JBCR Journal of Biomedical & Clinical Research
Journal of Medical University of Pleven

Journal of Biomedical and Clinical Research 17(2): 177–186 (2024)
DOI: 10.3897/jbcr.e128440

Research Article

Association between rhythm-conduction disorders and COVID-19 infection

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Summary

The paper highlights the correlation between COVID-19 and the heightened susceptibility to arrhythmia - a cardiac rhythm disturbance, in hospitalised individuals. The data indicates a significant positive link between the quantity of COVID-19 instances and the number of patients with rhythm pathology. The COVID-19 infection is linked to an increased risk of arrhythmia. The precise ways in which COVID-19 harms the heart tissue and leads to rhythm and conduction disturbances are not completely known and necessitate additional investigation. Moreover, research indicates that a previous SARS-CoV-2 infection may increase the likelihood of rhythm complications.

Key words: Arrhythmias, atrial fibrillation, AV block, COVID-19, heart damage

Introduction

The SARS-CoV-2 virus, which is the cause of the novel COVID-19 pandemic, which spread around the world in 2019, is a threat. SARS-CoV-2 belongs to the viral Group Coronaviridae subfamily, specifically as the eighth strain. Of these viruses, SARS- CoV-1, MERS, and SARS-CoV-2 can cause severe infections in humans and animals, while HKU1, NL63, OC43, and 229E are associated with minor respiratory symptoms (Akkawi and Ghazal 2021). The name of the Coronaviridae family viruses is associated with the spikes on their surface, which form a crown-like structure. It is established that SARS-CoV-2 can lead to severe and occasionally fatal respiratory infections in humans (Nanshan et al. 2020). Conversely, acute respiratory infections contribute to complications in cardiovascular diseases (CVD), and the presence of CVD can, in turn, complicate and exacerbate the progression of the infectious disease (Dhainaut et al. 2005; Cowan et al. 2018). SARS-CoV-2 causes acute respiratory syndrome, which sets off a strong inflammatory response and a cytokine storm. Damages are caused to many organ systems, especially the cardiovascular system, which is a key factor in the worsening of CVD (Mladenova et al. 2020; Zhou et al. 2020). The virus specifically binds to the zinc peptidase angiotensin-converting enzyme 2 (ACE2), serving as a receptor for viral entry. ACE2 is a surface molecule present in vascular endothelial cells, arterial smooth muscle, and cardiac myocytes (Mendoza-Torres et al. 2015; Xu et al. 2020).

Academic editor: Galya Stavreva
Received: 29 May 2024
Accepted: 15 August 2024
Published: 12 November 2024

Citation: Yovchev Z, Gospodinov K, Todorova Y, Tisheva-Gospodinova S (2024) Association between rhythm-conduction disorders and COVID-19 infection. Journal of Biomedical and Clinical Research 17(2): 177–186. https://doi.org/10.3897/jbcr.e128440

Given this interaction, some authors have proposed the concept of a distinct manifestation of vasculitis, particularly in specific strains. When SARS-CoV-2 attaches to ACE2 receptors on myocardial cells, it leads to the suppression of regulatory processes, followed by the accumulation of angiotensin II. Subsequently, adverse myocardial remodelling occurs, mediated by the action of angiotensin II on ACE1 receptors (Atri et al. 2020). This intricate process contributes to various heart complications, encompassing the onset or exacerbation of diverse arrhythmias. The array of arrhythmias observed in COVID-19 patients is broad, including atrial fibrillation (AF), atrial flutter, supraventricular tachycardia, monomorphic and polymorphic ventricular tachycardia, and various conduction disorders.

Mechanisms of arrhythmogenesis

Preliminary investigations indicate a heightened incidence of cardiac arrhythmias associated with coronavirus disease (COVID-19). Heart myocyte damage from the Coronavirus 2 infection and subsequent severe acute respiratory syndrome increases the likelihood of arrhythmias. An examination of available data suggests that the pathophysiology of COVID-19-related arrhythmia is intricate and may stem from tissue damage, often referred to as myocardial damage. This damage could be a result of inflammatory harm to the myocytes (myocarditis) or due to myocardial ischemia and/or necrosis, akin to myocardial infarction (Fig. 1). Another contributing factor to arrhythmia involves heightened pressure in the right ventricle due to pulmonary hypertension or pulmonary embolism.

