Case Series Open Access

Mid-term follow-up of COVID-19 patients with permanent pacemaker implantation due to bradyarrhythmia at the acute phase of infection

Javad Shahabi[1], Saeed Sadri[2*], Fereshteh Sattar[3*], Amirhossein Azhari[1]

1- Interventional Cardiology Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
2- Cardiac Rehabilitation Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran
3- Cardiology Department, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran

Correspondence:
Saeed Sadri;
Cardiac Rehabilitation Research Center, Cardiovascular Research Institute, Isfahan University of Medical Sciences, Isfahan, Iran;
Email:
dr.sadrikarami@gmail.com

Fereshteh Sattar;
Cardiology Department, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran;
Email:
fereshte_sattar@yahoo.com

Received: 13-09-2023
Accepted: 11-11-2024

How to cite this article:
Shahabi J, Sadri S, Sattar F, Azhari A. **Mid-term follow-up of COVID-19 patients with permanent pacemaker implantation due to bradyarrhythmia at the acute phase of infection.** ARYA Atheroscler. 2025; 21(1): 7-14.

DOI:
https://doi.org/10.48305/arya.2024.42107.2920

Abstract

BACKGROUND: Arrhythmia is one of the important cardiac manifestations of SARS-CoV-2 disease with possible mechanisms such as direct damage to the myocardium, hypoxia, myocardial damage, cytokine storm, and electrolyte imbalances. Bradyarrhythmia is a manifestation of conduction system involvement, which is associated with an unfavorable prognosis and sometimes requires treatments such as implanting a pacemaker. Whether bradycardia in the acute phase of the COVID pandemic is a transient complication of the virus or whether it will be permanent can affect the treatment approach.

Is the effect of SARS-CoV-2 on the conduction system of the heart temporary or permanent, and in the one-year follow-up, how many patients will need a pacemaker?

METHODS: The study population was among patients with symptomatic bradyarrhythmias who were referred to Chamran Heart Center, Isfahan, Iran, from the outbreak of SARS-CoV-2 (February 2020) until February 2022 and were diagnosed with COVID-19 based on the polymerase chain reaction (PCR) test. They underwent permanent pacemaker implantation and were monitored for 1 month and 12 months after device implantation.

RESULTS: The most common comorbid disease was hypertension. Systolic blood pressure and respiratory rate in hospitalized patients decreased significantly during discharge. Also, oxygen saturation and heart rate increased significantly during discharge ($P < 0.001$). In this study, high-degree atrioventricular block remained permanent in most patients and was not transient.

CONCLUSION: Based on the experiences gained from this study, the implantation of a permanent pacemaker for the treatment of bradyarrhythmia should be done based on the existing guidelines, regardless of the status of COVID-19.

Keywords: Permanent Pacemaker; Heart Block; COVID-19

Introduction

In December 2019, 27 cases of pneumonia were reported in Wuhan, China. This was later identified as COVID-19 caused by the SARS-CoV-2 virus[1,2].

COVID-19 has been linked to various heart-related complications, such as myocardial damage, atrial and ventricular arrhythmias, and pro-inflammatory and prothrombotic effects[3-7]. Hypertension is common in COVID-19 patients, appearing in 30% of severe cases. Other comorbidities include diabetes, obesity, and pulmonary, cardiovascular, and cerebrovascular diseases[8-10].

COVID-19 is associated with a wide array of heterogeneous arrhythmias, including sinus tachycardia and bradycardia, atrial and ventricular fibrillation, complete heart block, and pulseless electrical activity (PEA). In general, acute infection causes arrhythmia by various mechanisms such as direct damage of the virus on the myocardium, hypoxia, myocarditis, ischemia, cytokine storm, and electrolyte disturbances. Additionally, interruption of cardiac medications and the iatrogenic effects of certain medications used in the treatment of COVID-19 may contribute to arrhythmias[11-13].

Sinus tachycardia may occur during SARS-CoV-2 infection, but the risk of other arrhythmias is lower and depends on the severity of the infection and underlying diseases[14]. However, bradyarrhythmias worsen the prognosis[15].

COVID-19 patients with severe and critical symptoms often experience bradycardia and complete heart block. 76.3% of these patients need pacemaker implantation, with most of the pacemakers being permanent[16].

