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Original Article

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History of COVID-19 as a Risk Factor for Cardiac Arrhythmias: A Case-Control Study

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Abstract

Background: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was responsible for the coronavirus disease 2019 (COVID-19) pandemic and generated high morbidity and mortality rates worldwide, as well as several sequelae that persist and need to be evaluated. The aim of this study was to evaluate the association between a history of COVID-19 infection and the occurrence of cardiac arrhythmias in outpatients from a private clinic in Arequipa.

Methods: We conducted a retrospective, analytical, unmatched case-control study in a private cardiology clinic in Arequipa, Peru. A total of 252 adult patients who underwent 24-h Holter monitoring between October and December 2023 were included. Cases were defined as patients with documented cardiac arrhythmias; controls had no arrhythmic findings. The main exposure was a confirmed history of COVID-19. Age, sex, and additional Holter findings were also analyzed. Logistic regression was used to estimate crude and adjusted odds ratios (ORs) with 95% confidence intervals (CIs), adjusting for age and sex.

Results: Of the total sample, 68 patients were classified as cases and 184 as controls. A history of COVID-19 was more frequent among cases (70.6%) than among controls (50.5%) (P = 0.004). In unadjusted analysis, patients with prior COVID-19 had more than twice the odds of presenting arrhythmias (OR: 2.35; 95% CI: 1.29 - 4.26; P = 0.005). After adjusting for age and sex, the association remained statistically significant (OR: 2.12; 95% CI: 1.10 - 4.11; P = 0.025).

Conclusion: A prior history of COVID-19 was significantly associated with increased odds of cardiac arrhythmias. These findings highlight the importance of structured cardiac evaluation in patients with prior SARS-CoV-2 infection.

Keywords: COVID-19; Sequelae; Cardiac arrhythmia; Holter

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Introduction

Since its emergence in late 2020, the coronavirus disease 2019 (COVID-19) pandemic has been devastating in Latin America, with high morbidity and mortality rates [1]. In addition to its acute effects, this disease has resulted in multiple sequelae [2, 3] and has been linked to new hematological [4], pulmonary, cardiovascular [5, 6], renal, endocrine, gastrointestinal, dermatological, neuropsychiatric diseases, among others [7, 8].

The population has been affected by a growing number of cardiovascular complications among patients experiencing post-acute sequelae of COVID-19 [9]. Myocardial damage may be caused by direct viral involvement with a local inflammatory response or by an exaggerated systemic reaction characterized by cytokine release and a prothrombotic state. Along with vascular involvement, these factors can trigger thrombotic and ischemic events due to microvascular damage or destabilization of preexisting atheroma plaques [5].

Currently, there is an increasing number of myocarditis, arrhythmias, and myocardial injury cases, underscoring the urgent need for further research [10-12]. COVID-19 infection can worsen cardiovascular health, particularly in vulnerable groups such as pregnant women or patients with pre-existing cardiovascular disease [13]. Several mechanisms have been proposed to explain how severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) affects the heart, including direct viral cytotoxicity and an excessive immune response [14]. It has also been studied that the virus enters cells through angi-

otensin-converting enzyme 2 (ACE2) receptors, causing direct myocardial injury [15]. Another theory suggests that ACE2 downregulation prevents the cardioprotective effects of angiotensin 1-7, triggering an inflammatory cascade with tumor necrosis factor-alpha (TNF-α) production that may ultimately damage cardiomyocytes [16]. Likewise, the myocardial inflammation caused by SARS-CoV-2 may trigger cardiac arrhythmias through electrophysiological and structural remodeling or ion channel dysfunction [17]. The aim of this study was to evaluate the association between a history of COVID-19 infection and the occurrence of cardiac arrhythmias in outpatients from a private clinic in Arequipa.

Materials and Methods

Study design

An observational, analytical, and retrospective unmatched case-control study was conducted, based on the review of medical records and 24-h Holter cardiac monitoring reports from a private cardiology clinic in the city of Arequipa, Peru.

Population, sample, and sampling

The study population consisted of 252 adult patients who attended outpatient cardiology consultations during the specified period. All patients had been referred for 24-h Holter monitoring due to symptoms suggestive of cardiac arrhythmias, including palpitations, dizziness, syncope, atypical chest pain, or a prior history of cardiovascular disease. The arrhythmic findings were verified and confirmed by a qualified cardiologist. A non-probabilistic convenience sampling method was used, including all patients who met the eligibility criteria.

Eligibility criteria

Cases were defined as patients aged 18 years or older, of either sex, with documented evidence of cardiac arrhythmia in their Holter reports, based on established electrocardiographic criteria. All arrhythmic findings were verified and confirmed by a qualified cardiologist. Controls were defined as patients whose Holter reports showed no evidence of arrhythmia, according to the interpretation of the same cardiologist. Patients were excluded from the study if they had a permanent pacemaker or an implantable cardioverter-defibrillator (ICD), due to the inability of Holter devices to accurately assess intrinsic cardiac activity in such cases. Additionally, those with incomplete or technically invalid Holter data, or with missing essential clinical information, were also excluded.