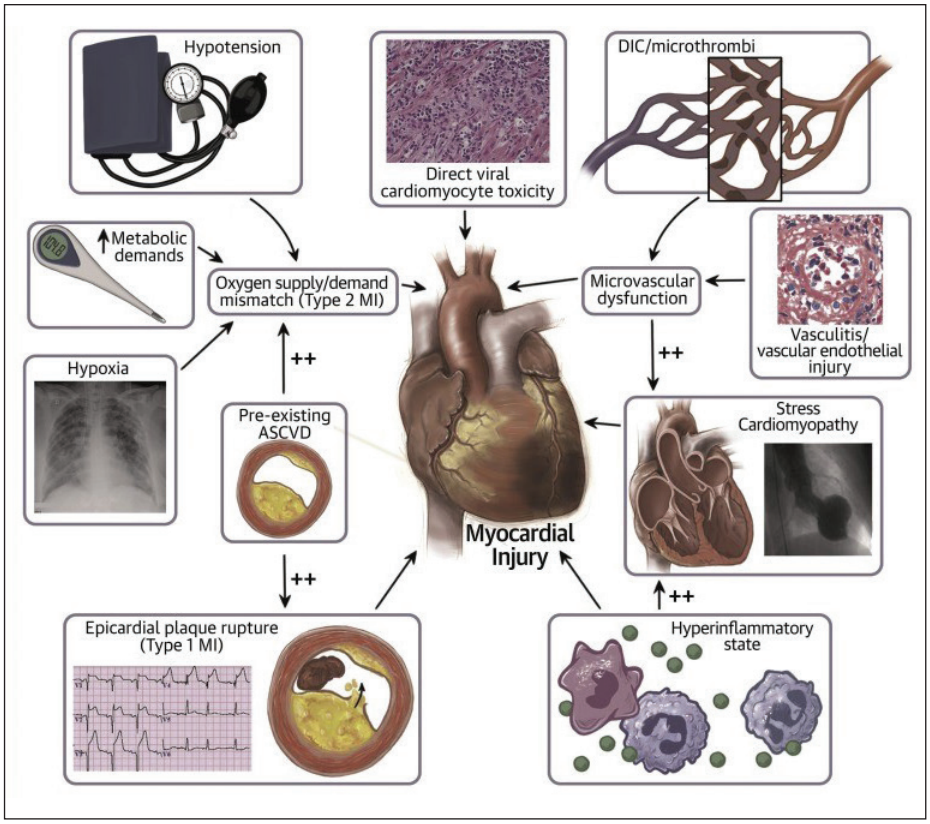

Figure 1. Potential mechanisms of myocardial injury in COVID-19. Adapted from Atri D et al. (2020, p. 518).

Additionally, arrhythmia can be induced by cell-mediated cytotoxicity from CD8+T lymphocytes migrating into the heart, causing myocardial inflammation. Overactivation of lymphocytes due to a cytokine storm leads to an excessive release of pro-inflammatory mediators, creating a positive feedback loop of immune activation and myocardial damage—a vicious circle. A case was presented by Sala et al. (2020) involving myocarditis confirmed simultaneously with magnetic resonance imaging (MRI) and endomyocardial biopsy in a 43-year-old woman with COVID-associated pneumonia, showcasing diffuse T-cell leachates. Notably, the presence of SARS-CoV-2 genomic material in the myocardium was absent, and revealed the myocardial changes in the cited case as morphologically opposite to Taco-Tsubo cardiomyopathy, with impairment observed in the basal and middle heart segments (Sala et al. 2020).

