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Holter Study for Post Coronavirus Disease Syndrome Patients Presented by Palpitation

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Abstract

Background: Post coronavirus disease (COVID) syndrome is described as persistent symptoms for several weeks after recovery from acute infection.

Aim: Of the study was to correlate the palpitation with Holter monitoring findings.

Patient and techniques: This research was happened at National Heart Institute in the period between February 2022 and August 2022. The study included 50 post-COVID syndrome patients and 48 symptomatic patients who had not infected by COVID-19 virus (control group).

Results: Supra-ventricular ectopic burden was abnormal in 2 (4%) patients in post-COVID group and in 1 (2.1%) patient in the control group and was normal in 48 (96%) patients in post-COVID group and in 47 (97.9%) patients in the control group (P value = 1.00). Total supraventricular ectopic had been greater in post-COVID group compared with control group (P value = 0.002). The ventricular ectopic burden was abnormal in 1 (2%) patient in post-COVID group and in 1 (2.1%) patient in the control group and was normal in 49 (98%) patients in post-COVID group and in 47 (97.9%) patients in the control group (P value = 1.00). Total ventricular ectopic beats were lower in post-COVID group compared with control group (P value = 0.011). Supra ventricular bigeminy, trigeminy, and Ventricular bigeminy were insignificantly different between both groups. Ventricular trigeminy was significantly lower in post-COVID group compared with control group (P value = 0.045).

Conclusion: Findings of research have demonstrated palpitation as a common symptom in survivors of COVID-19 after discharge. Our study did not find evidence of malignant or sustained arrhythmia. Palpitation was common corresponding to sinus rhythm tachycardia or nonmalignant arrhythmia like premature atrial contractions (PACs) or premature ventricular contractions (PVCs).

Keywords: Holter electrocardiography, Palpitation, Post covid

1. Introduction

World Health Organization declared Public Health Emergency of International Concern in March 2020 after receiving report from Wuhan, China, in December 2019 detailing epidemic of 27 cases of pneumonia caused by severe acute respiratory syndrome, Coronavirus (SARS-CoV). Since then, coronavirus 2019 (COVID 19) illness brought on by SARS-CoV2 virus has grown to pandemic proportions.¹ Considerable percentage of studied cases experience severe symptoms that

could result in ARDS and hypoxemic respiratory failure, shock, multi-organ failure, and death, while the majority of cases are typically asymptomatic or related to mild systemic and respiratory symptoms (fever, cough, lethargy).²

COVID 19 is a disease caused by the COVID 19 virus that causes a range of clinical presentations, from asymptomatic infection to acute respiratory failure, cytokine storm, and death. It has multiple implications for cardiovascular specialists. Early in the beginning of COVID 19 pandemic, no one would have thought that the disease might be

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chronic. Many patients have persistent symptoms after acute infection, known as long COVID or post COVID syndrome. Post-COVID syndrome is described as persistent symptoms for several weeks after recovery from acute infection. reported symptoms cover a wide range of cardiac and neurological issues, containing dysautonomia, tiredness, palpitations, chest discomfort, and shortness.³

Goal of the research was to correlate palpitation with Holter monitoring findings.

2. Patients and techniques

This research was carried out at National Heart Institute in the period between February 2022 and August 2022. The study included 50 post-COVID syndrome patients and 48 symptomatic patients who have not infected by COVID-19 virus (control group).

The study included 50 patients who got infected by COVID-19 virus (mild and moderate cases) confirmed by PCR swap and complaining of palpitations after complete recovery from the acute illness and control group consisting of 48 symptomatic patients not infected by COVID-19 virus and was indicated for Holter monitoring.

2.1. Exclusion criteria

History of cardiovascular disease before having COVID-19 infection, previous history of any type of arrhythmia before having COVID-19 infection and patients refusing consent for enrollment.

2.2. Methods

2.2.1. History taking

History was performed, and each risk factor for cardiovascular disease was assessed as hypertension, diabetes, cholesterol, dyslipidemia, smoking, BMI, history of COVID 19 infection and arrhythmogenic drugs.

2.2.2. Clinical examination

General examination: Post-COVID 19 syndrome cases had been subjected to complete physical test containing evaluation of general condition and vital signs as blood pressure, heart rate (HR) and Pulse oximetry resting and following mild exertion (10 m walk distance).

Local examination of the heart: Heart sounds, additional sounds as S3 or S4 and cardiac murmurs.

2.2.3. Laboratory investigations

Complete blood picture, thyroid profile, serum K, and hs troponin were measured for post-COVID 19 syndrome patients.

2.2.4. Standard 12-leads ECG

Was done for all studied cases.

