

Original Research Article

Electrocardiographic findings in COVID-19 patients

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ABSTRACT

Background: Severe acute respiratory syndrome coronavirus 2 (SARS CoV2) has caused the global pandemic, COVID-19. Though predominantly a respiratory illness, cardiac manifestations of COVID-19 significantly contribute to mortality. We wanted to determine whether admission electrocardiographic (ECG) characteristics provide prognostic information in COVID-19.

Methods: We performed a retrospective, cross-sectional observational study in a designated District COVID hospital. COVID-19 patient's medical records were converted into an electronic database which included demographic data, clinical characteristics and electrocardiogram recorded at/near the time of admission. Primary outcome assessed was the occurrence of severe COVID-19.

Results: Of 180 patients, the majority were males (67.8%) and aged 31-50 years (38.9%). The predominant comorbidity among patients who were discharged (non-severe COVID-19 disease) and those who got referred (severe COVID-19 disease) was hypertension (56.5% vs 43.3%), followed by diabetes mellitus (37.7% vs 36.7%). Fatigue (41.9%) and cough (18.5%) were the most frequent symptoms among non-severe cases. Of 32.8% of the patients with abnormal ECG, abnormal axis (25.5%), poor R wave progression (23.5%), T inversion (15.3%), left ventricular hypertrophy (12.2%) followed by ST segment depression (8.3%) were the frequent findings. Logistic regression analysis revealed that elderly patients (>60 years) ($\beta=2.276$, OR=9.737, $p=0.002$), Heart rate ($\beta=0.191$, OR=1.211, $p=0.045$) and ST segment depression ($\beta=9.986$, OR=21725.39, $p=0.022$) showed statistically significant positive association with Severe COVID-19.

Conclusions: ST segment abnormalities on admission ECG are markers of cardiac injury and may assist in prognostication of COVID-19. Early identification of these findings might play a crucial role in identifying patients likely to progress to severe COVID-19.

Keywords: COVID-19, Electrocardiogram, Cardiac manifestations

INTRODUCTION

In December 2019, a cluster of unusual cases of pneumonia were reported from Wuhan, China. Later on, the causative agent was identified as severe acute respiratory syndrome coronavirus 2 (SARS CoV2) which is a highly infectious single stranded RNA virus. This novel viral infection was subsequently termed as coronavirus disease 2019 (COVID 19).¹ Soon COVID19 had spread rapidly to most of the countries in the world.

Consequently, on 11th March 2020, the world health organisation (WHO) declared COVID-19 as "pandemic", an emergency of international concern, requiring urgent public health interventions to control the disease.

SARS CoV2 binds to Angiotensin converting enzyme (ACE2) receptors with help of its spike protein and thus gains access into the human cell.² ACE 2 receptors are expressed on pneumocytes (both type-1 and type-2) and endothelial cells. Though the virus mainly targets the

respiratory system, other organ involvement is also common.¹⁻³ Research demonstrates that various tissues, including the myocardium of the heart, express ACE2 protein on their cellular surface.³ The spectrum of the severity of the disease ranges from asymptomatic/mild form to severe/ life threatening disease. Elderly people and those with co-morbidities are at risk of developing moderate to severe disease. People with cardiac risk factors and established cardiovascular disease are more prone to develop severe disease.⁴ The variety of cardiac manifestations caused by COVID-19 comprises acute coronary syndromes, myocarditis, new onset or worsening of pre-existing heart failure, thromboembolic events, arrhythmia and sudden cardiac death.⁵⁻⁸ Several possible mechanisms are proposed for cardiac injury in COVID-19 disease, viz., ACE2-related signalling pathways causing direct injury, hypoxemia caused by COVID-19 resulting in damage to myocardial cells and diffuse systemic inflammatory response and cytokine storm.^{3,9,10}