In COVID-19 patients, bradyarrhythmias may require a permanent pacemaker to alleviate symptoms and restore a normal heart rhythm. However, it's unclear whether the bradyarrhythmia is temporary or permanent. Long-term follow-up studies are needed. For this purpose, this study has been designed to investigate the persistence or resolution of bradyarrhythmias in patients with COVID-19 who underwent pacemaker implantation in a 12-month follow-up.

Method

The study population consisted of patients with symptomatic bradyarrhythmias who were admitted to Shahid Chamran Heart Center, Isfahan, Iran, from February 2020 until February 2022 with SARS-CoV-2 infection. All cases were confirmed with a reverse-transcriptase PCR test on a nasopharyngeal specimen collected by a healthcare professional. Patients underwent a history and physical examination and a PA chest X-ray. A 12-lead electrocardiography (ECG) and echocardiography were performed for all patients, and blood tests including CBC, BUN, Cr, electrolytes, coagulation tests, and liver and thyroid function tests were conducted. All patients were Iranian, over 18 years old, and had stable evidence of documented bradyarrhythmia in ECG strips, including sinus bradycardia, advanced AV block, and complete heart block, while hospitalized. Some of them were also symptomatic due to bradyarrhythmias. They did not have advanced internal disease and had given informed consent to participate in the research. None of the patients had been vaccinated against SARS-CoV-2. Patients who did not return for follow-up visits or had incomplete clinical documents were excluded from the study. Patients who were candidates for permanent pacemaker implantation, regardless of whether they had positive PCR tests for COVID-19 infection, underwent the procedure following the isolation protocols related to COVID-19. On average, it took eight days from the diagnosis of COVID-19, based on PCR tests, until the implantation of cardiac implantable electronic devices (CIEDs).

Information related to the pacemaker device, including device type (single chamber or dual chamber), device mode (VVI, VOO, AAI, DDD, DDI, etc.), manufacturer, reason for implantation of the device (bradyarrhythmia type), intrinsic heart rate, percent of pacing, and pacemaker dependency, were all recorded in the device analysis form.

Due to the positive COVID-19 PCR test, the patients were monitored in the respiratory isolation ward of Chamran Heart Center, and in the absence of any complication, they were discharged with the recommendation of home quarantine after 24 hours of device implantation. Patients who were admitted to the intensive care unit (ICU) stayed longer in the hospital.

Thirty-two patients with these features were entered into the study as shown in Figure 1. Of these, two patients died during hospitalization due to acute respiratory failure (ARDS), one patient died 25 days after discharge due to pulmonary thromboembolism and cardiorespiratory failure, and one patient died 28 days after discharge due to cardiorespiratory failure. Ten of the remaining 28 patients were excluded due to transient bradyarrhythmia and lack of indication for permanent pacemaker implantation. Follow-up and periodic device analysis and generator data interrogation were performed for the 18 remaining patients.

Statistical analysis

In this study, continuous quantitative variables were reported as mean ± standard deviation, and qualitative variables were reported as frequency and percentage. The paired t-test was used if the data were normally distributed, and if not, the non-parametric Wilcoxon test was used to compare the para clinical findings at the beginning and at the time of discharge. A significant value of 5% was considered. SPSS version 24 software was used for analysis.

Results

Eighteen patients entered the study, six of them were men (33.3%) and twelve were women (66.7%). The average age of the patients was 68.78 ± 15.64 years, with a minimum age of 24 years and a maximum age of 89 years. There was no significant difference in the average age between the two genders (66.83 years for men vs. 69.75 years for women, $P > 0.05$). The most common symptoms in patients were weakness (83.3%) and dizziness (50%); others experienced syncope, chest pain, and dyspnea.

Ten patients (55.6%) had a history of hypertension, and four patients (22.2%) had a history of heart failure with reduced ejection fraction. Ten patients (55.6%) had a history of beta-blocker or calcium channel blocker drug consumption. Despite stopping these drugs and monitoring for a five-half-life passing, bradyarrhythmia remained unchanged even after the period passed. Twelve patients (66.6%) showed bilateral parenchymal infiltration, and two patients (11.1%) showed unilateral parenchymal infiltration in chest X-rays. Table 1 shows demographic, baseline clinical, and laboratory data.

