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The Impact of SARS-CoV-2 Virus on Acute Coronary Syndrome Patients Correlated with Clinical, Echocardiographic and Coronary Angiographic Outcome

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ORIGINAL ARTICLE

The Impact of SARS-CoV-2 Virus on Acute Coronary Syndrome Patients Correlated with Clinical, Echocardiographic and Coronary Angiographic Outcome

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Abstract

Background: **COVID-19 pandemic has led to many negative problems for all aspects of society. While respiratory symptoms are typically the most severe manifestations of COVID-19 infection, SARS-CoV-2 infection may be responsible for the diversity of potentially severe cardiovascular manifestations, mainly in studied cases with pre-existing cardiovascular conditions.**

Aim and objectives: **To evaluate the impact of SARS-CoV-2 virus on acute coronary syndrome patients and correlate it with clinical presentation, Echocardiography and coronary angiography outcome.**

Subjects and techniques: **This was case-control research, has been happened in the Department of Cardiology Al-Azhar University on 300 patients divided on three groups presented with the acute coronary syndrome, STEMI and NSTEMI. The first control group were presented only with STEMI. The second case under the study group was presented by STEMI and SARS-CoV-2 active infection. The last group were presented by 'NSTEMI or unstable angina' and SARS-CoV-2 active infection.**

Results: **Statistical analysis of 300 patients divided on three groups presented with acute coronary syndrome (STEMI, NSTEMI or unstable angina). That showed high variation among studied groups as regard EF (Ejection fraction), cardiogenic shock, respiratory failure and increased in-hospital mortality.**

Conclusion: **In studied cases infected with SARS-CoV-2, acute myocardial injury is related to in-hospital mortality and is an indicator of poor prognosis. careful indication of more invasive diagnostic tests and subsequent implementation of appropriate treatments without delay as time is muscle.**

Keywords: Acute coronary syndrome, Coronavirus, Covid-19

1. Introduction

Pandemic of Coronavirus disease 2019 is currently affecting 212 countries worldwide, with great morbidity and mortality rates.[1]

Individuals with underlying cardiovascular disease are at the greatest risk of severe disease and death, with fatality rate of 10.5%. Despite the fact that respiratory symptoms dominate clinical manifestations of COVID-19, some infected patients initially exhibit typical cardiovascular symptoms.[2]

The pathophysiology underlying cardiac involvement throughout COVID-19 infection is likely to be multifactorial more often. This is most likely because of coronavirus-related damage, which affects the heart other organs, causing parenchymal cell degeneration and necrosis.[3]

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Previous research has found greater rate of cardiogenic shock and in-hospital cardiac mortality in studied cases with SARS-CoV-2 infection and ACS (Acute Coronary Syndrome) during research period when compared to same time interval prior year. If studied cases delay and avoid treatment for life-threatening conditions as ACS due to pandemic fear, death rate can far exceed that directly related to SARS-CoV-2 infection.[4]

Aim of Work was to evaluate impact of SARS-CoV-2 virus on acute coronary syndrome patients and correlate it with clinical presentation, Echocardiography and coronary angiography outcome.

2. Studied cases and methods

This is cross sectional research which had been happened under supervision of Department of cardiology Al-Azhar University in collaboratin with Health Insurance Hospital. Study included 300 patients divided on three groups presented with acute coronary syndrome. First control group will presented only with STEMI. The second case under study group will present by STEMI and SARS-CoV-2 active infection. The last group had been present by 'NSTEMI or unstable angina' and SARS-CoV-2 active infection. Then all studied cases was matched for age and sex at a period of time.

2.1. Inclusion criteria

Studied cases more than eighteen years old who was presented with acute coronary syndrome STEMI, NSTEMI and unstable angina and SARS-CoV-2 infection.

2.2. Exclusion criteria

Stable coronary artery disease, systolic dysfunction (LVEF less than 40%), history of end-stage renal and liver disease, studied cases with active/chronic inflammatory disorder and declined informed consent.

2.3. Methods

2.3.1. History taking

History was performed, and each risk factor for coronary artery disease or previous events was assessed as hypertension, diabetes, cholesterol, dyslipidemia, smoking and BMI. The personal family history is also important to identify the genetic predisposition.

