DOI: 10.4274/MMJ.galenos.2023.87400
Medeni Med J 2023;38:236-242

Original Article

Interatrial Block and Electrocardiographic Markers of Repolarization in Patients Hospitalized with COVID-19: Classical and Bayesian Analysis

COVİD-19 Nedeni ile Hastaneye Yatırılan Hastalarda İnteratriyal Blok ve Repolarizasyonun Elektrokardiyografik Belirteçleri: Klasik ve Bayesyen Analiz

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ABSTRACT

Objective: Coronavirus disease-2019 (COVID-19) is associated with atrial fibrillation (AF) and ventricular arrhythmias. Several electrophysiological abnormalities on surface electrocardiography (ECG) are associated with AF and ventricular arrhythmias, either as markers of abnormal interatrial conduction or abnormal repolarization. The present study sought to understand whether such ECG markers are more common in patients hospitalized with COVID-19 infection during the pandemic.

Methods: A total of 87 COVID-19 patients formed the study group, whereas 64 patients who were hospitalized for any reason other than COVID-19 infection served as controls. The frequency of partial and advanced interatrial block (IAB), QT and corrected QT (QTc) durations, QT dispersion (QTd), and T peak-to-end duration (Tpe) were measured from ECGs at admission.

Results: Both partial and advanced IAB were more common in patients with COVID-19, although statistical significance was only observed for advanced IAB (11.5% in COVID-19 patients vs. 0.0% in controls, p=0.005). There were no differences between the groups for QTc, QTd or Tpe. On Bayesian analyses, there was strong evidence favoring an association between COVID-19 and advanced IAB (BF_{10}:16), whereas there was no evidence for an association for partial IAB, QTc, QTd, or Tpe (BF_{10}<1 for all).

Conclusions: Patients hospitalized with COVID-19 were more likely to have advanced IAB, which may explain why AF is more frequent in these patients.

Keywords: COVID-19, atrial fibrillation, interatrial block, ventricular arrhythmia, electrocardiography

ÖZ

Amaç: Koronavirüs hastalığı-19 (COVİD-19) atriyal fibrilasyon (AF) ve ventriküler aritmiler ile ilişkilidir. Yüzey elektrokardiyografisindeki (EKG) çeşitli elektrofizyolojik anormallikler, anormal interatriyal iletimin veya anormal repolarizasyonun belirteçleri olarak AF ve ventriküler aritmilerle ilişkilendirilmiştir. Bu çalışmada, pandemi sırasında COVİD-19 enfeksiyonu ile hastaneye yatırılan hastalarda bu tür EKG belirteçlerinin daha yaygın olup olmadığı anlaşılmaya çalışılmıştır.

Yöntemler: Toplam 87 COVİD-19 hastası çalışma grubunu oluştururken, COVİD-19 enfeksiyonu dışında herhangi bir nedenle hastaneye yatırılan 64 hasta kontrol grubu olarak çalışmaya dahil edildi. Kısmi ve ileri interatriyal blok (İAB) sıklığı, QT ve düzeltilmiş QT (QTc) süreleri, QT dispersiyonu (QTd) ve T tepeden uca süresi (Tpe) başvuru EKG'lerinden ölçüldü.

Bulgular: COVİD-19 hastalarında hem kısmi hem de ileri İAB daha yaygındı, ancak istatistiksel anlamlılık yalnızca ileri İAB için gözlendi (COVİD-19 hastalarında %11,5'e karşı kontrollerde %0,0, p=0,005). QTc, QTd veya Tpe için gruplar arasında fark yoktu. Bayesyen analizlerde, COVİD-19 ile ileri İAB (BF_{10}: 16) arasında bir ilişkiyi destekleyen güçlü kanıtlar bulunurken, kısmi İAB, QTc, QTd veya Tpe için bir ilişki olduğuna dair kanıt yoktu (hepsi için BF_{10}<1).

Sonuçlar: COVİD-19 ile hastaneye yatırılan hastalarda ileri İAB görülme olasılığı daha yüksektir ve bu durum AF'nin bu hastalarda neden daha sık görüldüğünü açıklayabilir.

