Electrocardiographic manifestations of COVID- 19

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Abstract:

Coronavirus disease of 2019 (COVID-19) is a lower respiratory tract infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This disease can impact the cardiovascular system and lead to abnormal electrocardiographic (ECG) findings. Emergency clinicians must be aware of the ECG manifestations of COVID-19.

Introduction:

In December 2019, an outbreak of a lower respiratory tract disease caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was first reported in Wuhan, China **(1).** This was sub- sequently termed coronavirus disease of 2019 (COVID-19) by the World Health Organization and has been declared a global pandemic, infecting millions worldwide **(2).**

While much of the focus has been on the respiratory system, COVID-19 can also cause a variety of cardiac complications and a range of electrocardiographic abnormalities. Studies have described COVID-19 related myocarditis, cardiac tamponade, Brugada-like pattern, transient ST segment elevations, as well as medication-induced cardiac dysrhythmias **(3).**

Myocardial involvement in COVID-19 has been associated with poor outcomes, especially in myocardial injury which occurs in 22–44% of critical or severe cases. Therefore, it is crucial that the emergency clinician recognize the electrocardiographic (ECG) manifestations of COVID-19 **(4).**

❖ **Supraventricular tachycardias (SVT)**

Sinus tachycardia is the most common supraventricular tachycardia encountered in the ill COVID-19 patient, resulting from the usual causes, including hypovolemia, hypoperfusion, hypoxia, elevated body temperature, pain, and anxiety. After sinus

tachycardia, atrial fibrillation (Fig. 1) is the next most commonly seen SVT **(5).**

Atrial fibrillation can present in a number of fashions, including new-onset, recurrence of a pre-existing dysrhythmia, and persistence of permanent atrial fibrillation with new rapid ventricular response. In the majority of patients with significant COVID-19 infection and atrial fibrillation, the rate will be quite rapid, thus the ECG will demonstrate atrial fibrillation with rapid ventricular response. Both sinus tachycardia and atrial fibrillation are independent predictors of illness severity, myocardial injury, and poor outcomes in COVID-19 **(5).**

One study conducted in New York hospitals found atrial fibrillation/flutter was present in 14.3% of patients at admission and occurred in 10.1% of patients during hospitalization **(6).**

Another study found atrial fibrillation/flutter to be present in 22% of critically ill patients requiring mechanical ventilation **(7).**

Atrial fibrillation is more common in patients following an inflammatory insult such as cardiomyopathy from COVID-19 and occurs in up to half of patients admitted to the ICU **(8).**

Two case reports demonstrated the highly variable impact that COVID-19 has on the heart in one case the patient experienced both atrial flutter and atrial fibrillation, eventually reverting to sinus rhythm 48 h later **(9).**
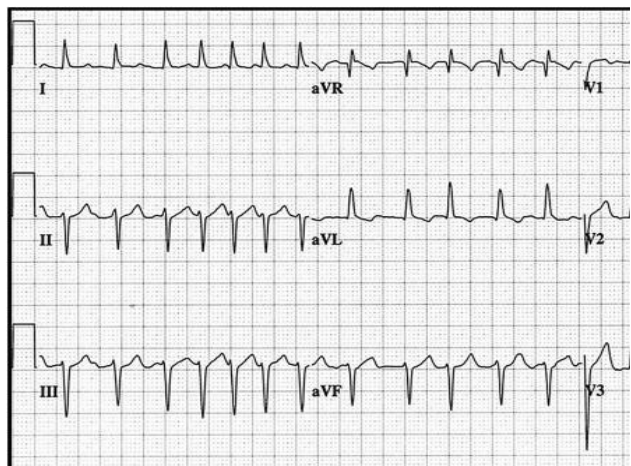
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Figure (1): Atrial fibrillation with rapid ventricular response in a COVID-19-infected 76 year-old female with new-onset atrial fibrillation **(9).**

