

Electrocardiographic Profile of Adult Patients with Coronavirus Disease (COVID-19) who were Given Remdesivir and Admitted in the University of the Philippines-Philippine General Hospital (UP-PGH)

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ABSTRACT

Background and Objective. Severe Acute Respiratory Syndrome - Coronavirus-2 (SARS-CoV-2) was initially known to affect the respiratory system and has been reported to also involve the cardiovascular system leading to myocardial damage. Remdesivir is one of the approved treatments for COVID-19, wherein viral replication is inhibited by terminating the RNA transcription prematurely. According to studies, the primary electrocardiographic effect of remdesivir in COVID-19 patients are sinus bradycardia and QT prolongation. The use of electrocardiogram (ECG) is an essential diagnostic tool in assessing the electrical conditions of the heart. The objective of this study is to describe the electrocardiographic profile of adult patients with COVID-19 who were given remdesivir and admitted in the University of the Philippines-Philippine General Hospital (UP-PGH). To this date, this is the only study done locally identifying the electrocardiographic profiles of adult patients with COVID-19 who were given remdesivir.

Methods. This was a retrospective descriptive study involving adult patients with COVID-19 who were given remdesivir and admitted in UP-PGH from June 2021 to June 2022. Demographic profiles and 12-lead ECG done during the hospital admission were gathered. Descriptive statistics was used to summarize the clinical characteristics and the electrocardiographic findings of the patients.

Results. There were 412 confirmed COVID-19 patients who were given remdesivir (mean age 56 years old; female 52%) included in this study. The most common comorbidities were hypertension, diabetes mellitus, and stroke. Majority of the patients had severe (58%) to critical (22%) COVID-19 infection. Most of the patients had sinus rhythm (94%), normal rate (72%), and normal axis (93%). The most common baseline ECG findings were non-specific ST-T wave changes (42%). Some patients had atrioventricular blocks (3.4%), bundle branch blocks (3.6%), prolonged QT interval (1.9%). Among those with repeat 12-L ECG (136 patients) during admission, ECG changes observed were sinus bradycardia (6%), prolonged QT interval (4%), and both (1.5%).



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Conclusion. Based on this retrospective review, which to our knowledge is the only study done locally investigating the effects of remdesivir on ECG of adult Filipino patients with COVID-19 infection, majority of the patients had sinus rhythm, normal rate, and axis. The most common ECG finding was non-specific ST-T wave changes. This study demonstrated a low incidence of adverse ECG changes that would preclude the administration of remdesivir when indicated. These include sinus bradycardia and QT interval prolongation which did not require further interventions. ECG remains to be useful, low-cost noninvasive tool that can help monitor electrophysiologic adverse events of remdesivir.

Keywords: *electrocardiogram, COVID-19, remdesivir*

INTRODUCTION

The World Health Organization (WHO) declared COVID-19 last March 12, 2020, as a global pandemic.¹ Since the onset of the pandemic, a lot of efforts has been made to control the spread of infection. Several therapeutic drugs have been investigated and experimented for the treatment of COVID-19; one of these antiviral drugs is remdesivir. In a study of Beigel et al., they concluded that remdesivir was superior to placebo in decreasing the time of recovery in patients infected with COVID-19.² However, after the initiation of remdesivir infusion, it was noted that patients developed bradycardia and/or prolongation of QT interval.²⁻⁹

Review of Related Literature

Severe Acute Respiratory Syndrome - Coronavirus-2 (SARS-CoV-2), was the virus responsible for coronavirus disease-2019 (COVID-19), that caused an outbreak in Wuhan, China last December 2019 and was declared by WHO as a pandemic last March 2020. The disease continued to rapidly increase worldwide and is associated with high mortality rate, especially in patients with known comorbidities.¹⁰ COVID-19 is initially thought to affect the respiratory system but it has also been reported to also involve the cardiovascular system leading to myocardial damage. Different mechanisms have been proposed for the myocardial damage, namely the cytokine release syndrome and direct myocardial cell injury.¹¹ According to Lugo et al., young, healthy individuals who have the delta COVID-19 variation are more likely to experience life-threatening cardiac problems.¹²