Anahtar kelimeler: COVİD-19, atriyal fibrilasyon, interatriyal blok, ventriküler aritmiler, elektrokardiyografi

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Received: 02 August 2023
Accepted: 11 September 2023
Online First: 10 October 2023

Cite as: Cetin Guvenc R, Guren AK, Engur B, Celik S, Demirtunc R. Interatrial Block and Electrocardiographic Markers of Repolarization in Patients Hospitalized with COVID-19: Classical and Bayesian Analysis. Medeni Med J 2023;38:236-242

INTRODUCTION

Coronavirus disease-2019 (COVID-19), which is caused by the novel coronavirus severe acute respiratory syndrome coronavirus 2 (SARS-COV-2), has affected more than 750 million people and caused nearly 7 million deaths since its initial dissemination from Wuhan, China, in 2019[1]. Although the pandemic is over, endemic infections are common worldwide, and the long-term consequences of SARS-COV-2 infection are largely unknown. Because the disease affects microvascular circulation and increases thrombogenicity during the acute phase, COVID-19 is associated with atherothrombotic and thromboembolic events, including myocardial infarction[2-4]. Moreover, there have been sporadic cases of myocarditis, and the viral genome has been isolated from myocardial cells from autopsy specimens[5-7]. Arrhythmias, particularly atrial fibrillation (AF), are frequent in hospitalized cases with a prevalence ranging between 11% and 19%[8]. Ventricular tachyarrhythmias, albeit less common, are still seen in a sizable part of the patients[9].

A particular concern for patients hospitalized with COVID-19 is to predict their predilection for cardiovascular complications. P-wave prolongation and reversal on surface electrocardiography (ECG) indicate partial or advanced interatrial block (IAB). The presence of IAB indicates abnormal atrial depolarization due to a dysfunctional Bachmann's bundle, and IAB is a risk factor for atrial tachyarrhythmias, particularly AF[10]. Corrected QT (QTc) duration and global QT dispersion (QTd) reflect prolongation of ventricular repolarization and local differences in the duration of repolarization, and both markers are linked to an increased predisposition to ventricular arrhythmias[11,12]. All these markers may assist in predicting the tendency for atrial and ventricular arrhythmias in COVID-19 patients.

Unfortunately, data on surface ECG markers for arrhythmias in COVID-19 patients are scarce. Therefore, we aimed to understand whether IAB, QTc, or QTd was more common in patients hospitalized with COVID-19 than in controls and to determine the strength of evidence favoring these associations.

MATERIALS and METHODS

This was a cross-sectional observational study. Patients admitted to the study center between January 2020 and June 2020 and subsequently hospitalized with either SARS-COV-2 nucleic acid test positivity with symptoms compatible with acute upper or lower respiratory track infection together with systemic inflammatory response or viral pneumonia compatible with COVID-19 disease were consecutively enrolled. Patients under 18 years old, those with a pacemaker rhythm at the time of ECG recording, and those who had an acute coronary event, pulmonary embolism, or any other type of cardiac emergency were excluded from the study. In addition, patients with a diagnosis of AF (previous or current), those with conditions associated with repolarization abnormalities (pacemaker rhythm, bundle branch block), and those with a rhythm other than sinus were excluded. After applying these inclusion/exclusion criteria, 87 out of 95 patients with COVID-19 who were initially screened were included in the analysis. 64 patients who were hospitalized for a reason other than COVID-19 served as controls. Patients' demographic and clinical data were collected via direct interviews and/or measurements or using the institutional electronic medical database.

The study was conducted according to the principles of the 1975 Declaration of Helsinki, and all patients provided informed consent before enrollment. The study was approved by the University of Health Sciences Turkey, Haydarpasa Numune Training and Research Hospital Ethics Committee (decision no: HNEAH-KAEK 2020/152, date: 31.08.2020).

Recording and Interpretation of Electrocardiograms

A 12-lead ECG was obtained at admission from all patients and recorded on a thermal ECG paper with a sweep speed of 25 mm/s with the standard gain setting using standard methods (10 mv/mm)[12]. All ECGs were interpreted by an experienced cardiologist (R.C.G.) and measurements were taken as described before[13,14]. IABs were classified into partial and advanced IAB according to the P-wave duration and reversal of the terminal portion of the P-wave in the inferior leads[15]. QT and T wave peak-to-end (Tpe) intervals were measured as described previously. The Bazett formula was used to calculate the QTc interval. QTd was calculated by extracting the maximum QT interval from the minimum QT interval.