As the knowledge concerning pathophysiology and management of COVID-19 disease is evolving, there is a necessity to identify prognostic markers that can assist clinicians in rapid triage of the patients. Prognostic markers are also essential for appropriate clinical decision making, estimation of the projected disease course and informed decision making with patients and their families. Electrocardiogram (ECG) is a widely available non-invasive diagnostic test requiring minimal health personnel to perform. It can be quickly done at the bedside, thus, reducing the exposure of health personnel to SARS CoV2. The potential cardiac injuries triggered by COVID-19 can be detected as various patterns in an Electrocardiogram (ECG). ECG has demonstrated incremental prognostic value in many population-based studies and in patients with a spectrum of underlying cardiovascular diseases, including hypertension, making it an appropriate non-invasive test for cardiovascular assessment during this pandemic.¹¹⁻¹⁴ There are many studies published from the Indian sub-continent providing information regarding epidemiology, demographics, and clinical characteristics of COVID-19, but very few studies concerning the Electrocardiographic findings and clinical outcomes. We thus wanted to determine whether ECG characteristics of patients on admission to hospital provide prognostic information, concerned with progression to severe COVID-19.

METHODS

A retrospective, hospital based observational, cross-sectional study was conducted in Gayatri Vidya Parishad Institute of healthcare and medical technology (GVP IHC and MT), designated as district COVID hospital in Visakhapatnam, India. The Institutional Ethical Committee approval was obtained to conduct the study [reference no: GVPIHCMT/IRC/200907/1]. Patients admitted to the Hospital during the period 1st July 2020

to 31st August 2020 were included in the study. Patients who 1) were above 18 years of age 2) tested positive for COVID-19 by real-time reverse transcriptase-polymerase chain reaction assay from nasal or pharyngeal swab specimens, 3) Fulfil the criteria for diagnosis of COVID-19.¹⁵ and 4) Underwent ECG at or near the time of admission were included in the study. Patients with incomplete case sheet details and investigational data were excluded from the study. Based on the patients admitted during the study period, a convenient sample size was taken for the study.

Data collection

It was a fully case-sheet based study. Patients fulfilling the inclusion criteria were identified by one of the investigators and corresponding medical records were studied. Requisite data was entered in the research proforma. The inpatient ID was converted into study ID (SID) and the principal investigator received the electronic database with SID only. As a result, patient's anonymity was maintained. Also, the requirement of informed consent was waived off. The clinical data which were extracted from the electronic database included demographic data, existing comorbidities, symptomatology and electrocardiogram (ECG) obtained at/near the time of admission into the hospital

Electrocardiographic evaluation

A 12 lead ECG which was done at/near the time of admission for each patient was personally reviewed and interpreted by two clinicians who were blinded to the study design and clinical status of the patients. Any disagreement in interpretation between clinicians was resolved by consensus. Data extracted from each ECG findings included heart rate, rhythm, categorized as sinus rhythm (normal/tachycardia/bradycardia) or atrial fibrillation/flutter, atrioventricular block, PR interval, QRS duration and the Bazett-corrected QT interval (in milliseconds), the presence of atrial premature contractions (APCs), ventricular premature contractions (VPCs), axis deviation (Left/Right), right bundle branch block (RBBB), left bundle branch block (LBBB) or left anterior hemi block (LAHB), presence of left or right ventricular hypertrophy, ST segment (ST elevation/depression) and T-wave changes (T-wave inversion) and other changes (poor R progression).

Outcome

Our institute was designated as district COVID hospital by the State Government of Andhra Pradesh, to deal with mild to moderately severe cases of COVID-19 infection.¹⁶ As per the state COVID-19 protocol, patients who progressed to severe disease were referred to State COVID hospital for further management.¹⁷ Accordingly, the primary outcome of our study was referral to the state COVID centre in view of progression to severe

COVID-19 disease during the study period. Referral criteria for COVID-19 patients from our hospital to State COVID hospital include any of the following: 1) respiratory distress RR>30/min, 2) PR>125/min 3) oxygen saturation levels (SpO₂)<90% 4) hypotension (systolic blood pressure<90mm Hg and/or diastolic Blood pressure<60mmHg) 5) altered sensorium 6) critical care complications like multi organ failure, sepsis, acute respiratory distress syndrome and requirement of mechanical ventilation and ICU care.^{16,17} The outcome was ascertained based on the review of case sheet data and information abstracted from the electronic database provided to the principal investigator.

