ORIGINAL ARTICLE

The Effect of COVID-19 Infection on Ventricular Pacing Threshold among Patients with Pacemakers: A Retrospective Observational Study

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Objective: To study the effect of COVID-19 infection on ventricular pacing threshold among patients with pacemakers.

Materials and Methods: Patients with pacemakers were retrospectively recruited at the device clinic, Central Chest Institute of Thailand between January 2022 and September 2023. Those patients were classified into two groups according to a history of previous COVID-19 infection. The primary outcome was the proportion of patients with high ventricular pacing threshold, and the secondary outcome was the mean ventricular pacing threshold between both groups. The chi-square or Fisher's exact test was used to compare the primary outcome between both groups. The independent t-test was used to compare the secondary outcome between both groups.

Results: One hundred twenty-two patients were enrolled. The average age was 69.4 years. Nearly 60% of these patients were implanted with pacemakers because of atrioventricular block. Of the 122 patients, there were 54 patients in the COVID-19 group and 68 patients in the non-COVID-19 group. The present study demonstrated that two patients (3.7%) in the COVID-19 group and two patients (2.9%) in the non-COVID-19 group had high ventricular pacing threshold, respectively. There were more patients with high ventricular pacing threshold in the COVID-19 group than those in the non-COVID-19 group with no statistical significance (adjusted odds ratio 1.55, 95% confidence interval [CI] 0.19 to 12.81, p=0.69). Compared with the non-COVID-19 group, there were comparable mean ventricular pacing threshold in the COVID-19 group, with a mean absolute difference of –0.03 (95% CI –0.14 to 0.09, p=0.65).

Conclusion: Patients with pacemakers had no significant increase in ventricular pacing threshold after recovering from COVID-19 infection.

Keywords: Capture threshold; Long COVID; Pacemakers; Pacing threshold; Post-COVID-19

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The sequelae of coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), are increasing in the world. There are many cardiovascular diseases associated with COVID-19 such as myocarditis, heart failure (HF), cardiac arrhythmia, acute coronary syndromes (ACS), and pulmonary embolism[1,2]. The possible causes of myocardial injury in patients with COVID-19 were cytokine storm and myocardial dysfunction resulting from the direct effect of SARS-CoV-2 infection[3].

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A previous study demonstrated that 5.5% had acute ischemic heart disease, and 5.4% had acute HF, which were the most common cardiac events, while 0.3% of those patients had acute myocarditis or pericarditis during COVID-19-associated hospitalization[4]. In addition, previous trials also demonstrated that patients with COVID-19 were associated with increased risk of atrial fibrillation (AF) during hospitalization as well as patients with cardiac implantable electronic devices (CIEDs) had increased AF episodes in high COVID-19 prevalence states in the United States of America (USA) during COVID-19 pandemic[5,6]. However, a recent study showed that patients receiving CIEDs had no significant increased risk of subclinical AF three months after COVID-19 infection[7].

Of note, patients recovering from COVID-19 infection had several cardiovascular consequences[8,9]. Previous trials found evidence of myocarditis and late gadolinium enhancement (LGE) reflecting myocardial scar after recovery from COVID-19 infection[10,11]. However, data about the effect of

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ventricular pacing threshold after recovering from COVID-19 infection are lacking. The present trial was conducted to study the effect of COVID-19 infection on ventricular pacing threshold among patients with pacemakers.

Materials and Methods

Consecutive patients with pacemakers were retrospectively recruited from the database of the device clinic at Central Chest Institute of Thailand between January 2022 and September 2023. Those patients with previous cardiac surgery, pacing threshold of 2.0 volts (V) or greater at 0.4 milliseconds (ms), new ventricular lead implantation/replacement within three months, conduction system pacing, acute myocarditis, recent ACS within one month, cardiac sarcoidosis, cardiac amyloidosis, stress-induced cardiomyopathy, pregnancy, and concealed study participation were excluded.

Demographic and clinical data of the study patients were retrieved from electronic medical records. Those patients were classified into two groups according to a history of previous COVID-19 infection based on antigen test kits (ATK) results or patient self-report when ATK data were unavailable. Patients with previous COVID-19 infection (the COVID-19 group) were enrolled after three months of the onset of COVID-19 infection. Those patients without previous COVID-19 infection (the non-COVID-19 group) were enrolled during follow-up visit at the device clinic. Baseline demographic data such as age, sex, medical history, indication of pacemakers, renal function, and left ventricular function were collected. Patients' device interrogation data were collected during follow-up visit at the device clinic for at least six months after enrollment in the non-COVID-19 group and after the onset of COVID-19 infection in the COVID-19 group.

