



Does Cardiac Arrhythmia Predict Worse Outcome in Mild or Moderate Covid-19 Infection?

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Abstract

Covid-19 is a multisystem disease with the lungs being predominantly affected. Cardiac involvement is mostly seen as a rise in troponins, arrhythmias, and ventricular dysfunction. This study aimed to estimate the incidence of arrhythmias seen in Covid-19 infection and assess if arrhythmias predict worsening or mortality. Prospective observational study involving patients with mild to moderate Covid illness admitted in a tertiary care centre. Among the 85 patients (Mean age 45.8 + 14.1 years; 75.31% men), worsening of Covid-19 illness was seen in 29 (34.1%) patients. New onset arrhythmias were detected on Holter in 9 (10.5%) patients. Supraventricular tachycardia was seen in 7 (8.2%) patients of whom 6 showed worsening which was statistically significant (p -value-0.006). Risk factors associated with worsening on univariate analysis were male gender (OR [95%CI]=6.93(1.49–32.31), p -value – 0.014), new onset supraventricular tachycardia (OR [95% CI] = 14.35 [1.64–125.94], p -value – 0.016) and D-dimer elevation (OR [95% CI] = 1.00(1.00–1.01), p -value – 0.02). On multivariate analysis D-dimer (OR [95% CI]= 1.00(1.00–1.01; p -value 0.046) and supraventricular arrhythmias (OR [95% CI] = 11.12 (1.22–101.14); p -value – 0.033) were independently associated with worsening. Covid-19 infection can lead to cardiac arrhythmias. The development of supraventricular tachycardia in patients with Covid-19 infection predicts higher morbidity and worsening.

Keywords Covid-19 · Arrhythmias · Worsening · Deterioration

Background

The coronavirus disease—2019 (Covid-19) pandemic has so far affected more than 660 million individuals and claimed more than 6.69 million lives across the globe as of January 2023 [1]. Covid-19 has been recognized as a multisystem

disease with predominant pulmonary involvement [2]. Cardiac manifestations include a rise in troponin levels, arrhythmias, and ventricular dysfunction [3, 4]. Studies have suggested increased mortality in Covid-19 patients with cardiac involvement [5, 6]. Hospitalised patients with Covid-19 have an increased incidence of cardiac arrhythmias, many of whom required intensive care [7, 8]. However, secondary infections, hypoxia, and use of inotropes in critically ill

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patients could result in arrhythmias that were not primarily due to Covid-19 infection. We conducted a prospective observational study to estimate the incidence of arrhythmias in patients with mild to moderate Covid illness and sought to study its association with morbidity and mortality.

Methods

Study Population

This was a prospective observational cohort study. Adults (> 18 years) admitted to our tertiary care centre with Covid-19 infection (diagnosed based on a positive RT-PCR test from a nasal swab) from December 2020 to June 2021 were included. Criteria for mild and moderate Covid-19 as followed in our institution at the time of study approval were as follows — Mild COVID-19: There was no clinical or radiological evidence of pneumonia and RR < 24/min, SpO₂ > 94% on room air, BP > 90 mm Hg systolic. Moderate COVID-19: RR < 30/min, SpO₂ > 90%, systolic BP < 90 systolic (not requiring inotropic support). The following patients were excluded: (i) patients with severe Covid-19 disease requiring ICU admission, (ii) patients with pre-existing arrhythmias or known to have had treatment for arrhythmias, (iii) patients who did not provide informed consent, (iv) those with skin disease over the chest precluding Holter patch application, and (v) technical difficulties in pairing the Holter patch with mobile phone app (described below). Patients with pre-existing arrhythmias were identified based on their clinical history, prior ECGs and health record documentation.

Hospital Course and Treatment

All patients were treated according to the institutional protocol for Covid-19 illness which was constantly modified, based on the latest evidence prevalent during the course of the study. On admission, informed consent was obtained from the subjects who were willing to participate in the study. Demographic and clinical data for all patients were collected. All patients underwent baseline ECG, D-dimer, CRP, and Cardiac biomarker [Troponin T] as part of standard care.

