**Journal Of Medical Science And Clinical Research**
IGM Publication                    An Official Publication Of IGM Publication

# A Study on electrographic abnormalities and troponin elevation in COVID-19 patients: A Bangladeshi Perspective

Authors
**Dr Syeda Masuma Kawsar*[1], Dr Amirul Islam Bhuyan[2]**
[1]MBBS, MD (Cardiology), Consultant Cardiologist, New Labaid Hospital, Brahmanbaria, Bangladesh
[2]MBBS, MS (CVTS), Consultant Cardiac Surgery Department, Labaid Cardiac Hospital, Dhanmondi, Dhaka, Bangladesh
Corresponding Author
**Dr. Syeda Masuma Kawsar**
MBBS, MD (Cardiology), Consultant Cardiologist, New Labaid Hospital, Brahmanbaria, Bangladesh

**Abstract**
**Background:** *In Bangladesh, COVID-19 patients frequently present with electrographic abnormalities and elevated troponin levels, indicative of cardiac stress. These observations highlight the virus's potential impact on cardiovascular health, necessitating vigilant monitoring and timely interventions to mitigate complications.*

**Study Objective:** *This research aims to assess electrographic abnormalities and troponin elevation in COVID-19 patients within Bangladesh.*

**Methods:** *Conducted retrospectively at a tertiary medical college and hospital, our study enrolled 100 consecutive adult COVID-19 patients. We analyzed baseline characteristics, lab results, and serial ECGs. Troponin I concentrations were measured using the Abbott Architect method (normal range: ≤0.05 ng/mL). Troponin levels were categorized as normal (0.00-0.05 ng/mL), mildly elevated (0.05-1 ng/mL), and significantly elevated (>1 ng/mL). ECGs were reviewed and interpreted.*

**Results:** *The majority of patients (71%) were males aged 41-50. Common COVID-19 symptoms included dyspnea and cough (75%), followed by fever, fatigue, and malaise (80%). Gastrointestinal symptoms were seen in 20%, and 10% experienced anosmia or dysgeusia. Comorbidities included diabetes mellitus (65%), hypertension (60%), CKD (10%), coronary artery diseases (13%), and other cardiovascular diseases (5%). Abnormal LFTs were observed in 30%. Initial troponin levels averaged 0.18 ± 1.75 ng/mL, with a maximum of 2.55 ± 17.90 ng/mL. Mortality occurred in 25% of cases.*

**Conclusion:** *Our findings suggest that ECG repolarization abnormalities may serve as markers of clinical severity, even in cases with mildly elevated troponin values in COVID-19 patients.*

**Keywords:** *covid-19, electrographic abnormalities, troponin.*

## Introduction

Coronavirus disease 2019 (also known as COVID-19) is an urgent situation that commonly affects the cardiovascular system and is rapidly spreading. People who are hospitalized with COVID-19 infection and who already have coronary artery disease or who have immediate cardiac damage have a considerable chance of passing away. This risk is significantly increased for those who experience acute cardiac damage. [1-2] However, the effect of cardiac involvement on short-term outcomes caused by COVID-19 infection is not fully understood at this time. During the bedside assessment of COVID-19-infected individuals, echocardiography is used to a significant degree, and abnormalities in echocardiographic findings are usually identified.[3-4]

Cardiac symptoms have been linked to a variety of causes, including injury to the myocardium caused by acute coronary syndromes, myocarditis, and takotsubo cardiomyopathy.

As a result of COVID-19, the procedures for cardiac imaging have been streamlined to protect both the sonography experts and the patients. Bedside echocardiograms have been used to investigate ventricular size and systolic function, the presence or absence of left ventricular regional wall motion abnormalities, and the presence of hemodynamically important valvular heart disease or pericardial effusion. In addition, bedside echocardiograms have been used to look for left ventricular regional wall motion anomalies.[5-7]

However, because of these modifications to a traditional comprehensive echocardiographic examination, the utility of echocardiography may be diminished when used to the classification of clinical risks. In this research, our main goal is to evaluate the electrographic abnormalities and troponin elevation in COVID-19 patients in Bangladesh.

