Clinical Characteristics and Outcomes of Patients Hospitalized for COVID-19 Pneumonia Who Developed Bradycardia

Fernando Stancampiano, MD, Mohamed Omer, MD, Dana Harris, MD, Jose Valery, MD, Michael Heckman, MS, Launia White, MS, and Claudia Libertin, MD

Objective: To assess the clinical characteristics and clinical outcomes of bradycardic patients with coronavirus disease 2019 (COVID-19) pneumonia.

Methods: The electronic medical records of 221 consecutive patients hospitalized for COVID-19 pneumonia between June and September 2020 were retrospectively reviewed. Patient characteristics, electrocardiographic data, and clinical and laboratory information were retrospectively collected. Patients not treated with drugs that blunt chronotropic response (nodal) were analyzed separately.

Results: Only patients whose heart rate was <60 beats per minute (bpm) (136/221, 61.5%) were included. Serial electrocardiography revealed that most patients (130/137, 97.7%) remained in sinus rhythm. The heart rate was between 50 and 59 bpm in 75% of the patients, while 18.4% were in the 40 to 49 bpm range, and 6.6% were <40 bpm. Medians for development of bradycardia after swab polymerase chain reaction positivity and duration of bradycardia were 41 hours and 5 days, respectively. Bradycardia resolved in 81 patients (59.6%). There were no statistically significant differences in outcomes according to degree of bradycardia (<50 vs 50–59, all $P \geq 0.073$). No significant differences were noted for the overall cohort when comparing COVID-19 treatments according to resolution of bradycardia; however, when considering only the patients who were not receiving a nodal agent or antiarrhythmic, treatment with lenzilumab was more common in patients with resolution of bradycardia than patients without resolution of bradycardia (12.2% vs 0.0%, $P = 0.030$).

Conclusions: Sinus bradycardia occurs frequently in patients with severe COVID-19, but the degree of bradycardia does not correlate with clinical outcomes. Lenzilumab may be associated with the resolution of bradycardia.

Key Words: bradycardia, COVID-19, lenzilumab, pneumonia

Coronavirus disease 2019 (COVID-19) has been linked to a variety of acute cardiovascular abnormalities, including myocardial infarction, new-onset congestive heart failure, myocarditis, and ventricular arrhythmias, even in patients without structural heart disease.[1–5] Evidence of persistent inflammation of the myocardium also has been observed in patients who recovered from the disease.[6] An interesting phenomenon is the presence of bradycardia in the acute phase of COVID-19, in spite of conditions usually associated with an elevated heart rate, such as fever and hypotension. Other groups have reported bradycardic rhythms in COVID-19 patients, but their work was limited to a small number of subjects and included patients with mild or "relative" bradycardia.[7,8] Our study was designed to describe the clinical characteristics and outcomes of patients admitted to a tertiary care medical center with the diagnosis of COVID-19 pneumonia, who presented with bradycardia in the course of their hospitalization. The primary aims of this study were to describe characteristics and outcomes in COVID-19 patients with bradycardia and make comparisons of interest according to the degree of bradycardia and the resolution of bradycardia.

Key Points

- Bradycardia is highly prevalent in hospitalized patients with coronavirus disease 2019 pneumonia.
- The degree of bradycardia does not appear to correlate with clinical outcomes.
- Bradycardic rhythms may develop in the absence of ventricular dysfunction and predate the onset of severe respiratory disease.
- Lenzilumab may be associated frequently with the resolution of bradycardia than other anticoronavirus disease 2019 treatments.

From the Division of Community Internal Medicine, the Clinical Research Unit, the Division of Biomedical Statistics and Informatics, and the Division of Infectious Diseases, Mayo Clinic Florida, Jacksonville.

Correspondence to Dr Fernando Stancampiano, Mayo Clinic Florida, 4500 San Pablo Rd, Cannady 3-W, Jacksonville, FL 32224. E-mail: stancampiano.f@mayo.edu. To purchase a single copy of this article, visit sma.org/smj. To purchase larger reprint quantities, please contact reprintsolutions@wolterskluwer.com.

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Off-label use: Lenzilumab is being used on a compassionate basis as an investigational new drug in the treatment of patients with severe COVID-19 pneumonia and is also being administered as part of Phase III protocols (see also https://www.sciencedirect.com/science/article/pii/S0025619620309897?via%3Dihub).