Arrhythmia Monitoring

A commercially available disposable adhesive patch with an embedded biosensor (WebCardio, Gadgeon Medical Systems, Kochi, Kerala, India) was applied to the patient's anterior chest on the left side within 24 h of hospital admission. This biosensor continuously recorded two ECG channels and sends the data every 12 h to its companion app on

the patient's phone, with which the sensor was paired. The app transferred the data to the cloud. The system was used to monitor heart rhythm for up to 7 days. The data were analysed manually by trained technicians and a report was generated. Important events on the Holter were tagged. The whole recording was available online for review by the treating physician. This FDA-approved device has been found to be comparable if not superior to the standard Holter in terms of electrogram quality and arrhythmia detection [9, 10].

Outcomes

An event was either clinical deterioration or death in hospital. Clinical deterioration was defined as the need for vasopressors, oxygen supplementation, and non-invasive or invasive mechanical ventilation. Arrhythmias were defined as at least 1 episode of supraventricular tachycardia (SVT), new-onset atrial fibrillation (AF)/atrial flutter (AFL) lasting longer than 30 s, sinus pauses lasting longer than 2 s, second- or third degree atrioventricular block, ventricular tachycardia, and/or ventricular fibrillation [11, 12]. AF and AFL were considered as SVT for the analysis. Arrhythmias that occurred after the patient developed a secondary infection or while on inotropes or invasive ventilation were excluded from the analysis. Standard definitions were used for diabetes mellitus, hypertension, acute respiratory distress syndrome, obesity, chronic kidney disease, chronic respiratory disease and immunocompromised state [13–19].

Statistical analysis

Continuous data was reported as mean \pm standard deviation or as median (interquartile range [IQR]). Number of patients and percentages were presented for categorical data. Based on the normality of the data, *t* test and non-parametric Mann–Whitney *U* test were used to compare the differences between the two groups. Pearson Chi-square test was used to assess association between the categorical variables. Binary logistic regression was used to assess association of the factors on worsening status. Results of logistic regression were reported as odds ratio (OR) with 95% confidence interval (CI). A two-sided alpha of 0.05 was taken as the level of significance. All analyses were done using SPSS software Version 21.0 (Armonk, NY: IBM Corp).

Results

A total of eighty-five patients were included in the study. Mean age was 45.8 ± 14.1 years and 64 (75.3%) were men. The baseline characteristics of the study population are shown in Table 1. The demographics of the study

Table 1 Patient characteristics

	Number = 85 n (%)
Age in years; Mean (SD)	45.8 (14.1)
Male	64 (75.3)
BMI (kg/m ²); Mean (SD)	27.0 (4.4)
DM	28 (32.9)
HT	25 (29.4)
Ischemic heart disease	3 (3.5)
OSA	1 (1.2)
Obesity	55 (64.7)
Chronic respiratory illness	7 (8.2)
CKD	2 (2.4)
Heart failure	1 (1.2)
RHD	2 (2.4)
Cancer	3 (3.5)
Immunocompromised state	1 (1.2)
Significant medical history	4 (4.7)
Outcome – worsening	29 (34.1)
New onset arrhythmias	9 (10.6)

population are shown in Table 2. Five patients out of 85 had pre-existing heart disease (5.9%). This included 3 patients with ischemic heart disease (2 with double vessel coronary artery disease, 1 who was treadmill positive), 2 patients with rheumatic heart disease (1 had mitral stenosis while the other patient who had a recent heart failure admission had earlier undergone a mitral valve replacement for mitral regurgitation). Two out of these 5 patients who had pre-existing heart disease developed arrhythmia during the study. One of these patients had coronary artery disease and went on to clinically worsen before he eventually recovered. The other patient who developed an arrhythmia was the patient post MVR. He never had any clinical worsening despite the arrhythmia.