Two patients (11.1%) were prescribed Remdesivir infusion based on current SARS-CoV-2 guidelines for 3 to 5 days, and implantation was performed after stopping the infusion for more than 2 days. Additionally, these two

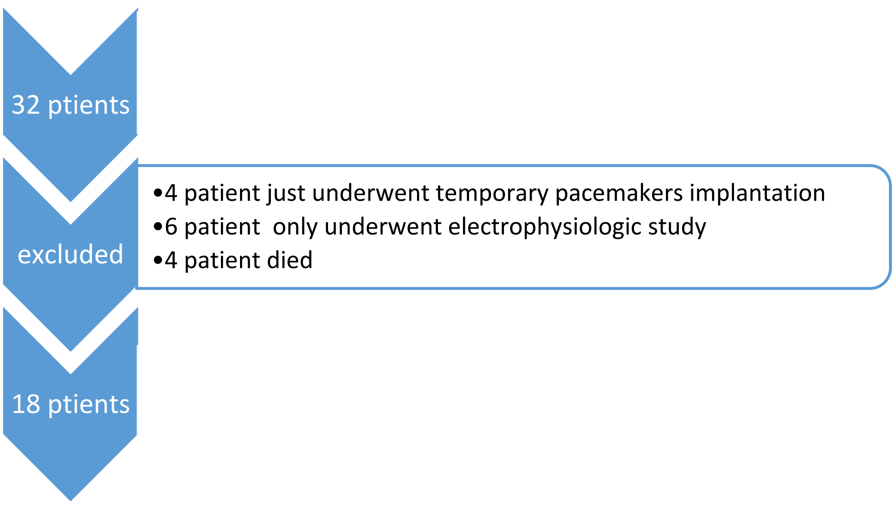

Figure 1. SARS-CoV2 positive patients who referred to Chamran hospital electrophysiology laboratory

Table 1. Demographic, baseline clinical and laboratory data

Data	Mean ± SD	min	max	Normal range
Demographic				
Age	68.78±15.64	24	89	-
Sex (male, N, %)	6 (33.3%)	-	-	-
Laboratory/Imaging Evaluation				
Hemoglobin (g/dl)	13.93±1.83	9.60	17.20	Male:13.8-17.2 Female:12.1-15.1
GFR by EPI (ml/min/1.73m²)	60.54±23.68	15.50	100	90-120
WBC	8566.67±3814.37	4400	18500	4-11×10⁹/L
Lymphocyte	19.14±10.55	7	40.40	1.5-4.5×10⁹/L
Potassium (meq/L)	4.23±0.65	3.50	6.20	3.5-5.2
Sodium (meq/L)	138.05±4.44	133	152	135-145
Magnesium (mg/dL)	2.08±0.18	1.80	2.60	1.7-2.2
LVEF (%)	46.61±12.78	20	60	50-70%
Troponin(positive)	1(5.6%)			
Medical History				
Hypertension (N, %)	10(55.6%)	-	-	-
Diabetes (N, %)	3(16.7%)	-	-	-
Kidney failure (N, %)	3(16.7%)	-	-	-
Heart failure (N, %)	4(22.2%)	-	-	-
Stroke (N, %)	1(5.6%)	-	-	-
Pulmonary embolism (N, %)	1(5.6%)	-	-	-
Pulmonary obstruction (N, %)	1(5.6%)	-	-	-
Ischemic heart (N, %)	1(5.6%)			
Imaging of chest X-ray			N (%)	
Bilateral infiltration			12(66.7%)	
Unilateral infiltration			2(11.1%)	
Normal			4(22.2%)	

Table 2. Vital signs changes from the first day of admission until discharge

Vital signs	Baseline	Discharge	P-value
Systolic Blood Pressure (mmHg)	152.56±21.18	127.94±15.70	<0.001
Diastolic Blood Pressure (mmHg)	79.78±11.90	75.05±8.61	0.117
Heart rate (bpm)	36.67±9.11	65.78±6.59	<0.001
Oxygen Saturation (%)	89.72±5.23	95.33±1.94	<0.001
Breathing rate (breaths per minute)	19.83±2.23	17.11±0.76	<0.001

patients received intravenous corticosteroids (dexamethasone) during hospitalization and were discharged with an oral corticosteroid (prednisolone) for 2 weeks.

