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Unveiling Subclinical Arrhythmias After COVID-19: Insights from 24-Hour Holter Monitoring

COVID-19 Sonrası Subklinik Aritmilerin Ortaya Çıkışı: 24 Saatlik Holter Monitörizasyonundan Elde Edilen Bulgular

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ABSTRACT

Aim: Coronavirus disease 2019 (COVID-19) has been linked to various arrhythmias, including atrial and ventricular ectopy, atrial fibrillation, and conduction disturbances. Proposed mechanisms include myocardial injury, systemic inflammation, autonomic dysfunction, and endothelial damage. This study aimed to assess arrhythmic burden and its predictors in patients with prior COVID-19 infection using 24-hour Holter monitoring.

Materials and Methods: We retrospectively analyzed 153 patients who underwent Holter electrocardiography (ECG) between January 2021 and June 2023. Participants were divided into COVID-19 positive (n=62) and control (n=91) groups. Demographic, clinical, and laboratory characteristics were compared. Ventricular ectopic beats (VES) were quantified, and ROC analysis identified 571 VES/day as the threshold for high arrhythmic burden. Patients were stratified accordingly, and predictors of high burden were determined using multivariate logistic regression.

Results: VES counts were significantly higher in the COVID positive group (p<0.001). Patients with >571 VES had a higher frequency of fragmented QRS (75.0% vs. 7.6%) and prior COVID-19 infection (60.4% vs. 31.4%) (p<0.001 for both). In multivariate analysis, fragmented QRS [odds ratio (OR): 26.99, p<0.001] and COVID-19 history (OR: 10.30, p<0.001) independently predicted high arrhythmic burden. COVID-19 vaccination was associated with a reduced risk (OR: 0.13, p=0.006).

Conclusion: COVID-19 infection is significantly associated with increased ventricular ectopy and fragmented QRS on Holter ECG, indicating persistent arrhythmic risk. Fragmented QRS and COVID-19 history independently predict high arrhythmic burden, while vaccination appears protective. Post-COVID rhythm surveillance may help identify high-risk individuals.

Keywords: COVID-19, cardiovascular arrhythmia, ventricular extra systole

ÖZ

Amaç: Koronavirüs hastalığı-2019 (COVID-19), atriyal ve ventriküler ektopi, atriyal fibrilasyon ve ileti bozuklukları dahil olmak üzere çeşitli aritmilerle ilişkilendirilmiştir. Öne sürülen mekanizmalar arasında miyokardiyal hasar, sistemik enflamasyon, otonom disfonksiyon ve endotel hasarı yer almaktadır. Bu çalışma, COVID-19 enfeksiyonu öyküsü olan hastalarda 24 saatlik Holter monitörizasyonu kullanılarak aritmi yükünü ve bunun belirleyicilerini değerlendirmeyi amaçlamıştır.

Gereç ve Yöntem: Ocak 2021 ile Haziran 2023 tarihleri arasında Holter elektrokardiyografi (EKG) uygulanmış 153 hasta retrospektif olarak analiz edildi. Katılımcılar COVID-19 pozitif (COVID+) (n=62) ve kontrol (n=91) gruplarına ayrıldı. Demografik, klinik ve laboratuvar özellikleri karşılaştırıldı. Ventriküler ektopik atımlar (VES) sayıldı ve ROC analiziyle günlük 571 VES değeri yüksek aritmi yükü için eşik olarak belirlendi. Hastalar bu değere göre sınıflandırıldı ve yüksek aritmi yükünün öngörücüsü olan değişkenler çok değişkenli lojistik regresyonla analiz edildi.

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Bulgular: VES sayıları COVID pozitif grubunda anlamlı derecede yüksekti (p<0,001). Günde 571'den fazla VES olan hastalarda, fragmented QRS (%75,0 vs. %7,6) ve COVID-19 öyküsü (%60,4 vs. %31,4) daha sık görüldü (her ikisi için p<0,001). Çok değişkenli analizde, fragmented QRS [olasılık oranı (OR): 26,99, p<0,001] ve COVID-19 öyküsü (OR: 10,30, p<0,001), bağımsız olarak yüksek aritmi yükünü öngördü. COVID-19 aşılaması, daha düşük risk ile ilişkilendirildi (OR: 0,13, p=0,006).

Sonuç: COVID-19 enfeksiyonu, Holter EKG'de artmış ventriküler ektopi ve fragmented QRS ile anlamlı şekilde ilişkilidir ve bu durum kalıcı aritmik riske işaret eder. Fragmented QRS ve COVID-19 öyküsü yüksek aritmi yükünün bağımsız belirleyicileridir; aşılanma ise koruyucu bir etkiye sahip olabilir. Post-COVID döneminde ritim izlemi, yüksek riskli bireylerin belirlenmesine katkı sağlayabilir.