There is a paucity of literature evaluating the presence of other types of SVT, such as atrioventricular nodal reentry tachycardia (AVNRT), in COVID-19 patients. In most instances of AVNRT from the pre-COVID-19 era, patients with AVNRT are frequently younger and lack significant acute and chronic cardiorespiratory ailments; this typical AVNRT presentation likely explains the relative paucity of literature on this dysrhythmia in the COVID-19 patient. Fig. 2: shows the ECG of a 19-year-old patient with palpitations and dyspnea who presented with AVNRT. The patient was appropriately treated with adenosine but experienced persistent dyspnea after uncomplicated conversion to sinus tachycardia. Further evaluation revealed that she was positive for COVID-19, and she was admitted to the hospital with persistently low oxygen saturations. She ultimately did well and was discharged from the hospital 5 days later, without AVNRT recurrence **(9).**
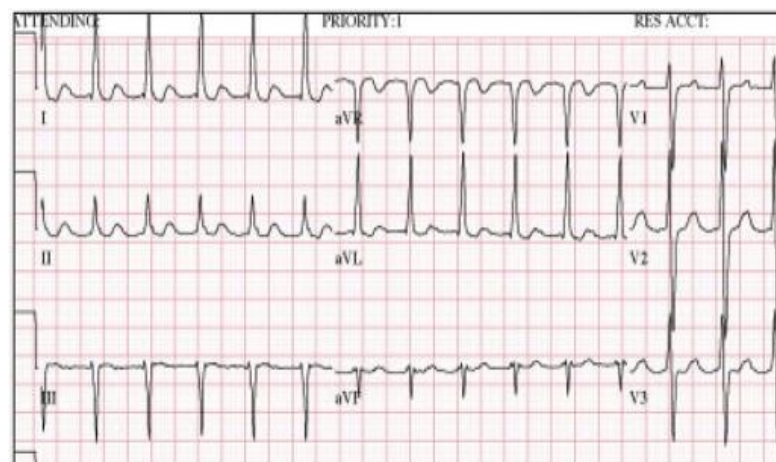

Figure (2): Atrioventricular nodal re-entrant tachycardia in a 19 year-old patient with palpitations and dyspnea who presented with AVNRT. The patient was appropriately treated with adenosine but experienced persistent dyspnea after uncomplicated conversion to sinus tachycardia; further evaluation revealed that she was positive for COVID-19 **(9).**

❖ **Malignant ventricular dysrhythmias: ventricular tachycardia and ventricular fibrillation.**

Malignant ventricular arrhythmias are a known complication of viral myocarditis and cardiomyopathy, with ventricular tachycardia (VT) and/or ventricular fibrillation (VF) occurring in 1–6% of patients **(10).**

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In patients with COVID-19, these arrhythmias may be due to a combination of QT interval prolonging medications, metabolic abnormalities, and myocardial inflammation **(10).**

COVID-19 patients with elevated troponin have been shown to have a higher incidence of VT than those with normal troponins **(8).**

VT with a pulse is seen with both monomorphic and polymorphic presentations. Monomorphic ventricular tachycardia (MVT) is the most frequent form of VT seen in the COVID-19 patient, frequently resulting from structural heart disease such as acute coronary syndrome with STEMI, myocardial injury, or myocarditis; patients with pre-existing structural heart disease of many types can provide the substrate for MVT during periods of extreme physiologic stress due to the range of issues encountered in the COVID-19-infected patient. Polymorphic VT (PVT), including the PVT subtype torsade de pointes, results from functional (i.e., non-structural) heart disease and is likely less common; it most often occurs in situations involving medication toxicities, electrolyte abnormality, and various pro-arrhythmic states (e.g. Brugada syndrome, long QT syndrome). Cardiac arrest dysrhythmias include the traditional 4 rhythm scenarios: the "shockable" (pulseless VT and VF) and "non-shockable" (pulseless electrical activity [PEA] and asystole) dysrhythmias. Specific dysrhythmia diagnosis in the cardiac arrest patient, whether out-of-hospital or hospital-based, is unchanged in the COVID-19 pandemic. During the pandemic, the occurrence of both out-of-hospital cardiac arrest (OHCA) and hospital-based cardiac arrest have increased based on recent data **(11).**

A study conducted in Italy found close to a 60% increase in out-of-hospital cardiac arrest OHCA in 2020 compared to 2019 ,a second study in France found a 52% increase in OHCA between February and April 2020, compared to 2019 **(11).**

This unfortunate increased rate of OHCA has also been seen in the United States. For example, in New York City there was a three-fold higher rate of patients undergoing resuscitation in the out-of-hospital setting when compared to a similar 2019 pre-pandemic period; the odds ratio (OR) of encountering "non-shockable" dysrhythmias increased significantly with an OR for PEA of 3.50 and an OR for asystole of 1.99, the mortality rate of patients in cardiac arrest increased in all three studies **(12).**

There are several factors that may contribute to this increased occurrence with more frequent poor outcome, including the COVID-19 infection itself as well as the delays in seeking care related to the pandemic, and lower rates of important bystander care in the pre-arrival period of cardiac arrest management **(13)**.

