Effect of Pre-existing Ischemic Heart Disease on 30-Days Mortality in COVID-19 Patients

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Abstract

Background: COVID-19 has emerged as a multisystem inflammatory disorder with significant cardiovascular implications. Patients with pre-existing cardiovascular conditions may be particularly vulnerable to severe outcomes, yet data from developing countries regarding the impact of pre-existing ischemic heart disease (IHD) on COVID-19 outcomes remains limited.

Objective: To investigate the effect of pre-existing ischemic heart disease on 30-day mortality in COVID-19 patients at a tertiary care hospital in Bangladesh.

Methods: This case-control study was conducted at Dhaka Medical College Hospital over twelve months, enrolling 120 RT-PCR-confirmed COVID-19 patients (60 with pre-existing IHD, 60 without IHD). Clinical characteristics, laboratory parameters, and treatment modalities were assessed. Outcomes included in-hospital mortality, complications, and 30-day mortality. Data analysis included chi-square tests, t-tests, and multivariable logistic regression.

Results: COVID-19 patients with pre-existing IHD showed significantly higher rates of critical disease (30.0% vs 11.6%, p=0.002), cardiac complications including acute myocardial infarction (45.0% vs 0%, p=0.001), and heart failure (28.3% vs 6.66%, p=0.001). The IHD group required more intensive care (71.6% vs 36.6%, p<0.001) and demonstrated higher in-hospital mortality (36.6% vs 10%, p<0.001) and 30-day mortality (60.0% vs 16.6%, p<0.001). After adjusting for confounders, pre-existing IHD independently predicted 30-day mortality (OR=7.25, 95% CI=2.17-24.23).

Conclusion: COVID-19 patients with pre-existing IHD experience significantly worse outcomes, including higher rates of complications and mortality. These findings emphasize the need for enhanced monitoring and aggressive management strategies for COVID-19 patients with pre-existing IHD in Bangladesh.

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Introduction

The coronavirus disease 2019 (COVID-19) pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has emerged as one of the most significant global health challenges of the 21st century. The initial cases were reported to the World Health Organization (WHO) by China Health Authority on December 31, 2019, presenting as pneumonia of unknown etiology in Wuhan City, Hubei Province.[1] Following its identification and classification, the virus demonstrated unprecedented transmission efficiency, rapidly spreading beyond China to multiple countries including Italy, the United States, and Germany.[2] By March 11, 2020, the WHO declared a global pandemic, with case numbers escalating to over 70 million and deaths exceeding 1.5 million within nine months.[3]

SARS-CoV-2 transmission primarily occurs through respiratory droplets between individuals in close proximity (within 2 meters), particularly during coughing or sneezing episodes.[4] While the disease typically manifests with fever, dry cough, dyspnea, and fatigue, severe cases can progress to viral pneumonia and acute respiratory distress syndrome (ARDS), potentially leading to mortality.[5] Diagnosis is confirmed through reverse transcription-polymerase chain reaction (RT-PCR), established by WHO as the gold standard for SARS-CoV-2 detection.[6]

Although initially characterized as primarily a respiratory illness, COVID-19 has been recognized as a multisystem inflammatory and thrombotic disorder with significant cardiovascular implications. The pathophysiological mechanism involves the binding of the virus's spike protein to angiotensin-converting enzyme 2 (ACE-2) receptors on vascular endothelium, initiating a cascade of events that leads to reduced ACE-2 receptor density and activity. This results in angiotensin II accumulation, promoting a state of vasoconstriction, fibrosis, and hypertrophy.[7] Severe cases frequently present with venous thromboembolism, arterial thrombosis, and microvascular thrombi, potentially culminating in multiple organ dysfunction.[8] The inflammatory activation triggers coagulation system abnormalities, measurable through D-dimer plasma levels.[9]

The intersection of COVID-19 and cardiovascular health presents particular concerns for patients with pre-existing ischemic heart disease (IHD). The pandemic has created substantial barriers to IHD management, resulting in increased frequency of adverse clinical events, hospitalizations, and mortality risk. This relationship appears to be mediated through a COVID-related Cardiometabolic Syndrome (CIRCS), characterized by abnormal adiposity, dysglycemia, dyslipidemia, and hypertension. Management complexity is further heightened by considerations regarding cardiovascular medications, although retrospective studies suggest potential protective effects of calcium channel blockers and beta-blockers in COVID-19 patients with heart disease.[10]

The characteristic presentation of IHD, angina pectoris, is typically managed through various pharmaceutical interventions, including beta-blockers, calcium antagonists, and nitrates.[11] However, the high incidence of cardiovascular complications in COVID-19 patients, attributed to systemic inflammatory responses and immune system disruption, presents unique challenges in disease management. The potential role of ACE2-related signaling pathways in both viral infection and heart injury suggests a mechanistic link between COVID-19 and IHD that warrants further investigation.