## Methodology

One hundred consecutive adult patients hospitalized with COVID-19 illness were included in this retrospective research conducted at a tertiary medical institution and hospital in Dhaka. Baseline characteristics, laboratory data, and serial electrocardiograms were collected by reviewing patient medical records. The Abbott Architect technique (Abbott, Abbott Park, Illinois) was used to measure troponin I concentrations, with a 99th percentile of 0.05 ng/mL for a healthy population. Troponin levels were classified as either normal (1 ng/mL), slightly raised (1 ng/mL), or considerably increased (>1 ng/mL) for descriptive analysis. Electrocardiograms were examined and analyzed. Rate, rhythm (sinus or AF), bundle-branch blocks (AV, RB, L), nonspecific intraventricular conduction block (QRS duration >120 ms), and ST segment/T-wave changes (localized ST elevation, localized T-wave inversion, or other nonspecific repolarization abnormalities) were all taken into account when analyzing the ECGs.

IBM SPSS Statistics 26 was used for the statistical analysis, and GraphPad Prism 8 was used to create the graphics. Categorical variables are shown as counts (percentages), whereas continuous variables are presented as means and standard deviations.

## Results

Table-1 demonstrates that most of the patients (71%), are in the (41-50) age range. The following diagram is explained in further depth below:

**Table 1:** Age distribution of the patients

| Age group | Percentage (%) |
|---|---|
| 41-50 | 71% |
| 51-60 | 19% |
| 61-70 | 10% |

In figure-1 shows the gender distribution of the patients where out of 100 patients 54% were male and 46% were female. The following figure is given below in detail:
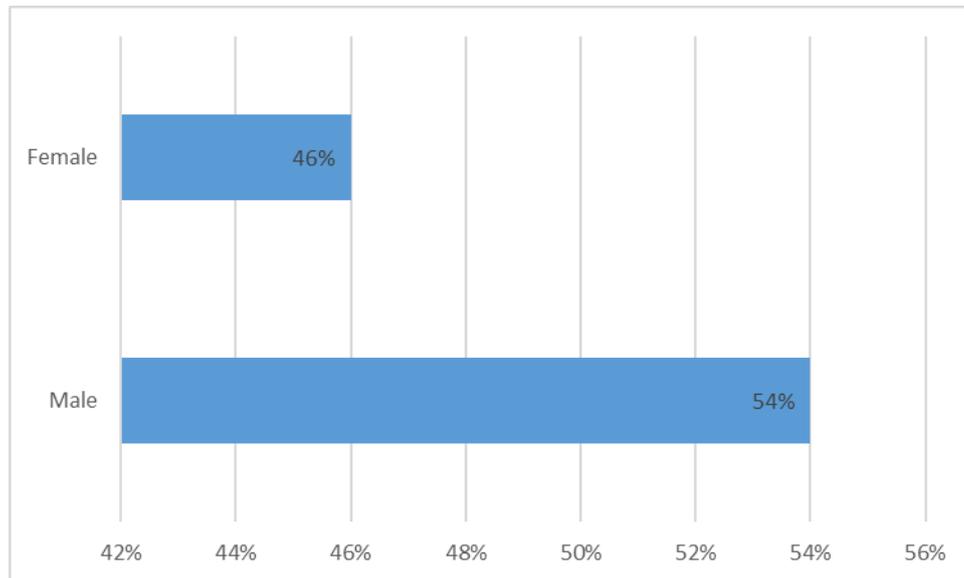
**Figure 1:** Gender distribution in the patients.

Table 2 provides an overview of COVID-19 symptom distribution among patients. Notably, dyspnea and cough were prevalent, affecting 75% of cases, while fever, fatigue, and malaise were reported in 80% of cases. Additionally, gastrointestinal symptoms were noted in 20% of patients, and 10% experienced anosmia and dysgeusia. For a detailed breakdown, please refer to the following figure:

**Table 2:** Patient Distribution Based on COVID-19 Symptoms

| COVID-19 symptoms | Percentage (%) |
|---|---|
| Dyspnea, cough | 75% |
| Fever, fatigue, malaise | 80% |
| Gastrointestinal symptoms | 20% |
| Anosmia, dysgeusia | 10% |

Table 3 presents the clinical status of COVID-19 patients, revealing that 65% had diabetes mellitus, 60% had hypertension, 10% had chronic kidney disease (CKD), 13% had coronary artery diseases, and 5% had other cardiovascular conditions. For a comprehensive breakdown, please refer to the detailed table below:

**Table 3:** Clinical Profile of COVID-19 Patients

| Clinical status | Percentage (%) |
|---|---|
| Diabetes mellitus | 65% |
| Hypertension | 60% |
| CKD | 10% |
| Coronary artery disease | 13% |
| CHF | 20% |
| Cardiovascular disease (stroke/coronary artery disease) | 5% |