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Methods

Study Subjects

A total of 136 consecutive patients who were diagnosed as having COVID-19 pneumonia at the Mayo Clinic in Jacksonville, Florida between June 2020 and September 2020, and who also had absolute bradycardia defined as a heart rate of <60 beats per minute (bpm), were included in this retrospective study. An additional 85 patients admitted for COVID-19 pneumonia during the same time period did not meet the criteria for bradycardia and were excluded. Information was collected regarding patient characteristics, electrocardiogram patterns, severity of bradycardia, biomarkers, outcomes, and resolution of bradycardia. All of the data collected by the investigators remain stored in secure, encrypted Mayo Clinic servers. Data recording was completed with the use RedCap software, as Mayo Clinic is a member of the RedCap Consortium. The study was conducted after receiving approval by the Mayo Clinic institutional review board.

Statistical Analysis

All of the analyses were performed in both the overall cohort of 136 patients and the subset of 90 patients who were not receiving a nodal agent or antiarrhythmic. Continuous variables were summarized with the sample median and range. Categorical variables were summarized with the number and percentage of patients. Comparisons of outcomes and biomarkers according to degree of bradycardia (50–59 vs <50) were made using a Wilcoxon rank-sum test (continuous and ordinal variables) or the Fisher exact test (categorical variables). Comparisons of COVID-19 treatments according to resolution of bradycardia were made using the Fisher exact test. $P < 0.05$ was considered statistically significant. All of the statistical tests were two-sided. The statistical analyses were performed using SAS version 9.4 (SAS Institute, Cary, North Carolina).

Results

A summary of patient characteristics is shown in Table 1. The median age was 67 years (range 19–101 years), male sex was most common (56.6%), and 98 patients (72.1%) were White. The most common COVID-19 treatments were corticosteroids (81.6%) and remdesivir (77.2%).

Bradycardia information is provided in Table 2. The degree of bradycardia was 50 to 59 in 102 patients (75.0%), 40 to 49 in 25 patients (18.4%), and <40 in 9 patients (6.6%). The median duration of bradycardia was 5 days (range 1–29 days). A history of bradycardia was noted in 42 patients (36.5%). The resolution of bradycardia occurred in 81 patients (59.6%).

Comparisons of outcomes and biomarkers according to degree of bradycardia are shown in Table 3 for all of the patients and the subset of 90 patients who were not receiving a nodal agent or antiarrhythmic. No statistically significant differences were observed (all $P \geq 0.065$). A comparison of COVID-19 treatments according to resolution of bradycardia is shown in Table 4 for all of the patients and the subgroup who were not receiving a nodal agent or antiarrhythmic. Comparisons of outcomes and biomarkers according to race in all patients and in patients not treated with a nodal agent/antiarrhythmic are shown in Supplemental Digital Content Tables 5a (http://links.lww.com/SMJ/A227) and 5b (http://links.lww.com/SMJ/A227) respectively.

Table 1. Patient characteristics

Variable	N	Median (minimum, maximum) or no. (%) patients: All patients (N = 136)	Patients not on a nodal agent/ antiarrhythmic (n = 90)
Age, y	136	67 (19–101)	66 (19–101)
Male sex (%)	136	77 (56.6)	48 (53.3)
Race (%)	136		
White		98 (72.1)	63 (70.0)
Black		27 (19.9)	19 (21.1)
Asian		10 (7.4)	7 (7.8)
Other		1 (0.7)	1 (1.1)
BMI, kg/m^2 (range)	136	29.1 (18.8–54.8)	28.3 (18.8–54.8)
Length of stay, d (range)	136	7 (1–55)	7 (1–32)
Chronic respiratory disease (%)	136	33 (24.3)	22 (24.4)
CAD (%)	136	24 (17.6)	9 (10.0)
CHF (%)	136	16 (11.8)	6 (6.7)
Diabetes mellitus (%)	136	41 (30.1)	23 (25.6)
Organ transplant (%)	136	9 (6.6)	4 (4.4)
Immunosuppressive therapy (%)	136	15 (11.0)	9 (10.0)
COVID-19 treatment (%)			
Remdesivir	136	105 (77.2)	70 (77.8)
Corticosteroids	136	111 (81.6)	73 (81.1)
Lenzilumab	136	9 (6.6)	6 (6.7)
Anti-SARS-CoV-2 convalescent plasma	136	26 (19.1)	15 (16.7)
Tocilizumab	136	12 (8.8)	7 (7.8)
Anakinra	136	3 (2.2)	1 (1.1)
Supportive care only	136	23 (16.9)	14 (15.6)
EKG rhythm (%)			
NSR	133	130 (97.7)	87 (100.0)
Atrial fibrillation/flutter	133	3 (2.3)	0 (0.0)
Junctional	133	0 (0.0)	0 (0.0)
MAT	133	0 (0.0)	0 (0.0)
Wenckebach	133	0 (0.0)	0 (0.0)
Mobitz	133	0 (0.0)	0 (0.0)
Third-degree block	133	0 (0.0)	0 (0.0)
Other	133	0 (0.0)	0 (0.0)

BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; COVID-19, coronavirus disease 2019; EKG, electrocardiogram; MAT, multifocal atrial tachycardia; NSR, normal sinus rhythm; SARS-CoV-2, severe acute respiratory syndrome-coronavirus-2.

Table 2. Bradycardia information

Variable	N	Median (minimum, maximum) or no. (%) patients	
		All patients (N = 136)	Patients not receiving a nodal agent/ antiarrhythmic (N = 90)
Degree of bradycardia, bpm (%)	136		
50–59		102 (75.0)	62 (68.9)
40–49		25 (18.4)	21 (23.3)
<40		9 (6.6)	7 (7.8)
HR max (range)	136	109 (68–198)	110 (74–189)
HR low (range)	136	47 (18–59)	46 (18–59)
Duration of bradycardia, d (range)	136	5 (1–29)	5 (1–28)
History of bradycardia (%)	115	42 (36.5)	27 (30.0)
Nodal agent/ antiarrhythmic (%)	136	46 (33.8)	0 (0.0)
β-Blocker (%)	136	38 (27.9)	0 (0.0)
CCB (%)	136	2 (1.5)	0 (0.0)
Amiodarone (%)	136	7 (5.1)	0 (0.0)
Other medication (%)	136	3 (2.2)	0 (0.0)
Associated conduction delay (%)	136	28 (20.6)	16 (17.8)
LBBB	136	3 (2.2)	2 (2.2)
RBBB	136	14 (10.3)	9 (10.0)
IVCD	136	14 (10.3)	6 (6.67)
Congestive heart failure (%)	136	9 (6.6)	4 (4.4)
NT-pro-BNP (10-263 pg/mL) (range)	91	305 (20–19,674)	236 (20–16,141)
Pneumonia (%)	136	122 (89.7)	79 (87.8)
Unilobar	136	6 (4.4)	4 (4.4)
Multilobar	136	113 (83.1)	74 (82.2)
Interstitial	136	63 (46.3)	43 (47.8)
Ground glass	136	64 (47.1)	39 (43.3)
Thrombotic complication (%)	136	11 (8.1)	7 (7.8)
Venous	136	3 (2.2)	3 (3.3)
Arterial	136	9 (6.6)	5 (5.6)
DVT	136	1 (0.7)	1 (1.1)
PE	136	5 (3.7)	3 (3.3)
Myocardial infarction (%)	136	40 (29.4)	3 (3.3)
Treatment of bradycardia (%)			
Anticholinergic	40	1 (2.5)	0 (0.0)
Adrenergic	40	3 (7.5)	2 (66.7)
Discontinuation of nodal agent/antiarrhythmic	40	36 (90.0)	0 (0.0)
Pacemaker	40	1 (2.5)	1 (33.3)
PCR-bradycardia time, h (range)	136	41 (1–624)	39 (1–560)
Resolution of bradycardia (%)	136	81 (59.6)	49 (54.4)
Mechanism of resolution			
After COVID-19 medication	81	69 (85.2)	42 (85.7)
After discontinuation of nodal agent	81	29 (35.8)	1 (2.0)
Spontaneous	81	8 (9.9)	7 (14.3)
Other cardiac diagnoses (%)			
Myocarditis	136	1 (0.7)	1 (1.1)
Pericarditis	136	2 (1.5)	2 (2.2)
Pericardial effusion	136	7 (5.1)	2 (2.2)
Acute coronary event	136	4 (2.9)	2 (2.2)
Time from bradycardia to worsening oxygenation, h (range)	136	4 (0–120)	4 (0–120)
HR on admission (range)	136	88 (48–154)	91 (48–131)
Lymphocytopenia (%)	136	89 (65.4)	62 (68.9)

Continued next page

Table 2. *(Continued)*

bpm, beats per minute; CCB, calcium-channel blocker; COVID-19, coronavirus disease 2019; DVT, deep vein thrombosis; HR, heart rate; IVCD, intraventricular conduction delay; LBBB, left bundle branch block; NT-pro-BNP, N-terminal pro b-type natriuretic peptide; PCR, polymerase chain reaction; PE, pulmonary embolism; RBBB, right bundle branch block.