Variables, instruments, and procedures

The dependent variable was the diagnosis of cardiac arrhythmia, based on findings reported in the 24-h Holter recording. Standard electrocardiographic criteria were used to classify arrhythmic events. Holter monitoring was performed using a three-channel device, and the recordings were analyzed using manufacturer-validated automated software, followed by manual verification by an experienced cardiologist. Only complete and clinically validated reports were included.

The exposure variable was a history of confirmed COVID-19 infection, defined exclusively through documentation in the patients' medical records, based on positive results of either molecular (reverse transcription polymerase chain reaction (RT-PCR)) or antigen tests recorded during the clinical course.

In addition, the following covariates were obtained from the clinical records and Holter reports: age (years), sex (male/female), and additional Holter findings, including the number and type of ventricular and supraventricular events, as described above. Ventricular extrasystoles (VEs) were defined as premature beats with wide QRS complexes (≥ 120 ms) and no preceding P wave [18]. Supraventricular extrasystoles (SVEs) were defined as premature beats with narrow QRS complexes (< 120 ms), preceded by an abnormal P wave of different morphology from the baseline rhythm [18]. Ventricular tachycardia (VT) was defined as three or more consecutive ventricular beats with a heart rate ≥ 100 beats per minute (bpm) and wide QRS complexes and further classified as sustained (> 30 s) or non-sustained (< 30 s) [18]. Comorbidities and clinical indications for Holter monitoring were not consistently available and were therefore not included in the analysis.

Statistical analysis

The information from the cardiac Holter was extracted into a database of the Microsoft Excel 2019 program to be subsequently verified by a cardiologist. Then, it was exported to the statistical program STATA v16.0 (StataCorp, TX) for their respective analyses. First, in the univariate analysis, qualitative variables were reported using frequencies and percentages. Quantitative variables were reported using measures of central tendency and dispersion after evaluation of their normality. Second, bivariate analyses were performed according to the presence of a history of COVID-19. When comparing categorical variables, the Chi-square test or Fisher's exact test was used. The Student's *t*-test was used to compare numerical variables according to the groups formed.

Additionally, binary logistic regression was performed to estimate crude and adjusted odds ratios (ORs) with 95% confidence intervals (CIs), evaluating the association between a history of COVID-19 and the presence of arrhythmia. The model was adjusted for age and sex. Statistically significant differences were considered to exist if the P-value was less than 0.05.

Ethical guidelines

This study was approved by the favorable opinion 160-2023

Table 1. General Characteristics According to Diagnosis of Arrhythmia in Cardiac Patients in Arequipa

Characteristics	Cases (n = 68)	Controls (n = 184)	P-value
Age (years), mean ± SD	62.97 ± 19.13	44.69 ± 15.32	< 0.001[a]*
Sex, n (%)			0.329[b]
Female	33 (48.53%)	102 (55.43%)	
Male	35 (51.47%)	82 (44.57%)	
COVID-19 history, n (%)			0.004[b]*
No	20 (29.41%)	91 (49.46%)	
Yes	48 (70.59%)	93 (50.54%)	

[a]Chi-square test. [b]Student's *t*-test. *P < 0.05. SD: standard deviation.

of the Institutional Research Ethics Committee of the Universidad Catolica de Santa Maria in Arequipa, for adhering to the ethical standards of the institution, as well as to the principles established in the Declaration of Helsinki.

Results

A total of 68 cases of adult patients with a diagnosis of cardiac arrhythmia and 184 controls without cardiac arrhythmia were included. Patients with cardiac arrhythmias were significantly older than those without arrhythmias (mean age: 62.97 ± 19.13 vs. 44.69 ± 15.32 years; P < 0.001). There were no significant differences in sex distribution between cases and controls (P = 0.329). A history of COVID-19 was more common among cases than controls (70.59% vs. 50.54%, respectively; P = 0.004) (Table 1).

When evaluating the findings in cardiac Holter monitoring according to the history of COVID-19, we found that when evaluating heart rate variability, the maximum heart rate was significantly lower for those who had a history of COVID-19 compared to those who did not (P = 0.046). Likewise, no statistically significant differences were found for any other variable (Table 2).

When evaluating the association between a history of COVID-19 infection and cardiac arrhythmia, the crude model showed that patients with a history of COVID-19 infection were significantly more likely to have arrhythmia compared to those without such a history (OR: 2.35; 95% CI: 1.29 - 4.26; P = 0.005). Then, after adjusting for age and sex, the association remained statistically significant (OR: 2.12; 95% CI: 1.10 - 4.11; P = 0.025) (Table 3).