Data analysis

The electronic database was organized into Microsoft excel sheet and Statistical Package for social sciences (SPSS) software version-26 was used to conduct statistical analysis. Data are presented as mean±standard deviation (SD) for continuous variables and frequencies and percentages for categorical variables. The frequency of various ECG characteristics based on outcome status were graphically depicted as a bar diagram. The relationship between clinical, ECG characteristics and outcome was examined using logistic regression analyses. Logistic regression analysis was conducted to examine (1) the association of various factors like age, gender, comorbidities with outcome (2) the relationship

between ECG characteristics and outcome. The odds ratio (OR) along with the 95% confidence interval (CI) were reported. P<0.05 was considered statistically significant for all analyses.

RESULTS

A total of 180 case sheets were retrospectively analysed in the present study. Of 180 cases, the majority were males [n=122(67.8%)] and belonged to the age group of 31-50years [n=70 (38.9%)]. Mean age in the study population was 44.97±15.39 years. Patients who were referred to State COVID hospital in view of severe disease constituted about 10.5% (n=19) of the total cases. About 38.9% (n=70) of the cases had comorbidities. The predominant comorbidity among patients who were discharged (non-severe COVID-19 disease) and those who got referred (Severe COVID-19 disease) was hypertension (56.5% vs 43.3%), followed by diabetes mellitus (37.7% vs 36.7%). Fatigue (41.9%) was the most common symptom at presentation, followed by cough (18.5%) and fever (17.3%), among cases which were discharged from the hospital. However, patients with severe disease had breathlessness (36.6%) as their principal presenting complaint. The demographic and clinical characteristics of the patients in the present study, by the outcome status are depicted in Table 1.

Table 1: Demographic and clinical characteristics of patients (n=180) admitted with COVID-19 by outcome.

Variable	Total (%)	Discharged/Non severe (n=161)	Referred/Severe (n=19)	Pearson's chi square	P
		Frequency (%)	Frequency (%)		
Gender					
Male	122 (67.8)	110 (68.3)	12 (63.2)	0.208	0.649
Female	58 (32.2)	51 (31.7)	7 (36.8)		
Age group (in years)					
18-30	41 (22.8)	40 (24.8)	1 (5.3)	23.274	0.000**
31-50	70 (38.9)	67 (41.6)	3 (15.8)		
51-70	64 (35.6)	52 (32.3)	12 (63.2)		
>70	5 (2.8)	2 (1.2)	3 (15.8)		
Age (Mean ±SD)	44.97±15.39	43.25±14.576	59.58±14.656	F=2.62	0.000
Comorbidities					
Hypertension	52 (28.9)	39 (56.5)	13 (43.3)	16.160	0.000**
Diabetes mellitus	37 (20.6)	26 (37.7)	11 (36.7)	18.136	0.000**
Chronic kidney disease	4 (2.2)	1 (1.4)	3 (10)	17.995	0.000**
Coronary artery disease	6 (3.3)	3 (4.3)	3 (10)	18.136	0.001*
Symptomatology					
Fever	43 (17.3)	38 (18.4)	5 (12.2)	0.069	0.793
Cough	46 (18.5)	40 (19.3)	6 (14.6)	0.405	0.524
Breathlessness	21 (8.5)	6 (2.9)	15 (36.6)	93.306	0.000*
GI symptoms	23 (9.3)	18 (8.7)	5 (12.2)	3.493	0.062
Fatigue	104 (41.9)	97 (46.9)	7 (17.1)	3.817	0.051
Chest pain	11 (4.4)	8 (3.9)	3 (7.3)	3.468	0.063

*p<0.05, **p<0.01 (statistically significant)

Table 2: Electrocardiographic characteristics of patients admitted with COVID 19, overall and by outcome status (n=180).