Table 4 offers insights into the laboratory profile of COVID-19 patients, highlighting that 30% exhibited abnormal liver function tests (LFTs). The initial troponin level averaged 0.18 ± 1.75 ng/mL, with a maximum of 2.55 ± 17.90 ng/mL. Furthermore, mortality was observed in 25% of cases. A comprehensive breakdown is provided in the detailed table below:

**Table 4:** Laboratory Analysis of COVID-19 Patients

| Initial creatinine (mg/dL) | 1.2 ± 2.0 |
|---|---|
| Abnormal LFTs | 30% |
| Initial troponin (ng/mL) | 0.18 ± 1.75 |
| Maximum troponin | 2.55 ± 17.90 |
| Troponin group | % |
| ≤0.05 | 55% |
| 0.05-1.00 | 33% |
| >1.00 | 12% |
| Mortality | 25% |

Table 5 unveils the factors influencing mortality, as revealed through univariate and multivariate Cox regression analyses. Clinical indicators associated with death encompassed age and elevated Troponin, while gender, race, and cardiovascular comorbidities (CAD, CHF, HTN) showed no significant impact. The mortality rate exhibited an upward trend with increasing troponin levels. For comprehensive information, consult the detailed table below:

Univariate Regressions:

**Table 5:** The factors influencing mortality

|  | HR | 95% CI | P value |
|---|---|---|---|
| Age (yrs) | 1.09 | 1.05 - 1.1 | <0.01 |
| Gender | 1.08 | .56 - 2.13 | 0.80 |
| CAD | 1.07 | .49 - 2.41 | 0.84 |
| HTN | 1.38 | .78 - 2.48 | 0.26 |
| CKD | 2.3 | 1.12 - 5.11 | 0.02 |
| DM | 1.77 | 1.01 - 3.1 | 0.05 |
| CHF | 2.08 | .65 - 6.73 | 0.22 |
| Initial creatinine (mg/dL) | 1.25 | 1.08 - 1.43 | <0.01 |
| Abnormal LFTs | 0.83 | .42 - 1.7 | 0.64 |
| Initial troponin (ng/mL) | 0.98 | .84 - 1.16 | 0.91 |
| Maximum troponin | 1.02 | 1. - 1.02 | 0.02 |
| Positive troponin (>0.05) | 4.25 | 2.28 - 7.86 | <0.01 |
| Multivariate Regression: | | | |
| Age (yrs) | 1.05 | 1.04 - 1.1 | <0.01 |
| CKD | 1.52 | .65 - 3.6 | 0.22 |
| DM | 0.95 | 53 - 1.86 | 0.92 |
| Positive troponin (>0.05) | 3.21 | 1.71 - 6.05 | <0.01 |

In figure-2 shows, mortality rate stratified by Troponin elevation groups. The following figure is given below in detail:
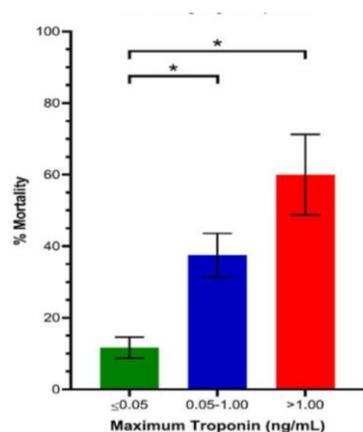


**Figure 2:** Mortality rate stratified by Troponin elevation groups[8]

In figure-3 shows KM survival according to ECG changes stratified by Troponin elevation group. The following figure is given below in detail:
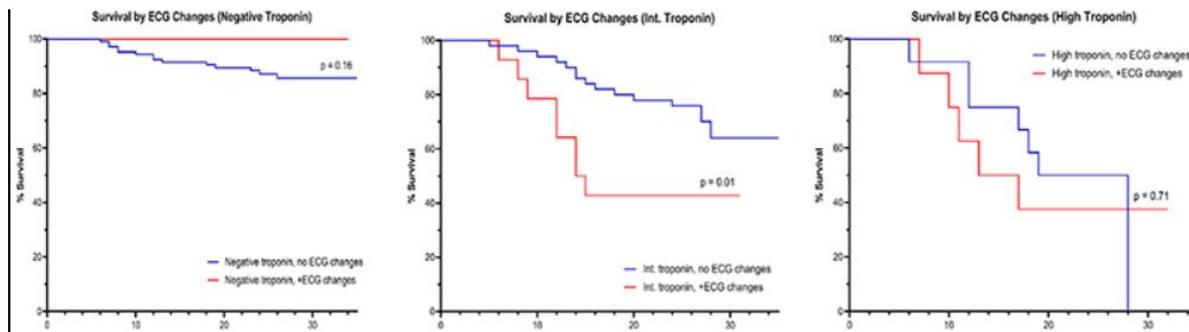