According to the Department of Health, patients without evidence of pneumonia or hypoxia and without risk factors are categorized to have mild disease. While infected patients who are elderly and/or with co-morbidities are considered to have moderate COVID-19. Severe COVID-19 patients are those with pneumonia with signs of respiratory distress and oxygen desaturation requiring oxygen supplementation. Lastly, critical COVID-19 are patients with impending acute respiratory failure and/or in septic shock.¹³

The Philippine Society for Microbiology and Infectious Diseases recommended the use of Remdesivir with Dexamethasone in COVID-19 patients requiring oxygen supplementation.¹⁴ Furthermore, remdesivir is also approved by the Food and Drug Administration (FDA) as a treatment for COVID-19. Remdesivir is able to inhibit viral replication by terminating the RNA transcription prematurely.¹⁰ It is found to be an effective antiviral agent against other virus families including Filoviridae, Paramyxoviridae, Pneumoviridae, and Orthocoronavirinae.¹² Remdesivir also has showed in vitro activity against SARS-CoV-2. In a study by Sheahan et al., it was noted that the use of remdesivir and IFN- β have superior antiviral activity in vitro, and in mice when used prophylactically and therapeutically, remdesivir improved pulmonary function and reduced lung viral loads and severe lung pathology.¹⁵ Remdesivir is a nucleoside analogue that commonly target viral replication, particularly the viral DNA or RNA polymerase.¹⁶ In a literature review done by Nabati and Parsaei on the potential cardiotoxic effects of remdesivir, it shows that remdesivir can possibly cause cardiotoxicity by binding to human mitochondrial RNA polymerase and can induce QT prolongation as well as Torsade de point by increasing the potential duration with decreased Na⁺ peak amplitudes in a dose-dependent manner.⁵

The use of electrocardiogram (ECG) is an essential diagnostic tool in assessing the electrical and muscular conditions of the heart. Based on a recently published case report by Gupta et al., the primary cardiac adverse effects of Remdesivir in COVID-19 patients are primarily bradycardia, hypotension, and QT prolongation.⁸ Adults with sinus bradycardia are diagnosed when their sinus node discharges at a rate of less than 50 beats per minute. While prolonged QT interval, through Bazett formula, is considered for corrected QT interval of more than 460 msec for women and more than 450 msec for men. Barkas et al. noted in their case report the occurrence of sinus bradycardia within three days of remdesivir infusion with resolution after discontinuation.⁶ According to other case reports, adverse effects of remdesivir are noted after three doses of infusion with spontaneous resolution after discontinuation.⁵ Another case study by Day et al. mentioned that bradycardia occurred immediately after remdesivir infusion and with improvement in heart rate after discontinuing the drug within 48 hours.⁷

Remdesivir has been recommended for use in patients with moderate to critical COVID-19 infection. Data suggest that it reduces time to recovery and risk of mechanical ventilation. Given the frequent use of remdesivir among patients with COVID-19, it is essential to consider the possible cardiac adverse effects to our patients with the use of ECG monitoring.

OBJECTIVES

This study aimed: (1) to describe the demographic and clinical profile of patients admitted in UP - PGH with COVID-19 infection who were given remdesivir; (2) to determine the baseline electrocardiographic findings of patients with COVID-19; (3) to identify the electrocardiographic changes during remdesivir administration, and (4) to describe the frequency of bradycardia and QT prolongation according to the doses of remdesivir infusion.

MATERIALS AND METHODS

This was a retrospective descriptive study involving adult patients with COVID-19 who were given remdesivir and admitted in UP-PGH from June 2021 to June 2022. Demographic profiles and electrocardiographic findings during the hospital admission were gathered from the UP-PGH DCVM ECG study Group database. COVID-19 patients who are not confirmed by SARS-CoV-2 RT-PCR, had no ECG tracing, and did not receive remdesivir during hospitalization were excluded in the study (Figure 1).