2.3.2. Clinical examination

General examination: All participants were subjected to thorough physical examination, which included evaluation of their overall health, as well as vital signs, such as blood pressure and heart rate. Heart examination on local level: Heart sounds, additional sounds such as S3 and S4, and cardiac murmurs are all possible and heart failure patients classified according killip classification.

2.3.3. Laboratory investigations

Complete blood picture, serum creatinine, liver enzymes, HS-CRP, D-dimer and cardiac biomarkers, including troponin and CKMB were measured for all patients. Five millimeter was drawn from each paient once.

2.3.4. Standard 12-leads ECG

Had been done within 10 min of 1st medical contact to detect ST segment and Twave abnormalities of acute coronary syndrome (Unstable angina, NSTEMI and STEMI).

2.3.5. Resting transthoracic echocardiography (TTE)

Done using standard echocardiographic views, EDD, ESD, PWD, IVSD, FS, LVEF, LA dimension and RV function by TAPSE. Measures protocol using 2D, M mode, modified simpson's rule and Regional wall motion score index (WMSI) based on the 16 segments LV model taken by two independent echo experts who were blind to the cases for all subjects according to ASE recommendations.

2.3.6. CT chest

All patient underwent CT chest to confirm the viral ground-glass pneumonia and the results will be interpreted by professional radiologist under supervision of Chest consultant who were blind to the cases. There is epidemiological history, as well as clinical symptoms (fever and dry cough). Main CT characteristics opacity, and lesion distribution are defined.

2.3.7. Coronary angiography

Professional interventionists who were blind to cases performed right and left coronary angiography using multiple projections. Fifty percent luminal diameter stenosis of at least 1 major epicardial coronary artery is described as angiographic CAD. Patients with acute coronary syndrome underwent coronary angiography for presenting with unstable angina with (severe chest pain not responding to medical therapy, serious ventricular arrhythmias), NSTEMI and STEMI.

Table 1. Comparison between studied groups as regard demographic data.

	STEMI group ($n = 100$)	STEMI and SARS-CoV-2 ($n = 100$)	NSTEMI and SARS-CoV-2 ($n = 100$)	test	*P*
Age					
Range	43–88	44–83	46–88	F = 0.593	0.553
Mean ± SD	64.96 ± 13.92	64.93 ± 11.14	66.58 ± 11.53		
Sex	No (%)	No (%)	No (%)		
Female	24 (24.0)	24 (24.0)	20 (29.0)	$\chi^2 = 0.609$	0.738
Male	76 (76.0)	76 (76.0)	80 (71.0)		
Smoking					
Current	50 (50.0)	40 (40.0)	40 (40.0)	$\chi^2 = 2.741$	0.602
Ex-smoker	16 (16.0)	20 (20.0)	20 (20.0)		
Non-smoker	34 (34.0)	40 (40.0)	40 (40.0)		
Hypertension					
No	43 (43.0)	35 (35.0)	45 (45.0)	$\chi^2 = 2.315$	0.314
Yes	57 (57.0)	65 (65.0)	55 (55.0)		
Dyslipidemia					
No	48 (48.0)	47 (47.0)	56 (56.0)	$\chi^2 = 1.947$	0.378
Yes	52 (52.0)	53 (53.0)	44 (44.0)		
Diabetes					
No	58 (58.0)	53 (53.0)	54 (54.0)	$\chi^2 = 0.566$	0.754
Yes	42 (42.0)	47 (47.0)	46 (46.0)		
Height					
Range	159–185	158–185	158–185	F = 0.009	0.991
Mean ± SD	172.1 ± 7.06	172.23 ± 7.52	172.2 ± 7.4		
BMI					
Range	28.2–37.1	28.1–36.9	28.4–37.1	F = 1.881	0.154
Mean ± SD	32.33 ± 2.83	32.37 ± 2.69	32.99 ± 2.54		

F, Oneway ANOVA test; χ^2, Chi-square test.
p: *P* value to compare among variance groups.
*Statistically significant at $P \leq 0.05$.
BMI, Body Mass Index; NSTEMI, non ST segment elevation myocardial infarction; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SD, standard deviation; STEMI, ST segment elevation myocardial infarction.

2.3.8. Follow up

All patients followed up for:

Acute (in-hospital) outcome including: Clinically: Duration of hospitalization, acute pulmonary edema, cardiogenic shock, arrhythmias and sudden cardiac death. Echocardiographic: Systolic dysfunction and mechanical complications of acute coronary syndrome. Coronary angiography outcomes: Revascularization success and in-stent thrombosis.