Anahtar Kelimeler: COVID-19, kardiyovasküler aritmi, ventriküler ekstra sistol

INTRODUCTION

Severe acute respiratory syndrome coronavirus 2 infection has been associated with a wide range of cardiovascular complications, including arrhythmias[1]. Post-Coronavirus disease (COVID) cardiac manifestations are increasingly recognized, with ventricular arrhythmias being one of the concerning sequelae[2]. The potential mechanisms underlying this increased arrhythmic burden include direct myocardial injury, systemic inflammation, and autonomic dysfunction. Emerging evidence also suggests that survivors of Coronavirus disease 2019 (COVID-19) may develop autonomic nervous system disturbances for instance, persistent sympathetic activation and reduced heart rate variability have been observed – which could facilitate post-COVID arrhythmogenesis[3]. Identifying patients at higher risk of developing significant arrhythmias may help in optimizing post-COVID-19 cardiac care[4]. However, data regarding the long-term ventricular arrhythmic burden in relatively young, non-hospitalized post-COVID populations remain limited. This study aimed to evaluate the arrhythmic burden in patients with a history of COVID-19 infection and determine the clinical predictors of increased ventricular ectopic activity (VEA).

MATERIALS AND METHOD

This retrospective study included 153 patients who underwent 24-hour electrocardiography (ECG) Holter monitoring, classified into two groups: COVID-19 positive (COVID+) (n: 62) and control (n: 91). Between January 2021 and January 2022, patients were selected for inclusion if they had a confirmed COVID-19 diagnosis by positive reverse transcription polymerase chain reaction and subsequently presented with cardiac symptoms such as palpitations, presyncope, or chest pain warranting Holter monitoring. Symptoms such as presyncope, syncope, palpitations were recorded. None of the patients had complete syncope. Patients in the control group also underwent Holter monitoring for similar cardiac symptoms such as palpitations, syncope, or chest pain, despite having no history of COVID-19. Exclusion criteria comprised any history of structural heart disease, pacemaker implantation, or severe electrolyte imbalances, to avoid confounding factors that could independently affect arrhythmia propensity. Patients' vaccination history was recorded, however, due to the heterogeneity in vaccine types (inactivated and mRNA-based) and variable dose numbers (ranging from one to four doses), vaccination status was analyzed categorically as vaccinated or unvaccinated. Baseline clinical characteristics, laboratory parameters, and ECG Holter recordings were compared between groups. A ROC curve analysis was performed to determine the optimal threshold for high arrhythmic burden based on ventricular extra systole (VES) counts. A cut-off value of 571 VES was identified. Patients were then further categorized into two subgroups: VES >571 (n: 48) and VES ≤571 (n: 105). Univariate and multivariate logistic regression analyses were conducted to identify independent predictors of high VES burden. The study was carried out in compliance with the Declaration of Helsinki and received approval from the Institutional Committee on Human Research and Ethics. All participants provided written informed consent before enrollment. Ethical approval for this research was granted by The Ethics Committee for Scientific Research, Faculty of Medicine, Trakya University (decision no: 02/17, date: 18.12.2021).

Statistical Analysis

Statistical analyses were conducted using SPSS version 25.0 (SPSS, Chicago, IL). Continuous variables were summarized as mean ± standard deviation, while categorical variables were reported as frequencies and percentages. The Shapiro-Wilk test was applied to evaluate the normality of distributions. Group comparisons for continuous variables were performed using the independent t-test, whereas categorical variables were analyzed with the Pearson's chi-square test. Correlations were evaluated with spearman correlation test. ROC curve analysis was utilized to determine the area under the curve (AUC) and optimal cut-off values for diagnostic performance. Logistic regression analysis was employed to identify independent predictors. All statistical tests were two-sided, with a significance level set at p<0.05.

RESULTS

Baseline clinical and demographic characteristics are presented in Table 1. mean age was similar between the COVID+ and control groups (38.57±15.14 vs. 37.12±16.03 years, p: 0.903). Female patients were more prevalent in the COVID+ group (41.93% vs. 25.27%, p: 0.030). Thyroid dysfunction was significantly higher in the COVID+ group (24.19% vs. 7.69%, p: 0.030), while rates of hypertension (43.54% vs. 43.95%, p: 0.848) and diabetes mellitus (37.09% vs. 29.67%, p: 0.336) showed no significant differences (Table 1). No difference was found in terms of the medication (antihypertensive or antiarrhythmic) used by the patients (Table 1). COVID+ patients had significantly elevated high-sensitivity-C-reactive protein (hs-CRP) levels (2.47±3.33 mg/dL vs. 1.46±1.95 mg/dL, p: 0.002) and a higher incidence of fragmented QRS (46.77% vs. 10.98%, p<0.001). The COVID+ group also exhibited a significantly higher burden of VEA (VES count, p<0.001). The analysis of ECG Holter parameters is presented in Table 2. ROC curve analysis identified 571 VES as the optimal threshold for high arrhythmic burden (AUC: 0.82, 95% confidence interval: 0.76-0.88, p<0.001) (Table 3).