Patients hospitalized with COVID-19 are at increased risk of cardiac arrest, but the rhythms associated with cardiac arrest vary in

the inpatient population. A study focusing on hospitalized patients in New York City noted that asystole occurred most often, followed by PEA and then pulseless VT/VF **(6).**

Another study found 9 patients out of 700 experienced cardiac arrest, with only one having a shockable rhythm **(14).**

A study of 136 Chinese inpatients with COVID-19 complicated by cardiac arrest during hospitalization found most arrests were respiratory in origin with non-shockable rhythms (90% asystole and 4% PEA); only 3% survived to 30 days, with just 1% having intact neurologic function **(15).**

❖ **Bradycardia and atrioventricular block**

Bradycardias and AVB are less commonly encountered as compared to tachydysrhythmias, though they may account for up to 11.8% of cardiac dysrhythmias **(16).**

A case report details a patient with COVID-19 presenting with first-degree atrioventricular block (AVB); during hospitalization, the rhythm transitioned to Mobitz type 1 second-degree AVB with further evolution ultimately to third-degree AVB **(16).**

Other cases describe older patients with multiple cardiac risk factors experiencing progression to high-grade AVB (second-degree type II and third-degree AVB) and/or intra-ventricular conduction block; many of

these patients developing these more concerning conduction abnormalities progressed rapidly to cardiac arrest **(16).**

Three of these dysrhythmias (sinus bradycardia, junctional rhythm, idioventricular rhythm) occurred immediately prior to cardiac arrest onset – thus, in these cases, the development of significant bradycardia in the critically ill COVID-19 patient is a marker of risk of impending cardiovascular collapse **(17).**

❖ **Interval and axis abnormalities**

QT interval prolongation may occur in over 13% of patients with COVID-19 infection, and several medications previously used for COVID-19 may prolong the QT interval, including chloroquine, hydroxychloroquine, and azithromycin **(18).**

QT interval prolongation is associated with more critical illness requiring ICU admission, as well as cardiac injury and mortality **(19).**

Left and right bundle branch blocks may occur in up to 12% of patients at the time of admission or during hospitalization. Significant QT interval prolongation may lead to torsade des pointes. Fig. 3 is the ECG of a patient with COVID-19-related myocarditis; the ECG reveals atrial fibrillation with a mean rate of approximately 100 bpm, significant QRS complex widening, right axis deviation, poor R wave

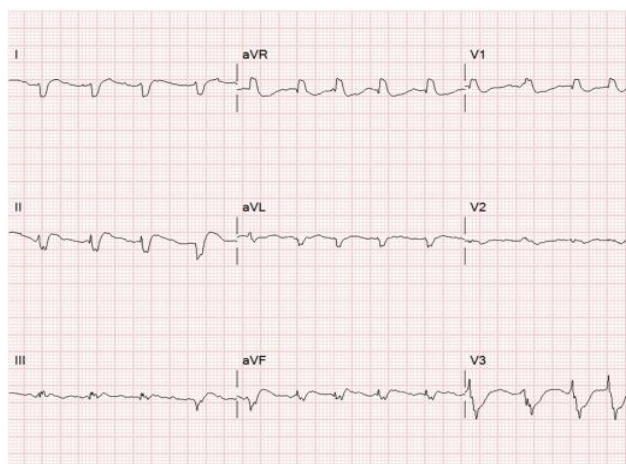progression, and ST segment elevation in the anterolateral leads **(6).**

Figure (3): COVID-19-related myocarditis with atrial fibrillation, QRS complex widening, right axis deviation, poor R wave progression, and ST segment elevation **(6).**