The primary outcome in the present study was the proportion of patients with high ventricular pacing threshold between both groups. The ventricular pacing threshold was defined as the minimum amount of energy needed to electrically capture the myocardial tissue[12]. High ventricular pacing threshold was defined as increased ventricular pacing threshold for 0.5 V or more, absolute ventricular pacing threshold of 2.0 V or more at 0.4 ms or requirement of higher pulse width for pulse amplitude pacing threshold testing. The secondary outcome was the mean ventricular pacing threshold between both groups.

The present study protocol was approved by the Human Research Ethics Committee of Central Chest Institute of Thailand (No. 011/2567). The study was conducted in compliance with the Declaration of Helsinki and the International Conference on Harmonization for Good Clinical Practice Guidelines.

Statistical analysis

The author specified 0.05 for type I error and 0.10 for type II error, so the power of this study was 90%. The author estimated 0.1 and 0.4 for the proportion of patients with high ventricular pacing threshold in non-COVID-19 and COVID-19 groups, respectively. Missing data were expected for 20%. The author compared two independent proportions of study patients using a chi-square test and a sample size of 102 patients or more was estimated.

Descriptive statistics were used to analyze baseline demographic and clinical data. Categorical data were analyzed using a chi-square test or Fisher's exact test and continuous data were analyzed using an independent t-test. The categorical data were presented as numbers and percentages, and the continuous data were presented as mean and standard deviation (SD). The chi-square test or Fisher's exact test was used to compare the primary outcome between COVID-19 and non-COVID-19 groups and reported as adjusted odds ratio (OR) and 95% confidence interval (CI). Independent t-test was used to compare the secondary outcome between both groups. A p-value less than 0.05 was considered statistically significant.

Results

One hundred twenty-two patients were recruited at the device clinic, Central Chest Institute of Thailand between January 2022 and September 2023. The average age was 69.4 years. About one-third of these patients were males. Most of these patients had hypertension and dyslipidemia. About one-fifth of these patients had diabetes mellitus and chronic kidney disease. Nearly 60% of these patients were implanted with pacemakers because of atrioventricular block. The average left ventricular ejection fraction (LVEF) was 65.9%. The average follow-up time was 10.7 months.

Of the 122 patients, there were 54 patients in the COVID-19 group and 68 patients in the non-COVID-19 group (Figure 1). Patients in the non-COVID-19 group had more hypertension, and dyslipidemia than those in the COVID-19 group. Baseline characteristics are shown in Table 1.

There were two patients (3.7%) in the COVID-19

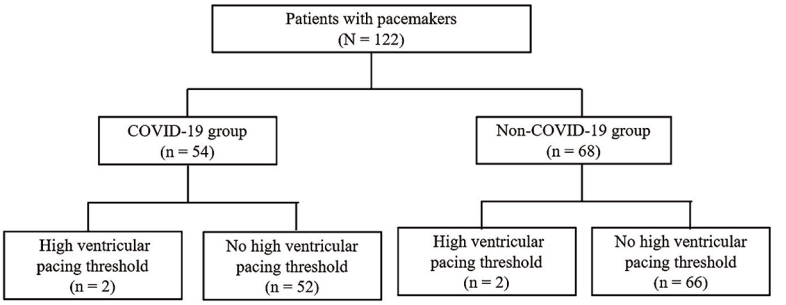

Figure 1. Flow diagram of study patients.

Table 1. Baseline characteristics of the study patients

Demographic data	Total (n=122)	COVID-19 (n=54)	Non-COVID-19 (n=68)	p-value
Age (years); mean ± SD	69.4±14.8	66.9±17.2	71.4±12.3	0.09
Male sex; n (%)	42 (34.4)	15 (27.8)	27 (39.7)	0.24
Medical history; n (%)				
Diabetes mellitus	25 (20.5)	11 (20.4)	14 (20.6)	>0.99
Hypertension	85 (69.7)	32 (59.3)	53 (77.9)	0.04*
Dyslipidemia	79 (64.8)	25 (46.3)	54 (79.4)	<0.01*
Atrial fibrillation	10 (8.2)	6 (11.1)	4 (5.9)	0.34
CAD	12 (9.8)	3 (5.6)	9 (13.2)	0.27
CKD	29 (23.8)	15 (27.8)	14 (20.6)	0.48
Previous stroke/TIA	5 (4.1)	4 (7.4)	1 (1.5)	0.17
History of HF	3 (2.5)	1 (1.9)	2 (2.9)	>0.99
Pulmonary disease	3 (2.5)	3 (5.6)	0 (0.0)	0.08
Indication of pacemakers; n (%)				
Sick sinus syndrome	52 (42.6)	19 (35.2)	33 (48.5)	0.20
AV block	70 (57.4)	35 (64.8)	35 (51.5)	0.20
Implantation duration (years); mean±SD	9.5±5.4	9.1±4.6	9.8±5.9	0.47
Serum creatinine (mg/dL); mean±SD	0.9±0.3	1.0±0.3	0.9±0.3	0.43
eGFR (mL/min/1.73 m^2); mean±SD	74.4±22.5	74.2±25.0	74.6±20.4	0.93
LVEF (%); mean±SD	65.9±11.1	66.3±11.4	65.6±11.0	0.75