Statistical Analysis

Continuous variables are given as mean ± standard deviation or as median and interquartile range, whereas categorical variables are given as percentages. Patterns of distribution and equality of variance for continuous variables were analyzed using Shapiro-Wilk and Levene's tests, respectively. For continuous variables, comparisons between groups were performed using the t-test or Mann-Whitney U test as appropriate, while chi-squared or Fisher's Exact tests were used for categorical variables. Bayesian tests were performed to understand the evidence supporting the association of COVID-19

with a given variable. To understand the independent associations between COVID-19 with any type of IAB, QTc, QTd and Tpe, logistic and linear regression models were constructed to adjust for age, gender, presence of coronary artery disease or heart failure, creatinine and electrolytes sodium and potassium. Prior to these analyses, the association between COVID-19 and the aforementioned variables was interrogated using univariate analysis, and regression models were constructed only if the initial association had a p-value <0.1 on univariate analysis. For all frequentist analyses, a p-value <0.05 was set as the limit for significance. For Bayesian analyses, a BF_{10} between 1 and 3 showed anecdotal evidence supporting the alternative hypothesis, whereas a BF_{10} approximately 3-10 and more than 10 indicated moderate and strong evidence, respectively. All statistical analyzes were performed using Jamovi 2.3.22 (The Jamovi project. Jamovi 2.3 for MacOS. Retrieved from htpss://www.jamovi.org) and R [R Core Team (2021). R: A Language and Environment for Statistical Computing. Version 4.1 for MacOS. Retrieved from https://cran.r-project.org.]

RESULTS

The mean age of the study sample was 56.5±14.2, and 36.4% (n=55) cases were females. 25.8% (n=39) and 6.6% (n=10) of the study sample had coronary artery disease and heart failure, respectively. Partial and advanced IAB were present in 9.3% (n=14) and 6.6% (n=10) of the cohort, while none of the patients had any degree of atrioventricular block.

Table 1 summarizes the baseline characteristics of the study groups. Patients in the COVID-19 group were older and more likely to be female, but significant cardiac conditions and cardiovascular risk factors such as hypertension, diabetes, and coronary artery disease were more frequent in the control group. Notably, inflammatory parameters such as C-reactive protein

Table 1. Baseline characteristics of the study groups.

characteristics	COVID-19 patients (n=87)	Controls (n=64)	p-value
Age (years)	58.3±16.7	54.0±9.6	**0.047**
Gender (% female)	36 (41.4%)	19 (29.7%)	0.14
Heart rate (beats/minute)	88.0 (75.0-100.0)	75.0 (68.0-83.0)	**<0.001**
Smoking (%)	4 (4.6%)	11 (17.1%)	**0.01**
Regular alcohol consumption (%)	1 (1.1%)	2 (3.1%)	0.57
Hypertension (%)	31 (35.7%)	49 (76.5%)	**<0.01**
Diabetes (%)	22 (25.3%)	28 (43.7%)	**0.02**
Coronary artery disease (%)	12 (13.8%)	27 (42.1%)	**<0.001**
Heart failure (%)	5 (5.8%)	5 (7.8%)	0.74
Chronic obstructive lung disease (%)	6 (6.9%)	1 (1.5%)	0.24
Asthma (%)	6 (6.9%)	2 (3.1%)	0.47
Diagnosis of COVID-19			
COVID-19 NAT (% positive)	40 (46.0%)		
Unilateral pneumonia (%)	16 (18.4%)		
Bilateral pneumonia (%)	64 (73.6%)		
Blood urea nitrogen (mg/dL)	19.0 (14.0-30.8)	15.0 (13.0-19.0)	**0.02**
Creatinine (mg/dL)	0.90 (0.78-1.19)	0.84 (0.74-1.01)	0.09
Sodium (mEq/L)	138.0 (136.0-141.0)	138.0 (137.0-139.0)	0.90
Potassium (mEq/L)	4.2 (4.0-4.6)	4.2 (4.0-4.5)	0.37
C-reactive protein (mg/dL)	3.4 (0.80-8.5)	1.7 (1.3-3.0)	0.37
Aspartate aminotransferase (IU/L)	25.0 (19.0-38.0)	19.0 (17.0-22.0)	**<0.001**
Alanine aminotransferase (IU/L)	22.5 (15.0-30.8)	22.0 (16.3-29.0)	0.89
Hemoglobin (g/dL)	12.8 (11.8-14.4)	14.5 (13.1-15.3)	**<0.001**
White blood cell count (/10^3)	7.9 (5.7-11.2)	7.5 (6.2-8.8)	0.20
Platelet count (/10^6)	217 (179-272)	252 (216-304)	**0.005**