Other potential mechanisms for arrhythmia involve the use of arrhythmogenic drugs in the treatment of COVID-19. Some of the initially recommended medications during the pandemic have a definite pro-arrhythmogenic effect, as do some antibiotics widely used for treatment. Electrolyte imbalance in hospitalised patients with severe forms is a sufficient prerequisite for rhythm disturbances. Another cause may also be the endogenous catecholamine adrenergic status. A study by Guo et al. (2020) highlighted that patients with underlying cardiovascular disease (CVD) had elevated troponin-T (TnT) levels, leading to more frequent development of complications, including malignant arrhythmias such as ventricular tachycardia and fibrillation (Xiong et al. 2020).

It has been established that patients without a previous history of CVD also have high levels of TnT and develop malignant arrhythmias, albeit with a lower frequency (TnT levels are elevated in 13.2% of patients without underlying CVD versus 54.5% of patients with CVD; malignant arrhythmias are observed in 5.2% of patients without CVD versus 11.5% in patients with CVD).

Cardiac arrhythmias are twice as common as elevated troponin levels. In a global study conducted at 29 centres worldwide, 827 of the 4,526 COVID-19 hospitalised patients developed arrhythmia (Guo et al. 2020). AF was the most common rhythm disturbance, present in 80% of these patients; 20.7% developed ventricular arrhythmias, and 22.6% had bradyarrhythmia (Guo et al. 2020). Arrhythmias were proven to be associated with high morbidity and mortality among these patients: 43% of patients who developed arrhythmia were mechanically ventilated, and 51% survived hospital discharge (Guo et al. 2020; Coromilas et al. 2021). In another study by Wang et al. (2020a), COVID-19 patients were observed, with 23 (16.7%) developing arrhythmias and 16 requiring transfer to an intensive care unit (ICU) due to this complication. Cardiac arrhythmias were more common in patients requiring admission to an ICU (44.4% vs. 6.9%). COVID-19 has been reported to cause myocardial damage according to the mechanisms discussed above. In most cases, myocardial damage is caused by an increased myocardial oxygen demand associated with systemic infection, ongoing hypoxia caused by severe pneumonia or acute respiratory distress syndrome (ARDS), as well as electrolyte disorders. These factors can potentiate cardiac arrhythmias. The authors believe it is very likely that the virus can lead to atrial or ventricular arrhythmias after cases of acute myocarditis with cardiogenic shock (Lakkireddy et al. 2020; Wang et al. 2020a).

Bhatla et al. (2020) analysed the clinical data of 700 patients admitted to the hospital with COVID-19 from March 6, 2020, to May 19, 2020. Out of the total, 9 individuals experienced cardiac arrest, with 6 cases of pulseless arrhythmia,

one case of torsades de pointes, and 2 patients with asystole. Out of the total, 25 individuals had atrial fibrillation and needed treatment with diltiazem and amiodarone. There were 10 cases of non-sustained ventricular tachycardia and 9 clinically significant bradyarrhythmias (Driggin et al. 2020). The authors have proposed that the incidence of arrhythmic events in COVID-19 individuals had not been caused by the viral infection but rather by a combination of factors, with the severity of the disease course being significant.

A study organised by the Heart Rhythm Society (HRS) showed that atrial fibrillation (AF) was the most common arrhythmia in hospitalised patients with COVID-19, presented in 142 (21%), atrial flutter in 37 (5.4%), prolonged atrial tachycardia in 24 (3.5%), and paroxysmal supraventricular tachycardia in 39 (5.7%) of the 683 patients studied. The study also reported ventricular arrhythmias, in which monomorphic arrhythmias were the most common form, reported in 36 people. Other ventricular arrhythmias have been reported, including polymorphic, non-sustained ventricular tachycardia, ventricular fibrillation (VF), cardiac arrest, and electrical activity without a pulse (electromechanical dissociation (EMD)). Regarding conduction disorders in COVID-19 patients, of the 663 most frequently studied, sinus bradycardia and complete AV block were reported for 51 (8%) and 51 (8%), respectively. In these patients, AV block, first or second-degree, bundle branch block, or delay in intraventricular conduction have also been reported (Bhatla et al. 2020).