Figure 1. Patients with VES >571 had a higher prevalence of fragmented QRS (75.0% vs. 7.62%, p<0.001) and prior COVID-19 infection (60.41% vs. 31.42%, p<0.001). VEA, age, ejection fraction (EF) and hs-CRP were evaluated by correlation analyses (Figure 2). A weak positive correlation was found between VEA and advanced glycation end-products. There is a negative, weak and statistically significant correlation between VEA and EF (r=-0.2313, p=0.004). There was a negative, moderate and statistically significant correlation between CRP and EF variables (Figure 2). Vaccination rates were significantly lower in the high VES group (52.08% vs. 90.47%, p<0.001). Elevated hs-CRP levels correlated with high VES burden (p: 0.002). Although the EF was slightly lower in the high VES group (54.45% vs. 55.58%, p: 0.314), the difference was not statistically significant.

Table 1. The Clinical characteristics of patients of the study population in comparison with COVID-19 and Control group

Variables	COVID+ (n: 62)	Control (n: 91)	p
Age, years	38.57±15.14	37.12±16.03	0.903
Sex, female, n (%)	26 (41.93)	23 (25.27)	0.030
BMI kg/m^2	25.34±5.11	25.93±3.69	0.796
Smoker (no) n (%)	25 (40.32)	33 (36.26)	0.075
HT n (%)	27 (43.54)	40 (43.95)	0.848
DM n (%)	23 (37.09)	27 (29.67)	0.336
CAD n (%)	14 (22.58)	21 (23.07)	0.426
Thyroid dysfunction	15 (24.19)	7 (7.69)	0.03
Vaccination	39 (62.90)	81 (71.37)	<0.001
Neutrophil/lymphocyte ratio	4.02±2.65	3.62±1.99	0.302
Hemoglobin (g/dL)	11.05±1.65	11.69±1.72	0.962
Na (mmol/L)	138.51±2.77	139.75±5.13	0.084
K (mmol/L)	4.60±0.59	4.38±0.81	0.074
hs-CRP (mg/dL)	2.47±3.33	1.46±1.95	0.002
Creatinine (mg/dL)	0.93±0.29	0.85±0.59	0.258
Frag QRS	29 (46.77)	10 (10.98)	<0.001
Ejection fraction (%)	54.11±8.87	55.82±5.18	0.135
Presyncope (%)	3 (3.29)	3 (4.83)	0.630
Syncope	0	0	-
Palpitation	62 (100)	91 (100)	-
Hospitalization	0	0	-
Chest pain	7 (11.29)	11(12.08)	0.721
ACEi	21(33.87)	30 (32.96)	0.671
Beta blockers	10 (16.12)	17 (18.68)	0.272
CCB-dihydropyridine Non dihydropyridine	10 (16.12) 2 (3.22)	16 (17.58) 4 (4.39)	0.568 0.128

ACEi: Angiotensin converting enzyme inhibitor, CAD: Coronary artery disease, CCB: Ca canal blockers, DM: Diabetes mellitus, HT: Hypertension, hs-CRP: High-sensitive C-reactive protein, BMI: Body-mass index, Na: Sodium, K: Potassium, COVID-19: Coronavirus disease-19

Table 2. The ECG Holter parameters of patients of the study population in comparison with COVID-19 and Control group

Variables	COVID+ (n: 62)	Control (n: 91)	p
Atrial extra systole	103 (31-401)	80 (30-189)	0.001
Ventricular extra systole	796 (361-950)	110 (65-180)	<0.001
Non-sustained ventricular tachycardia	3 (4.83)	4 (4.39)	0.758
Supraventricular tachycardia	11 (17.74)	19 (19.78)	0.129
QRS (msn)	93.45±18.04	91.82±12.41	0.547
QT (msn)	373.67±35.77	391.54±42.44	0.012
QTc	441.14±24.05	430.47±24.50	0.020
QTd	52.58±16.68	40.87±16.78	<0.001
QTcd	50.50±11.85	46.29±17.45	0.003
QRS (msn)	93.82±18.51	90.89±12.04	0.641
MQTc	437.09±22.02	427±25.53	0.047

COVID-19: Coronavirus disease-19, ECG: Electrocardiography, COVID+: COVID-19 pozitif

Table 3. Diagnostic performance of VES for group of COVID+

Diagnostic Performance of VES for COVID+					
	Cut-off	AUC (95% CI)	Sensitivity	Specificity	p-value
VES	571	0.82 (0.76-0.88)	0.77 (0.65-0.86)	0.91 (0.83-0.95)	<0.001