Demographic Profile

The demographic profiles of the included patients were gathered wherein the data collected are the following: age, sex, comorbidities, current medications, baseline laboratory results, SARS-CoV2 infection category upon admission, baseline vital signs, and COVID-19 treatments received.

Baseline Electrocardiographic Findings

The electrocardiographic findings were determined. Data collected included rate, rhythm, conduction abnormalities such as QT prolongation and atrioventricular block.

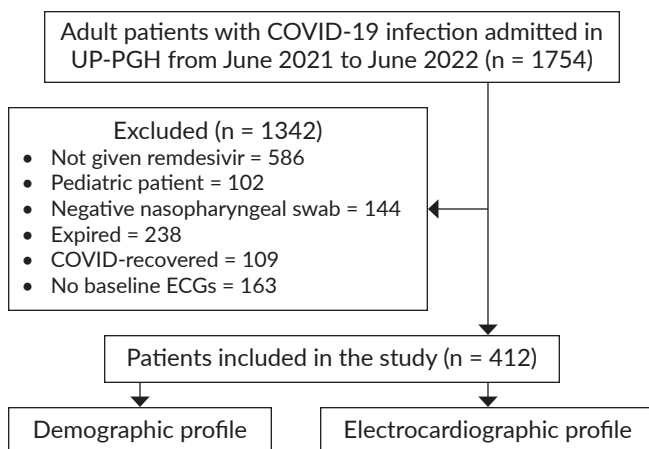


Figure 1. Flow diagram of inclusion and exclusion criteria.

Electrocardiographic Changes during Remdesivir Administration

Serial ECG tracings of patients during hospitalization were collected. The multiple ECG tracings were compared (baseline versus during remdesivir infusion). Rate, rhythm and conduction abnormalities were identified.

Data Analysis

Descriptive statistics were used to summarize the demographic and clinical characteristics of the patients. Frequency and proportion were used for categorical variables; median and interquartile range for non-normally distributed continuous variables; mean and SD for normally distributed continuous variables. Shapiro-Wilk test was used to test the normality of the continuous variables. Missing values were neither replaced nor estimated. STATA 13.1 was used for data analysis.

Ethical Considerations

The protocol of the study was approved by the UP Manila Research Ethics Board (UPM-REB) with code 2022-0451-01. This study was conducted by the primary author and co-investigators following the set guidelines of the Philippine National Ethical Guidelines for Health Research and in accordance with the Declaration of Helsinki.

RESULTS

Demographic and Clinical Profile

A total of 412 confirmed COVID-19 patients were included in this retrospective descriptive study. The demographic and clinical profiles are shown in Table 1, with mean age of 56 ± 17 years old. The majority of the patients were female (52.67%). The most common comorbidities were hypertension (58.98%), diabetes mellitus (28.4%), and stroke (6.07%). Majority of patients had severe (58.01%) to critical (21.84%) COVID-19 infection while 16.75% had moderate COVID-19 infection on admission.

Vital signs and oxygen support

Upon admission, mean systolic blood pressure was 128 ± 21 mmHg while the mean diastolic blood pressure was 79 ± 14 mmHg. The mean heart rate was 92 ± 19 bpm. None of the patients were bradycardic at baseline but 21 patients (5.19%) were tachycardic. The mean respiratory rate was 25 ± 13 cpm. A total of 299 patients (74.01%) were noted to be tachypneic. The mean temperature was 36.52 ± 0.76 with 14 patients (3.61%) febrile upon admission. Lastly, mean oxygen saturation was 90 ± 10 . Majority of the patients with a total of 171 (41.5%) were in need of oxygen support via nasal cannula and 113 (27.43%) needed oxygenation via facemask, 12 (2.91%) were intubated and hooked to mechanical ventilator while 97 (23.54%) did not require oxygen supplementation.