A study by Wang et al. (2020b) showed that the antiviral drug Remdesivir effectively inhibited SARS-CoV-2 in vitro. Wang et al. (2020b) demonstrated that patients receiving Remdesivir had faster clinical improvement than those receiving a placebo, but the difference was not statistically significant. In addition, studies have shown a lack of benefit in treating COVID-19 patients with chloroquine, hydroxychloroquine, or azithromycin; therefore, they are not recommended as standard therapy for COVID-19 hospitalised patients, as they may increase the risk of arrhythmias. The combination of hydroxychloroquine and azithromycin significantly prolongs the QTc interval in patients with COVID-19. This extension may be responsible for a life-threatening arrhythmia. This risk requires careful consideration of the therapy. Close monitoring of the QTc interval should be performed if a regimen including these medications is administered (Wang et al. 2020c).

Patients with COVID-19 are prone to developing arrhythmias, as per the study by researchers (Parwani et al. 2021). A total of 113 consecutive patients (mean age 64.1 ± 14.3 years, 30 (26.5%) women) with a positive PCR test for SARS-CoV2, as well as radiographically confirmed pulmonary involvement, were admitted to intensive care from March to May 2020. They were observed for 2321 days, during which fifty episodes of prolonged atrial tachycardia, five episodes of prolonged ventricular arrhythmias, and thirty bradycardia events were documented. Persistent new-onset atrial arrhythmias were associated with hemodynamic deterioration in 13 cases (35.1%). Older patients with newly occurring atrial arrhythmias showed higher levels of TnT and NT-proBNP and had a more severe course of the disease. Five ventricular arrhythmias (two ventricular tachycardias, two episodes of ventricular fibrillation, and one tachycardia-type torsade de pointes) were observed in four patients. All episodes could be terminated by immediate defibrillation or cardioversion. Five events of bradycardia were associated with deterioration of hemodynamics. Accelerating factors could be identified in 19 of 30 episodes (63.3%), and no patients required electrocardiostimulation (Carpenter et al. 2020). Baseline characteristics were sim-

ilar across patients who experienced bradycardia incidents and those who did not. Arrhythmias frequently occur in COVID-19 patients and are linked to a more severe disease progression, requiring specialised treatment (Fig. 2).