*Late (*3 months*) outcome including*: Clinically: Character and severity of chest pain, development of serious arrhythmias, stroke and re-infarction. Echocardiographic: Heart failure and regional wall motion abnormalities. Coronary angiography outcomes: In-stent restenosis and re-infarction.

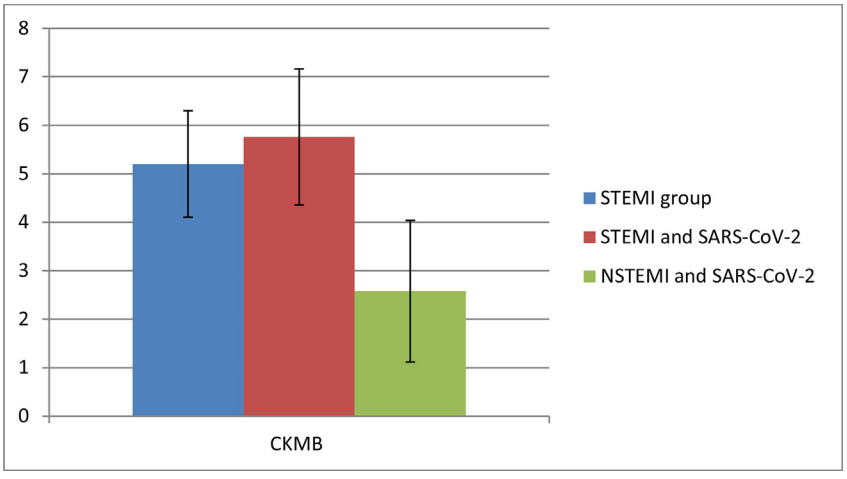

Fig. 1. Comparison between studied groups as regard CK-MB.

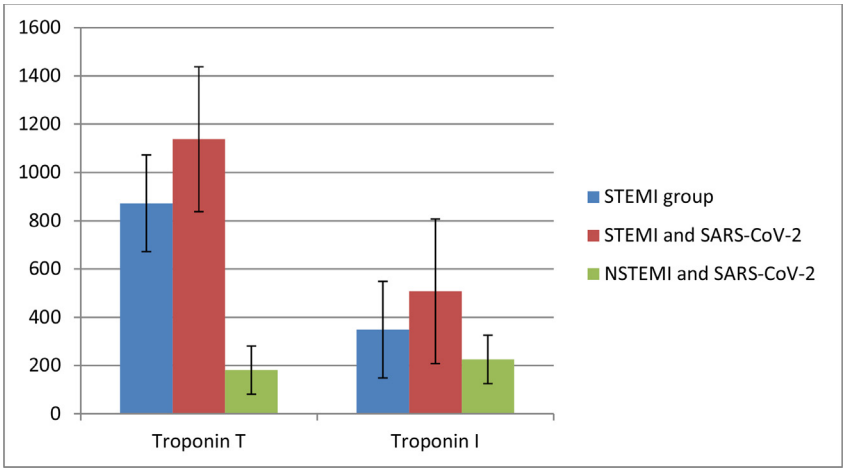

Fig. 2. Comparison between studied groups as regard troponin.

Ethical considerations: All of studied cases signed written informed consent forms. Every studied case was assigned unique file with code number. All investigations and research findings were only used for scientific purposes, with studied case privacy protected. Any unexpected risks that arose throughout course of study were promptly communicated to cases and ethical committee. All infection control procedures was considedrd to avoid the spread of the virus.

2.4. Data management and statistical analysis

Data entry, processing and statistical analysis had been happened using SPSS version 20 **(USA Statistical Package for Social Sciences).** The following tests of importance had been used: Kruskal-Wallis, Wilcoxon's, Chi square, logistic regression analysis, and Spearman's relationship. Data had been presented, and appropriate analysis had been performed based on type of data (parametric and nonparametric) achieved for each variable. *P* values of less than 0.05 (five percent) had been deemed statistically significant. *P* value: level of significance: $P > 0.05$ indicates nonsignificant, $P < 0.05$ indicates significant, and $P < 0.01$ indicates greatly significant.