Variable	n=98 N (%)	Discharged/Non severe (n=161)	Referred/Severe (n=19)	Pearson's chi square	p
		Frequency (%)	Frequency (%)		
Atrial premature complex	2 (2.0)	2 (2.9)	0	0.239	0.625
Ventricular premature complex	4 (4.1)	3 (4.3)	1 (3.6)	0.904	0.342
AV block	1 (1.0)	0	1 (3.6)	8.521	0.004*
RBBB	2 (2.0)	2 (2.9)	0	0.239	0.625
LBBB / LAHB	2 (2.0)	1 (1.4)	1 (3.6)	3.333	0.068
Left ventricular hypertrophy	12 (12.2)	8 (11.4)	4 (14.3)	7.065	0.008*
ST elevation	4 (4.1)	0	4 (14.3)	34.665	0.000**
ST depression	8 (8.3)	2 (2.9)	6 (21.4)	36.827	0.000**
T inversion	15 (15.3)	10 (14.3)	5 (17.9)	8.992	0.003*
Poor R wave progression	23 (23.5)	21 (30)	2 (7.1)	0.097	0.756
Abnormal axis deviation	25 (25.5)	21 (30)	4 (14.3)		
Left axis	23 (23.4)	19 (27.1)	4 (14.3)	0.911	0.340
Right axis	2 (2.0)	2 (2.9)	0		
Variable	Overall Mean±SD	Mean±SD		F	P
Heart rate	82.81±18.224	80.59±15.294	101.63±28.383	2.785	0.000**
PR Interval	143.23±17.219	143.28±17.472	142.83 ±15.209	0.794	0.841
QRS	85.67±11.697	85.30±8.624	88.79 ±26.231	1.086	0.357
QTc	417 ±19.675	415.72±18.628	436±19.411	1.726	0.005*

*p<0.05, **p<0.01 (statistically significant), AV block–atrioventricular block, RBBB–right bundle branch block, LBBB–left bundle branch block, LAHB–left anterior hemiblock

Patients who were referred to State COVID hospital in view of progression to severe disease had statistically significant association with higher age group (p≤0.001), presence of comorbidities such as hypertension (p≤0.001), diabetes mellitus (p≤0.001), Chronic Kidney Disease (p≤0.001) and coronary artery disease (p=0.001), and presence of breathlessness(p≤0.001) as the presenting complaint.

Analysis of the characteristics of the patients' Electrocardiogram, done at/near the time of admission revealed abnormal ECG in 32.8% (n=58) of the patients. Mean values of Heart rate (bpm) and Corrected QT interval (QTc) (milliseconds) were 82.81±18.224 and 417±19.675 respectively. Abnormal axis deviation [25.5% (left axis deviation 23.5% vs right axis deviation

2.0%)], Poor R wave progression (23.5%), T inversion (15.3%), left ventricular hypertrophy (LVH) (12.2%) followed by ST segment depression (8.3%) were major findings observed in the study population. Higher Heart rate (p≤0.001), Prolonged QTc (p=0.005), Presence of LVH (p=0.008), ST segment elevation (p≤0.001), ST segment depression (p≤0.001) and T inversion (p=0.003) showed statistically significant association with Severe COVID-19 disease (Table 2). Abnormal ECG characteristics of the study population are represented in Figure 1.

Binary logistic regression analysis of demographic and clinical factors predicting severe COVID-19 revealed that age above 60 years had statistically significant positive association with Severe disease (β=2.276, OR=9.737, p=0.002) (Table 3).

Table 3: Binary logistic regression of clinical factors associated with referred (severe COVID-19) outcome.

