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A STUDY OF CARDIAC COMPLICATIONS BASED ON ECG CHANGES IN COVID 19 POSITIVE PATIENTS

Cardiology

T V SUSHMA* Assistant professor AIMSR&C *Corresponding Author
S N Ravishankar Professor & HOD AIMSR&C

ABSTRACT

Aims & Objectives
- To study the cardiac complications based on ECG changes in COVID 19 positive patients.

Materials And Methods
- It is a Prospective case study with sample size of 500 done in East point Medical College and Research hospital, Bangalore.

Results: 300 patients had normal sinus rhythm.130 patients had tachycardia, 70 patients had bradycardia. 30 patients had arrhythmia, 27 patients had evidence of cardiac injury.154 patients had evidence of myocarditis. 40 patients had evidence of heart failure and 15 patients had cardiomyopathy. 50 patients had an evidence of venous thromboembolic events. **Conclusion:** Cardiac involvement is very common in SARS –Cov-2 infection. Myocarditis is more common, though it may not be fatal in most cases, it can be problematic in case of associated co-morbid conditions

KEYWORDS

INTRODUCTION

Corona virus disease (COVID-19) is an infectious disease caused by a novel corona virus, SARS-CoV-2.Most people infected with the COVID-19 virus will experience mild to moderate respiratory illness and recover without requiring special treatment. Older people and those with underlying medical problems like cardiovascular disease, diabetes, chronic respiratory disease, and cancer are more likely to develop serious illness.

MATERIALS AND METHODS

Source Of Data

Patients admitted in Hospital East Point Medical College and Research hospital, Bangalore

Methods Of Collection Of Data

1. **Study Design:** Prospective study
2. **Study Period:** July 2020 to November 2020
3. **Place Of Study:** East point Medical College and Research hospital, Bangalore
4. **Sample Size: 500**

Inclusion Criteria

a. Patient/ attender willing to give informed consent
b. Patient of either Sex with age more than 18 years
c. Covid 19 RT-PCR Positive Patients admitted in EPMC

Exclusion Criteria

a. Patient not willing to give informed consent
b. Age less than 18 years
c. Patients with HIV infection, HbsAg, HCV positive status, EBV, CMV,Herpes or Enterovirus infections
d. Pregnant patients
e. Patients with malignancy on chemotherapy

RESULTS

1. Sinus Rhythm

Sinus rhythm defined as heart rate 60-100 bpm.
300 patients had normal sinus rhythm.130 patients had tachycardia, 70 patients had bradycardia.

2. Arrhythmia

- Arrhythmia defined as heart Rate of 350 to 650 beats per minute ; slow-to-rapid ; can be too low or too high depending upon ventricular response to chaotic atrial arrhythmia, with Irregular rhythm, absent P wave and QRS complex <0.12 seconds.

30 patients had arrhythmia who fulfil above criteria.

3. Acute Cardiac Injury

Acute cardiac injury defined as ST segment depression, depression and inversion of the T wave, and Q waves, along with elevated cardiac biomarker.
15 patients had ST elevation, among them 5 patient had elevated cardiac biomarker.12 patient had T inversion among them 5 patient had dyselectrolytemia, and 2 patient had elevated cardiac biomarker.

4. Myocarditis

Prior viral illnesses, including Middle East respiratory syndrome corona virus (MERS-CoV), have been associated with myocardial injury and myocarditis with troponin elevation, thought to be due to increased cardiac physiologic stress, hypoxia, or direct myocardial injury.ECG may show following findings.[1]

- **Sinus Tachycardia:** Indicators include a heart rate over 100 beats per minute, regular rhythm, identical P waves before each QRS, PR intervals between 0.12 and 0.20 seconds, and QRS under 0.12 seconds.
- **Conduction Defects:** Presentations vary depending on the specific defect. Branch blocks, for example, have identical P waves before each QRS, PR intervals between 0.12 and 0.20 seconds, QRS of 0.12 seconds or less, and unique characteristics, such as ST elevation or RSR' in V1.