In the context of the pandemic's impact on Bangladesh, where virus spread since March 8, 2020, has positioned the country among the most affected globally,[12] understanding the relationship between pre-existing cardiovascular conditions and COVID-19 outcomes becomes crucial. With limited data available in our regional context regarding this critical intersection, this study aims to investigate the effect of pre-existing ischemic heart disease on 30-day mortality in COVID-19 patients.

Methods

This case-control study was conducted in the Department of Cardiology at Dhaka Medical College Hospital (DMCH), Bangladesh, over a twelve-month period. The study population comprised COVID-19 patients admitted to the cardiology department who met the study criteria. Exposed were defined as RT-PCR-confirmed COVID-19 patients with pre-existing ischemic heart disease (IHD), while unexposed were RT-PCR-confirmed COVID-19 patients without pre-existing IHD. A total of 120 patients were enrolled using purposive sampling, with equal distribution between exposed and unexposed.Male and female patients above 18 years with RT-PCR-confirmed COVID-19 who provided informed consent were included. Patients with severe chronic conditions, including advanced COPD, pulmonary fibrosis, liver cirrhosis, end-stage neurologic conditions, cardiac devices, recent major surgeries, severe dementia, metastatic malignancy, pregnancy, and those unable to provide consent were excluded.

The study evaluated demographic characteristics, diagnostic parameters including COVID-19 severity and IHD diagnosis, cardiovascular risk factors, and clinical outcomes including mortality and hospital course. Participants underwent comprehensive clinical evaluation, including detailed history, physical examination, and relevant investigations conducted at DMCH and BSMMU laboratories. ECG and echocardiography were performed using standardized equipment at DMCH. Follow-up assessments were conducted at 30 days post-admission through in-person visits or telephone interviews. Data collection utilized a pretested Bengali questionnaire gathering information on clinical characteristics, socioeconomic factors, and medical history. COVID-19 severity was classified according to standard criteria.

Statistical analysis was performed using SPSS version 24. Baseline characteristics were reported using appropriate descriptive statistics. Between-group comparisons were conducted using t-tests and χ^2 tests for continuous and categorical variables, respectively. Regression analysis examined risk factors for mortality. Statistical significance was set at $p<0.05$ with 95% confidence intervals.

The study protocol received approval from the Ethical Review Committee of DMCH. Written informed consent was obtained from all participants following detailed explanation of the study procedures. Participant confidentiality was maintained throughout the study period. All procedures were conducted following the guidelines of the Declaration of Helsinki.

Results

The sociodemographic characteristics analysis revealed that the mean age of COVID-19 patients with pre-existing IHD was 64.2 ± 8.6 years, while those without IHD averaged 62.7 ± 8.5 years. Half of the patients with IHD were above 60 years. Males predominated in both groups (66.7% vs 75.0%). Regarding education, graduation completion was higher among IHD patients (40.0% vs 30.0%). Most participants belonged to lower middle

socioeconomic status in both groups (40.0% vs 51.7%). No significant differences were observed in sociodemographic parameters between the groups (Table I).

Table I: Sociodemographic profile of participants (n=120)

Characteristics	COVID with pre-existing IHD (n=60) n (%)	COVID without IHD (n=60) n (%)	p-value
Age, mean±SD	64..2 ± 8.6	62.7 ± 8.5	0.325
Age groups			
< 40	5 (8.33)	4 (3.33)	0.205
41 – 50	11 (18.3)	10 (16.6)	
51 – 60	14 (23.3)	18 (30.0)	
> 60	30 (50.0)	28 (46.7)	
Sex			
Male	40 (66.7)	45 (75.0)	0.431
Female	20 (33.3)	15 (25.0)	
Education			
Illiterate	3 (5.0)	3 (5)	0.311
Primary complete	2 (3.3)	2 (3.3)	
Secondary complete	10 (16.7)	16 (26.7)	
Higher secondary complete	17 (28.3)	13 (21.7)	
Graduation completed	24 (40.0)	18 (30.0)	
Socio-economic status			
Poor	10 (16.7)	7 (11.7)	0.323
Lower middle	24 (40.0)	31 (51.7)	
Upper middle	17 (28.3)	16 (26.7)	
Rich	9 (15.0)	6 (10.0)	