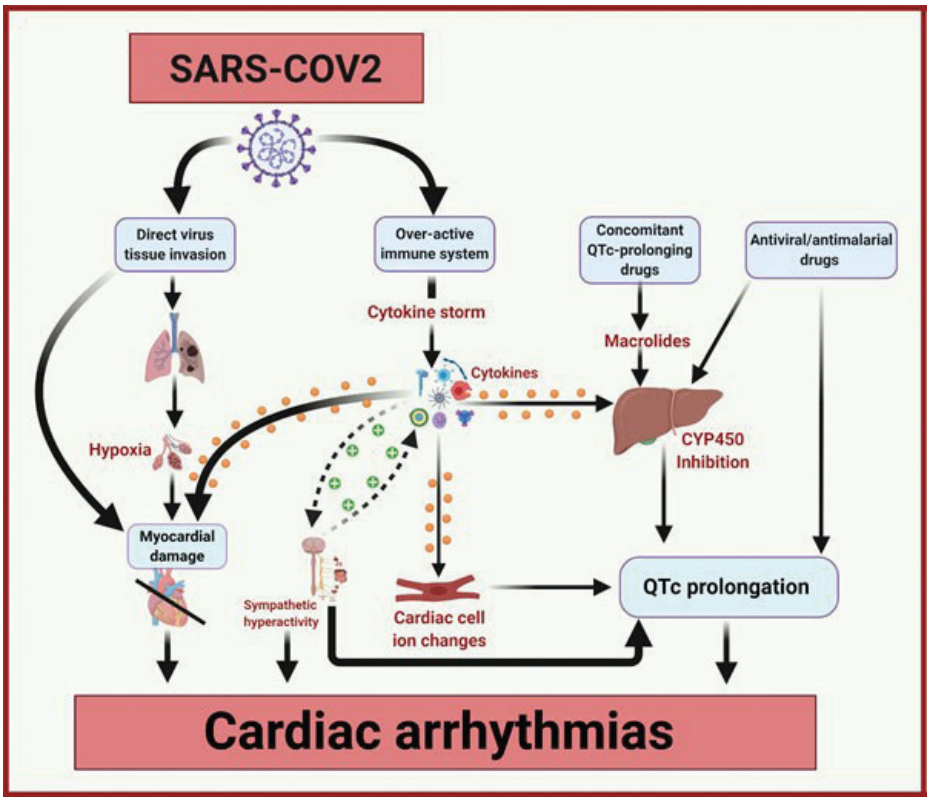

Figure 2. The mechanisms of cardiac arrhythmias in COVID-19. Adapted from Taqi et al. (2020) COVID-19 coronavirus disease-2019, CYP450 cytochrome P450 enzymes, QTc heart rate, corrected QT interval, SARS-COV-2 severe acute respiratory syndrome-coronavirus-2.

Role of direct myocardial damage

The role of direct myocardial damage in COVID-19 patients has been extensively studied, revealing an incidence rate of approximately 2,096 cases among the affected population. This damage is characterised by an increase in troponin levels above the 99th percentile and is strongly correlated with a higher mortality rate (Parwani et al. 2021). Two primary mechanisms contribute to this direct myocardial damage: direct viral invasion and immune-mediated damage (Shi et al. 2020). The first mechanism involves the binding of the virus's H-protein to the ACE2 receptor, allowing the virus to enter cardiomyocytes. As a result, the number of ACE2 receptors decreases, potentially leading to the predominance of angiotensin P action on type 1 angiotensin receptors and subsequent myocardial remodelling. Simultaneously, the presence and replication of the virus lead to cellular damage (Siripanthong et al. 2020). The second mechanism involves the infiltration of activated T lymphocytes into the myocardium, triggering an inflammatory response and subsequent damage to the myocardium.

Additionally, cytokine storm conditions further stimulate T lymphocyte activation and contribute to additional myocardial damage (Shi et al. 2020). These processes have a significant potential to induce arrhythmias due to the direct cytopathic effect, necrosis, changes in intercellular contacts, and alterations in

the expression of ion channels in cardiomyocytes. Consequently, structural and electrical remodelling of the myocardium occurs primarily through disruptions in calcium homeostasis and potassium channel activity. These changes create conditions for trigger activity expression, ultimately leading to the manifestation of arrhythmias with a re-entry mechanism. In some cases, the potential for arrhythmias persists even after the inflammatory process has subsided, particularly if the myocardium has formed fibrotic zones (Vaduganathan et al. 2020).

Supraventricular tachycardia (SVT)

Supraventricular tachycardia, a condition that causes a rapid heart rate, needs to be addressed differently in the context of COVID-19. It is important to identify and treat any underlying conditions that may be causing the heart rhythm disturbance. These underlying conditions can include metabolic and electrolyte disorders, myocardial ischemia, and respiratory deficiency, resulting in low oxygen levels. If a supraventricular tachycardia episode occurs, it is suggested that adenosine be used to stop it. However, it is important to note that the effectiveness and safety of adenosine in COVID-19 patients have not been confirmed. If a patient is unstable and adenosine is ineffective, synchronised electrocardioversion may be considered. Early initiation of maintenance therapy is crucial, and beta-blockers or non-dihydropyridine calcium antagonists are recommended. However, it is important to avoid bradycardia, as it can lead to QT interval prolongation (Peretto et al. 2019).

Atrial Fibrillation (AF) and Flutter

For all individuals diagnosed with AF and atrial flutter, the primary objectives of the treatment are to attain control over ventricular frequency, prevent thromboembolic complications, and, when necessary, manage rhythm. Despite the challenges posed by COVID-19, these core goals remain pertinent, albeit with certain distinctions dictated by the specifics of the infectious disease. In cases of newly occurring or recurring AF where patients exhibit stable hemodynamics, it is advisable to contemplate discontinuing antiarrhythmic therapy and initiating a frequency control approach. This recommendation holds for most antiarrhythmic medications utilised in our region, including potential amiodarone, owing to the significant risk of drug interactions with antivirals.