Factors	B (coefficient)	Standard error	OR (95% CI)	P
Age>60 yrs (vs<60 yrs)	2.276	0.726	9.737 (2.345-40.437)	0.002*
Male (vs female)	1.223	0.743	3.397 (0.791-14.584)	0.100
Hypertension	0.697	0.635	2.007 (0.578-6.975)	0.273
Diabetes mellitus	1.168	0.650	3.216 (0.899-11.500)	0.072
Chronic kidney disease	2.475	1.474	11.880 (0.661-213.510)	0.093
Coronary artery disease	1.368	1.048	3.9275 (0.503-30.656)	0.192

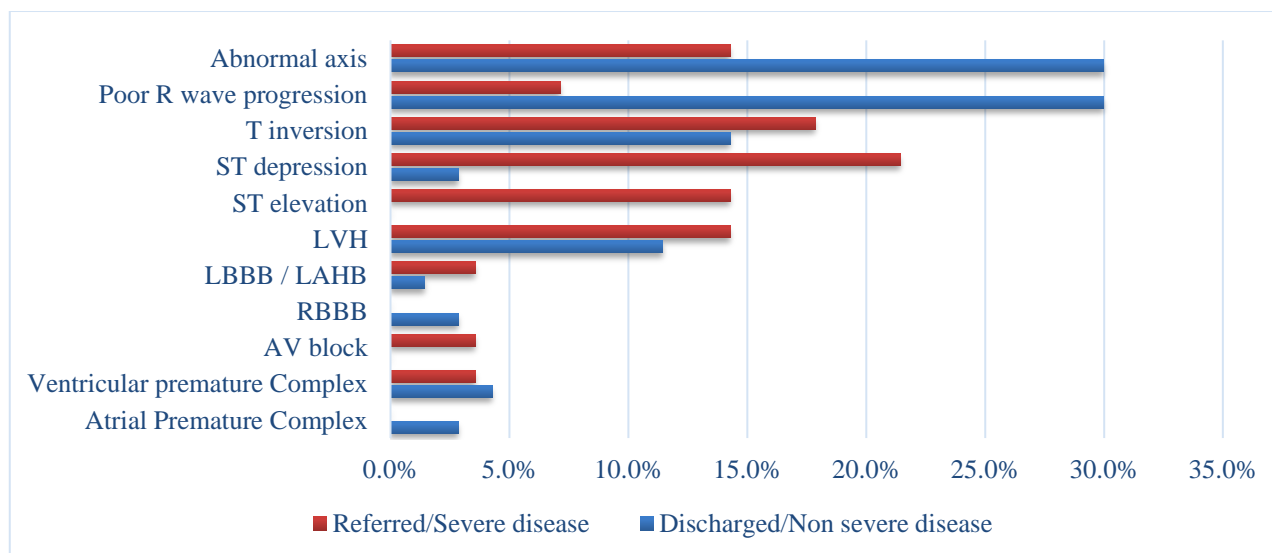


Figure 1: ECG characteristics of COVID19 patients at admission in the present study.

Table 4: Binary logistic regression of ECG characteristics associated with referred (Severe COVID-19) outcome.

ECG characteristic	B (Coefficient)	Standard error	OR (95%CI)	P
Heart rate	0.191	0.096	1.211(1.004-1.460)	0.045*
PR Interval	-0.095	0.054	0.909(0.819-1.010)	0.077
QRS	0.027	0.097	1.027(0.850-1.242)	0.781
QTc	0.074	0.055	1.077(0.966-1.201)	0.181
Atrial premature complex	-21.645	23912.097	0.000	0.999
Ventricular premature complex	2.913	73.529	18.411(0.000-7.134)	0.968
RBBB	-16.898	28378.089	0.000	1.000
LBBB / LAHB	-19.574	26548.375	0.000	0.999
Left ventricular hypertrophy	-4.939	3.383	0.0071(0.000-5.430)	0.144
ST elevation	28.149	17338.470	1678997735921.250	0.999
ST depression	9.986	4.352	21725.391(4.292-109979669)	0.022*
T inversion	4.298	2.314	73.563(0.789-6857.156)	0.063
Poor R wave progression	2.864	2.439	17.529(0.147-2087.556)	0.240
Abnormal axis	2.135	2.352	8.4597(0.084-850.290)	0.364