2.4.1. Descriptive statistics

For parametric numerical data, mean, standard deviation, and range are used, while median and interquartile range are used for nonparametric numerical data, and frequency and percentage are used for non-numerical data.

2.4.2. Analytical statistics

Kruskal-Wallis test was used to determine statistical significance of nonparametric variable

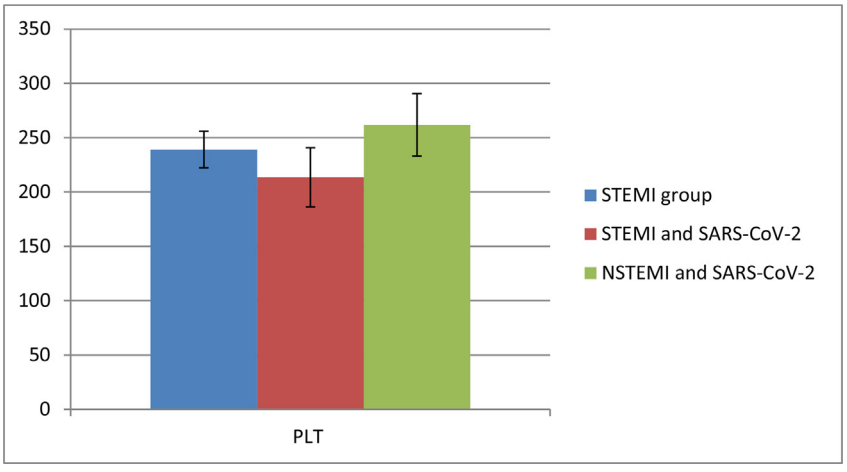

Fig. 3. Comparison between studied groups as regard Platelets.

variation among more than two study groups. 1 –way ANOVA for normally distributed continuous variables. Following ANOVA, post hoc had been conducted using Tukey test, followed by post hoc analysis using Mann-Whitney *U* test.

3. Results

Our research had been case-control study, on 300 patients divided equally on three groups presented with acute coronary syndrome. First control group were presented only with STEMI. The second case under study group was presented by STEMI and SARS-CoV-2 active infection. Last group were presented by 'NSTEMI or unstable angina' and SARS-CoV-2 active infection. our results had been as following.

This table finds that there had been no statistically difference among studied groups as regard demographic data Table 1.

There had been high differences among studied groups as regard CKMB, Troponin T and I, TLC and Platelets (Figs. 1–3).

There had been high statistically differences among studied groups as regard sounds and statistically significant difference as regard chest pain (Fig. 4).

There had been high statistical differences among the studied groups as regard ST segment and EF Table 2.

There had been no variation among the covid studied groups as regard Symptoms of COVID-19 (Fig. 5).

There had been great difference among studied groups as regard Killip classification, in hospitable stay, cardiogenic shock and mechanical ventilation and statistically difference as regard acute pulmonary edema and arrhythmias Table 3.

This table finds that there had been high statistically differences among studied groups as regard EF and chest pain Table 4.

4. Discussion

Despite fact that respiratory symptoms predominate in all clinical manifestations of Covid-19, preliminary research suggests that some of studied cases can develop severe cardiovascular damage, although others with underlying CV diseases may be at higher risk for death.[5]

Screening and elective therapies for coronary artery disease are underestimated in context of overburdened healthcare system, implying that dealing with acute coronary syndromes has become more complicated and appears to be less common. ACS remains major reason for morbidity and mortality worldwide, accounting for more than one million hospital admissions in United States each year, whereas ischemic heart disease accounts for nearly 1.8 million annual deaths, and 20% of all deaths in Europe.[6]

In current research we found that there had been high variation among studied groups as regard CKMB, Troponin T, I, TLC and Platelets.

In agreement with our research, Shi et al.[7] illustrated that Serum troponin levels showed positive linear relationship with *C*-reactive protein and NT-pro BNP levels in retrospective research of 187 studied cases hospitalized with COVID-19, indicating important relation among myocardial injury and ventricular stress with systemic inflammation, which is primarily present in more severe stages of disease. As result, with $P < 0.001$, these results suggest that troponin is important prognostic indicator in studied cases infected withSARS-CoV-2.