Among our 500 patients, 130 patients had tachycardia, among them 100 had sinus tachycardia and 30 of them had arrhythmia.24 patients had conduction defects.

5. Cardiac Arrest

The predictive value of ECG for sudden cardiac arrest in ECG include pathologic Q waves (as with prior scar tissue or myocardial infarction) and RSR' patterns.

8 patient had cardiac arrest among them 5 were revived and 3 couldn't be revived and declared dead.

6. Myocardial Infarction (MI)

Severe systemic inflammation increases the risk of atherosclerotic plaque disruption and AMI [2]

The ACC essential patterns indicative of MI, include—anterior/ inferior/posterior STEMI and STEMI with right- and left-bundle branch blocks.

6 patients had STEMI, 2 anterior wall MI, 3 inferior wall MI AND 1 STEMI with LBBB.

7. Acute Heart Failure And Cardiomyopathy

Acute heart failure can be the primary presenting manifestation of COVID-19 infection. 40 patients had evidence of heart failure and 15 patients had cardiomyopathy.

8. Venous Thromboembolic Event

Patients with COVID-19 are also at an increased risk of VTEs [3,4].

Systemic inflammation, abnormal coagulation status, multiorgan dysfunction, and critical illness are all potential contributing factors to the increased risk of VTE [3,4]. Studies suggest significant coagulation pathway abnormalities in patients with COVID-19, including elevated D-dimer [3,4]. 50 patients had evidence of venous thromboembolic events.

9. Medication Interactions

Many of the newly studied medications interact extensively with other cardiovascular drugs, including antihypertensives, antiarrhythmics, anticoagulants, antiplatelets, and statins [1]. Current medications under study include antivirals (e.g., remdesivir, ribavirin, lopinavir/ritonavir, favipiravir), antimalarials (e.g., chloroquine, hydroxychloroquine), azithromycin, corticosteroids, and biologics (tocilizumab) [1] .Lopinavir/ritonavir may cause QT and PR prolongation, particularly in those with baseline QT prolongation or in those taking medications that may cause QT prolongation [5]. These medications can also affect anticoagulant medications, antiplatelet agents, and statins [5]. Chloroquine and hydroxychloroquine affect the intracellular pH, which can result in electrolyte abnormalities, cardiotoxicity, and prolonged QT intervals; they may also interact with antiarrhythmic agents [5,6]. Methylprednisolone can cause electrolyte derangements, fluid retention, and hypertension. A summary of the mechanism of action and effect of these medications is located in Table 1 .

Table 1 Medications and the cardiovascular system [1].

Medication	Mechanism	Cardiovascular effects and medication interactions
Remdesivir	Nucleotide-analog inhibitor of RNA polymerases	- May cause hypotension, arrhythmias
Ribavirin	Inhibits RNA and DNA virus replication	- Interacts with anticoagulants - May cause severe hemolytic anemia
Lopinavir/Ritonavir	Lopinavir inhibits protease Ritonavir inhibits CYP3A metabolism	- Interacts with anticoagulants, antiplatelets, statins, antiarrhythmics - May result in prolonged QTc, AV blocks, Torsades de pointes
Favipiravir	Inhibits RNA-dependent RNA polymerases	- Interacts with anticoagulants, statins, antiarrhythmics - May cause severe hemolytic anemia
Chloroquine and Hydroxychloroquine	Changes endosomal/organelle pH	- Interacts with antiarrhythmics - May cause direct myocardial toxicity; worsen cardiomyopathy; alter cardiac conduction; result in bundle branch block, AV block, ventricular arrhythmias, Torsades de pointes
Azithromycin	Interferes with protein synthesis, binds to 50s ribosome	- Interacts with anticoagulants, statins, antiarrhythmics, other QT prolonging agents - May result in dysrhythmias, prolonged QTc, Torsades de pointes
Interferon	Immune system activation	- May cause direct myocardial toxicity; worsen cardiomyopathy; alter cardiac conduction; cause hypotension or cardiac ischemia
Methylprednisolone	Reduces inflammation	- Interacts with anticoagulants - May cause fluid retention, hypertension, electrolyte changes
Tocilizumab	Inhibits IL-6	- May increase medication metabolism such as statins - May cause hypertension