QRS complex axis deviation has been reported, usually involving situations presenting with RV strain; such patients are frequently experiencing acute respiratory failure from multi-lobar pneumonia and/or pulmonary embolus with large clot burden. Electrocardiographically, these patients present with right axis deviation along with prominent R waves in leads V1 and V2 and ST segment depression/T wave inversion in leads II, III, aVF, and V1 to V4. Fig. 4 shows the inferior and right to mid precordial leads with T wave inversion, indicative of RV strain, in a patient with significant pulmonary embolus **(4).**
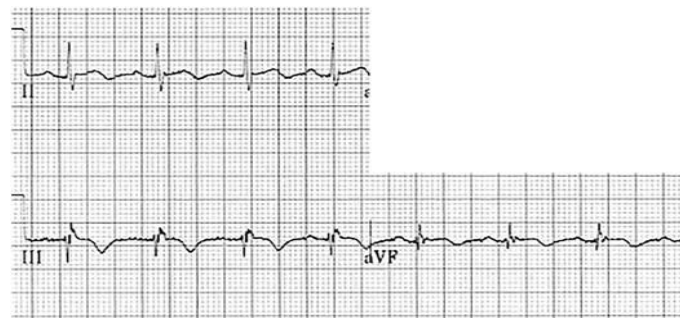

Figure (4): Right ventricular strain with T wave inversions in the inferior and right to mid precordial leads **(4).**

❖ **Morphologic presentations – ST segment, T wave, and QRS complex abnormalities.**

The interpretation of the ECG in the patient with chest pain or a similar presentation is more challenging in the COVID-19 pandemic. Emergency clinicians must consider STEMI, myocardial injury, and myocarditis, as all three clinical entities present with ST segment and T wave abnormalities. COVID-19 infection associated with myocardial injury may demonstrate ST segment deviations (elevation or depression), T wave inversion, and pathologic Q waves **(19).**

One study found ST segment and T wave changes to be the most common abnormality in patients requiring ICU admission, occurring in 40% of patients **(12).**

Another investigation noted that nonspecific <u>repolarization</u> changes including ST segment and T wave abnormalities were encountered in 41% of patients; reportedly, these findings resulted from myocardial injury and are associated with poor outcomes, including increased need for ICU admission, more frequent mechanical ventilatory support, and increased mortality **(5).**

A case series included 18 patients with COVID-19 infection and ST segment elevation, 10 of which had ST segment elevation at the time of presentation and 8 developed ST segment elevation during hospitalization **(20).**

Ultimately, <u>acute myocardial infarction</u> (AMI) was diagnosed in 8 patients, while 10 demonstrated nonocclusive (i.e., not involving ACS) myocardial injury **(20).**

Myocarditis and/or myopericarditis associated with COVID-19 infection can demonstrate ECG findings resembling occlusive AMI, such as focal ST segment elevation with reciprocal change **(21).**

In the patient with ST segment elevation, the distinction between STEMI, myocardial injury, and myocarditis may not be possible strictly based upon ECG interpretation, which can make this particularly challenging. In the aforementioned case series of ST segment elevation in COVID-19-infected patients **(20).**

STEMI was suggested with focal ST segment elevations, while myocardial injury demonstrated diffuse or widespread ST segment elevation. The clinical presentation can provide diagnostic clues and aid in the distinction of STEMI versus myocardial injury/myocarditis. Chest pain is a frequent presentation in the STEMI patient, while it is less common with myocardial injury/myocarditis which more often involves dyspnea and other symptoms consistent with acute viral infection **(1).**

Echocardiographic findings such as focal wall motion abnormalities with depressed <u>left ventricular function</u> suggest STEMI while myocardial injury more often demonstrates normal left ventricular function or diffuse dysfunction **(22).**
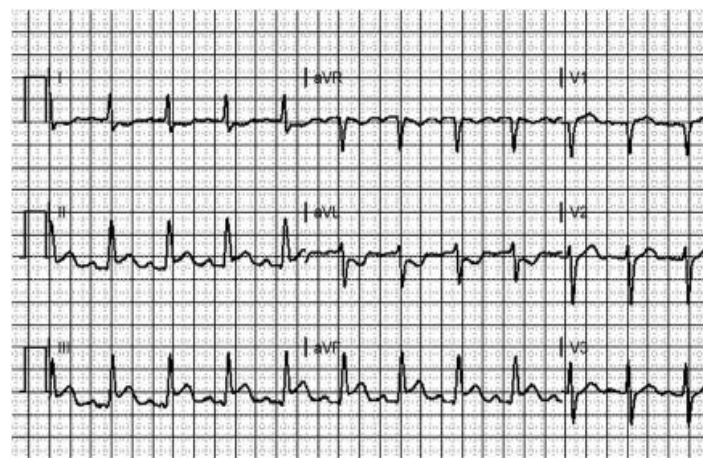