Clinical deterioration was noticed in 29 (34.1%) patients (Table 1). 83.5% of patients had mild disease and 16.5% had moderate disease. Worsening was noted in 23.9% of mild cases and 85.7% of moderate cases. Holter was normal in 76 (89.4%) patients. It was normal in 23 of the 29 patients [79.3%] who subsequently developed worsening of their illness. Mean age in both the worsening and non-worsening groups was similar. Male gender was strongly associated with clinical deterioration (OR [95% CI] = 6.93 [1.49–32.31]; *p*-value = 0.014). New onset SVT predicted worsening (OR [95% CI] = 14.35 [1.64–125.94]; *p*-value = 0.016) and so did D-dimer elevation (OR [95% CI] = 1.00 (1.00–1.01); *p*-value of 0.02) (Table 3). Troponin T predicted worsening in the univariate analysis but was not found to be significant in the multivariate analysis. NT-pro BNP was elevated in the worsening group, but it did not reach statistical significance (*p*-value = 0.158).

On multivariate analysis, D-dimer (OR [95% CI] = 1.00 (1.00–1.01); *p*-value of 0.046) and supraventricular arrhythmias (OR [95% CI] = 11.12 [1.22–101.14]; *p*-value = 0.033) were independently associated with worsening.

New onset arrhythmias detected on Holter were seen in 9 (10.5%) patients. Of these 9 patients, 2 patients had no clinical deterioration. One of these patients had transient CHB and the other had atrial fibrillation. There were 7 patients with arrhythmia who clinically deteriorated of which 2 had died. One patient died after being shifted to the ICU while the other passed away in the high dependency unit area. Of the 9 patients with arrhythmia, 7 had SVT, 1 had VT and 2 had AV blocks. One patient had more than one type of arrhythmia. This patient had NSVT and ill sustained SVT, not associated with any symptoms. He subsequently deteriorated briefly requiring oxygen supplementation [2 L/min by mask] but did not require invasive or non-invasive ventilation. He was closely monitored and given supportive care. His general condition improved over the next 3 days with no further arrhythmia. He was stabilised and discharged subsequently and is doing well on clinical follow-up. Nonsustained VT and atrial flutter [AFL] were seen in 1 (1.2%) patient each. Atrial fibrillation [AF] was seen in 2 (2.4%) patients of whom 1 worsened and 1 remained stable. Supraventricular tachycardia (SVT) was seen in 7 (8.2%) patients of whom 6 showed worsening of disease which was statistically significant with a *p*-value of 0.006. Two participants developed AV-block (2.4%), of which Type II second-degree AV block with intermittent complete heart block [CHB] was seen in 1 (1.2%) patient who had no comorbidities and did not show any clinical outcome worsening. Another patient with obesity, hypertension, diabetes and sleep apnoea had a 2: 1 AV-block with intermittent CHB (1.2%) and subsequently worsened. The former patient with no comorbidities and complete heart block had not been on any medication while the latter had been on calcium channel blocker (amlodipine) for hypertension. There was no correlation noticed between arrhythmia development and duration of hospital stay.

Discussion

In this prospective observational study among patients hospitalized with mild to moderate Covid-19 infection, new onset arrhythmias were noted in 9 (10.6%) patients, most of which were SVTs. This is lower than that reported in previous studies, which ranged from 16.7 to 28.0% [7, 20]. In one study, atrial arrhythmias were more frequent in mechanically ventilated patients (17.7% vs 1.9% otherwise) [21]. Another study showed that AF-related symptoms were the most common reason for electrophysiology consultations during the pandemic peak in New York City at Columbia University