VES: Ventricular extra systole, AUC: Area under the curve, CI: Confidence interval, COVID: Coronavirus disease

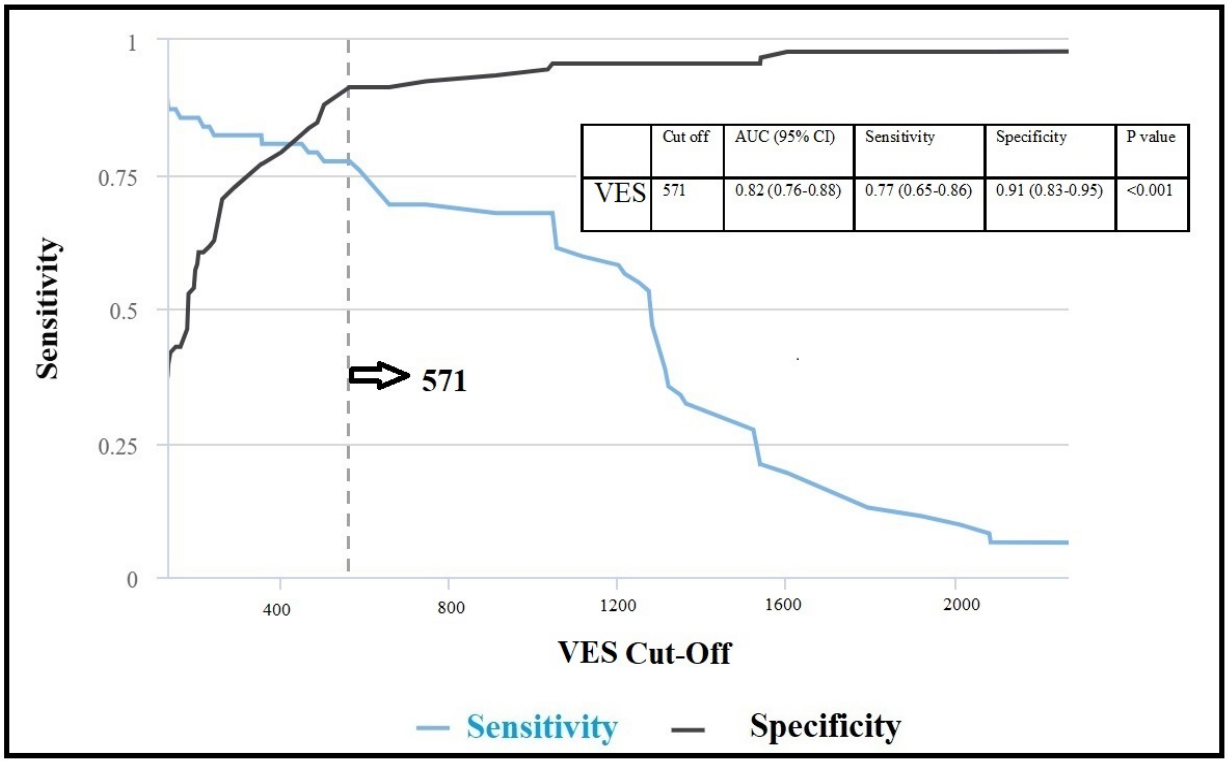

Figure 1. Sensitivity and specificity chart of VES

VES: Ventricular extra systole, AUC: Area under the curve, CI: Confidence interval

