accelerated atherosclerosis.

Erdoğan et al.^[16] reported similar results in their retrospective cohort study, finding that a history of COVID-19 was associated with a higher likelihood of atherosclerotic soft plaque (OR: 2.05, 95% CI: 1.32-3.11), mixed plaque (OR: 2.48, 95% CI: 1.39-4.43), and high-risk plaque (OR: 2.75, 95% CI: 1.98-3.84). Our study's findings of increased plaque presence in the COVID-19 group corroborate these observations, suggesting that COVID-19 may indeed accelerate atherosclerotic processes. The

trend toward a higher prevalence of soft plaques (17.86% vs. 9.72%, $p = 0.132$) and mixed plaques (10.71% vs. 2.78%) in our COVID-19 group, although not statistically significant, aligns with the observations of *Erdoğan et al.*^[16].

Our study found that the median CAC score was significantly higher in the COVID-19 group [0 (0 - 41.75)] than in the non-COVID-19 group [0 (0 - 9.25)] ($p = 0.030$). This finding is particularly noteworthy and consistent with several recent studies examining the relationship between COVID-19 and coronary calcification.

Delfiner et al.^[17] reported higher mean CAC scores in non-survivors (406 AU) than survivors (319 AU) of COVID-19 ($p = 0.02$). While our study did not specifically examine mortality outcomes, the higher CAC scores in our COVID-19 group suggest a potential link between the infection and accelerated coronary calcification.

Similarly, *Braun et al.*^[18] found that individuals who died during their study period had significantly higher median CAC scores (592 AU) than survivors (142 AU) ($p = 0.0001$). Their findings indicated that a CAC score >143 AU was associated with a 3.82 times higher relative risk of mortality (95% CI 1.60 – 9.11; $p = 0.0025$).

Our results also align with those of *Fovino et al.*^[19], who reported that a CAC score ≥ 400 was associated with higher in-hospital mortality and more frequent myocardial infarction in COVID-19 individuals. While our study did not stratify patients based on specific CAC score thresholds, the overall higher CAC scores in the COVID-19 group suggest an increased CAV risk profile.

Our study found that individuals with previous COVID-19 infections demonstrated a significantly higher incidence of arrhythmia (17.86%) than those without COVID-19 (2.78%) ($p = 0.017$, RR = 6.43, 95% CI: 1.32-31.23). This finding is consistent with several other studies that have reported increased arrhythmia incidence in COVID-19 individuals.

Coromilas et al.^[20] conducted a large international study of 4526 patients, finding that 18.2% of COVID-19 patients developed arrhythmias. Similar to our findings, they reported that the majority (81.8%) of arrhythmias were atrial. Our study's higher relative risk (RR = 6.43) for arrhythmia in COVID-19 individuals than in non-COVID-19 individuals underscores the potential long-term arrhythmogenic effects of the infection.

Liao et al.^[21] conducted a meta-analysis reporting an overall arrhythmia incidence of 16.8% (95% CI: 12.8–21.2%) in COVID-19 patients, which closely aligns with our finding of 17.86%. They also noted a higher mortality

rate (20.3%) in COVID-19 patients who developed arrhythmias, highlighting the clinical significance of this complication.

Our observation of increased acute myocardial injury in the COVID-19 group (7.14% vs. 1.39%), although not statistically significant ($p = 0.188$), is consistent with the findings of *Wen et al.*^[22]. Their meta-analysis showed that arrhythmia was significantly associated with severely ill COVID-19 patients, with a pooled odds ratio of 17.97 (95% CI 11.30-28.55, $p < 0.00001$).

The occurrence of heart failure and cardiac arrest only in our COVID-19 group, while not reaching statistical significance, aligns with the observations of *Degano et al.*^[23]. Their large-scale study found an increased risk of heart failure in females (HR 1.73, 95% CI: 1.23-2.43) and atrial fibrillation/flutter/tachycardia in males (HR 1.61, 95% CI: 1.10-2.36) during the period from 4 months to the end of follow-up post-COVID-19.