Cardiovascular and COVID-19 medications on admission

Cardiovascular and COVID-19 medications given to the patients on admission are shown in Table 2. The most common cardiovascular drugs given to patients on admission were angiotensin II receptor blocker (23.54%), beta blocker (11.41%), calcium channel blocker (18.69%) and statin (9.47%). The most commonly administered COVID-19 medication aside from remdesivir were steroid (58.01%) and tocilizumab (32.77%).

Baseline Laboratory Profile of the Patients

Baseline laboratory profile of the patients are shown in Table 3. Majority of the included patients had anemia (60.78%) but with normal platelet (77.4%) and white blood cell (63.41%) counts. Most of the patients included had normal creatinine, with mean eGFR of 88. Majority of the blood chemistries like potassium, calcium, and magnesium were normal. Only 50 (12%) patients had troponin determination, 46 (92%) had normal result while 4 (8%) had elevated troponin result. 47% of the patients had elevated NT-proBNP and 90.49% had elevated D-dimer. Mean PaO₂/FiO₂ ratio (P/F ratio) observed is 221; 30.67% of the patients had normal P/F ratio while 30.4% was in moderate acute respiratory distress syndrome (ARDS), 25.33% with mild ARDS and 13.6% with severe ARDS on admission.

Baseline electrocardiographic findings

The baseline electrocardiographic findings included in this study are shown in Table 4. Majority of the ECG result had normal rate (72%). Only 3.41% of the ECG had sinus bradycardia. Most of the ECGs were in sinus rhythm (93.93%) with less than 5% seen with atrial fibrillation and atrial flutter combined. Conduction abnormalities were rare; first degree atrioventricular block was seen in 11 patients (2.67%), second-degree AV block in two patients (0.48%) and third-degree AV block in one patient (0.24%), as well as in right bundle branch block comprising only 2.67%. Left ventricular hypertrophy was seen in 7.04% and left atrial abnormality in 8.74% compared with right ventricular hypertrophy and right atrial abnormality each consisting of less than 1%. The most common electrocardiographic abnormalities seen were non-specific ST-T wave changes (42.23%), while ST-elevation myocardial infarction was seen in 2.67%, ST depression in 0.97% and T wave inversion in 1.7%. Majority had normal QT interval (96.84%) while prolonged and shortened QT interval was seen in 1.94% and 1.21%, respectively.

ECG changes observed during remdesivir infusion

Among the 412 patients included in this study, 136 (33%) had multiple ECGs done during hospitalization. Sinus bradycardia was observed in 6% followed by prolonged QT interval with 4% and those with both sinus bradycardia

Table 1. Demographic and Clinical Profile of the Patients (n=412)

	Frequency (%); Mean ± SD
Age	56 ± 17
Sex	
Male	195 (47.33)
Female	217 (52.67)
Comorbidities	
Hypertension	243 (58.98)
Diabetes mellitus	117 (28.4)
Dyslipidemia	60 (14.56)
Heart failure	10 (2.43)
Ischemic heart disease	16 (3.88)
Valvular disease	4 (0.97)
Stroke	25 (6.07)
Chronic kidney disease (non-dialysis requiring)	21 (5.1)
End-stage renal disease (dialysis requiring)	2 (0.49)
Asthma	24 (5.83)
COPD	5 (1.21)
Pulmonary tuberculosis	17 (4.13)
Cancer	11 (2.67)
Autoimmune	5 (1.21)
COVID-19 Severity	
Mild	5 (1.21)
Moderate	69 (16.75)
Severe	239 (58.01)
Critical	90 (21.84)
Asymptomatic	9 (2.18)

Table 2. Cardiovascular and COVID-19 Medications on Admission (n=412)

On admission	Frequency (%); Mean ± SD
Steroid	279 (67.72)
Tocilizumab	69 (16.75)
Anticoagulant	97 (23.54)
Antiplatelet	32 (7.77)
Angiotensin receptor blocker	97 (23.54)
ACE inhibitor	15 (3.64)
ARNI	4 (0.97)
Calcium channel blocker	77 (18.69)
Beta blocker	47 (11.41)
Ivabradine	2 (0.49)
Mineralocorticoid receptor antagonist	4 (0.97)
Diuretic	14 (3.4)
Statin	39 (9.47)

and prolonged QT interval was seen in 1.5%. Remdesivir infusion was discontinued in 2.2% due to the occurrence of sinus bradycardia. No active interventions done nor deemed necessary by the attending physicians for patients who developed sinus bradycardia, prolonged QT interval or both. One patient developed first-degree AV block and another patient had second degree AV block Mobitz type 1.