P-values below 0.05 were given in bold. COVID-19: Coronavirus disease-2019, NAT: Nucleic acid test

concentration and white blood cell count were not different between the groups.

Electrocardiographic variables are given in Table 2. The P-wave duration was significantly higher in patients with COVID-19, and advanced IAB was significantly more frequent in this group. Although partial IAB was also more common in COVID-19 patients, this finding did not reach statistical significance (Figure 1).

On univariate regression analysis, the presence of COVID-19 infection had a statistically significant association with any degree of IAB (p<0.004), whereas the p-value was >0.05 for all other associations with QTc, QTd and TPe. Multivariate logistic regression analysis of the association between COVID-19 and any degree of IAB is summarized in Table 3. After adjustment for covariates, COVID-19 remained an independent predictor of IAB [p=0.005, odds ratio (OR): 9.45, 95% confidence interval (CI): 1.95-45.76].

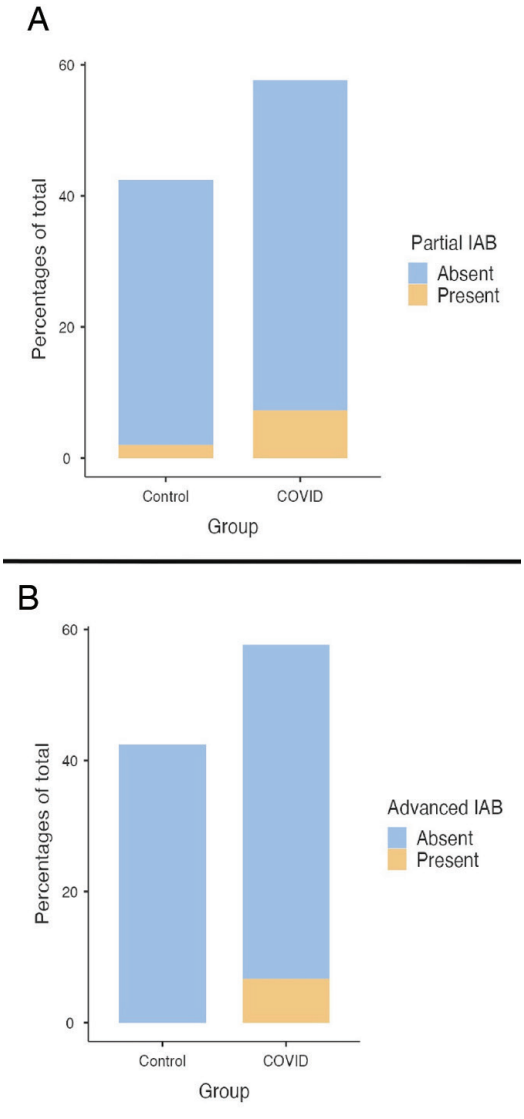

Figure 1. Stacked bar charts showing the frequency of partial **(A)** and advanced **(B)** interatrial block in COVID-19 patients and in controls.

COVID-19: Coronavirus disease-2019, IAB: Interatrial block

On Bayesian analyses, there was strong evidence to suggest an association between COVID-19 and advanced IAB (BF_{10}: 16.05, logOR: 2.57, 95% CI: 0.77-5.87), as well as an association between COVID-19 and any type of IAB (BF_{10}: 74.94, logOR: 1.70, 95% CI: 0.65-3.00); however, there was no evidence to suggest an association between COVID-19 and partial IAB (BF_{10}: 0.87, logOR: 0.95, 95% CI: -0.19-2.31) (Figure 2). Likewise, there was no evidence to suggest a higher QTc, QTd, or Tpe in patients with COVID-19 than in controls (BF_{10}<1 for all).