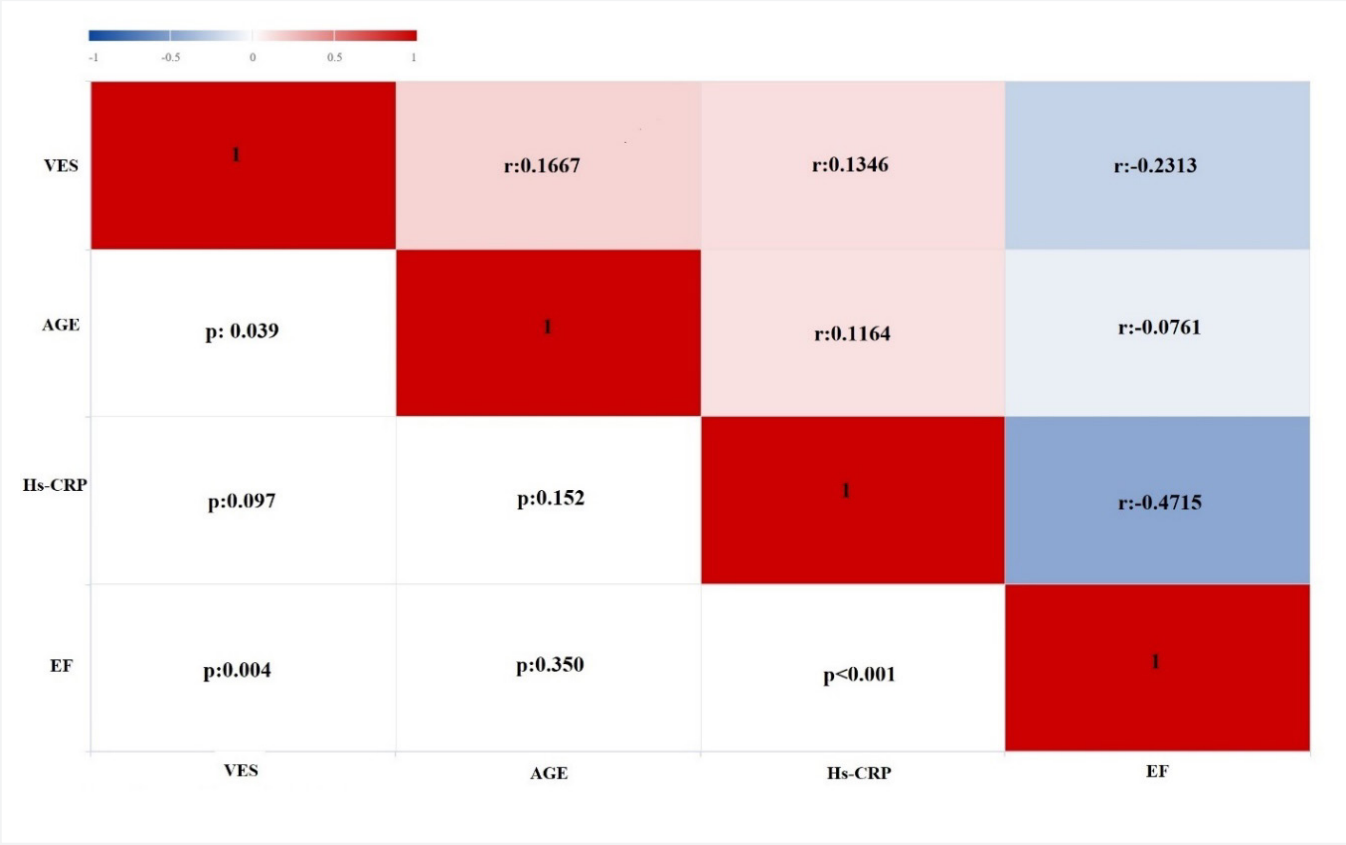

Figure 2. Correlogram table constructed as a result of correlation analyses

VES: Ventricular extra systole, AGE: Advanced glycation end-products, hs-CRP: High-sensitivity-C-reactive protein, EF: Ejection fraction

Table 4. The clinical characteristics of patients of the study population in comparison with VES count (571)

Değişkenler	VES >571 (n: 48)	VES <571 (n: 105)	p
Age	39.06±14.18	38.15±15.89	0.753
Gender, female n (%)	23 (47.92)	26 (24.76)	0.004
Smoker (no) n (%)	20 (41.66)	38 (36.19)	0.135
HT n (%)	17 (35.41)	36 (34.28)	0.297
DM n (%)	23 (37.09)	27 (29.67)	0.336
CAD n (%)	8 (16.66)	24 (22.85)	0.385
Thyroid dysfunction	10 (20.83)	9 (8.57)	0.041
COVID-19	29 (60.41)	33(31.42)	<0.001
Vaccination	25 (52.08)	95 (90.47)	<0.001
NLR	4.15±2.99	3.82±2.05	0.360
hs-CRP	2.17±2.75	1.73±2.58	0.343
Fragmented QRS	36 (75.00)	8 (7.62)	<0.001
Ejection fraction (%)	54.45±9.87	55.58±6.18	0.314

CAD: Coronary artery disease, DM: Diabetes mellitus, HT: Hypertension, hs-CRP: High-sensitive C-reactive protein, NLR: Neutrophil/lymphocyte ratio, COVID-19: Coronavirus disease-19, VES: Ventricular extra systole

Table 5. Univariate, multivariate and stepwise binary logistic regression analysis for the VEA >571

Log reg	Univariate model			Multivariate model		
	OR	95% CI	p	OR	95% CI	p
Fragmented QRS	36.37	13.74-96.23	<0.001	26.99	6.98-99.73	<0.001
Gender (female)	0.78	0.35-2.40	0.208	0.264	0.05-1.28	0.098
Thyroid dysfunction	2.80	1.05-7.44	0.038	0.578	0.05-5.44	0.632
COVID+	29.75	11.17-79.61	<0.001	10.30	3.07-34.51	<0.001
Vaccination	0.11	0.05-0.27	<0.001	0.13	0.03-0.57	0.006
Age	1.158	0.91-1.25	0.428	1.01	0.97-1.05	0.595

OR: Odds ratio, CI: Confidence interval, COVID: Coronavirus disease, VEA: Ventricular ectopic activity, COVID: Coronavirus disease, COVID+: COVID-19 pozitif