Table 2 Demographics and clinical characteristics

	Total (n = 85)	Worsening (n = 29)	Non-worsening (n = 56)	p-value
Age (years) ‡	45.75 (14.08)	48.48 (16.05)	44.34 (12.8)	0.235
Male (%)	64 (75.3)	27 (93.1)	37 (66.1)	0.007
BMI ‡	27.04 (4.4)	27.31 (4.17)	26.89 (4.61)	0.684
DM (%)	28 (32.9)	10 (34.5)	18 (32.1)	0.828
HT (%)	25 (29.4)	7 (24.1)	18 (32.1)	0.443
Pre-existing heart disease (%)	5 (5.9)	3 (10.3)	2 (3.57)	0.23
Chronic respiratory illness (%)	8 (9.41)	3 (10.3)	5 (8.9)	1.000
Obesity (%)	55 (64.7)	16 (55.1)	39(69.6)	0.186
CKD (%)	2 (2.4)	1 (3.4)	1 (1.8)	1.000
Immunocompromised State (%)	4 (4.71)	2 (6.9)	2 (3.57)	0.603
Significant medical history (%)	4 (4.7)	1 (3.4)	3 (5.36)	1.000
Medications				
ACE inhibitors (%)	4(4.7)	2 (6.9)	2 (3.5)	0.603
ARBs (%)	9 (10.6)	2 (6.9)	7 (12.5)	0.712
Statins (%)	14 (16.5)	6 (20.7)	8 (14.3)	0.45
Beta-blockers (%)	10 (11.8)	4 (13.8)	6 (10.7)	0.729
CCBs (%)	17 (20)	5 (17.2)	12 (21.4)	0.647
Amiodarone (%)	1 (1.2)	1 (3.4)	0 (0)	0.341
Investigations				
Hb‡	13.22 (2.084)	13.55 (1.8)	13.05 (2.2)	0.299
Troponin-T [‡]	7 (4.5–11.0)	9 (6–11.5)	6 (4–10)	0.044
D-Dimer [‡]	448 (324.5–920)	618 (403.5–1153)	430.5 (296–786)	0.017
NT pro-BNP [‡]	52(17.5–204.5)	141 (29.5–304)	42.5 (14.5–155.75)	0.158
CRP [‡]	27 (8–75)	63 (45–109)	14 (3.25–57.5)	0.566
ECG				
PR interval (ms) [‡]	143.84 (26.40)	142.38 (31.54)	144.59 (27.24)	0.717
QTc Interval (ms) [‡]	432.95 (31.66)	440.55 (38.19)	429.02 (27.24)	0.759
Holter				
Nil findings	76 (89.4)	23 (79.3)	53 (96.4)	0.028
NSVT	1 (1.2)	1 (3.4)	0 (0)	0.341
Atrial fibrillation	2 (2.4)	1 (3.4)	1 (1.7)	1.000
Atrial flutter	1 (1.2)	1 (3.4)	0 (0)	0.341
SVT	7 (8.2)	6 (20.7)	1 (1.7)	0.006
Type II 2 nd degree AV block	1 (1.2)	0 (0)	1 (1.7)	1.000
2:1 AV block	1 (1.2)	1 (3.4)	0 (0)	0.341
Complete AV block	2 (2.4)	1 (3.4)	1 (1.7)	1.000

Values are presented as number (percentage) and the *p*-value is obtained from chi-square or Fisher's exact test

‡ Values are presented as Mean (SD) and *p* value is obtained from *t* test

‡ Values are presented as Median (25th percentile, 75th percentile) and *p*-value is obtained from Mann–Whitney *U* test

Table 3 Factors associated with outcome

	Univariate		Multivariate	
	Odds ratio (95 CI)	<i>P</i> value	Adjusted odds ratio (95 CI)	<i>P</i> value
Gender	6.93 (1.49, 32.31)	0.014	4.49 (0.93, 21.69)	0.062
SVT	14.35 (1.64, 125.94)	0.016	11.12 (1.22, 101.14)	0.033
Troponin -T	1.02 (0.98, 1.05)	0.238	1.018 (0.98, 1.05)	0.238
D-Dimer	1.01 (1.00, 1.02)	0.020	1.001 (1.00, 1.02)	0.046

for Covid-19 patients (31%), with only 13% of these having history of AF [22]. The comparatively lower incidence of SVT of 8.2% in our study is probably because the patients were younger and had less severe disease at admission. Six of the seven patients with documented SVT of undetermined aetiology, clinically deteriorated in our study. This included the 1 patient who developed AFL. AF was seen in 2 patients (2.4%) of whom 1 had clinical worsening. In a larger study on 700 patients hospitalized with Covid-19, incident AF was detected among 25 patients on cardiac telemetry. This study however included patients with more severe systemic illness requiring ICU care and hence a higher likelihood of developing cardiac arrhythmias due to causes other than the infection per se [23].