Incidence of sinus bradycardia and prolonged QT interval during remdesivir infusion

The incidence of sinus bradycardia and prolonged QT interval during remdesivir infusion are shown in Table 5. Among patients who were given five doses of remdesivir at most, sinus bradycardia occurred in seven patients and prolongation of the QT interval was seen in three patients.

Table 3. Baseline Laboratory Profile of the Patients (n=412)

	Valid observation	Median (IQR)	Frequency %		
			Normal	Low	Elevated
<i>Hemoglobin</i>	408	130 (119 to 141)	38.24	60.78	0.98
<i>Platelet</i>	407	234 (163 to 310)	77.40	17.20	5.41
<i>WBC</i>	399	8.5 (6.1 to 11.9)	63.41	8.02	28.57
<i>Creatinine</i>	400	7 (52 to 93.5)	50.25	31.50	18.25
<i>eGFR</i>	114	88 (63 to 106)	-	-	-
<i>Potassium</i>	392	4 (3.6 to 4.4)	76.28	18.11	5.61
<i>Calcium</i>	344	2.16 (2.08 to 2.25)	67.73	29.36	2.91
<i>Albumin</i>	307	35 (32 to 38)	51.79	47.23	0.98
<i>Magnesium</i>	332	0.89 (0.8 to 0.97)	74.40	7.83	17.77
<i>NT-proBNP</i>	102	264.32 (41 to 1279)	52.94	-	47.06
<i>Troponin</i>	51	0.066 (0.018 to 0.24)	92.16	-	7.84
<i>D-dimer</i>	410	1.31 (0.78 to 2.65)	9.51	-	90.49
<i>PaO₂/FiO₂ ratio</i>	375	221 (140 to 333)			
<i>Normal</i>		30.67%			
<i>Mild ARDS</i>		25.33%			
<i>Moderate ARDS</i>		30.40%			
<i>Severe ARDS</i>		13.60%			

Table 4. Baseline Electrocardiographic Findings (n=412)

Baseline ECG findings	Frequency (%)
Rate	
Bradycardia	14 (3.41)
Tachycardia	101 (24.57)
Rhythm	
Sinus	387 (93.93)
Atrial fibrillation	16 (3.88)
Axis	
Left axis deviation	23 (5.58)
Right axis deviation	7 (1.7)
ST segment elevation MI	11 (2.67)
ST segment depression	4 (0.97)
T wave inversion	7 (1.7)
Left ventricular hypertrophy	29 (7.04)
Left atrial enlargement	36 (8.74)
Non-specific ST-T wave changes	174 (42.23)
Low voltage complexes	31 (7.52)
QTc interval	
Prolonged	8 (1.94)
Shortened	5 (1.21)

Table 5. Incidence of Sinus Bradycardia and Prolonged QT Interval during Remdesivir Infusion

Number of Remdesivir doses	Number of patients who had sinus bradycardia	Number of patients who had Prolonged QT interval
1		
2	4	1
3	1	1
4	1	
5	1	1
6		
7		
8	1	
9		
10	1	2

On the other hand, among those patients who received more than five doses of remdesivir, two patients developed sinus bradycardia and two had prolonged QT interval. However, it is important to take note that only seven patients received more than five doses of Remdesivir.