The clinical characteristics of the study population in comparison with VEA count (571) are presented in Table 4. ECG Holter analysis showed that COVID+ patients had a higher burden of atrial extrasystoles (987.22±208.85 vs. 463.55±221.33, p: 0.042), prolonged QTc intervals (430.47±24.50 ms vs. 441.14±24.05 ms, p: 0.020), and increased QT dispersion (40.87±6.78 ms vs. 52.58±16.68 ms, p<0.001) compared to controls. Multivariate logistic regression analysis identified fragmented QRS [odds ratio (OR): 26.99, p<0.001] and COVID-19 infection (OR: 10.30, p<0.001) as independent predictors of high VES burden (Table 5). Vaccination was associated with a significantly lower risk of arrhythmia (OR: 0.13, p: 0.006).

DISCUSSION

COVID-19 infection has been implicated not only in acute cardiac injury but also in long-term cardiac sequelae, including inflammatory and arrhythmogenic manifestations. In a previously published case, we reported the development of constrictive pericarditis in a young individual following COVID-19 and vaccination exposure, raising awareness of the virus's potential to induce delayed cardiac inflammation and remodeling, even in structurally normal hearts[5]. COVID-19 infection has been linked to various cardiac arrhythmias, including atrial and ventricular ectopy, atrial fibrillation, and conduction abnormalities. Proposed mechanisms include direct myocardial injury, systemic inflammation, autonomic dysfunction, and endothelial damage, all contributing to altered cardiac electrophysiology. Recent studies indicate that post-COVID-19 patients, especially those with severe illness, have an increased risk of ventricular arrhythmias and QT prolongation, highlighting the need for long-term cardiac surveillance[6]. In a recently published review, the relationship between COVID-19 and the cardiovascular system has been thoroughly evaluated. This study discusses the mechanisms by which COVID-19 induces arrhythmias, which are associated with hypoxia, myocarditis, and secondary causes. Specifically, it highlights that hypoxia can trigger anaerobic glycolysis through cellular damage, increase cytosolic calcium levels, and facilitate arrhythmogenesis by inducing early and late depolarizations[7]. Unlike prior studies focusing on hospitalized COVID-19 patients, our study uniquely highlights the persistent arrhythmic risk even in a relatively young outpatient population.

The findings of this study highlight three key results: 1. prior COVID-19 infection is significantly associated with increased VEA and fragmented QRS, suggesting a potential link between myocardial injury and arrhythmic risk. 2. hs-CRP levels were significantly elevated in patients with high arrhythmic burden, reinforcing the role of systemic inflammation in post-COVID arrhythmogenesis. 3. vaccination was associated with a significantly lower risk of high VES burden, indicating a potential protective effect against post-COVID cardiovascular complications.

The association between COVID-19 infection and increased ventricular arrhythmic burden has been supported by previous studies. For example, Turagam et al.[8] demonstrated that post-COVID patients, particularly those with severe disease, exhibited a higher frequency of ventricular ectopy. The f-QRS pattern may have significant prognostic implications in COVID-19 patients. There are publications in the literature suggesting that this condition may exhibit temporal and mechanistic variations, and our study supports these findings[9]. Our findings align with these studies, suggesting that myocardial injury and fibrosis, as reflected by fragmented QRS patterns, may contribute to sustained arrhythmic risk[10]. The presence of fragmented QRS has been well-documented as a marker of myocardial scarring, which predisposes patients to malignant arrhythmia.

However, it should be noted that truly malignant ventricular arrhythmias (such as sustained ventricular tachycardia or fibrillation) appear to be relatively infrequent in post-COVID patients without critical illness. In fact, clinical data indicate that ventricular tachyarrhythmias in COVID-19 are mainly observed in the presence of severe metabolic derangements suggesting that profound electrolyte imbalances or other

metabolic factors during acute illness are often required to precipitate life-threatening arrhythmias[11].

Elevated hs-CRP levels in patients with high VES burden further emphasize the role of systemic inflammation in arrhythmogenesis. Prior studies have shown that post-COVID inflammatory responses, including cytokine-mediated myocardial stress, can alter cardiac electrophysiology. In line with this, Marques et al.[12] reported that elevated inflammatory markers, particularly CRP and interleukin-6, were associated with an increased risk of ventricular arrhythmias post-COVID[13]. Our results support this concept, as heightened systemic inflammation may exacerbate myocardial excitability, leading to increased ectopic activity. The association between autonomic dysfunction and inflammation in long-COVID patients, as indicated by elevated CRP and impaired heart rate recovery, aligns with our findings of increased VEA and prolonged QT parameters in post-COVID individuals[14]. These results further support the hypothesis that persistent systemic inflammation and autonomic imbalance may contribute to heightened arrhythmic risk in COVID-19 survivors, underscoring the need for long-term cardiac monitoring

The observed protective effect of vaccination against high arrhythmic burden is an important finding. Previous research has demonstrated that COVID-19 vaccination reduces the severity of systemic inflammatory responses, which are known contributors to arrhythmia development. Studies such as Pari et al.[15] reported a lower incidence of post-COVID cardiovascular complications among vaccinated individuals, likely due to the mitigation of endothelial dysfunction and myocardial inflammation. Our findings reinforce these observations, suggesting that vaccination may play a role in reducing post-infectious arrhythmic risk.