DISCUSSION

COVID-19 remains a disease with many unknowns, particularly with respect to cardiovascular involvement and outcomes. The present findings suggest a strong association between COVID-19 infection and IAB (particularly advanced IAB), which may explain why AF is so frequent in patients with COVID-19. In contrast, we have not observed an association between COVID-19 and various ECG parameters considered as risk markers for ventricular tachyarrhythmias, despite the use of various statistical methods to analyze the dataset.

Although the existence of an interconnecting muscle bridge that allows the fast conduction of impulses from right to left atrium - the so-called Bachmann bundle - was long known, the understanding that IAB may be a cause of atrial arrhythmias, particularly AF, is a relatively recent development[16]. Although left atrial enlargement (LAE), hence prolonged P-wave duration on ECG, is a major risk factor for AF, the association between IAB and AF is largely independent of LAE[17,18]. The present findings suggest an independent association between COVID-19 disease and IAB, thus providing a potential mechanistic explanation for the higher than expected prevalence of AF in this population. A similar finding was previously noted by Yenerçağ et al.[19], although in their study, the investigators only assessed P-wave duration without a detailed analysis of the patterns of LAE of IAB. Thus, the present findings are incremental over these past results and suggest that IAB is at least partly responsible for this increase in P-wave duration. However, because we have not collected echocardiographic data for the purposes of this study, it remains unclear to what degree this increase in P-wave duration can be attributable to IAB or LAE.

Contrary to the findings on IAB, the present study did not suggest a difference in indices of ventricular repolarization in patients with or without COVID-19. Indeed, no statistical differences were found between the groups in adjusted or unadjusted analyzes nor was there any evidence on Bayesian analyzes that would support an increased duration or heterogeneity of repolarization in the COVID-19 group. This is in contrast with previous reports, where it has been suggested that measures of repolarization (including QTc, QTd and Tpe) were significantly prolonged in COVID-19 patients and this finding correlated with overall mortality[20,21]. A possible explanation for this finding is that the control group was used to assess the statistical significance of changes in the COVID-19 patients. In contrast to other studies, which used healthy volunteers or patients who were presumably less sick than COVID-19 patients, the control group in the present study consisted of patients who either had already established cardiovascular diseases or were at a high risk for cardiovascular disease. Thus, the present findings suggest that most of the variability in the ECG markers of repolarization can be attributable to coexisting conditions in COVID-19 patients, as suggested by others[22].

The present findings have practical implications. Given that IAB provides a substrate for the development of AF, it would be reasonable to closely follow-up COVID-19 patients with IAB and sinus rhythm to prevent the devastating consequences of AF. Diuretics and ventricular resynchronization may reverse IAB in patients with heart failure; however, it is unclear whether IAB is reversible in other circumstances[23,24]. Thus, patients with a history of COVID-19 with IAB may be at an increased life-long risk of AF. Given that other physiological abnormalities, including autonomic dysfunction, are also prevalent in patients with past COVID-19 infection, this risk may be even more pronounced than anticipated[25]. Conversely, we do not recommend the routine use of indices of

Table 2. Electrocardiographic characteristics of the study groups.

characteristics	COVID-19 patients (n=87)	Controls (n=64)	p-value
P duration (ms)	80.0 (80.0-100.0)	80.0 (60.0-82.5)	0.04
P voltage (mV)	0.13 (0.10-0.16)	0.15 (0.10-0.18)	0.31
Any interatrial block	21 (24.7%)	3 (4.7%)	**0.001**
Partial interatrial block	11 (12.6%)	3 (4.7%)	0.09
Advanced interatrial block	10 (11.5%)	0 (0.0%)	**0.005**
PR interval (ms)	160.0 (140.0-165.0)	160.0 (140.0-190.0)	0.27
QRS duration (ms)	80.0 (60.0-80.0)	70.0 (60.0-80.0)	0.21
Corrected QT interval (ms)	320.0 (300.0-360.0)	360.0 (320.0-360.0)	**0.04**
QT dispersion (ms)	40.0 (40.0-60.0)	40.0 (40.0-60.0)	0.57
Peak to end for T wave (ms)	50.0 (40.0-70.0)	60.0 (40.0-60.0)	0.94

P-values below 0.05 were given in bold. COVID-19: Coronavirus disease-2019.