No electrolyte disturbance or ischemia was found to be the cause of bradyarrhythmia. Dual chamber pacemakers were the most frequent, with nine patients (50%) receiving them. The most frequent inflammatory factor, C-reactive protein (CRP), was +2 in six patients (33.3%).

Table 2 shows vital sign changes from the first day of admission until discharge. Systolic blood pressure decreased significantly between the first measurement and the time of discharge ($P < 0.001$). The difference between the initial and discharge heart rate was statistically significant, with the heart rate increasing during this period ($P < 0.001$). Additionally, the respiratory rate decreased at the time of discharge, and oxygen saturation increased at the time of discharge.

The main results of the study are shown in Table 3, which represents follow-up data. All patients were visited at the first and 12th months after the implantation of cardiac CIEDs, and their performance was checked with device analysis. According to the analysis, the percentage of pacing at the first month follow-up was 88.15% and 79.78% after 12 months from implantation;

Table 3. Cardiac implantable electronic device follow-up data

Variables	**Mean ± SD**
The percentage of ventricular pacing in the first month	88.15±30.10
Percentage of ventricular pacing in the 12th month	79.78±35.95
Percentage of atrial pacing in the first month	1.96±3.07
Percentage of atrial pacing in the 12th month	2.65±5.56
Variables	Frequency (%)
Rhythm analysis of the first month	
Atrioventricular block (Complete heart block,2:1 AV block)	16(88.9%)
junctional bradycardia	1(5.6%)
Left bundle branch block	3(16.6%)
Rhythm analysis of the 12th month	
Atrioventricular block (Complete heart block,2:1 AV block)	15(83.3%)
Sinus bradycardia	1(5.6%)
Left bundle branch block	3(16.6%)
A ventricular episode during 12 months	
Ventricular Tachycardia	2(11.1%)
An episode of atrial arrhythmia during 12 months	
Atrial tachycardia	3(16.7%)
Atrial fibrillation	1(5.6%)
Persistence of bradycardia	15(83.3%)
Total dependence on the device	2(11.1%)

the programmed lower rate of the pacemaker was 60 beats per minute (bpm).

Atrioventricular block (consisting of second and third-degree AVB) was the most frequent rhythm at the first month evaluation (88.9%) and also at the 12th month (83.3%). Bradyarrhythmia was persistent in 15 patients (83.3%).

Discussion

In this study, the majority of patients who underwent permanent pacemaker implantation due to symptomatic bradyarrhythmia had persistent bradyarrhythmia after one year of follow-up (83.3%). The percent of pacemaker pacing at the first month follow-up was 88.15% and 79.78% after 12 months from implantation (the programmed lower rate of the pacemaker was 60 beats per minute (bpm)), which indicates permanent bradyarrhythmia. However, 2 patients were completely dependent on the device pacing. It should be mentioned that dependency on a pacing device in this study is described as a native heart rate lower than 30 bpm.

Shahabi J. et al.'s study in Chamran Heart Center showed no significant difference in the rate of permanent and temporary pacemaker implantation following bradyarrhythmia from the beginning of the COVID-19 pandemic to the end of 2021 compared to the same period in 2019[17]. This finding was also observed by the study of Akhtar et al., and no significant difference was found in the rate of high-grade atrioventricular block during the pandemic compared to before the pandemic, and high-grade heart block remained persistent in patients with permanent pacemakers[13]. Of course, it should be noted that the impossibility of regular follow-ups during the COVID-19 pandemic can affect the findings[18].

Our results were in contrast with previous studies in the field of transient conduction block in patients with SARS-CoV-2, including Dagher et al. and Mahdawi T et al.[19,20]. In these studies, hospitalized patients infected with SARS-CoV-2 had transient and temporary high-grade conduction disorder; only one case required a temporary pacemaker, and the other cases resolved after the acute phase of the disease.

It is assumed that the COVID-19 infection has revealed an underlying abnormal cardiac conduction system in our study population and COVID is not necessarily the cause of bradyarrhythmia. Patients in our study and

Akhtar et al. were older than those of Dagher et al. and therefore more likely to have had pre-existing senile abnormality and fibrosis in the conduction system[21]. The infection may be considered a precipitating factor for increased cardiac demand; this assumption exists that COVID-19 infection brought to our attention patients with pre-existing bradycardia who had previously gone undiagnosed.