Beyond these primary results, certain findings warrant further discussion. The significantly prolonged QTc interval and increased QT dispersion in the COVID+ group suggest potential autonomic dysregulation or direct myocardial electrophysiological alterations post-infection[16]. In addition to autonomic dysregulation, potential contributors to QTc prolongation may include subclinical electrolyte imbalances or the use of QT-prolonging medications, although major disturbances were part of our exclusion criteria. These parameters are known predictors of ventricular arrhythmias and sudden cardiac death, necessitating further research on long-term QT dynamics in post-COVID populations[17]. Additionally, the high prevalence of thyroid dysfunction in the COVID+ group raises questions regarding the interplay between endocrine abnormalities and arrhythmic risk, as thyroid dysfunction is a known contributor to ventricular ectopy. Although thyroid dysfunction was more prevalent in the COVID+ group, this association may be incidental, and no direct causal link can be established based on our data. Future prospective studies should investigate whether these abnormalities persist over time and their potential therapeutic implications in post-COVID cardiac care[18]. The observed increase in atrial extrasystoles, prolonged QTc intervals, and greater QT dispersion in the COVID+ group suggests a potential impact of COVID-19 on cardiac electrophysiology. QT dispersion, a known marker of ventricular repolarization heterogeneity, has been associated with an increased risk of malignant arrhythmias and sudden cardiac death in various clinical settings. Previous studies have reported that prolonged QT dispersion in post-viral myocarditis and systemic inflammatory states may contribute to long-term arrhythmic complications, emphasizing the need for close monitoring in post-COVID-19 patients[19].

Correlation analysis further supported these findings by revealing a weak but significant inverse relationship between VES burden and left ventricular EF, suggesting that even subtle reductions in systolic function may contribute to increased VEA. Additionally, the observed moderate inverse correlation between hs-CRP and EF highlights the interplay between systemic inflammation and myocardial performance, potentially linking inflammatory burden to both arrhythmic risk and subclinical cardiac dysfunction. Although the correlation between age and VES burden was weak, it may reflect an age-related increase in myocardial irritability or autonomic imbalance in post-COVID patients.

From a clinical standpoint, our results underscore the importance of vigilant cardiac follow-up in recovered COVID-19 patients. Implementing routine 24-hour Holter monitoring for post-COVID individuals with palpitations, syncope, or other worrisome cardiac symptoms could facilitate early detection of significant arrhythmias and guide timely intervention.

Study Limitations

Despite these insights, certain limitations must be acknowledged. This study is retrospective in nature, and the sample size remains relatively small. Furthermore, the long-term arrhythmic risk beyond the study period is unknown. Future research should focus on larger, prospective cohorts with longer follow-up durations to better elucidate the long-term cardiovascular effects of COVID-19. We could not classify COVID-19 positive patients according to clinical severity (e.g., asymptomatic, mild, moderate, severe) due to lack of standardized symptom documentation. This may have influenced arrhythmic outcomes. Future studies should consider subgroup analysis based on COVID-19 severity to better delineate its prognostic impact. Additionally, the exact time interval between COVID-19 infection and Holter monitoring could not be uniformly determined due to

retrospective data collection. This temporal uncertainty limits the ability to distinguish between transient and persistent arrhythmic patterns.

CONCLUSION

COVID-19 infection is significantly associated with an increased burden of VEA. Fragmented QRS and a history of COVID-19 infection are independent predictors of high arrhythmic burden, whereas vaccination appears to be protective. These findings highlight the need for close cardiac monitoring and risk stratification in post-COVID-19 patients, particularly those exhibiting fragmented QRS on ECG. Vaccination appears to have a protective effect against post-COVID arrhythmias, suggesting its potential role in reducing cardiovascular complications. Larger-scale studies with extended follow-up periods are needed to better understand the long-term clinical significance of these findings.

Ethics

Ethics Committee Approval: Ethical approval for this research was granted by The Ethics Committee for Scientific Research, Faculty of Medicine, Trakya University (decision no: 02/17, date: 18.12.2021).