No significant differences were noted for the overall cohort; however, when considering only the subjects who were not receiving a nodal agent or antiarrhythmic, randomization to lenzilumab versus placebo was more common in patients with resolution of bradycardia than patients without resolution of bradycardia (12.2% vs 0.0%, $P = 0.030$).

Discussion

Our study showed that bradycardia, defined as a heart rate of <60 bpm, appears to be a common occurrence (136/221 = 61.5%) in patients with COVID-19 pneumonia; however, the degree of bradycardia did not correlate with the clinical outcome. We included patients with a heart rate of <60 bpm only, of whom the majority (68.9%) remained in the 50 to 59 range, and 23.3% between 40 and 49 bpm. Those who were not receiving nodal agents such as calcium channel blockers and β-blockers or antiarrhythmics were analyzed separately and compared with the total study population and found to have similar clinical outcomes. Relative bradycardia associated with hyperthermia (sphygmothermic

Table 3. Comparisons of outcomes and biomarkers according to degree of bradycardia

		All patients			Subset of patients not receiving a nodal agent/antiarrhythmic		
Variable	N	Degree of bradycardia: 50–59 (n = 102)	Degree of bradycardia: <50 (n = 34)	*P*	Degree of bradycardia: 50–59 (n = 62)	Degree of bradycardia: <50 (n = 28)	*P*
Transfer to ICU (%)	136	16 (15.7)	6 (17.6)	0.79	8 (12.9)	6 (21.4)	0.35
In-hospital mortality (%)	136	4 (3.9)	4 (11.8)	0.11	2 (3.2)	4 (14.3)	0.073
Discharged on O_2 (%)	128	20 (20.4)	6 (20.0)	1.00	13 (21.7)	4 (16.7)	0.77
Disposition (%)							
Home	136	83 (84.7)	25 (83.3)	1.00	51 (82.3)	20 (71.4)	0.27
Rehab center	136	4 (4.1)	2 (6.7)	0.62	2 (3.2)	1 (3.6)	1.00
SNF	136	8 (8.2)	3 (10.0)	0.72	7 (11.3)	3 (10.7)	1.00
LTAC	136	2 (2.0)	0 (0.0)	1.00	0 (0.0)	0 (0.0)	1.00
Hospice	136	1 (1.0)	0 (0.0)	1.00	0 (0.0)	0 (0.0)	1.00
Highest level of oxygenation required during hospitalization (%)				0.23			
Room air	136	17 (16.7)	1 (2.9)		10 (16.1)	1 (3.6)	0.083
2–6 L by nasal cannula	136	50 (49.0)	19 (55.9)		31 (50.0)	13 (46.4)	
>6 L via oxygen mask	136	0 (0.0)	3 (8.8)		0 (0.0)	3 (10.7)	
High-flow oxygen	136	19 (18.6)	4 (11.8)		14 (22.6)	4 (14.3)	
Bipap	136	3 (2.9)	3 (8.8)		1 (1.6)	3 (10.7)	
Mechanical ventilation	136	13 (12.7)	4 (11.8)		6 (9.7)	4 (14.3)	
CRP (≤8.0 mg/dL) (range)	132	72.6 (2.5–450.0)	84.4 (11.2–193.3)	0.97	74.1 (2.5–450.0)	84.4 (16.9–157.8)	0.92
Procalcitonin (≤0.08 ng/mL) (range)	129	0.2 (0.1–141.4)	0.1 (0.1–3.7)	0.065	0.1 (0.1–141.4)	0.1 (0.1–3.7)	0.33
D-dimer (≤500 ng/mL) (range)	130	796 (210–38,158)	996 (210–14,117)	0.44	888 (228–24,133)	996 (210–14,117)	0.91
IL-6 (≤1.8 pg/mL) (range)	122	19.0 (1.6–424.0)	18.0 (2.9–600.0)	0.80	18 (2–424)	18 (4–600)	0.87
Ferritin (24–336 (males), 11–307 (females) µg/L (range)	132	411 (36–14,820)	411 (35–3003)	0.87	327 (36–7417)	424 (57–3003)	0.39
LDH (122–222 U/L) (range)	131	294 (14–1841)	281 (113–519)	0.22	307 (129–930)	277 (113–519)	0.26

The sample median (minimum, maximum) is given for continuous variables. P values result from the Fisher exact test (categorical variables) or a Wilcoxon rank-sum test (continuous and ordinal variables).