Table 3. Multivariable predictors of any degree of IAB in the study sample.

parameters	Odds ratio (95% confidence interval)	p-value
Age (per year increase)	1.01 (0.98-1.06)	0.60
Gender (being female)	1.52 (0.55-4.23)	0.42
Hypertension (presence of)	2.30 (0.67-7.84)	0.19
Diabetes (presence of)	0.66 (0.18-2.46)	0.54
Smoking (presence of)	2.41 (0.37-15.90)	0.36
Coronary artery disease (presence of)	1.04 (0.25-4.28)	0.96
Heart failure (presence of)	1.04 (0.16-20.56)	0.64
Creatinine (per mg/dl increase)	1.39 (0.86-2.24)	0.17
Sodium (per mEq/L increase)	1.14 (0.97-1.34)	0.11
Potassium (per mEq/L increase)	0.33 (0.12-0.91)	**0.03**
COVID-19 (presence of)	9.45 (1.95-45.76)	**0.005**

P-values below 0.05 were given as bold. COVID-19: Coronavirus disease-2019, IAB: Interatrial block

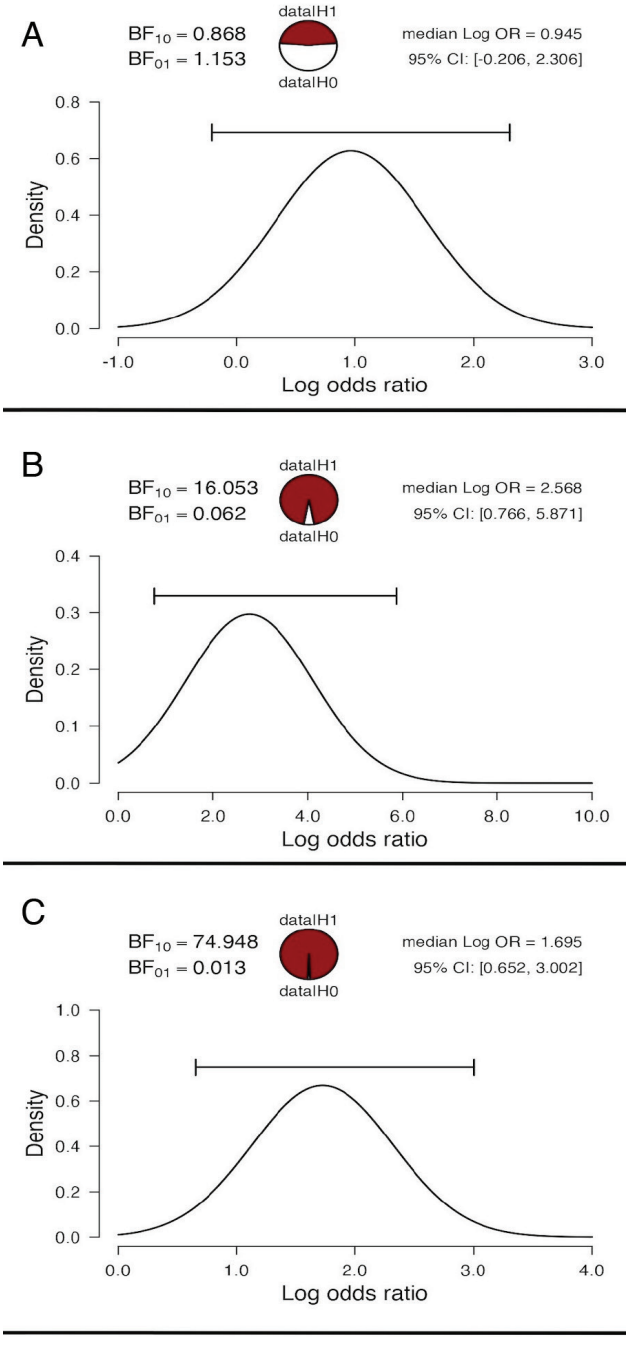

Figure 2. Probability density distributions for partial **(A)**, advanced **(B)** and any type of **(C)** interatrial block in COVID-19 patients as compared to controls. Pie charts above distribution plots show Bayesian factors supporting alternative and null hypotheses.