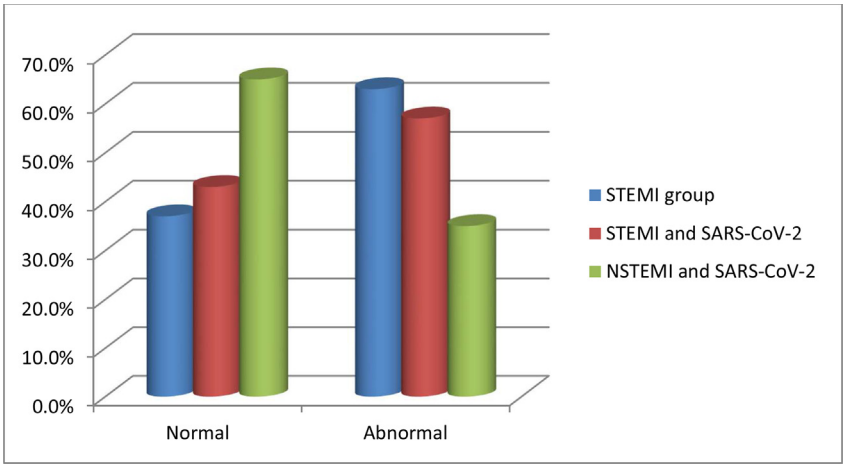

Fig. 4. Comparison between studied groups as regard Heart sounds.

Table 2. Comparison between studied groups as regard ECG and Echo.

	STEMI group (n = 100) No. (%)	STEMI and SARS-CoV-2 (n = 100) No. (%)	NSTEMI and SARS-CoV-2 (n = 100) No. (%)	Test	*P*
ST segment					
Normal	7 (7.0)	5 (5.0)	10 (10.0)	$\chi^2 = 275.759$	<0.001[a]
Depressed	0 (0.0)	0 (0.0)	90 (90.0)		
Elevated	93 (93.0	95 (95.0)	0 (0.0)		
Post-Hoc	*P*1 = 0.552, *P*2<0.001[a], *P*3<0.001[a]				
EF %					
Range	37–42	37–42	45.3–52	F = 903.551	<0.001[a]
Mean ± SD	39.52 ± 1.56	39.4 ± 1.53	48.29 ± 1.96		
Post-Hoc	*P*1 = 0.611, *P*2<0.001[a], *P*3<0.001[a]				
LVEDD					
Range	39.3–68.3	39.3–67.7	39.4–68	F = 0.355	0.702
Mean ± SD	53.19 ± 8.2	52.54 ± 8.73	52.49 ± 8.25		
LVESD					
Range	23.3–60.5	23.1–60.5	23.1–60.5	F = 0.566	0.569
Mean ± SD	41.96 ± 10.23	42.27 ± 11.01	41.02 ± 11.36		
RMWA	No. (%)	No. (%)	No. (%)		
No	1 (1.0)	3 (3.0)	2 (2.0)	$\chi^2 = 1.020$	0.600
Yes	99 (99.0)	97 (97.0)	98 (98.0)		
RV function					
Good	90 (90.0)	82 (82.0)	78 (78.0)	$\chi^2 = 5.375$	0.068
Reduced	10 (10.0)	18 (18.0)	22 (22.0)		
MR					
I	70 (70.0)	68 (68.0)	70 (70.0)	$\chi^2 = 6.902$	0.330
II	20 (20.0)	25 (25.0)	15 (15.0)		
III	8 (8.0)	4 (4.0)	9 (9.0)		
IV	2 (2.0)	3 (3.0)	6 (6.0)		

F, Oneway ANOVA test; χ2, Chi-square test.
p: *P* value to compare among variance categories.
*P*1: *P* value to compare among STEMI group and STEMI and SARS-CoV-2.
*P*2: *P* value to compare among STEMI group and NSTEMI and SARS-CoV-2.
*P*3: *P* value to compare among STEMI andSARS-CoV-2 group and NSTEMI andSARS-CoV-2.
LVEDD, left ventricular end-diastolic diameter.
[a] Statistically significant at P ≤ 0.05.

Lippi *et al.*[8] found that analysis of 341 studied cases linked myocardial injury to severeCOVID-19infection because studied cases with great troponin serum levels needed more intensive care.

In current research we found that there had been high variation among studied groups as regard (ST segment, EF, LVEDD, LVESD, RMWA, RV function and MR).