2.2.5. Echocardiography

Was done for post-COVID 19 syndrome patients only to assess the following: Left ventricular systolic function by EF. The EF was estimated by Teicholz equation in this study. Left atrial size assessed by the M mode from a parasternal long axis view using American Heart Association (AHA) recommendation of echocardiography. A diameter less than 40 mm and a left atrium/aortic root less than 1.3 are considered normal.

2.3. HOLTER monitoring with heart rate variability

Was done for all patients using Holter contact 3 channels for 48 h to assess the following:

Minimum and maximum HR (normal HR 60–100 beat/min^{4,5}). HR variability which is a noninvasive technique for assessing control of autonomic nervous system on cardiac sinus node. Normal HR variability ratio (LF/HF) is 1.5–2. Low LF/HF ratio (<1.5) indicates parasympathetic dominance and a great LF/HF ratio (>two) reflects sympathetic dominance.⁶ The supraventricular ectopic burden was defined by dividing a total number of supraventricular ectopic beats by the total number of beats recorded through Holter monitoring. Burden greater than 10% was considered abnormal. Ventricular ectopic burden s was defined by dividing total number of ventricular ectopic beats by total number of beats recorded throughout Holter monitoring. Burden greater than 10% was considered abnormal.⁷

2.4. Statistical analysis

Version 20 of the Statistical Program for Social Science was used for data analysis (SPSS Inc., Chicago, IL, USA). Mean and standard deviation were used to characterize quantitative variables. Number and percentage were used to indicate qualitative factors. Student *t*-test was used to compare parametric quantitative variables among two groups. When frequencies fell below 5, the chi-square (χ^2) test or Fisher's exact test was used

to compare qualitative variables. In order to evaluate relationship among 2 normally distributed variables, Pearson correlation coefficients were used. *P* value of less than 0.05 or lower is regarded as significant when variable was not normally distributed.

2.5. Ethical considerations

National Heart Institute and Al Azhar University's department of medicine's Institutional Review Board both gave their approval to research. research is unfunded. There are no conflicts of interest between candidate and any supervisors. Treatment of studied cases follows guidelines set forth in Helsinki Declaration.

3. Results

There were 58 (59.1%) female and 40 (40.8%) males. The mean age was 41.9 years with a standard deviation of 16.5 years. The youngest age presented was 14 years old and the oldest year old was 88 (Table 1). There was no variation among groups concerning minimum HR using Fischer's exact test and Pearson χ^2 test. Regarding the maximum heart rate there was no variation among both groups using Fischer's exact test and Pearson χ^2 test. One patient in the control group developed a single episode of Narrow complex tachycardia with rapid

Table 1. Describe baseline characteristics of the studied patients.

	Total patients (n = 98)
Age (y)	
Mean \pm SD	41.9 \pm 16.5
Range	14–88
Sex	
Men	Forty (40.8 %)
Women	Fifty-eight (59.1%)
COVID status	
Positive	50 (51.02%)
Negative	48 (48.98%)

Table 2. Minimum and maximum heart rate between the studied groups.

	Post COVID group (n = fifty)	Control group (n = forty-eight)	<i>P</i> value	Test
Minimum HR				
Bradycardia	34 (68%)	27 (56.2%)	0.230	$\chi^2 = 1.438$
Normal	16 (32%)	21 (43.7%)		
Mean \pm SD	56.4 \pm 10.85	59.6 \pm 12.57	0.089	T = 1.36
Range	34–83	33–99		
Maximum HR				
Tachycardia	34 (68%)	26 (54.1%)	0.160	$\chi^2 = 1.974$
Normal	16 (32%)	22 (45.8%)		
Mean \pm SD	117.7 \pm 29.03	114.8 \pm 32.5	0.320	T = -0.47
Range	61–179	66–179		

onset and offset mostly supraventricular tachycardia (SVT) that lasted for 5 min (Table 2). HR Variability Ratio (LF/HF ratio) was insignificantly different between both groups (*P* = 0.326) (Table 3). Supraventricular ectopic burden was abnormal in 2 (4%) patients in post-COVID group and in 1 (2.1%) patient in the control group and was normal in 48 (96%) patients in post-COVID group and in 47 (97.9%) patients in control group (*P* = 1.0). Regarding the post-COVID syndrome patients group the mean cumulative total supraventricular ectopic beats was 1194.2 beats with standard deviation \pm 4806.1 beats. While in the control group the mean cumulative of total supraventricular ectopic beats was 1189.396 beats with standard deviation \pm 3820.367 beats. Total supra-ventricular ectopic was greater in post-COVID group compared with control group (*P* value = 0.002) (Table 4). Ventricular ectopic burden was abnormal in 1 (2%) patient in post-COVID group and in 1 (2.1%) patient in control group and was normal in 49 (98%) patients in post-COVID group and in 47 (97.9%) patients in control group (*P* = 1.0). Regarding the post-COVID syndrome patients group the main cumulative total ventricular ectopic was 1591.44 beats with standard deviation \pm 4092.6 beats. While in the control group, the main cumulative of total ventricular ectopic was 1966.3 beats with standard deviation \pm 4552.379 beats. Total ventricular ectopic beats were lower in post-COVID group compared with control group (*P* value = 0.011) (Table 5). Supraventricular bigeminy, trigeminy, and Ventricular bigeminy were insignificantly different between both groups. Ventricular trigeminy was lower in post-COVID group compared with the control group (*P* value = 0.045) (Table 6).