Out of the 9 patients who developed sinus bradycardia during remdesivir infusion, two of the patients also had ongoing propofol and fentanyl drip, which are possible confounding causes for the occurrence of sinus bradycardia. Sinus bradycardia was purely attributed to Remdesivir in 77% (7 out of 9) of the patients. One of the patients who had sinus bradycardia died after five days of hospitalization due to septic shock and this was two days after administration of two doses of Remdesivir. Another patient who had both sinus bradycardia and prolonged QT interval died due to sepsis from COVID-19 and the demise was 16 days after completion of 10 doses of Remdesivir.

Out of the five patients who developed QT interval prolongation during Remdesivir infusion, three patients were also on sedatives like propofol and fentanyl or being given azithromycin, which could also cause QT prolongation. Only two cases of QT prolongation could be purely attributed to remdesivir infusion.

DISCUSSION

A total of 412 COVID-19 patients who were given remdesivir and admitted from June 2021 to June 2022 were included in this study. Majority of the patients were female with mean age of 50 years old. Almost all of the patients had comorbidities with hypertension and diabetes mellitus as the most common. Compared to the study of Hajimoradi et al., in Iran, patients had similar demographic and clinical profiles, none of the patients included in both studies were hypotensive, and around 30% required oxygen supplementation upon admission, but in contrast to the study of Hajimoradi et al., our study included respiratory rate and majority of them were tachypneic composing of 74%.⁹ According to the NIH COVID-19 treatment guidelines, remdesivir is one of the drugs approved by the Food and Drug Administration for the treatment of COVID-19 and it is given to patients with moderate to critical infection.¹⁰ In our study, majority of the patients had severe to critical infection, hence the need for remdesivir treatment. In addition to remdesivir as COVID-19 treatment, most of the patients included also received steroids and tocilizumab. On the other hand, the most common cardiovascular medications received by the patients were angiotensin II receptor blocker, beta blocker, calcium channel blocker, and statin, given that most patients had hypertension, diabetes mellitus, and dyslipidemia. Anemia was a common CBC abnormality finding in our patients with normal platelet and WBC counts. According to the study of Tao et al., anemia is an independent risk factor for the severity of COVID-19 infection.¹⁷ It is known that electrolyte imbalances can cause electrocardiogram changes;

hyperkalemia can cause bradycardia or hypocalcemia may cause QT interval prolongation. However, in our study, baseline electrolytes were mostly within normal limits. One of the limitations of this study is that electrolytes during remdesivir administration were not analyzed as a possible confounding factor for the ECG changes. Notably, in this study, 90.49% of the patients had elevated D-dimer result.

The baseline electrocardiographic results in this study were consistent with the findings in the study done by McCullough et al., showing that, majority of the patients had normal rate, normal axis, and sinus rhythm, and that at baseline, atrioventricular blocks and bundle branch blocks are rare.¹⁸ Furthermore, the most common ECG abnormality are non-specific ST-T wave changes, and that myocardial infarctions, ischemia, and prolonged QT interval are rare at baseline. In this study, the most common ECG changes observed during remdesivir administration was sinus bradycardia (25.81%), consistent with the study of Kumar et al. which reported 28.7% and the study of Hajimoradi et al. which reported that 27% developed bradycardia after receiving remdesivir.^{9,19} Since this study is of descriptive design, the statistically significant incidence of bradycardia after remdesivir administration was not analyzed. Compared with the findings of Attena et al. and Hajimoradi et al., wherein there was a statistically significant incidence of sinus bradycardia after administration of five doses of remdesivir.^{4,9} On the other hand, the study of Bistrovic and Lucijanic which investigated 14 patients diagnosed with COVID-19 with a baseline ECG and observed changes two hours after remdesivir infusions demonstrated that rightward T wave inversion and T-axis deviation were predictors for sudden cardiac death and nonfatal cardiac events in elderly.³ These findings were not noted in our study. Additionally, their study was not able to demonstrate QT prolongation and bradycardia among patients given remdesivir within their specified time period.