Significant conduction system disease was seen in 2 of our patients. One of these patients had transient findings [Mobitz type II second degree and CHB] that did not persist beyond 24 h. On follow-up, the conduction abnormalities recovered completely, and the patient is asymptomatic on 1 year follow-up. The patient who had persistent findings was the one with more comorbidities (diabetes mellitus, hypertension, obesity, sleep apnoea). This patient with 2:1 AV-block and CHB suffered further clinical deterioration. These findings suggest that the conduction disease could be secondary to transient myocarditis that has the potential to resolve completely in those with less comorbidities.

Multiple factors have been identified as risk factors for worsening and mortality in previous studies. Frequently identified risk factors were obesity, diabetes, hypertension, troponin elevation, and D-dimer [4, 6, 8]. Except for D-dimer, none of these factors were significantly associated with worsening in our study (Table 4). The plausible reasons could be a younger population with lesser comorbidities, a small sample size, a low event rate, and a different definition of worsening in our study. NT-pro BNP and Troponin T though elevated in the worsening group, did not reach statistical significance on multivariate analysis. The elevation could have been due to transient heart failure secondary to Covid myocarditis. Echocardiography and a 6-min walk test if performed would have added value towards ascertaining how sick the patients in the study cohort were. Unfortunately, since these tests would require more personnel who perform them to be potentially exposed to the risk of Covid, they were not done. Vaccination against Covid-19 was beginning to be available during the latter part of the study period. However, none of the patients included in the study had prior vaccination. Hence the possible confounding effect of Covid vaccination on causing carditis/arrhythmia in the study population did not exist.

The present study was prospective and included patients with Covid-19 illness without ICU requirements on admission. By doing so, we were able to identify arrhythmias that could primarily be attributed to Covid-19 and not secondary

Table 4 Risk factors for arrhythmias

	Total (n=85)	Arrhythmias (n=9)
Age (years) [‡] (mean/SD)	45.75 (14.08)	65.5 (11.013)
Male (%)	64 (75.3)	7 (10.9)
BMI (mean/SD) [‡]	27.04 (4.4)	27.89 (5.798)
DM (%)	28 (32.9)	5 (55.6)
HT (%)	25 (29.4)	3 (33.3)
CAD (%)	3 (3.5)	1 (11.1)
OSA (%)	1 (1.2)	1 (11.1)
Obesity (%)	55 (64.7)	5 (55.6)
Chronic respiratory illness (%)	7 (8.2)	3 (33.3)
CKD (%)	2 (2.4)	0
Heart failure (%)	1 (1.2)	0
RHD (%)	2 (2.4)	1 (11.1)
Cancer (%)	3 (3.5)	0
Immunocompromised state (%)	1 (1.2)	0
Significant medical history (%)	4 (4.7)	0
Medications		
ACE inhibitors (%)	4 (4.7)	1 (11.1)
ARBs (%)	9 (10.6)	1 (11.1)
Statins (%)	14 (16.5)	2 (22.2)
Beta-blockers (%)	10 (11.8)	2 (22.2)
CCBs (%)	17 (20)	3 (33.3)
Amiodarone (%)	1 (1.2)	0
Investigations		
Chest infiltrates (%)	45 (52.9)	8 (88.9)
Haemoglobin	13.22 (2.084)	12.89 (1.616)
Troponin ^ϕ	7 (4.5–11)	11 (8.5–20.50)
D-Dimer ^ϕ	448 (324.5–920)	533(392–1187)
NT pro-BNP ^ϕ	52 (17.5–204.5)	133 (29.5–297.50)
CRP ^ϕ	27 (8–75)	58.5 (27–80.5)

Values are presented as number (percentages)

[‡] Values are presented as Mean (SD)

^ϕ Values are presented as Median (25th percentile, 75th percentile)

to inotropes or ventilatory disturbances. This was unlike most other studies which were retrospective and predominantly included ICU patients. The second major strength of this study was the use of the 7-day Holter which monitored patients during the entire period of hospitalisation and thereafter.