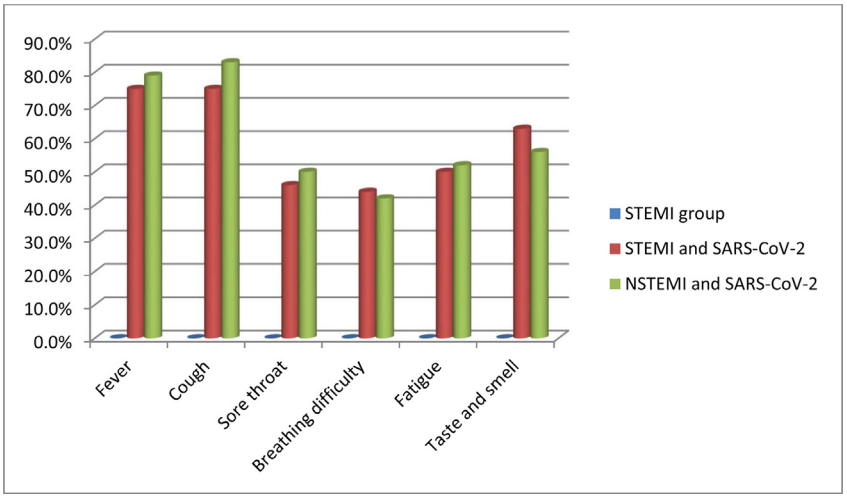

Fig. 5. Comparison between studied groups as regarding symptoms of COVID-19.

Table 3. Comparison between studied groups as regard early clinical follow up.

	STEMI group ($n = 100$)	STEMI and SARS-CoV-2 ($n = 100$)	NSTEMI and SARS-CoV-2 ($n = 100$)	Test	*P*
In hospitable stay					
Range	6–18	6–18	4–14	F = 20.986	<0.001[a]
Mean ± SD	11.97 ± 3.83	12.01 ± 3.84	9.13 ± 3.1		
Post-Hoc	*P*1 = 0.938, *P*2<0.001[a], *P*3<0.001[a]				
	No. (%)	No. (%)	No. (%)		
acute pulmonary edema	14 (14.0)	34 (34.0)	23 (23.0)	$\chi^2 = 11.108$	0.004[a]
Post-Hoc	*P*1<0.001[a], *P*2 = 0.101, *P*3 = 0.085				
cardiogenic shock	7 (7.0)	27 (27.0)	26 (26.0)	$\chi^2 = 15.875$	<0.001[a]
Post-Hoc	*P*1<0.001[a], *P*2<0.001[a], *P*3 = 0.873				
Arrhythmias	13 (13.0)	24 (24.0)	11 (11.0)	$\chi^2 = 7.292$	0.026[a]
Post-Hoc	p1 = 0.045[a], p2 = 0.663, p3 = 0.016[a]				
Sudden cardiac death	13 (13.0)	18 (18.0)	14 (14.0)	$\chi^2 = 1.098$	0.578
Cardiac	2 (15.4)	3 (16.7)	2 (14.3)	$\chi^2 = 0.245$	0.993
Covid (chest)	10 (76.9)	13 (72.2)	11 (84.6)		
Others	1 (7.7)	2 (11.1)	1 (7.7)		
Acute heart failure	16 (16.0)	20 (20.0)	16 (16.0)	$\chi^2 = 0.744$	0.689
mechanical ventilation	4 (4.0)	24 (24.0)	19 (19.0)	$\chi^2 = 16.399$	<0.001[a]
Post-Hoc	*P*1<0.001[a], *P*2<0.001[a], *P*3 = 0.390				
Killip classification					
I	63 (63.0)	19 (19.0)	35 (35.0)	47.23	<0.001[a]
II	16 (16.0)	20 (20.0)	16 (16.0)		
III	14 (14.0)	34 (34.0)	23 (23.0)		
IV	7 (7.0)	27 (27.0)	26 (26.0)		
Post-Hoc	*P*1<0.001[a], *P*2<0.001[a], *P*3 = 0.062				

F, One way ANOVA test; χ^2, Chi-square test.
p: *P* value to compare among variance categories.
*P*1: *P* value to compare among STEMI group and STEMI and SARS-CoV-2.
*P*2: *P* value to compare among STEMI group and NSTEMI and SARS-CoV-2.
*P*3: *P* value to compare among STEMI andSARS-CoV-2 group and NSTEMI andSARS-CoV-2.
[a] Statistically significant at P ≤ 0.05.