4. Discussion

Post COVID syndrome is described as persistent symptoms for several weeks after recovery from

Table 3. Heart rate variability ratio (low frequency/high frequency) between the studied groups.

	Post COVID group (n = fifty)	Control group (n = forty-eight)	P value	Test
HR Variability Ratio (LF/HF)				
Parasympathetic	35 (70%)	33 (68.75%)	0.326	$\chi^2 = 2.24$
Sympathetic	7 (14%)	11 (22.9%)		
Normal	8 (16%)	4 (8.3%)		
Mean \pm SD	1.35 \pm 0.49	1.50 \pm 1.07	0.441	0.595
Range	0.4–4.9	0.43–5.4		

Table 4. Total supra-ventricular ectopic and supra-ventricular burden between the studied groups.

	Post COVID group (n = 50)	Control group (n = 48)	P value	Test
Supra-ventricular burden and Total supra-ventricular ectopic beats				
Abnormal	2 (4%)	1 (2.1%)	1.00	–
Normal	48 (96%)	47 (97.9%)		
Mean \pm SD	1194.2 \pm 4806.2	1189.3 \pm 3820.4	0.002*	9.64
Range	0–30161	0–23976		

*: Statistically significant at $p \leq 0.05$.

Table 5. Total ventricular ectopic beats and ventricular ectopic burden between the studied groups.

	Post COVID group (n = fifty)	Control group (n = forty-eight)	P value	Test Kruskal
Total ventricular ectopic and ventricular ectopic burden				
Abnormal	1 (2%)	1 (2.1%)	1.00	–
Normal	49 (98%)	47 (97.9%)		
Mean \pm SD	1591.4 \pm 4092.6	1966.3 \pm 4552.4	0.011*	6.38
Range	0–17099	0–34268		
Range	0–2760	0–725		

Table 6. Bigeminy and trigeminy among studied groups.

	Post Covid group (n = fifty)	Control group (n = forty-eight)	P value	Test Kruskal
Supra ventricular bigeminy				
Mean \pm SD	8.28 \pm 37.1	10.2 \pm 56	0.066	3.38
Range	0–225	0–390		
Supra ventricular trigeminy				
Mean \pm SD	25.2 \pm 166.3	21.7 \pm 109.1	0.126	2.33
Range	0–1176	0–726		
Ventricular bigeminy				
Mean \pm SD	34.9 \pm 154.8	48.9 \pm 258.8	0.112	2.52
Range	0–1020	0–1755		
Ventricular trigeminy				
Mean \pm SD	10.04 \pm 52.5	28.3 \pm 127.2	0.045*	4.002
Range	0–363	0–860		

acute infection. reported symptoms cover wide range of cardiac and neurological issues, containing dysautonomia, tiredness, palpitations, chest discomfort, and shortness.³

Palpitation is one of the most common problems which are seen among the outpatients who present to internists and cardiologists. Although it is usually benign, it is occasionally a manifestation of potentially life-threatening conditions. Establishing the

cause of palpitations may be difficult, because the historical clues are not always accurate.⁸

Our study aimed to correlate the palpitation with Holter monitoring findings in patients with and without previous COVID-19 virus infection.

This cross-sectional study was carried out on 50 patients who infected by COVID-19 virus (mild and moderate cases) confirmed by PCR swap and complaining of palpitations after complete recovery from the acute illness. The control group included 48 patients who have no history of covid 19 infection and no history of cardiovascular diseases. All patients complain of palpitations.

To determine source of palpitations, ambulatory Holter electrocardiography (ECG) monitoring is typically performed; nevertheless, diagnostic yield of this technique is limited in studied cases whose symptoms occur infrequently.⁹

Most common Holter monitoring period is 24 h, however studies have indicated that extending recording period to 48 h improves technique's diagnostic precision. Studied cases find it uncomfortable when Holter monitoring is extended further. Furthermore, it is more challenging to document cardiac arrhythmia paroxysms throughout course of 24- or 48 h period due to their extremely varied frequency. For instance, it has been demonstrated that the daily variability of ventricular arrhythmias can approach 37%.^{10,11}

In our present study, HR Variability Ratio (LF/HF ratio) was insignificantly different between both groups ($P = 0.326$). Parasympathetic overactivity occurred in 35 (70%) patients in post COVID group and in 33 (68.75%) patients in control group while Sympathetic overactivity occurred in 7 (14%) patients in post-COVID group, and in 11 (22.9%) patients in control group ($P = 0.326$).