P-value was determined by chi-square test and Fisher's exact test where appropriate

Analysis of comorbidities and COVID-19 severity demonstrated that hypertension (86.6% vs 33.3%, p=0.002), diabetes mellitus (76.8% vs 41.2%, p=0.003), dyslipidemia (68.3% vs 38.0%, p=0.001), and family history of IHD (65% vs 28.3%, p=0.001) were significantly higher in patients with pre-existing IHD. Notably, COVID-19 severity differed significantly between groups (p=0.002), with critical cases being more prevalent in IHD patients (30.0% vs 11.6%) (Table II).

Table II: Comorbidities and COVID-19 severity among participants (n=120)

Characteristics	COVID with pre-existing IHD (n=60) n (%)	COVID without IHD (n=60) n (%)	p-value
Smoking or chewing tobacco	34 (57.7)	25 (41.6)	0.132
Hypertension	52 (86.6)	20 (33.3)	0.002
DM	46 (76.8)	25 (41.2)	0.003
Dyslipidemia	41 (68.3)	23 (38.0)	0.001
Obesity	34 (56.6)	28 (46.6)	0.273
Family H/O IHD	39 (65)	17 (28.3)	0.001
COPD/Asthma	29 (48.38)	22 (36.2)	0.196
COVID-19 severity			
Mild	10 (16.3)	28 (46.6)	0.002
Moderate	20 (33.3)	18 (30)	
Severe	12 (20.0)	17 (28.3)	
Critical	18 (30.0)	7 (11.6)	

P-value was determined by chi-square test

Laboratory and radiologic parameters showed that patients with pre-existing IHD had significantly higher levels of cardiac biomarkers, metabolic parameters, and lipid profiles. ECG abnormalities were more frequent in IHD patients, particularly ST-T changes (53.3% vs 6.6%, $p<0.001$) and ventricular fibrillation with cardiac arrest (8.3% vs 0%, $p=0.028$). Echocardiographic findings revealed significantly higher prevalence of cardiac dysfunction in IHD patients, including global hypokinesia (16.6% vs 0%, $p<0.001$) and mild LV systolic dysfunction (40.0% vs 6.66%, $p<0.001$) (Table III).

Table III: Comparison of laboratory and radiologic parameters between the groups (n=120)

Characteristics	COVID with pre-existing IHD; (n=60); n (%)	COVID without IHD; (n=60); n (%)	p-value
Troponin I (ng/ml)	0.293±0.145	0.03±0.023	0.001
Fasting glucose (mmol/l)	7.6±1.23	5.7±1.49	0.002
Total cholesterol (mg/dl)	222.5±71.3	160.8±45.3	0.002
LDL cholesterol (mg/dl)	127.7±28.2	86.23±20.8	0.003
HDL cholesterol (mg/dl)	28.2±12.3	45.7±11.6	0.002
Triglycerides (mg/dl)	292.3±27.34	169.3±26.56	0.003
HbA1c (%)	8.8±0.61	6.87±0.68	0.001
S. Creatinine (mg/dL)	0.9± 0.2	0.7 ± 0.28	0.342
ECG abnormalities			
ST-T abnormality	32 (53.3)	4 (6.6)	<0.001
Sinus tachycardia	14 (23.3)	22 (36.6)	0.111
Atrial fibrillation	7 (11.6)	2 (3.33)	0.083
Ventricular tachycardia (VT)	4 (6.66)	0 (0.0)	0.059
Ventricular fibrillation and cardiac arrest	5 (8.3)	0 (0.0)	0.028
Echocardiography findings			
Global hypokinesia	10 (16.6)	0 (0.0)	<0.001
Mild LV systolic dysfunction	24 (40.0)	4 (6.66)	<0.001
Moderate LV systolic dysfunction	12 (20.0)	0 (0.0)	<0.001
Severe LV systolic dysfunction	3 (5.0)	0 (0.0)	0.122
Pericardial effusion	16(26.6)	4 (6.66)	<0.001

P-value was determined by independent samples *t*-test, Chi-square test and Fisher's exact test where appropriate

Assessment of treatment modalities revealed that IHD patients received significantly more cardiovascular medications, including anti-lipid therapy (85.0% vs 23.3%, p=0.001), anti-platelets (86.6% vs 23.3%, p=0.001), and ACEi/ARB (61.6% vs 26.6%, p=0.001). Oxygen requirements were higher in IHD patients, with more requiring high-flow nasal cannula (30.0% vs 11.6%, p=0.013). HDU/ICU admission rates were significantly higher in the IHD group (71.6% vs 36.6%, p<0.001) (Table IV).