Informed Consent: All participants provided written informed consent before enrollment.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ç.K., M.E., F.K., S.A., N.K., Concept: Ç.K., M.E., F.K., S.A., N.K., Design: Ç.K., M.E., F.K., S.A., N.K., Data Collection or Processing: Ç.K., M.E., F.K., S.A., N.K., Analysis or Interpretation: Ç.K., M.E., F.K., S.A., N.K., Literature Search: Ç.K., M.E., F.K., S.A., N.K., Writing: Ç.K., M.E., F.K., S.A., N.K.

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REFERENCES

1. Aktoz M, Altay H, Aslanger E, Atalar E, Atar İ, Aytekin V, et al. Türk Kardiyoloji Derneği uzlaşı raporu: COVID-19 pandemisi ve kardiyovasküler hastalıklar konusunda bilinmesi gerekenler. [Turkish Cardiology Association consensus report: COVID-19 pandemic and cardiovascular diseases. Turk Kardiyol Dern Ars. 2020;48:1-87.
2. Goerlich E, Chung TH, Hong GH, Metkus TS, Gilotra NA, Post WS, et al. Cardiovascular effects of the post-COVID-19 condition. Nat Cardiovasc Res. 2024;3:118-29.
3. Qin M, Lee K, Yoo SJ. The impact of long COVID on heart rate variability: a cross-sectional study. BMC Infect Dis. 2025;25:261.
4. Xiong TY, Redwood S, Prendergast B, Chen M. Coronaviruses and the cardiovascular system: acute and long-term implications. Eur Heart J. 2020;41:1798-800.
5. Kaya Ç, Yalta K. Is constrictive pericarditis associated with long COVID, its vaccine, or both? Balkan Med J. 2024;41:411-2.
6. Tanni SE, Tonon CR, Gatto M, Mota GAF, Okoshi MP. Post-COVID-19 syndrome: cardiovascular manifestations. Int J Cardiol. 2022;369:80-1.
7. Kounis NG, Gogos C, de Gregorio C, Hung M-Y, Kounis SN, Tsounis EP, et al. "When,""where," and "how" of SARS-CoV-2 infection affects the human cardiovascular system: a narrative review. Balkan Medical Journal. 2024;41:7.
8. Turagam MK, Musikantow D, Goldman ME, Bassily-Marcus A, Chu E, Shivamurthy P, et al. Malignant arrhythmias in patients with COVID-19: ıncidence, mechanisms, and outcomes. Circ Arrhythm Electrophysiol. 2020;13:e008920.
9. Taylan G, Öztürk C, Yalta K. Fragmented QRS pattern in patients with COVID-19: further ınsights into ıts temporal and mechanistic features. Anatol J Cardiol. 2022;26:239-40.
10. Katkat F, Kalyoncuoğlu M, Karahan S, Abanus H, Turhan Çağlar FN, Karabulut D, et al. Does coexistence of fragmented QRS and cardiovascular disease have the ability to predict the mortality in hospitalized, critically ill patients with COVID-19? Anatol J Cardiol. 2021;25:803-10.
11. Podestà MA, Valli F, Galassi A, Cassia MA, Ciceri P, Barbieri L, et al. COVID-19 in chronic kidney disease: the ımpact of old and novel cardiovascular risk factors. Blood Purif. 2021;50:740-9.
12. Marques KC, Quaresma JAS, Falcão LFM. Cardiovascular autonomic dysfunction in "long COVID": pathophysiology, heart rate variability, and inflammatory markers. Frontiers In Cardiovascular Medicine. 2023;10:1256512.
13. Koc M, Sumbul HE, Gulumsek E, Koca H, Bulut Y, Karakoc E, et al. Disease severity affects ventricular repolarization parameters in patients with COVID-19. Arq Bras Cardiol. 2020;115:907-13.
14. Çoksevim M, Yenerçağ M. Investigation of autonomic dysfunction following COVID-19: the role of heart rate recovery ındices. Kafkas Tıp Bil Derg. 2024;14:296-303.
15. Pari B, Babbili A, Kattubadi A, Thakre A, Thotamgari S, Gopinathannair R, et al. COVID-19 vaccination and cardiac arrhythmias: a review. Curr Cardiol Rep. 2023;25:925-40.
16. Kole C, Stefanou E, Karvelas N, Schizas D, Toutouzas KP. Acute and post-acute COVID-19 cardiovascular complications: a comprehensive review. Cardiovasc Drugs Ther. 2024;38:1017-32.
17. Lu D, Hu J, Qian J, Cheng F. Temporal variations in QTc interval during and after COVID-19 infection: a retrospective study. BMC Cardiovasc Disord. 2024;24:738.
18. Wu K, Van Name J, Xi L. Cardiovascular abnormalities of long-COVID syndrome: pathogenic basis and potential strategy for treatment and rehabilitation. Sports Med Health Sci. 2024;6:221-31.
19. Jang SW. QTc dispersion predicts prognosis in COVID-19 disease. Korean Circ J. 2021;51:863-5.