COVID-19: Coronavirus disease-2019, OR: Odds ratio, CI: Confidence interval

ventricular repolarization for COVID-19 patients who are not otherwise at risk of QT prolongation. This recommendation was not solely based on the current findings, as there are significant methodological concerns about the usefulness of these parameters for predicting ventricular arrhythmias[26].

The present study has several limitations that should be stated. Patients were enrolled from a single center, and the sample size was rather limited. Because this was an all-comers study and prior data on the prevalence of ECG abnormalities in COVID-19 patients were limited, an a priori power analysis was not feasible. However, a post-hoc power analysis found that the study had a power (1-β) of 0.94 and 0.99 to detect a significant difference between the advanced and any IAB groups, respectively. The diagnosis of COVID-19 was based on nucleic acid tests only in approximately one-third of the sample, whereas the diagnosis was based on the presence of clinical and imaging findings for viral pneumonia in the remaining cases. Although the study was conducted during the peak of the COVID-19 pandemic, some cases might have been infected with an agent other than SARS-COV-2. No data on the severity of COVID-19 were collected; therefore, it was not possible to analyze whether the degree of ECG abnormalities was related to the severity of COVID-19. Although predictive usefulness and prognostic implications of IAB have been demonstrated, no follow-up data were collected for the purposes of the present study; therefore, the outcomes related to IAB in COVID-19 patients remain unknown[18].

CONCLUSION

Both partial and advanced IAB are more common in hospitalized COVID-19 patients, which may provide a mechanistic explanation for the higher incidence of AF in this patient population. Because of the proven connection of IAB with AF in other patient populations, a close follow-up is reasonable for patients with COVID-19 and IAB (particularly advanced IAB). The prognostic implications of IAB specific to patients with COVID-19 or the long-term implications of IAB in this patient population remain unknown, and further research is warranted on these topics.

Ethics

Ethics Committee Approval: The study was approved by University of Health Sciences Turkey, Haydarpasa Numune Training and Research Hospital Ethics Committee (decision no: HNEAH-KAEK 2020/152, date: 31.08.2020).

Informed Consent: All patients provided informed consent before enrollment.

Peer-review: Externally and internally peer-reviewed.