DISCUSSION

The COVID-19 infection can have multisystem involvement. Cardiac involvement has been reported in patients with COVID-19, which may be reflected in electrocardiographic (ECG) changes.

Several possible mechanisms were proposed for cardiac involvement. First, angiotensin-converting enzyme 2 (ACE2) has been identified as a functional receptor for coronaviruses,[7] which is highly expressed in the heart and lungs. Therefore, ACE2-related signaling pathways might have played a role in cardiac injury. Second, hypoxemia caused by COVID-19 may cause damage to myocardial cells. Third, systemic inflammatory response and immune system disorders may be important factors[8]. The infection has been associated with multiple direct and indirect cardiovascular complications including acute myocardial injury, myocarditis, arrhythmias and venous thromboembolism.

The underlying mechanisms of these ECG abnormalities in the severe stage of COVID-19 may be attributed to hypoxia and inflammatory damage incurred by the virus. The overall case fatality rate for COVID-19 patients has stayed relatively stable at 2.3%, while individuals with cardiovascular disease (CVD) face more than quadruple the risk at 10.5%. CVD patients also face a higher risk of getting infected with the novel corona virus. Disease-triggered cardiometabolic demand and associated complications can worsen prognoses for patients with underlying heart conditions, especially those with cardiovascular disease (CVD).

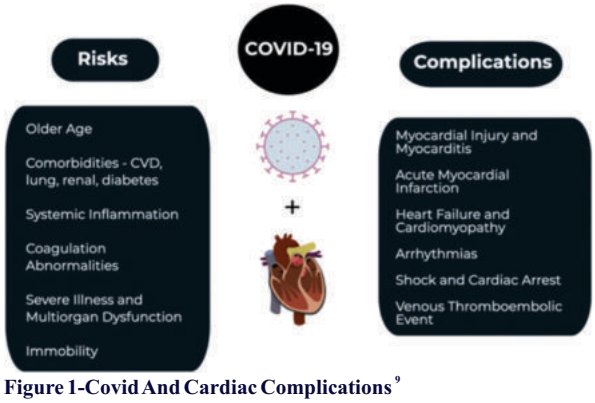

Figure 1-Covid And Cardiac Complications [9]

A meta-analysis of 1527 patients with COVID-19 found that the prevalence of hypertension was 17.1% and cardiac disease was 16.4%, and that these patients were more likely to require critical care [10]. Another study of 44,672 patients with COVID-19 found that a history of CVD was associated with a nearly five-fold increase in the case fatality rate when compared with patients without CVD (10.5% vs. 2.3%)[11] .

One study found that acute heart failure may be present in 23% of patients in their initial presentation for COVID-19, with cardiomyopathy occurring in 33% of patients [12]. Another study found that heart failure was present in 24% of patients and was associated with an increased risk of mortality [13]. Among those with heart failure, nearly half did not have a known history of hypertension or CVD [13] .It is currently unknown if heart failure is due to new cardiomyopathy versus an exacerbation of previously undiagnosed heart failure [14]. It is important to be conscious of this potential cardiac dysfunction when administering intravenous fluids and avoid overaggressive fluid replacement. Importantly, right heart failure may also occur, particularly among those with ARDS and acute lung injury [1].

CONCLUSION

Cardiac involvement is very common in SARS –Cov-2 infection. Myocarditis is more common, though it may not be fatal in most cases, it can be problematic in case of associated co-morbid conditions.

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