Discussion

Main findings

Our findings showed that patients with arrhythmias had a higher proportion of previous COVID-19 infection (70.6% vs. 50.5%). Logistic regression analysis showed that a history of COVID-19 was associated with more than twice the odds of presenting arrhythmias, even after adjusting for age and sex. Additionally, maximum heart rate was significantly lower in patients with a COVID-19 history.

Comparison with other studies

According to reviews of previous studies, cardiac complications are typically present in 20% to 30% of COVID-19 patients [19], with arrhythmias reported in approximately 10% to 20% of cases [20], which is consistent with the findings of our study. However, although some reports indicate that atrial fibrillation is the most common arrhythmia in patients with a history of COVID-19 [21], our data showed a higher prevalence of sinus rhythm disturbances and other tachyarrhythmias. Furthermore, Mooren et al [22] found a significant association between SARS-CoV-2 infection and cardiac rhythm disorders, which aligns with our finding of a significantly lower maximum heart rate in individuals with a history of COVID-19 (P = 0.046), supporting the hypothesis of rhythm dysfunction in this population.

Studies using Holter monitoring in convalescent COVID-19 patients have also documented a substantial arrhythmic burden. For example, Ingul et al (2022) reported that 27% of patients had arrhythmias on a 24-h Holter monitor 3 months after hospitalization, with the most common findings being frequent premature ventricular contractions (in 18%) and non-sustained ventricular tachycardia (in 5%) [23]. This high prevalence likely reflects a population with more severe acute illness and more intensive follow-up. In contrast, the Italian ARCA post-COVID registry, which included both hospitalized and non-hospitalized patients, found a lower overall arrhythmia prevalence (about 6% on Holter), suggesting that reported frequencies vary depending on patient selection and timing of evaluation [24].

Interpretation of results

The higher prevalence of arrhythmias in patients with a history of COVID-19 suggests possible direct and indirect myocardial damage, including inflammatory and prothrombotic processes that foster rhythm disturbances [19]. Furthermore, multifactorial toxicity in cardiac tissue may explain the early emergence of arrhythmias as one of the first post-infection

Table 2. Findings in Cardiac Holter Monitoring According to the History of COVID-19 in Cardiac Patients in Arequipa

Characteristics	COVID-19 history				P-value
	No		Yes		
	n	%	n	%	
Ventricular events					0.303[b]
No	39	35.45	59	41.84	
Yes	71	64.55	82	58.16	
Supraventricular events					0.496[b]
No	11	10.00	18	12.77	
Yes	99	90.00	123	87.23	
Heart rate variability[a]					
Average heart rate		75.7 ± 11.0		73.1 ± 12.3	0.076[c]
Maximum heart rate		128 (116 - 143)		123 (105 - 140)	0.046[d]*
Minimal heart rate		49 (44 - 55)		49 (44 - 55)	0.056[d]
Atrial fibrillation[a]					0.111[e]
No	15	75.00	44	91.67	
Yes	5	25.00	4	8.33	
Supraventricular tachycardia[a]					0.066[b]
No	9	45.00	33	68.75	
Yes	11	55.00	15	31.25	
Inappropriate sinus[a]					0.550[e]
No	20	100.00	45	93.75	
Yes	0	0.00	3	6.25	
Sinus tachycardia[a]					1.000[e]
No	17	85.00	41	85.42	
Yes	3	15.00	7	14.58	
Sinus rhythm[a]					0.487[e]
No	2	10.00	9	18.75	
Yes	18	90.00	39	81.25	
Ventricular tachycardia[a]					0.575[e]
No	18	90.00	46	95.83	
Yes	2	10.00	2	4.17	
Flutter					0.294[e]
No	19	95.00	48	100.00	
Yes	1	5.00	0	0.00	

[a]Using patients with arrhythmia as the denominator. [b]Chi-square test. [c]Student's *t*-test. [d]Mann-Whitney U test. [e]Fisher's exact test. *P < 0.05. Continuous variables were presented as mean ± SD or median (q1 - q3). SD: standard deviation.

Table 3. Association Between a History of COVID-19 and Cardiac Arrhythmias in Cardiac Patients in Arequipa

Exposure	Cardiac arrhythmia					
	Crude model[a]			Adjusted model[a,b]		
	OR	95% CI	P-value	OR	95% CI	P-value
COVID-19 history						
No	Ref.	-	-	Ref.	-	-
Yes	2.35	1.29 - 4.26	0.005	2.12	1.10 - 4.11	4.11

[a]Binary logistic regression model. [b]Adjusted for age and sex. CI: confidence interval; OR: odds ratio.

complications [19]. This phenomenon can be attributed to the direct infection of cardiomyocytes by SARS-CoV-2 through ACE2 receptors, as well as to indirect injury mediated by an exaggerated immune response, autonomic dysfunction, hypoxemia, and microthrombi formation, all of which can induce fibrosis and electrical instability [19]. Cardiac magnetic resonance imaging (MRI) studies have shown that even patients with mild COVID-19 may exhibit persistent myocardial inflammation weeks after infection [25, 26]. Nevertheless, reasonable doubts remain about the exact impact of COVID-19 on cardiac symptoms, the possible relationship with vaccination [27, 28], and the influence of infection severity [29].