Half of the Dagher et al. cohort had a broad-QRS escape rhythm as in our and Akhtar et al.'s patients with AV block; we believe that they should have been considered for pacemaker implantation. Experience from other clinical situations suggests that AV block initially thought to have a reversible cause often recurs despite correction of that cause[22-24]. Transient AV block has been previously described in patients with fibrous conduction tissue and may go on to produce persistent block[21].

Bradyarrhythmias in these patients did not relate to Remdesivir prescription because bradyarrhythmia associated with Remdesivir is transient and immediately resolves after stopping the drug as reported in James C.'s study[25]. In this study, for 2 patients, Remdesivir was prescribed but pacemaker implantation was postponed at least 2 days after the end of the drug infusion. Similar to our study, in Umeh's study, an association between Remdesivir and beta-blocker prescription and bradycardia was not seen in COVID-19 patients[26]. These 2 patients also received corticosteroids (intravenous dexamethasone) but this type of drug does not protect patients against developing bradycardia as seen in Umeh's study. In contrast with Umeh's study, steroids have been reported to cause bradycardia in non-COVID-19 patients, especially after using high or pulse-dose steroids[27-29]. The mechanisms of steroid protection against bradycardia in COVID-19 patients remain unclear, but inhibition of severe inflammation and cytokine storm is explained[30]. Another scenario is the effect of corticosteroids on beta-adrenergic receptors in the heart, leading to positive inotropic and chronotropic effects[30,31]. Thus, the elevated heart rate induced by corticosteroids may counteract bradycardia caused by COVID-19.

Chest X-ray is one of the instruments used to detect COVID-19 pneumonia severity. A common finding detected in more severe COVID-19 pneumonia was bilateral peripheral parenchymal infiltrations in the middle and lower lobes[32]. In this study, most patients (66.6%) showed bilateral parenchymal infiltration in chest X-rays as a marker of severity. In Bassi R.'s study, which was about COVID-19-induced complete heart block, bilateral parenchymal infiltration was also detected[33].

The data of the present study and Akhtar et al. based on permanent pacing during one year follow-up and lack of long-term follow-up in previous studies, show that atrioventricular block seems not to be completely reversible.

Conclusion

In this study, high-degree atrioventricular block remained persistent in most patients and was not transient. Based on the experiences gained from this study, the implantation of a permanent pacemaker for the treatment of bradyarrhythmias should be done based on the existing guidelines, regardless of the status of COVID-19.

Limitations

The emergence of new COVID-19 variants raises concerns about their potential effects on the cardiovascular system, including the initiation of arrhythmias. However, the impact of these new variants remains unknown due to the lack of examination of the variant type of diseases in patients and the small number of available samples. These limitations highlight the need for more research to better understand the potential impact of new COVID-19 variants on the cardiovascular system.

Acknowledgements

Foremost, I would like to express my sincere gratitude to my advisors, Dr. Javad Shahabi and Dr. Fereshteh Sattar, for their continuous support of my study and research, as well as for their patience, motivation, enthusiasm, and immense knowledge. Their guidance has been invaluable throughout my research and the writing of this thesis. In addition to my advisors, I would like to extend my deep gratitude to Prof. Masoume Sadeghi for her advice at the beginning of the study and, in particular, for her revision comments. My sincere thanks also go to my colleagues at Chamran Paraclinic, Ms. Mollagholi and Ms. Momeni, as well as the EP lab colleagues, for their assistance in enabling me to visit my participating patients.

Conflict of interests

The authors declare no conflict of interest.

Funding

There is no funding in this study.

Author's Contributions

Study Conception or Design: JS, FS
Data Acquisition: JS, SS, AA
Data Analysis or Interpretation: FS
Manuscript Drafting: FS
Critical Manuscript Revision: JS, SS, AA
All authors have approved the final manuscript and are responsible for all aspects of the work.