CRP, C-reactive protein; ICU, intensive care unit; IL, interleukin; LDH, lactate dehydrogenase; LTAC, long-term acute care; SNF, skilled nursing facility.

dissociation or Faget sign) has been reported in yellow fever, typhoid fever, and atypical pneumonia caused by *Chlamydia* species and Legionella.[9] The meaning of relative bradycardia is not universally accepted, but rather arbitrarily defined to describe an inappropriate chronotropic response to fever. Capoferri et al reported that patients with COVID-19-associated relative bradycardia (<90 bpm) were older than those with an appropriate heart rate response, but, in agreement with our findings, both groups had similar rates of admission to the intensive care unit, oxygen requirements, mechanical ventilation, and death.[10] The same group found the onset of low heart rate to have a median of 9 days from the beginning of symptoms, whereas our patients developed bradycardia much more rapidly, with an average of 41 hours from a positive nasal polymerase chain reaction result and a median duration of 5 days.

Most of our patients had lymphocytopenia (65.4%), a finding associated with poor clinical outcomes, and multilobar pneumonia (82.2%), and the overwhelming majority (97.7%) remained in sinus rhythm.[11] Coagulopathy and microangiopathy caused by endothelial disruption have been identified as risk factors for acute thrombotic complications in hospitalized COVID-19 patients.[12] In a retrospective cohort of 1114 patients, 2.6% of nonintensive care unit (ICU) patients and 35.6% of ICU counterparts experienced thrombosis, whereas in our study, the overall incidence was 8.1%, likely reflecting the small proportion of patients who required ICU care (15.6%) and mechanical ventilation (11.1%).[13]

Although the mechanism by which bradycardia develops in COVID-19 patients is not clearly established, it may be associated with a direct effect of the virus on the sinus node. In a study by Hu et al, sinus bradycardia progressively resolved, irrespective of the clinical course, as nucleic acid tests became negative.[8] Only 20.6% of our bradycardic patients and a smaller proportion of those not treated with nodal agents or antiarrhythmics (17.8%) had advanced associated intraventricular delay and wide QRS complex on electrocardiogram, likely reflecting a

Table 4. Comparison of COVID-19 treatments according to resolution of bradycardia

COVID-19 treatment	Resolution of bradycardia (n = 81) (%)	No resolution of bradycardia (n = 55) (%)	*P*
All patients			
Remdesivir	65 (80.2)	40 (72.7)	0.41
Corticosteroids	67 (82.7)	44 (80.0)	0.82
Lenzilumab	7 (8.6)	2 (3.6)	0.31
Convalescent plasma	18 (22.2)	8 (14.5)	0.37
Tocilizumab	7 (8.6)	5 (9.1)	1.00
Anakinra	2 (2.5)	1 (1.8)	1.00
Supportive care only			
Subset of patients not receiving a nodal agent/antiarrhythmic			
Remdesivir	40 (81.6)	30 (73.2)	0.45
Corticosteroids	40 (81.6)	33 (80.5)	1.00
Lenzilumab	6 (12.2)	0 (0.0)	0.030
Convalescent plasma	10 (20.4)	5 (12.2)	0.40
Tocilizumab	3 (6.1)	4 (9.8)	0.70
Anakinra	0 (0.0)	1 (2.4)	0.46
Supportive care only	7 (14.3)	7 (17.1)	0.78

P values result from the Fisher exact test. COVID-19, coronavirus disease 2019.

preferential viral effect on the sinus node rather than a generalized involvement of the intracardiac conduction system. Congestive heart failure may develop as a complication of COVID-19 and lead to severe disease and poor outcomes.[3] The most common echocardiographic abnormalities in patients with COVID-19 are right ventricular dilation, likely related to increasing pulmonary pressures as the disease worsens, and left ventricular dysfunction with preserved ejection fraction.[14,15] In our population, only 9 (6.6%) patients developed congestive heart failure during their hospitalization. In a series of 200 patients admitted to non-ICU departments, Pagnesi et al reported a prevalence of right ventricular dysfunction and pulmonary hypertension of 14.5% and 12%, respectively; however, only those with pulmonary hypertension had a more severe form of COVID-19 and worse clinical outcomes.[16] Severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2), the causative agent of COVID-19, enters human cells by coopting the peptidase angiotensin-converting enzyme 2.[17] Angiotensin-converting enzyme 2 receptors are highly expressed in cardiac cells and may represent a target for SARS-CoV-2 that leads to myocardial injury and clinical disease.[18] Consistent with a possible mechanism of direct viral invasion and damage to the heart, an autopsy study identified a high number of copies of SARS-CoV-2 in myocardial cells.[19] In the context of multisystem involvement, additional clinical and laboratory abnormalities such as hypoxemia, electrolyte abnormalities, and thyroid dysfunction also contribute to the development of bradycardia.[20] Treatment of COVID-19 with antivirals also has been reported as a cause of bradycardia.[21]