*p<0.05, **p<0.01 (statistically significant), AV block–atrioventricular block, RBBB-right bundle branch block, LBBB-left bundle branch block, LAHB–left anterior hemi block #age, gender, comorbidities kept constant

Among ECG characteristics, heart rate ($\beta=0.191$, OR=1.211, $p=0.045$) and ST segment depression ($\beta=9.986$, OR=21725.39, $p=0.022$) showed statistically significant positive association with severe COVID-19 (Table 4).

DISCUSSION

COVID-19 pandemic has affected millions of people worldwide. Though pulmonary involvement is common, COVID-19 predisposes to myocardial injury and thus exerts an adverse impact on the functioning of the cardiovascular system. Many common viruses, including coxsackie viruses, adenoviruses, and influenza viruses have been linked to myocarditis in humans. The suggested mechanisms of viral induced myocarditis include excessive immune-mediated destruction of the virus-infected cardiomyocytes by immune cells and/or by

circulating autoantibodies and autoreactive immune cells, and direct virus-induced cardiomyocyte injury.¹⁸ Myocarditis, if fulminant in nature, leads to severe diffuse inflammation of myocardium, causing ventricular arrhythmias, cardiogenic shock and mortality.¹⁹ Various mechanisms responsible for cardiac damage in COVID-19 include direct damage to myocardium by the virus and indirect damage secondary to hypoxia, hypotension, inflammatory reactions, ACE2 receptor down regulation. Another pivotal pathophysiological process in COVID-19 is the occurrence of pro-coagulative state resulting in systemic thrombosis, demand ischemia of the myocardium and vasospasm leading to acute coronary syndrome (ACS).²⁰ Direct myocardial damage by SARS CoV2 has been shown by Tavazzi et al by demonstration of virus particles in the endomyocardial biopsy.²¹ In patients with COVID-19, Huang et al. have shown increased levels of cytokines in patients requiring ICU

care compared to those who do not require ICU admission.²² Similarly, myocardial damage, as evidenced by elevated cardiac biomarkers can be a sinister sign of severe COVID-19.²³ The anticipated cardiac injury caused by COVID-19 could be reflected as various patterns of ECG changes. In a developing country like India, a non-invasive, rapid and basic diagnostic tool for assessing the cardiovascular system, such as ECG, can be of paramount importance for timely triage of the COVID-19 patients.

A variety of electrocardiographic manifestations have been documented in COVID-19. In patients with pre-existing heart disease, the spectrum of ECG changes, from common atrial arrhythmias, precipitated by acute systemic illness to conduction system abnormalities and life-threatening arrhythmias in severe disease, besides other manifestations such as acute coronary syndrome, myocarditis and heart failure, are noted.²⁴ In addition to the disease process, biochemical abnormalities like electrolyte imbalance, especially in critically ill patients and potential treatment modalities employed in COVID-19 management, such as hydroxychloroquine and other antiviral drugs, may have varied impact on the electrocardiographic parameters such as the QT interval.

In our study, only 3.3% of the patients had prior Coronary artery disease, yet about 32.8% of the patients showed abnormal ECG at admission in the hospital indicating an acute COVID-19 disease process as the potential cause of the electrocardiographic changes. In a case report by Zhung et al ECG changes documented in a COVID-19 patient with fulminant myocarditis were sinus tachycardia and Right bundle branch block. RBBB pattern without Significant ST-T wave abnormalities.²⁵ Kim et al have reported nonspecific interventricular conduction delay (IVCD) and premature complexes in a 21-year female with COVID-19 related myocarditis while in few other patients, ST elevation seen were in inferior leads.^{26,27} Diffuse ST segment elevation in inferolateral leads and ST depression with T inversion in V1 and aVR were the ECG findings in a study done by Inciardi et al.⁶