In correlation with our study, Asarcikli and colleagues studied the 24 h ambulatory electrocardiography recordings obtained greater than 12 weeks after the diagnosis of COVID-19 were compared with age–sex-matched healthy controls they found that patients with confirmed history of COVID-19 demonstrated increased HRV indices suggesting higher parasympathetic tone than controls.¹²

Our current research found that there was no variation among both groups concerning the minimum HR using Fischer's exact test and Pearson χ^2 test.

Our study revealed that regarding the maximum heart rate there was no variation among the groups using Fischer's exact test and Pearson χ^2 test. One patient in control group, however, develops a single episode of narrow complex tachycardia with sudden onset and offset mostly SVT that lasted for 5 min with maximum HR 197 bpm. Sinus tachycardia was common in the majority of patients in both groups.

In correlation with our results, another study¹³ found that the 2 h Holter monitoring showed 18/22 (81.8%) patients had cardiac arrhythmias. Sinus tachycardia was most prevalent arrhythmia, occurring in 17 out of 22 individuals (or 77.3%). In addition to these arrhythmias, 2/22 (9.1%) studied cases had atrial premature contraction, 1/22 (4.5%) had atrial tachycardia, and 1/22 (4.5%) had ventricular premature contraction. There were no dysrhythmias found. ⁶This study differs from our study in the following they use a very short period for Holter monitoring (2 h Holter monitoring), patient with structural heart diseases and chronic medical illness are not excluded. This study correlate palpitations to Psychological distress (anxiety and depression).

The major findings of our study revealed that Palpitation was common corresponding to sinus rhythm tachycardia or nonmalignant arrhythmia such as premature atrial contractions (PACs) or premature ventricular contractions (PVCs). In post COVID-19 patients the total PACs were significantly higher while PVCs and ventricular trigeminy were significantly lowers than in non-COVID-19 infected patients.

In correlation with our study another study¹⁴ found that total of 51 people, with mean years old of 42 and female to male ratio of 65%, were monitored 75 (IQR 34–126) days after positive COVID-19 test. The median monitoring duration was 13.2(IQR 10.5–13.8). Atrial fibrillation, atrial flutter, sustained supraventricular tachycardia, sustained ventricular tachycardia, and intranodal atrioventricular block were not seen in any subject. 1 subject had 15.4% ventricular ectopic and had 2.8% supraventricular ectopic burden. Nearly all patients (96%) had ectopic burden of less than 1%. ¹⁴This study differs from our study that they use a prolonged period for Holter monitoring. There was no control group in this study.

In our study hs troponin level measurement and echocardiography assessment were done to search for evidence of cardiovascular complication caused by COVID-19 virus. All post COVID patients show normal echocardiography assessment and have

normal hs troponin level. All patients in the control group were recruited based on elective base and having no history of cardiovascular diseases.

Researchers found that evaluation of post covid syndrome patient shows possible causes of arrhythmia that may persist in those patients. This causes include increased cardiometabolic demand, corticosteroid use and dysregulation of the renin–angiotensin–aldosterone system (RAAS). Myocardial fibrosis or scarring, and resultant cardiomyopathy from viral infection, can lead to re-entrant arrhythmias. COVID-19 may also sustain arrhythmias due to a heightened catecholaminergic state due to cytokines such as IL-6, IL-1 and tumor necrosis factor- α , which can prolong ventricular action potentials by modulating cardiomyocyte ion channel expression. Autonomic dysfunction.¹⁵

Researches done in the past on SARS studied cases who have recovered have revealed that palpitation symptoms might persist for very long period after discharge. Additionally, current researches have revealed that some COVID-19 studied cases continue to experience palpitations three months after being discharged.¹⁶

4.1. Limitations

4.1.1. Our study has several limitations

This was a single-center study with small sample size. None of our studied patients have evidence of cardiac complication e.g., myocarditis. Pulmonary function was not evaluated in the present study. Mild to moderate except severe degree of COVID-19 patients were included in our study. Only patients, who did undergo Holter monitoring, were included in the study population, so the results may not represent whole post-COVID population. Three cases with hypokalemia and three cases with severe anemia were not excluded from the study.

4.2. Conclusion

Palpitation was common corresponding to sinus rhythm tachycardia or non-malignant arrhythmia such as PACs or PVCs. In post-COVID 19 patients the total PACs were significantly higher while PVCs and ventricular trigeminy were significantly lowers than in non COVID-19 infected patients.

Disclosure

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Conflicts of interest

The authors declared that there were no conflicts of interest.

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