Similarly, patients experiencing atrial flutter can undergo treatment, but it is crucial to acknowledge that achieving frequency control in such cases may present increased complexities. Individuals with hemodynamic instability, those with heart failure, or severely ill patients are candidates for a rhythm control strategy, typically involving electrocardioversion. Rhythm control can also be achieved through the use of antiarrhythmic medications, with amiodarone being preferred. It is important to note that patients receiving amiodarone alongside antiviral drugs such as (hydroxy)chloroquine, azithromycin, or fingolimod should be subjected to close monitoring. In fact, according to specific guidelines applicable in the treatment of fingolimod patients in this scenario, rhythm control may be accomplished using propafenone or flecainide for a limited period (Traykov and Gelev 2021). In individuals diagnosed with COVID-19, the evaluation of embolic risk using the CHADS2 score is feasible. The decision to initiate anticoagulant therapy is based on the cumulative score. The choice between a rhythm control or frequency control strat-

egy should be reassessed post-recovery from COVID-19, and the continuation of anticoagulant therapy is contingent upon the CHADS2 Score sum.

Ventricular tachyarrhythmias alongside COVID-19

It is crucial to carefully identify and address factors that make patients more likely to suffer ventricular tachyarrhythmias in the overall therapy of COVID-19 patients. These may encompass factors such as hypoxia, metabolic disorders, dyselectrolytemias, or the potential proarrhythmic effects of medications. Patients experiencing cardiac arrest attributable to ventricular tachycardia (VT) or ventricular fibrillation (VF) should be managed following prevailing guidelines (Rattanawong et al. 2019). It is noteworthy that ventricular tachyarrhythmias can ensue during cardiopulmonary resuscitation (CPR) (Monsieurs et al. 2015). Accordingly, expeditious attention to febrile conditions in these patients becomes paramount. In instances where individuals with COVID-19 and Brugada's syndrome exhibit high-risk characteristics and persistent fever despite antipyretic administration, hospitalisation and vigilant monitoring are strongly advocated, particularly when the standard electrocardiogram (ECG) reveals a Type 1 Brugada pattern. Patients with a history of cardiogenic syncope or those manifesting spontaneous or induced fever with a Brugada type 1 ECG are also classified as high-risk. Additionally, individuals with a confirmed sodium channel mutation fall within this high-risk cohort. Timely and judicious interventions, coupled with continuous monitoring, assume pivotal roles in the effective management of such cases.

Conclusion

Upon reviewing the literature, an examination of the available information provides grounds to consider rhythm and conduction disturbances as outcomes of multifactorial influences stemming from COVID-19. Pinpointing the specific contribution of individual pathophysiological mechanisms to the genesis of these rhythm disturbances proves challenging. A cautious approach is necessary for statistical analysis aimed at summarising data from patients exhibiting a highly diverse clinical spectrum, ranging from severe respiratory syndrome and/or significant multiorgan damage to seemingly mild forms with symptoms such as rhinitis and mild discomfort. Simultaneously, the presence of various comorbidities further complicates the analysis.

It is imperative to discern whether a correlation exists among the severity of the clinical presentation, the extent of oxygen saturation reduction, pre-existing comorbidities, and the type and severity of rhythm and conduction disorders. The study of the factual data will remain a prolonged endeavour, posing a significant challenge in comprehending the electrogenesis of rhythm and conduction disorders.

Acknowledgements

Project No. 9/2022, Study of heart damage and rhythm-conduction disorders in patients who have had a history of COVID-19 infection and the relationship with the severity of the course of the disease, was implemented with financial resources from the Medical University of Pleven. The authors have declared that no competing interests exist.

Ethical statement

The authors declared that no clinical trials were used in the present study.
The authors declared that no experiments on humans or human tissues were performed for the present study.
The authors declared that no informed consent was obtained from the humans, donors or donors' representatives participating in the study.
The authors declared that no experiments on animals were performed for the present study.
The authors declared that no commercially available immortalised human and animal cell lines were used in the present study.

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