Our study had several limitations which included most importantly a small sample size. This could have underestimated the number and type of arrhythmias detected. It also limited its ability to identify predisposing factors for cardiac arrhythmias in Covid-19 infection. This was a single tertiary care centre study and so our findings may not be generalizable to all Covid-19 patients across the world. Some patients in the study had underlying conditions like pre-existing heart disease

that could make them prone to developing arrhythmias. However, these patients had no arrhythmias at or before the time of admission and hence satisfied inclusion criteria. Ideally these patients should have been excluded as it is possible that the arrhythmia they developed was secondary to their pre-existing heart disease and not Covid-related. Of the 3 patients with IHD, 1 patient developed an SVT and clinically worsened. Similarly, of the 2 patients with RHD the patient post MVR with prior heart failure admission developed SVT but had no clinical deterioration. Inclusion of these patients into the analysis could have influenced the outcome.

Another limitation of this study is that it lacks a control group containing patients with non-Covid illnesses. Ideally doing so would have helped us ascertain if there was a significant difference in the incidence of arrhythmias between the 2 groups. Although literature describes specific arrhythmias as poor prognostic factors for specific conditions, like ventricular arrhythmias in patients with polymyositis, there are no studies looking at arrhythmias in non-covid medical illness [24].

Conclusions

Covid-19 infection can lead to cardiac arrhythmias. The development of supraventricular tachycardia in patients with Covid-19 infection is associated with higher morbidity and clinical deterioration. Transient cardiac conduction defects that occur in Covid-19 patients do not warrant pacing. However, conduction defects that occur in patients with multiple comorbidities are likely to be persistent, requiring permanent pacing.

Abbreviations *RT-PCR*: Reverse transcriptase polymerase chain reaction; *SpO₂*: Saturation pressure of oxygen; *BP*: Blood pressure; *RR*: Respiratory rate; *CRP*: C reactive protein; *CK-MB*: Creatine kinase-MB; *ECG*: Electrocardiogram; *SVT*: Supraventricular tachycardia; *AF*: Atrial fibrillation; *AFL*: Atrial flutter; *ICU*: Intensive care unit; *IQR*: Interquartile range; *OR*: Odds ratio; *CI*: Confidence interval; *VT*: Ventricular tachycardia; *AV Block*: Atrioventricular block; *CHB*: Complete heart block; *MVR*: Mitral valve replacement; *IHD*: Ischemic heart disease; *CAD*: Coronary artery disease

Author contribution AS was involved in study design, data collection, interpretation, preparation of manuscript and technical support.

AM was involved in study design, data collection, interpretation, review of manuscript and technical support.

SCSP was involved in study design, data collection, interpretation, review of manuscript and technical support.

RK was involved in interpretation of study result and technical support, and was a major contributor in writing the manuscript.

RJ was involved in data collection, interpretation, and technical support.

MS was involved in data collection, interpretation, and technical support.

DC was involved in study design, interpretation, review of manuscript and technical support, guide, administrative and technical support.

JR was involved in study design, data collection, interpretation, review of manuscript and technical support, guide, administrative and technical support.

Data availability The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval Ethics approval was taken from the Institutional Review Board (Research and Ethics Committee, Registration No: ECR/326/INST/TN/2013 Re Reg – 2019 Issued under Rule 122D of the Drugs & Cosmetics Rules 1945, Govt. of India) of the Christian Medical College, Vellore, India, IRB Min no. 13379 dated 23.09.2020.

Consent to participate Consent taken to participate was in written form.

Competing interests The authors declare no competing interests.

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