Arrhythmias were reported more in the COVID-19 patients with severe disease than those with mild disease (44.4% vs 6.9% $p < 0.001$).²² While atrial premature complexes and ventricular premature complexes accounted for 2% and 4.1% of abnormal ECG changes in our study, conduction abnormalities such as Atrioventricular block (AV block), left bundle branch block (LBBB) and Right bundle branch block (RBBB) constituted only about 5%. In our study, sinus tachycardia was seen in 16.7% ($n=30$) of the patients. Sinus tachycardia is noted to be the frequent manifestation in patients with COVID-19. Wang et al in their study comparing ECG changes in severe and critically ill COVID-19 patients, found out sinus tachycardia to be the second most frequent ECG characteristic, more so in critically ill patients. Also, sinus tachycardia, along with atrial fibrillation were found to be an independent risk

factor for in hospital death and ventilator use in their study.²⁸ Sinus tachycardia may be a consequence of the inflammatory response or elevated body temperature and/or dehydration and volume depletion in acutely ill patients. It may also be the only manifestation of the ominous pulmonary embolism or underlying myocarditis.^{5,29} Of 180 patients, 9.4% ($n=17$) had sinus bradycardia. Severe bradycardia was identified in about 15% of patients with middle east respiratory syndrome (MERS).³⁰ Paediatric population were found to have bradycardia with coronaviruses that cause common cold.³¹ Our study revealed that patients with higher heart rate were more likely to progress to severe COVID-19 (OR=1.211, $p=0.045$). It was found out that mean QTc was higher in COVID-19 patients compared to normal person.³² In our study, mean QTc was higher among patients with severe disease and showed significance difference among severe and non-severe patient groups ($p=0.005$). No such difference was observed in a study by Ali et al.³³ The relationship between ST-T changes on ECG and myocardial damage with poor prognosis has been demonstrated.³⁴ Abnormal ST segment deviation (elevation/depression) and T inversion together, were the most common (27.7%) abnormal ECG manifestation in our study. Also, these changes had statistically significant difference among severe and non-severe groups ($p \leq 0.001$). Patients with ST segment depression on admission ECG were more likely to progress to severe disease needing referral to State COVID hospital. Similar results were found out by Wang et al in their study of severe and critically severe patient groups of COVID-19, in which ST-T abnormal changes were the most common ECG manifestation. They also noticed that such changes were more pronounced in critically severe patients compared to severe patients.²⁸ Similarly, in another study ST depression, T wave inversion were observed more in severe group than in non-severe group.³⁵ ST-T changes may have various pathological basis such as myocardial damage inflicted by SARS CoV2, hypocalcaemia, hypertension, or coronary heart disease. Thus, recognition of such abnormal ECG manifestations may guide the treating physicians in early identification of impending severe COVID-19 among infected patients.

Our study is probably one of the few studies focusing on electrocardiographic features of COVID-19 patients in our region of the country. Major limitations of our study are the retrospective nature of the study and data obtained from a single centre. Considering we did not have prior ECGs in all the patients, we could not draw a comparison between prior and admission ECGs for the occurrence of new findings. In addition, laboratory parameters and inflammatory markers which could have impacted the nature of the disease despite the presence or absence of ECG changes, were not studied. Also, the outcome of Severe COVID-19 patients referred to State COVID hospital could not be obtained. Only an association between ECG characteristics and severe COVID-19 could be ascertained. However, prospective studies

evaluating mortality and morbidity are needed to establish the causality of these results.

CONCLUSION

Our data showed that ECG can be a useful prognostic tool in COVID-19. Although major arrhythmias were not noted in our study population, repolarization abnormalities in the form of ST segment depression and T wave inversion showed significant association with Severe COVID-19 necessitating referral to higher centre in view requirement of ventilator support or multi-organ failure needing intensive care, thus indicating a poorer prognosis. Early identification of these ECG findings may play a crucial role in the management of COVID patients to reduce morbidity and mortality.

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