References

1. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. JAMA Cardiol. 2020 Jul 1;5(7):831-40. https://doi.org/10.1001/jamacardio.2020.1286
2. Hu B, Guo H, Zhou P, Shi ZL. Characteristics of SARS-CoV-2 and COVID-19. Nat Rev Microbiol. 2021 Mar;19(3):141-54. https://doi.org/10.1038/s41579-020-00459-7
3. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. JAMA. 2020 Mar 17;323(11):1061-9. https://doi.org/10.1001/jama.2020.1585
4. Cunha BA. The diagnostic significance of relative bradycardia in infectious disease. Clin Microbiol Infect. 2000 Dec;6(12):633-4. https://doi.org/10.1046/j.1469-0691.2000.0194f.x
5. Ikeuchi K, Saito M, Yamamoto S, Nagai H, Adachi E. Relative Bradycardia in Patients with Mild-to-Moderate Coronavirus Disease, Japan. Emerg Infect Dis. 2020 Oct;26(10):2504-6. https://doi.org/10.3201/eid2610.202648
6. Lakkireddy DR, Chung MK, Gopinathannair R, Patton KK, Gluckman TJ, Turagam M, et al. Guidance for cardiac electrophysiology during the COVID-19 pandemic from the Heart Rhythm Society COVID-19 Task Force; Electrophysiology Section of the American College of Cardiology; and the Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, American Heart Association. Heart Rhythm. 2020 Sep;17(9):e233-41. https://doi.org/10.1016/j.hrthm.2020.03.028
7. Babapoor-Farrokhran S, Rasekhi RT, Gill D, Babapoor S, Amanullah A. Arrhythmia in COVID-19. SN Compr Clin Med. 2020;2(9):1430-5. https://doi.org/10.1007/s42399-020-00454-2
8. Schiffrin EL, Flack JM, Ito S, Muntner P, Webb RC. Hypertension and COVID-19. Am J Hypertens. 2020 Apr 29;33(5):373-4. https://doi.org/10.1093/ajh/hpaa057
9. McGurnaghan SJ, Weir A, Bishop J, Kennedy S, Blackbourn LAK, McAllister DA, et al. Risks of and risk factors for COVID-19 disease in people with diabetes: a cohort study of the total population of Scotland. Lancet Diabetes Endocrinol. 2021 Feb;9(2):82-93. https://doi.org/10.1016/s2213-8587(20)30405-8
10. Hendren NS, de Lemos JA, Ayers C, Das SR, Rao A, Carter S, et al. Association of Body Mass Index and Age With Morbidity and Mortality in Patients Hospitalized With COVID-19: Results From the American Heart Association COVID-19 Cardiovascular Disease Registry. Circulation. 2021 Jan 12;143(2):135-44. https://doi.org/10.1161/circulationaha.120.051936
11. Dherange P, Lang J, Qian P, Oberfeld B, Sauer WH, Koplan B, Tedrow U. Arrhythmias and COVID-19: A Review. JACC Clin Electrophysiol. 2020 Sep;6(9):1193-1204. https://doi.org/10.1016/j.jacep.2020.08.002
12. Gopinathannair R, Merchant FM, Lakkireddy DR, Etheridge SP, Feigofsky S, Han JK, et al. COVID-19 and cardiac arrhythmias: a global perspective on arrhythmia characteristics and management strategies. J Interv Card Electrophysiol. 2020 Nov;59(2):329-36. https://doi.org/10.1007/s10840-020-00789-9
13. Akhtar Z, Leung LW, Kontogiannis C, Zuberi Z, Bajpai A, Sharma S, et al. Prevalence of bradyarrhythmias needing pacing in COVID-19. Pacing Clin Electrophysiol. 2021 Aug;44(8):1340-6. https://doi.org/10.1111/pace.14313