AV=atrioventricular; CAD=coronary artery disease; CKD=chronic kidney disease; eGFR=estimated glomerular filtration rate; HF=heart failure; LVEF=left ventricular ejection fraction; n=numbers; SD=standard deviation; TIA=transient ischemic attack

* $p<0.05$ indicates statistical significance

Table 2. Primary and secondary outcomes of patients with pacemakers between COVID-19 and non-COVID-19 groups

	COVID-19 (n=54)	Non-COVID-19 (n=68)	Adjusted OR (95% CI)	p-value
Primary outcome				
High ventricular pacing threshold; n (%)	2 (3.7)	2 (2.9)	1.55 (0.19 to 12.81)	0.69
Secondary outcome			Mean absolute difference (95% CI)	
Ventricular pacing threshold (V)¶, mean±SD	0.90±0.32	0.93±0.31	−0.03 (−0.14 to 0.09)	0.65

CI=confidence interval; n=numbers; OR=odds ratio; SD=standard deviation

¶ Ventricular pacing threshold indicates pulse amplitude pacing threshold testing at pulse width 0.4 ms

Variables for adjusted: hypertension, dyslipidemia

group and two patients (2.9%) in the non-COVID-19 group who had a high ventricular pacing threshold. There were more patients with high ventricular pacing threshold in the COVID-19 group than those in the non-COVID-19 group with no statistical significance (adjusted OR 1.55, 95% CI 0.19 to 12.81, p=0.69) (Table 2).

Compared with the non-COVID-19 group, there

were comparable mean ventricular pacing threshold in the COVID-19 group, with a mean absolute difference of –0.03 (95% CI –0.14 to 0.09, p=0.65) (Table 2).

Discussion

The present study was the first study to show no significant increase in ventricular pacing threshold after recovering from COVID-19 infection. Patients in the COVID-19 group had comparable ventricular pacing threshold compared to those in the non-COVID-19 group.

Previous studies showed myocarditis and LGE reflecting myocardial scars in patients after recovery from COVID-19 infection[10,11]. This may be the cause of increased ventricular pacing threshold. The present trial did not demonstrate significantly increased ventricular pacing threshold in these patients with pacemakers. Of note, myocardial inflammation/scar is usually found in left ventricular myocardium from cardiac magnetic resonance imaging, while the ventricular pacing leads are always implanted in the right ventricle (RV). However, the location of ventricular pacing leads may not be the same site of myocardial inflammation or scar because of localized myocardial inflammation/scar in RV. In addition, some patients recovering from COVID-19 infection may also undergo reverse remodeling of myocardial inflammation/scarring leading to no significantly increased ventricular pacing threshold in these patients.

The present study had several limitations. First, the present study was a retrospective study. There may be missing data about the history of COVID-19 infection based on patient self-report when ATK data were unavailable. In addition, a lower rate of ATK use could lead to lower detection rate of COVID-19 infection and patients may have been misclassified to the non-COVID-19 group. Second, there was a small number of patients in the present study leading to lower event rate than expected as demonstrated by the wide 95% CI. Third, there was no data of COVID-19 vaccination in this study. Lower severity of COVID-19 infection resulting from vaccination may reduce myocardial inflammation/scarring affecting the proportion of patients with high ventricular pacing threshold to lower than expected in the present study. However, the present study was the first study to show no significant increase in ventricular pacing threshold after recovery from COVID-19 infection. Lastly, there were only Thai patients in the present study leading to limited generalizability. A larger multinational study will be needed in the future.

Conclusion

Patients with pacemakers had no significant increase in ventricular pacing threshold after recovering from COVID-19 infection.

What is already known about this topic?

Data about effect of ventricular pacing threshold after recovering from COVID-19 infection are lacking.

What does this study add?

This study showed that patients with pacemakers had no significant increase in ventricular pacing threshold after recovering from COVID-19 infection.

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Conflicts of interest

The author declares no competing interests.

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