Figure (5): Inferior STEMI with focal ST segment elevation in leads II, III, and aVF and reciprocal <u>ST segment depression</u> in lead aVL in a COVID-19-infected patient **(22)**

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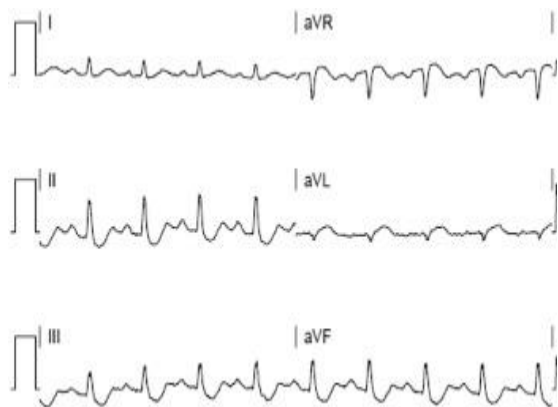
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Figure (6): Anterior STEMI with focal ST segment elevation in leads V1 to V4 and reciprocal ST segment depression in the inferior leads in a COVID-19-infected patient **(22)**
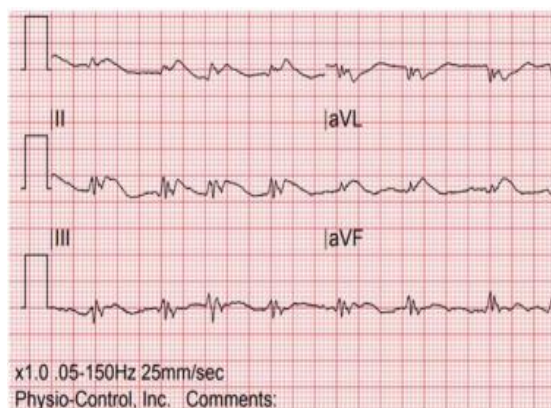

Figure (7): Myocardial injury in a COVID-19-infected patient with diffuse ST segment elevation in the inferior, lateral, and anterior regions. Bedside echocardiography revealed no focal wall motion abnormalities with only mild diffuse hypokinesis **(22),**
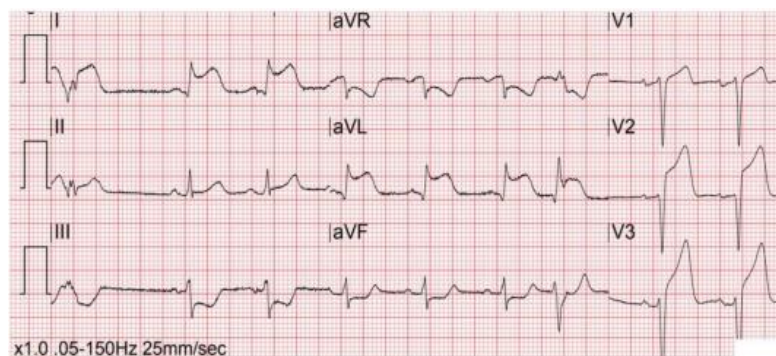

Figure (8): Myocardial injury in a COVID-19-infected patient with diffuse ST segment elevation in the anterior and lateral regions. This patient was initially taken to the cardiac catheterization laboratory with no coronary occlusive lesions noted. The serum troponin values were extremely elevated but in a plateau pattern, rather than the typical rise and fall patient of acute myocardial infarction **(22).**

COVID-19 may also result in an unmasked Brugada pattern on ECG in patients with existing pathology. A pseudo-Brugada type 1 pattern is associated with fever and changes in myocardial sodium channels. Type 1 is demonstrated by convex ST segment elevation > 2 mm in more than 1 lead of V1-V3, followed by a negative T wave **(23).**

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