There are reported studies showing that remdesivir treatment for COVID-19 patients causes bradycardia and QT prolongation which is consistent with our findings. Due to this complication, some health practitioners tend to discontinue remdesivir halting the benefit it provides to COVID-19 patients. As of today, there are still no set guidelines on the monitoring of COVID-19 patients undergoing remdesivir treatment. Given these findings, there is benefit of ECG monitoring during remdesivir administration to enable us to provide the optimal treatment for COVID-19 patients.

This study has limitations. Although all patients included in this study had baseline ECGs done during the admission, not all had regular monitoring of ECG during remdesivir administration. Performing a repeat ECG was upon the discretion of the attending physician. In addition, other factors like medications, electrolytes or hemodynamic instability that could cause ECG changes during remdesivir administration were not analyzed in this study.

CONCLUSION

Based on this retrospective review, which to our knowledge is the only study done locally investigating the effects of remdesivir on ECG of adult Filipino patients with COVID-19 infection, majority of the patients were in sinus rhythm, normal rate, and axis. The most common ECG finding was non-specific ST-T wave changes. This study has demonstrated a low incidence of adverse ECG changes that would preclude the administration of remdesivir when indicated. These include sinus bradycardia and QT interval prolongation which did not require further interventions. ECG remains to be useful, low-cost noninvasive tool that can help monitor electrophysiologic adverse events of remdesivir.

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Disclaimer

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Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