**Figure-3:** KM survival according to ECG changes stratified by Troponin elevation group [8]
source by: https://europepmc.org/article/PPR/PPR238336

## Discussion

Direct myocardial injury from COVID-19 has been linked to the inflammatory cascade or cytokine release, microvascular damage from disseminated intravascular coagulation and thrombosis, SARS-CoV-2 entering myocardial cells via binding to ACE2 receptors, and hypoxemia combined with the increased metabolic demands of acute illness. [9-11]

In this follow-up retrospective cohort research, we analyze how elevated Troponin levels relate to ECG irregularities. Our results show that 1) COVID-19 patients who are hospitalized often have moderate myocardial damage, as characterized by a low-level rise in troponin concentration. 2) An elevated Troponin level may be related with an increased risk of death in cases of severe myocardial damage. 3) Significantly higher mortality is related with ECG abnormalities in the group of individuals with modest Troponin elevation (0.05-1 ng/ml).

In spite of the fact that troponin elevation above the 99th percentile of the upper reference limit is considered the central marker of "myocardial injury," mild troponin elevation between 0.05 and 1 is often nonspecific and associated with non-vascular etiologies like strain, myocyte necrosis, and increased cell membrane permeability. [12]

The moderate rise of Troponin in our group of individuals with COVID-19 was indeed a common observation. Our findings imply that in-hospital risk classification of patients with modest Troponin increase may be improved by checking for the existence of ECG abnormalities. Last but not least, as COVID-19 is characterized by a prolonged fever, it is important for doctors to be aware of the phenomenon of fever caused Brugada pattern and not confuse it for ST elevation myocardial infarction.

## Conclusion

From the results of our research, we are able to draw the conclusion that anomalies in ECG repolarization may be a signal of clinical severity in individuals whose Troponin levels are only slightly elevated.

## Reference

1. Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020; 395: 497-506.
2. Coronavirus disease (COVID-19) pandemic. Geneva: World Health Organization. https://www.who.int/emergencies/diseases/novel-coronavirus-2019
3. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, Xiang J, Wang Y, Song B, Gu X, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020; 395: 1054-1062.

4. Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, Wang H, Wan J, Wang X, Lu Z. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol. 2020 March 27. https://doi.org/10.1001/jamacardio.2020.1017

5. Rautaharju PM, Kooperberg C, Larson JC, LaCroix A. Electrocardiographic predictors of incident congestive heart failure and all-cause mortality in postmenopausal women: the Women's Health Initiative. Circulation. 2006; 113: 481-489.

6. Daviglus ML, Liao Y, Greenland P, Dyer AR, Liu K, Xie X, Huang CF, Prineas RJ, Stamler J. Association of nonspecific minor ST-T abnormalities with cardiovascular mortality: the Chicago Western Electric Study. JAMA. 1999; 282: 530-536.

7. Park KC, Gaze DC, Collinson PO, Marber MS. Cardiac troponins: from myocardial infarction to chronic disease. Cardiovasc Res. 2017; 113: 1708-1718. 13

8. Chorin, E., Dai, M., Kogan, E., Wadhwani, L., Shulman, E., Nadeau-Routhier, C., ... & Jankelson, L. (2020). Electrocardiographic Abnormalities and Troponin Elevation in COVID-19. medRxiv.

9. Clerkin KJ, Fried JA, Raikhelkar J, et al. Coronavirus Disease 2019 (COVID-19) and Cardiovascular Disease. Circulation. 2020; 18.

10. Patel AB, Verma A. COVID-19 and Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers: What Is the Evidence? JAMA. 2020; 19.

11. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. JAMA Cardiology. 2020.

12. Korff S, Katus HA, Giannitsis E. Differential diagnosis of elevated troponins. Heart. 2006; 92(7): 987-993. https://doi.org/10.1136/hrt.2005.071282.