Table IV: Comparison of treatments provided between the groups (n=120)

Characteristics	COVID with pre-existing IHD (n=60) n (%)	COVID without IHD (n=60) n (%)	p-value
Anti- lipid	51(85.0)	14 (23.3)	0.001
Anti- platelets	52 (86.6)	14 (23.3)	0.001
Nitroglycerin/Nicorandil	30 (50.0)	0 (0.0)	0.001
Intravenous fluid	15 (25.0)	37 (61.6)	0.680
Antiviral	47 (78.3)	48 (80)	0.121
Steroid	53 (88.3)	55 (91.6)	0.131
Antibiotic	54 (90.0)	38 (63.3)	0.321
Diuretics	37 (61.6)	16 (26.6)	0.001
LMWH	55 (91.6)	45 (75)	0.143
Rivaroxaban/Apixaban	56 (93.3)	52 (86.6)	0.223
ACEi/ARB	37 (61.6)	16 (26.6)	0.001
O_2 therapy given through			
Nasal Prongs	10 (16.6)	28 (46.6)	<0.001
High Flow nasal cannula	18 (30.0)	7 (11.6)	0.013
Mask	20 (33.3)	18 (30)	0.694
Mask with reservoir	12 (20)	7 (11.6)	0.211
Flow of oxygen (L/min)			
1 – 5	10 (16.6)	28(46.6)	0.007
6 – 10	20 (33.3)	18 (30.0)	
11 – 15	12 (20.0)	17 (28.3)	
> 15	18 (30.0)	7 (11.6)	
HDU or ICU needed	43 (71.6)	22 (36.6)	<0.001
Non-invasive ventilation needed	3 (5.0)	1 (1.66)	0.309
Invasive ventilation needed	4 (6.66)	1 (1.66)	0.171

P-value was determined Chi-square test and Fisher's exact test where appropriate.
LMWH: Low molecular weight heparin; ACEi: Angiotensin Converting Enzyme Inhibitor; ARB: Angiotensin Receptor Blocker

Regarding complications and mortality outcomes, IHD patients experienced significantly higher rates of cardiac complications, particularly acute myocardial infarction (45.0% vs 0%, p=0.001) and heart failure (28.3% vs 6.66%, p=0.001). Mortality outcomes were notably worse in the IHD group, with higher in-hospital deaths (36.6% vs 10%, p<0.001) and total mortality (60.0% vs 16.6%, p<0.001). Among discharged patients, 30-day mortality remained significantly higher in the IHD group (36.84% vs 8.0%, p<0.001) (Table V).

Table V: Complication and mortality outcome between groups

Characteristics	COVID with pre-existing IHD (n=60) n (%)	COVID without IHD (n=60) n (%)	p-value
Cardiac complications			
AMI	27 (45.0)	0 (0.0)	0.001
Myocarditis	10 (16.6)	4 (6.66)	0.087
Heart Failure	17 (28.3)	4 (6.66)	0.001
Arrythmia	9 (15.0)	3 (5)	0.067
Pulmonary thromboembolism	3 (5.0)	0 (0)	0.121
Mortality outcomes			
Discharged & alive at 30 days post admission	38 (63.33)	50 (83.3)	0.013
In-hospital deaths	22 (36.6)	6 (10)	<0.001
Fatality after hospital discharge (30 days post admission)	14 (36.84)	4 (8.0)	<0.001
Total mortality (Mortality within and outside hospital)	36 (60.0)	10 (16.6)	<0.001

p-values were determined by Chi-square test and Fisher's exact test where appropriate

Multivariable logistic regression analysis showed that preexisting IHD (OR=7.25, 95% CI= 2.17-24.23) was the independent predictor for 30 days mortality in COVID-19 positive patients after adjusting DM, dyslipidemia, HTN, COPD.