Author Contributions

Surgical and Medical Practices: A.K.G., B.E., S.C., Concept: R.C.G., R.D., Design: R.C.G., A.K.G., B.E., S.C., R.D., Data Collection and/or Processing: R.C.G., A.K.G., B.E., S.C., Analysis and/or Interpretation: R.C.G., A.K.G., R.D., Literature Search: R.C.G., B.E., S.C., Writing: R.C.G., A.K.G., B.E., S.C., R.D.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. World Health Organization. WHO Coronavirus (COVID-19) Dashboard. https://covid19.who.int/
2. Çalışkan M, Baycan ÖF, Çelik FB, et al. Coronary microvascular dysfunction is common in patients hospitalized with COVID-19 infection. Microcirculation. 2022;29:e12757.
3. Zuin M, Rigatelli G, Battisti V, Costola G, Roncon L, Bilato C. Increased risk of acute myocardial infarction after COVID-19 recovery: A systematic review and meta-analysis. Int J Cardiol. 2023;372:138-43.
4. Del Prete A, Conway F, Della Rocca DG, et al. COVID-19, Acute Myocardial Injury, and Infarction. Card Electrophysiol Clin. 2022;14:29-39.
5. Siripanthong B, Nazarian S, Muser D, et al. Recognizing COVID-19-related myocarditis: The possible pathophysiology and proposed guideline for diagnosis and management. Heart Rhythm. 2020;17:1463-71.
6. Patone M, Mei XW, Handunnetthi L, et al. Risk of Myocarditis After Sequential Doses of COVID-19 Vaccine and SARS-CoV-2 Infection by Age and Sex. Circulation. 2022;146:743-54.
7. Escher F, Pietsch H, Aleshcheva G, et al. Detection of viral SARS-CoV-2 genomes and histopathological changes in endomyocardial biopsies. ESC Heart Fail. 2020;7:2440-7.
8. Li Z, Shao W, Zhang J, et al. Prevalence of Atrial Fibrillation and Associated Mortality Among Hospitalized Patients With COVID-19: A Systematic Review and Meta-Analysis. Front Cardiovasc Med. 2021;8:720129.
9. Woodruff RC, Garg S, George MG, et al. Acute Cardiac Events During COVID-19-Associated Hospitalizations. J Am Coll Cardiol. 2023;81:557-69.
10. Chhabra L, Devadoss R, Chaubey VK, Spodick DH. Interatrial block in the modern era. Curr Cardiol Rev. 2014;10:181-9.
11. Malik M, Batchvarov VN. Measurement, interpretation and clinical potential of QT dispersion. J Am Coll Cardiol. 2000 15;36:1749-66.
12. Okin PM, Devereux RB, Howard BV, Fabsitz RR, Lee ET, Welty TK. Assessment of QT interval and QT dispersion for prediction of all-cause and cardiovascular mortality in American Indians: The Strong Heart Study. Circulation. 2000;101:61-6.
13. Meek S, Morris F. ABC of clinical electrocardiography.Introduction. I-Leads, rate, rhythm, and cardiac axis. BMJ. 2002;324:415-8.
14. Meek S, Morris F. Introduction. II--basic terminology. BMJ. 2002;324:470-3.
15. de Luna AB, Massó-van Roessel A, Robledo LAE. The Diagnosis and Clinical Implications of Interatrial Block. Eur Cardiol. 2015;10:54-9.
16. Bachmann G. The Inter-auricular time interval. Am J Physiol. 1916;41:309-20.
17. Bayés de Luna A, Guindo J, Viñolas X, Martinez-Rubio A, Oter R, Bayés-Genís A. Third-degree inter-atrial block and supraventricular tachyarrhythmias. Europace. 1999;1:43-6.
18. Agarwal YK, Aronow WS, Levy JA, Spodick DH. Association of interatrial block with development of atrial fibrillation. Am J Cardiol. 2003;91:882.
19. Yenerçağ M, Arslan U, Şeker OO, et al. Evaluation of P-wave dispersion in patients with newly diagnosed coronavirus disease 2019. J Cardiovasc Med (Hagerstown). 2021;22:197-203.
20. Rubin GA, Desai AD, Chai Z, et al. Cardiac Corrected QT Interval Changes Among Patients Treated for COVID-19 Infection During the Early Phase of the Pandemic. JAMA Netw Open. 2021;4:e216842.
21. Mahmoudi E, Mollazadeh R, Mansouri P, et al. Ventricular repolarization heterogeneity in patients with COVID-19: Original data, systematic review, and meta-analysis. Clin Cardiol. 2022;45:110-8.
22. Cozzolino D, Romano C, Nevola R, et al. COVID-19 and arrhythmia: The factors associated and the role of myocardial electrical impulse propagation. An observational study based on cardiac telemetric monitoring. Front Cardiovasc Med. 2022;9:912474.
23. Song J, Kalus JS, Caron MF, Kluger J, White CM. Effect of diuresis on P-wave duration and dispersion. Pharmacotherapy. 2002;22:564-8.
24. Goyal SB, Neal S, Spodick DH, et al. Effect of cardiac resynchronization therapy on left atrial electromechanical function. Pacing Clin Electrophysiol. 2003;26:975. Abstract 187.
25. Asarcikli LD, Hayiroglu Mİ, Osken A, Keskin K, Kolak Z, Aksu T. Heart rate variability and cardiac autonomic functions in post-COVID period. J Interv Card Electrophysiol. 2022;63:715-21.
26. Pranata R, Yonas E, Vania R, Huang I. Markers of ventricular repolarization as an additional non-invasive electrocardiography parameters for predicting ventricular tachycardia/fibrillation in patients with Brugada Syndrome - A systematic review and meta-analysis. Indian Pacing Electrophysiol J. 2019;19:205-10.