14. Lilly LS. Braunwald's Heart Disease Review and Assessment E-Book: A Companion to Braunwald's Heart Disease. Elsevier Health Sciences; 2022 Aug 13:1751-65.
15. Chinitz JS, Goyal R, Harding M, Veseli G, Gruberg L, Jadonath R, et al. Bradyarrhythmias in patients with COVID-19: Marker of poor prognosis? Pacing Clin Electrophysiol. 2020 Oct;43(10):1199-204. https://doi.org/10.1111/pace.14042
16. Nagamine T, Randhawa S, Nishimura Y, Huang R, Leesutipornchai T, Benavente K, et al. Characteristics of bradyarrhythmia in patients with COVID-19: Systematic scoping review. Pacing Clin Electrophysiol. 2022 Apr;45(4):556-66. https://doi.org/10.1111/pace.14466
17. Shahabi J, Askari M, Azhari A, Kermani-Alghoraishi M. The survey of electrophysiology lab activity during the COVID-19 pandemic. J Arrhythm. 2021 Jun 24;37(4):899-903. https://doi.org/10.1002/joa3.12584
18. Akhtar Z, Montalbano N, Leung LWM, Gallagher MM, Zuberi Z. Drive-Through Pacing Clinic: A Popular Response to the COVID-19 Pandemic. JACC Clin Electrophysiol. 2021 Jan;7(1):128-30. https://doi.org/10.1016/j.jacep.2020.09.026
19. Dagher L, Wanna B, Mikdadi G, Young M, Sohns C, Marrouche NF. High-degree atrioventricular block in COVID-19 hospitalized patients. Europace. 2021 Mar 8;23(3):451-55. https://doi.org/10.1093/europace/euaa333
20. Eneizat Mahdawi T, Wang H, Haddadin FI, Al-Qaysi D, Wylie JV. Heart block in patients with coronavirus disease 2019: A case series of 3 patients infected with SARS-CoV-2. HeartRhythm Case Rep. 2020 Sep;6(9):652-6. https://doi.org/10.1016/j.hrcr.2020.06.014
21. Zoob M, Smith Ks. The Aetiology of Complete Heart-Block. Br Med J. 1963 Nov 9;2(5366):1149-53. https://doi.org/10.1136/bmj.2.5366.1149
22. Kennebäck G, Tabrizi F, Lindell P, Nordlander R. High-degree atrioventricular block during anti-arrhythmic drug treatment: use of a pacemaker with a bradycardia-detection algorithm to study the time course after drug withdrawal. Europace. 2007 Mar;9(3):186-91. https://doi.org/10.1093/europace/eul185
23. Yesil M, Bayata S, Arikan E, Yilmaz R, Postaci N. Should we revascularize before implanting a pacemaker? Clin Cardiol. 2008 Oct;31(10):498-501. https://doi.org/10.1002/clc.20280
24. Palmisano P, Ziacchi M, Ammendola E, Dell'Era G, Guerra F, Aspromonte V, et al. Long-term progression of rhythm and conduction disturbances in pacemaker recipients: findings from the Pacemaker Expert Programming study. J Cardiovasc Med (Hagerstown). 2018 Jul;19(7):357-65. https://doi.org/10.2459/jcm.0000000000000673
26. Umeh C, Giberson C, Kumar S, Aseri M, Barve P. A Multicenter Retrospective Analysis on the Etiology of Bradycardia in COVID-19 Patients. Cureus. 2022 Jan 16;14(1):e21294. https://doi.org/10.7759/cureus.21294
27. Taylor MR, Gaco D. Symptomatic sinus bradycardia after a treatment course of high-dose oral prednisone. J Emerg Med. 2013 Sep;45(3):e55-8. https://doi.org/10.1016/j.jemermed.2013.04.020
28. Akikusa JD, Feldman BM, Gross GJ, Silverman ED, Schneider R. Sinus bradycardia after intravenous pulse methylprednisolone. Pediatrics. 2007 Mar;119(3):e778-82. https://doi.org/10.1542/peds.2006-0029
29. Stroeder J, Evans C, Mansell H. Corticosteroid-induced bradycardia: Case report and review of the literature. Can Pharm J (Ott). 2015 Sep;148(5):235-40. https://doi.org/10.1177/1715163515597451
30. Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr Rev. 2000 Feb;21(1):55-89. https://doi.org/10.1210/edrv.21.1.0389
31. Durrani M, Haq IU, Kalsoom U, Yousaf A. Chest X-rays findings in COVID 19 patients at a University Teaching Hospital - A descriptive study. Pak J Med Sci. 2020 May;36(COVID19-S4):S22-6. https://doi.org/10.12669/pjms.36.covid19-s4.2778
32. Davies AO, Lefkowitz RJ. Regulation of beta-adrenergic receptors by steroid hormones. Annu Rev Physiol. 1984;46:119-30. https://doi.org/10.1146/annurev.ph.46.030184.001003
33. Bassi R, Ismail Z, Salabei JK, Charles K, Haider AA, Hussein A, et al. COVID-19-Induced Complete Heart Block: Case Series and Literature Review. Cureus. 2023 Apr 13;15(4):e37517. https://doi.org/10.7759/cureus.37517