Clinical relevance

Our findings have important clinical implications for the management of post-COVID patients, highlighting the need for rigorous cardiovascular follow-up, even in those who had mild illness but present with persistent symptoms such as palpitations, dizziness, or chest pain. Even patients who were not hospitalized for the infection should not be considered "low risk"; studies have shown that elevated cardiovascular risk is present even in mild cases, though it increases with disease severity [30]. In this context, expert consensus guidelines recommend a comprehensive cardiovascular evaluation in long COVID patients who report cardiac symptoms [31].

Another relevant clinical aspect is the role of COVID-19 vaccination in cardiovascular outcomes. Vaccination has been associated with a lower incidence of post-COVID cardiac complications. Recent large cohort data from the United Kingdom indicate that individuals who contracted COVID-19 after being vaccinated had a significantly lower risk of cardiovascular events in the post-acute phase compared to unvaccinated individuals [32]. In other words, by mitigating the severity of the infection, vaccines appear to indirectly protect against long-term cardiac sequelae of COVID-19. This finding reinforces public health messages promoting widespread vaccination not only to prevent acute COVID-19 hospitalizations and deaths but also to reduce the burden of long COVID [33], including arrhythmias and heart failure [34].

On the other hand, it is important to recognize and monitor the rare cardiovascular side effects of vaccination. mRNA COVID-19 vaccines have been associated with a small risk of myocarditis in young males, with an approximate incidence of 1 in 25,000 (around 40 cases per million second doses in men under 30 years) [31]. Fortunately, these cases have mostly been mild and self-limiting, with recovery occurring without serious complications [35]. The current consensus remains that the benefit-risk ratio of vaccination is highly favorable in terms of cardiovascular health [31]. Clinicians should be aware of both aspects: promoting preventive vaccination to reduce the long-term cardiac risks of COVID-19 and being prepared to evaluate and manage the rare cases of post-vaccine myocarditis.

Early identification of arrhythmias or cardiac dysfunction allows for timely interventions that can improve outcomes and quality of life in COVID-19 survivors. The results of our study reinforce the growing recognition of long-term cardiovascular problems following COVID-19 and support current recommendations for comprehensive cardiologic care in the post-acute phase.

Limitations and strengths

This study has several limitations. It was conducted in a single private institution in Peru, which may limit the external validity of the findings. The retrospective nature of data collection precluded assessment of infection severity, time since COVID-19 diagnosis, or presence of comorbid conditions such as hypertension or diabetes. Additionally, vaccination status and body mass index were not consistently recorded, preventing adjustment for these potentially relevant variables. Nonetheless, the systematic use of Holter monitoring and the consistency of our results with international literature constitute strengths that highlight the significant impact of COVID-19 on cardiovascular health, emphasizing the need for further investigation into pathophysiological mechanisms, as well as prevention and treatment strategies.

Conclusion

A history of COVID-19 infection was significantly associated with higher odds of cardiac arrhythmias among patients undergoing Holter monitoring. Although the types of arrhythmias did not differ markedly between groups, these findings support the need for targeted cardiac evaluation in post-COVID outpatients to detect potential rhythm disorders early and guide clinical management.

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None to declare.

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Conflict of Interest

The authors have no relevant financial or non-financial interests to disclose.

Informed Consent

All patients consented to be part of the study.

Author Contributions

MEMC and AMdCT conceptualized and designed the study. MEMC, AMdCT, JEBP, BAGT, GAC, JAST, and JKBM were involved in data acquisition, formal analysis, and data interpretation. MEMC, AMdCT, JEBP, BAGT, GAC, JAST, HJDCB, and JKBM contributed to the drafting of the original manuscript and provided input during the revision of the original draft. All authors participated in the review and approval of the final manuscript.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Abbreviations

ACE2: angiotensin-converting enzyme 2; bpm: beats per minute; CI: confidence interval; COVID-19: coronavirus disease 2019; ICD: implantable cardioverter-defibrillator; MRI: magnetic resonance imaging; OR: odds ratio; RT-PCR: reverse transcription polymerase chain reaction; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2; SVEs: supraventricular extrasystoles; TNF-α: tumor necrosis factor-alpha; VEs: ventricular extrasystoles; VT: ventricular tachycardia

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