Table VI: Multivariable logistic regression for 30 days mortality with confounding risk factors or comorbidities (n=120)

Predictor	OR	95% CI		p-value
		Lower	Upper	
IHD	7.25	2.17	24.23	0.001
DM	3.31	2.54	8.090	0.121
Dyslipidemia	0.543	0.220	5.045	0.146
HTN	0.740	0.208	2.626	0.641
COPD	2.321	1.479	5.63	0.531

IHD: Ischemic heart disease; DM: Diabetes mellitus; HTN: Hypertension;

Discussion

The risk of myocardial infarction has been shown to be proportional to the severity of acute respiratory infections. Since the emergence of SARS-CoV-2 in late 2019, COVID-19 has demonstrated significant cardiovascular implications, with acute viral pneumonia potentially resulting in various complications including heart failure, acute myocardial infarction, arrhythmia, and myocarditis.[13] Patients with cardiovascular involvement consistently demonstrate higher mortality rates compared to those without such involvement.

In our study, sociodemographic analysis revealed no significant differences between COVID-19 patients with and without pre-existing IHD regarding age, gender, educational status, and socioeconomic conditions (p>0.05). The mean age of patients with pre-existing IHD was 64.2±8.6 years, with male predominance (66.7%), which reflects the typical demographic profile seen in previous studies.[2]

Analysis of comorbidities showed significantly higher prevalence of diabetes mellitus (76.8% vs 41.2%, p=0.003), hypertension (86.6% vs 33.3%, p=0.002), dyslipidemia (68.3% vs 38.0%, p=0.001), and family history of IHD (65% vs 28.3%, p=0.001) in patients with pre-existing IHD. These findings align with the COVID-related Cardiometabolic Syndrome (CIRCS) described in recent literature, characterized by abnormal adiposity, dysglycemia, dyslipidemia, and hypertension.[14,15]

Laboratory parameters demonstrated significantly elevated levels of cardiac biomarkers, metabolic parameters, and adverse lipid profiles in IHD patients. ECG abnormalities, particularly ST-T changes (53.3% vs 6.6%, p<0.001), were more frequent in IHD patients. These findings correspond with the pathophysiological mechanism involving ACE-2 receptor binding and subsequent cardiovascular effects described bySouth et al.[16] Echocardiographic findings revealed significantly higher prevalence of cardiac dysfunction in IHD patients, including global hypokinesia and mild LV systolic dysfunction, suggesting direct myocardial involvement.

Treatment patterns showed significantly higher use of cardiovascular medications in IHD patients, including anti-lipid therapy and anti-platelets. This aligns with recent retrospective studies suggesting potential protective effects of certain cardiovascular medications like lipid lowering agents in COVID-19 patients with heart disease.[17] The present study found that COVID-19 patients with IHD required significantly more intensive care, with higher rates of HDU/ICU admission (71.6% vs 36.6%, p<0.001) and greater oxygen requirements. These findings correspond with Hasan et al.'s study, which identified cardiovascular disease as a significant risk factor for poor COVID-19 prognosis in Bangladesh.[18]

Most importantly, our study demonstrated significantly higher mortality rates in COVID-19 patients with pre-existing IHD, including increased in-hospital mortality (36.6% vs 10.0%) and total 30-day mortality (60.0% vs 16.6%). This aligns with previous findings showing increased cardiovascular complications in COVID-19 patients.[16,17] Logistic regression analysis confirmed IHD as an independent predictor for 30-day mortality (OR=7.25, 95% CI=2.17-24.23) after adjusting for other comorbidities. Notably, patients with moderate and severe LV dysfunction showed higher 30-day mortality compared to those with mild LV dysfunction (p<0.01), suggesting a correlation between pre-existing cardiac dysfunction and COVID-19 outcomes.

Limitations

The present study faced several methodological constraints that warrant consideration when interpreting the results. The single-center design at Dhaka Medical College Hospital may limit the generalized ability of findings to other healthcare settings or geographical regions. The relatively small sample size of 120 patients, while sufficient for preliminary analysis, may have impacted on the statistical power to detect subtle differences between groups or in subgroup analyses. Furthermore, the use of the purposive sampling technique rather than randomization could have introduced selection bias, potentially affecting the study's internal validity. These limitations underscore the need for larger, multicenter studies with randomized sampling to validate our findings.

Conclusion

This study demonstrated that COVID-19 patients with pre-existing IHD experienced significantly higher in-hospital mortality rates

and HDU/ICU admission compared to those without IHD. Multivariate logistic regression analysis confirmed IHD as an independent predictor for 30-day mortality in COVID-19 patients, even after adjusting for diabetes, dyslipidemia, hypertension, and chronic obstructive lung disease. While these findings largely align with previous international studies, larger multicenter research is warranted to further elucidate the impact of pre-existing IHD in COVID-19 patients and optimize clinical management strategies.

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