The mechanisms underlying the increased arrhythmia risk in COVID-19 patients are likely multifactorial. *Zhan et al.*^[24] proposed several potential pathways, including viral infection-induced changes in angiotensin-converting enzyme 2 expression, myocarditis, cytokine storm, cardiac injury, electrophysiological effects, hypoxemia, and electrolyte abnormalities. Our study's findings of increased CAD prevalence and higher CAC scores in COVID-19 patients may also contribute to the elevated arrhythmia risk, as underlying coronary disease is a known risk factor for arrhythmias.

This study has several limitations. The sample size was relatively small, which may limit the generalizability of the findings. The retrospective nature of the study introduces potential biases in data collection and interpretation. Single-center study, which may not represent diverse populations. The follow-up period of 6-12 months may be insufficient to fully assess the long-term impacts of COVID-19 on CAD progression. Additionally, the study did not account for potential confounding factors such as lifestyle changes, medication adherence, or variations in COVID-19 treatment protocols, which could influence CAD outcomes.

CONCLUSIONS

Our study provides important insights into the potential long-term CAV effects of COVID-19 infection. The observed increases in CAD prevalence, CAC scores, and arrhythmia incidence in patients with previous COVID-19 infection suggest that the virus may have lasting impacts on CAV health. These findings underscore the importance of long-term CAV monitoring and risk management in COVID-19 survivors.

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Nil

CONFLICTS OF INTEREST

The authors have no financial or proprietary interests in any material discussed in this article.

AUTHORS' CONTRIBUTIONS

SMH conceived and supervised the study, responsible for data collection. SMH analyzed and interpreted the data and provided comments on the manuscript at various stages of development.

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تسارع الإصابة بمرض الشريان التاجي في المرضى الذين تعرضوا لعدوى سابقة بفيروس كوفيد-١٩

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المقدمة: أثار جائحة كوفيد-١٩ مخاوف بشأن تأثيره على صحة القلب والأوعية الدموية.

هدف الدراسة: تقييم تسارع مرض الشريان التاجي لدى الأفراد الذين أصيبوا سابقاً بعدوى كوفيد-١٩.

الطرق: أجريت هذه الدراسة الاستيعادية على ١٠٠ مريض يعانون من مرض الشريان التاجي في مستشفيات جامعة الأزهر خلال الفترة من يوليو ٢٠٢٣ إلى ٢٠٢٤. خضع المشاركون لتقييمات شاملة شملت التاريخ المرضي، والفحص السريري، وتخطيط كهربية القلب، وتخطيط صدى القلب، وتصوير الأوعية التاجية.

النتيجة الأولية: حدوث مرض الشريان التاجي لدى مرضى كوفيد-١٩.

النتائج الثانوية: تطور مرض الشريان التاجي، والعلامات الالتهابية، ووظيفة بطانة الأوعية الدموية، وأحداث الشريان التاجي.

النتائج: كان معدل انتشار مرض الشريان التاجي أعلى بكثير لدى الأفراد المصابين سابقاً بكوفيد-١٩ (٥٧,١٤٪) مقارنةً بغير المصابين (٣٠,٥٦٪، قيمة الاحتمال = ٠,٠١٤)، مع زيادة في عدم انتظام ضربات القلب (١٧,٨٦٪ مقابل ٢,٧٨٪، قيمة الاحتمال = ٠,٠١٧)، وكانت درجات كالسيوم الشريان التاجي مرتفعة بشكل ملحوظ لدى مجموعة كوفيد-١٩ (قيمة الاحتمال = ٠,٠٣٠). لم يكن هناك فروق ذو دلالة إحصائية في المعايير الدموية والكيميائية الحيوية بين المجموعتين. ولوحظت علامات التهابية مرتفعة، ولكنها غير ذات دلالة إحصائية.

الاستنتاجات: يشير هذا البحث إلى تسارع في مرض الشريان التاجي لدى المرضى الذين سبق لهم الإصابة بكوفيد-١٩، والذي يتميز بزيادة انتشاره، ووجود لويحات دهنية أكبر، وارتفاع في نسبة الكالسيوم في الشرايين التاجية. تؤكد هذه النتائج على أهمية مراقبة الشرايين التاجية لدى الناجين من كوفيد-١٩.