Of significance, 36.5% of our patients and 30.0% of those who were not receiving a nodal agent on admission, had a history of bradycardia, which may have made them more susceptible to very low heart rates during the hospitalization. The discontinuation of medications with negative chronotropic activity led to resolution of bradycardia in only 35.8%, however, lending force to the idea of alternative pathophysiologic mechanisms. Our data suggest that the inflammatory storm that ensues during COVID-19 may play an essential role in the process of myocardial injury and bradycardia, perhaps mimicking the abnormalities commonly seen in other types of viral myocarditis.[22] Chinitz et al reported a series of 7 patients with severe bradycardia and high-grade atrioventricular block that required cardiac pacing, in whom inflammatory markers were significantly elevated in the absence of electrocardiographic changes consistent with ischemia.[23] None of their patients had a history of cardiovascular disease or echocardiographic evidence of left ventricular dysfunction during the hospitalization and/or within the previous 6 months. It also is noteworthy that bradycardia developed before the onset of respiratory symptoms.

In our cohort, there was no difference in the serum levels of several biomarkers (C-reactive protein, procalcitonin, D-dimer, interleukin-6, ferritin, and lactate dehydrogenase) in relation to the degree of bradycardia. White patients had significantly lower lactate dehydrogenase levels compared with Black and Asian patients ($P = 0.002$), and, although not statistically significant ($P = 0.078$), a lower rate of ICU care; the latter may be the result of fewer associated comorbidities, which are not analyzed in this study. Our patients received a variety of therapeutic interventions, including remdesivir, corticosteroids, anti-SARS-CoV-2 convalescent plasma, and monoclonal antibodies, aimed at different targets such as granulocyte-macrophage-colony-stimulating factor and interleukin-6. Remdesivir, an antiviral approved by the Food and Drug Administration on October 22, 2020 for the treatment of COVID-19 patients, has been shown to shorten the recovery time in patients with lower respiratory infection who did not require mechanical ventilation.[24] Despite the initial enthusiasm and high expectations placed on the administration of convalescent plasma, a 2020 study showed no benefit in terms of overall status or survival.[25] If a benefit exists, then convalescent plasma infusion should be given early in the course of the infection, a period of high viral replication.[26] Lenzilumab is a first-in-class granulocyte-macrophage-colony-stimulating factor neutralizing monoclonal antibody that was used on a compassionate basis in a small group of patients and demonstrated significant reduction in the levels of biomarkers as well as more rapid improvement of clinical parameters and oxygenation.[27] In our study, lenzilumab was associated with the resolution of bradycardia in 7 (8.6%) of the patients and absence of resolution in 2 (3.6%; $P = 0.31$). In contrast, in the group of patients not treated with a nodal agent or antiarrhythmic, all experienced resolution of bradycardia (6 patients, 12% of the cohort).

Several limitations of our study should be acknowledged. The retrospective design may have introduced biases into the

data collection. In addition, the sample size was relatively small, and therefore the possibility of a type II error (ie, a false-negative finding) caused by the relatively small sample size must be considered. Finally, our study was conducted in a single, tertiary care medical center with a predominantly White population, which may have affected the results. It has been reported that compared with non-Hispanic Whites, Blacks and African Americans have a higher rate of disease, hospitalization, and death (1.4, 3.7, and 2.8 times higher, respectively).[28]

Conclusions

Sinus bradycardia occurs frequently in patients hospitalized with COVID-19, even in those treated with nodal agents and antiarrhythmics. We found no correlation between the degree of bradycardia, biomarker levels, and clinical outcomes, including oxygen requirements and need for ICU care. The monoclonal antibody lenzilumab was the only medication associated with the resolution of bradycardia in patients not treated with nodal agents or antiarrhythmics. Additional studies are needed to further delineate the appropriate extent of cardiac testing in patients with COVID-19, particularly those who present with bradycardia but no evidence of pulmonary hypertension or ventricular dysfunction.

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