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REFERENCES

1. Coronavirus disease (COVID-19) pandemic. (2025, April 28). <https://www.who.int/europe/emergencies/situations/covid-19>.
2. Beigel JH, Tomashek KM, Dodd LE, Mehta AK, Zingman BS, Kalil AC, et al. Remdesivir for the Treatment of Covid-19 – Final Report. *N Engl J Med*. 2020 Nov 5;383(19):1813–26. doi: 10.1056/NEJMoa2007764.
3. Bistrovic P, Lucijanac M. Remdesivir might induce changes in electrocardiogram beyond bradycardia in patients with coronavirus disease 2019 – The pilot study. *J Med Virol*. 2021 Oct;93(10):5724–5. doi: 10.1002/jmv.27177. PMID: 34232520; PMCID: PMC8426664.
4. Attena E, Albani S, Maraolo AE, Mollica M, De Rosa A, Pisapia R, et al. Remdesivir-Induced Bradycardia in COVID-19: A Single Center Prospective Study. *Circ Arrhythm Electrophysiol*. 2021 Jul; 14(7):e009811. doi:10.1161/CIRCEP.121.009811. PMID: 34182791; PMCID: PMC8294658.
5. Nabati M, Parsaei H. Potential Cardiotoxic Effects of Remdesivir on Cardiovascular System: A Literature Review. *Cardiovasc Toxicol*. 2022 Mar 1;22(3):268–72. doi: 10.1007/s12012-021-09703-9. 6. Barkas F, Styli CP, Bechlioulis A, Milionis H, Liberopoulos E. Sinus Bradycardia Associated with Remdesivir Treatment in COVID-19: A Case Report and Literature Review. *J Cardiovasc Dev Dis*. 2021 Feb;8(2):18. doi: 10.3390/jcdd8020018. PMID: 33673216; PMCID: PMC7918811.
6. Barkas F, Styli CP, Bechlioulis A, Milionis H, Liberopoulos E. Sinus Bradycardia Associated with Remdesivir Treatment in COVID-19: A Case Report and Literature Review. *J Cardiovasc Dev Dis*. 2021 Feb;8(2):18. doi: 10.3390/jcdd8020018. PMID: 33673216; PMCID: PMC7918811.
7. Day LB, Abdel-Qadir H, Fralick M. Bradycardia associated with remdesivir therapy for COVID-19 in a 59-year-old man. *CMAJ*. 2021 Apr 26;193(17):E612–5. doi: 10.1503/cmaj.210300. PMID: 33903133; PMCID: PMC8101980.
8. Gupta AK, Parker BM, Priyadarshi V, Parker J, Gupta AK, Parker BM, et al. Cardiac Adverse Events with Remdesivir in COVID-19 Infection. *Cureus*. 2020 Oct 24;12(10). doi: 10.7759/cureus.11132. PMID: 33240723; PMCID: PMC7682945.
9. Hajmoradi M, Sharif Kashani B, Dastan F, Aghdasi S, Abedini A, Naghashzadeh F, et al. Remdesivir associated sinus bradycardia in patients with COVID-19: A prospective longitudinal study. *Front Pharmacol*. 2023;13. doi: 10.3389/fphar.2022.1107198. PMID: 36733376; PMCID: PMC9888491.
10. National Institutes of Allergy and Infectious Diseases - Rocky Mountain Labs. Information on COVID-19 Treatment, Prevention and Research [Internet]. COVID-19 Treatment Guidelines. [cited 2023 Aug 29]. Available from: <https://www.covid19treatmentguidelines.nih.gov/>.
11. Basu-Ray I, Almaddah N k, Adeboye A, Soos MP. Cardiac Manifestations of Coronavirus (COVID-19). In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 [cited 2023 Aug 29]. Available from: <http://www.ncbi.nlm.nih.gov/books/NBK556152/>. PMID: 32310612.
12. Brown AJ, Won JJ, Graham RL, Dinnon KH, Sims AC, Feng JY, et al. Broad spectrum antiviral remdesivir inhibits human endemic and zoonotic deltacoronaviruses with a highly divergent RNA dependent RNA polymerase. *Antiviral Res*. 2019 Sep 1;169:104541. doi: 10.1016/j.antiviral.2019.104541. PMID: 31233808; PMCID: PMC6699884.
13. Rementizo, E. (2022, August 2). Circular No. : 2022-004 – DOH Department Circular No. 2022-0002 – Advisory on COVID-19 Protocols for Quarantine and Isolation. People Management Association of the Philippines - the Official Website of PMAP. <https://pmap.org.ph/circular-no-2022-004/>.
14. Institute of Clinical Epidemiology, National Institutes of Health - UP Manila, Philippine Society for Microbiology and Infectious Diseases. Philippine COVID-19 Living Recommendations. 2021.
15. Sheahan TP, Sims AC, Leist SR, Schäfer A, Won J, Brown AJ, et al. Comparative therapeutic efficacy of remdesivir and combination lopinavir, ritonavir, and interferon beta against MERS-CoV. *Nat Commun*. 2020 Jan 10;11(1):222. doi: 10.1038/s41467-019-13940-6. PMID: 31924756; PMCID: PMC6954302.
16. Agostini ML, Andres EL, Sims AC, Graham RL, Sheahan TP, Lu X, et al. Coronavirus Susceptibility to the Antiviral Remdesivir (GS-5734) Is Mediated by the Viral Polymerase and the Proofreading Exoribonuclease. *mBio*. 2018 Mar 6;9(2):10.1128/mbio.00221-18. doi: 10.1128/mbio.00221-18. PMID: 29511076; PMCID: PMC5844999.
17. Tao Z, Xu J, Chen W, Yang Z, Xu X, Liu L, et al. Anemia associated with severe illness in COVID-19: A retrospective cohort study. *J Med Virol*. 2021;93(3):1478–88. doi: 10.1002/jmv.26444. PMID: 32813298; PMCID: PMC7461220.
18. McCullough SA, Goyal P, Krishnan U, Choi JJ, Safford MM, Okin PM. Electrocardiographic Findings in Coronavirus Disease-19: Insights on Mortality and Underlying Myocardial Processes. *J Card Fail*. 2020 Jul 1;26(7):626–32. doi: 10.1016/j.cardfail.2020.06.005. PMID: 32544622; PMCID: PMC7293518.
19. Kumar S, Arcuri C, Chaudhuri S, Gupta R, Aseri M, Barve P, et al. A novel study on SARS-COV-2 virus associated bradycardia as a predictor of mortality-retrospective multicenter analysis. *Clin Cardiol*. 2021;44(6):857–62. doi: 10.1002/clc.23622. PMID: 33964035; PMCID: PMC8207973.