In agreement with our research, Stefanini *et al.*[9] showed that In case series, 85.7% of studied cases had signs of infarction with STE as 1st symptomatic manifestation ofCOVID-19, while 39.3% had no evidence of obstructive disease. Studied case had STE, although it was not 1st symptom, and coronary artery angiography revealed no occlusion.

In alignment with our study, Schiavone *et al.*[10] noted that acute myocardial injury, as evidenced by STE and elevated troponin, could lead to one of

Table 4. Comparison between studied groups as regard late clinical and echo follow up.

	STEMI group ($n = 73$) No. (%)	STEMI and SARS-CoV-2 ($n = 67$) No. (%)	NSTEMI and SARS-CoV-2 ($n = 65$) No. (%)	Test	*P*
Chest pain	8 (11.0)	13 (19.4)	20 (30.8)	$\chi^2 = 8.456$	0.015[a]
Post-Hoc	*P*1 = 0.162, *P*2 = 0.004[a], *P*3 = 0.132				
Development of stroke	1 (1.4)	3 (4.5)	2 (3.1)	$\chi^2 = 1.195$	0.550
re-infarction	3 (4.1)	8 (11.9)	6 (9.2)	$\chi^2 = 2.927$	0.231
Heart failure	3 (4.1)	2 (3.0)	6 (9.2)	$\chi^2 = 2.887$	0.236
regional wall motion abnormalities	9 (12.3)	13 (19.4)	11 (16.9)	$\chi^2 = 1.342$	0.511
EF %					
Range	35–40	36–42	42.3–51	F = 567.432	<0.001[a]
Mean ± SD	38.41 ± 1.56	37.22 ± 1.53	46.31 ± 1.96		
Post-Hoc	*P*1<0.001[a], *P*2<0.001[a], *P*3<0.001[a]				
MR					
I	50 (68.5)	43 (64.2)	44 (67.7)	$\chi^2 = 4.426$	0.619
II	16 (21.9)	18 (26.9)	11 (16.9)		
III	6 (8.2)	4 (6.0)	6 (9.2)		
IV	1 (1.4)	2 (3.0)	4 (6.2)		

F, One way ANOVA test; χ^2, Chi-square test.
p: *P* value to compare among different categories.
EF, Ejection Fraction.
[a] Statistically significant at P ≤ 0.05.

many hypotheses: virus causing direct myocardial injury. Even so, MRI did not reveal any signs of mesocratic fibrosis, edoema, and necrosis, which contradicts former hypothesis. Due to late diagnosis and fact that coronary artery angiography revealed no thrombi and atherosclerotic procedure, another possibility is thrombosis with spontaneous lysis and microvascular injury - as hypercoagulability seen in COVID-19 in pro-inflammatory state predisposes to acute coronary events.

In present research we found that there had been high variation among studied groups as regard Killip classification, in hospitable stay, cardiogenic shock, mechanical ventilation, statistically difference as regard acute pulmonary edema and arrhythmias.

In alignment with our study, Puelles *et al.*[11] described thatSARS-CoV-2 RNA had been discovered in lungs at great concentrations by quantitative reverse transcription PCR in all twelve studied cases with COVID-19, and five studied cases had great viral RNA in heart, liver, and kidney. Likewise, SARS-CoV-2RNAwas found in lungs, heart, brain, liver, and kidneys of twenty seven studied cases who died from COVID-19, revealing that SARS-CoV-2has broad organ tropism.

Research by, **Paranjpe** *et al.*[12] who illustrated that variations in median survival time and mortality were more pronounced between mechanically ventilated studied cases (twenty one days versus nine days and 29.1% versus 62.7%).

Goyal *et al.*[13] stated that Among 393 consecutive COVID-19 studied cases hospitalized in New York, USA, up to fifty percent had hypertension (54% of ventilated studied cases), 36% had obesity (43% of ventilated studied cases), 25% had diabetes (28% of ventilated studied cases), and 14% had coronary artery disease (19% of ventilated studied cases).

Hoffmann *et al.*[14] showed that extra pulmonary manifestations and multiorgan failure noted in COVID-19 studied cases are not only the result of systemic inflammation and cytokine storm though may be the result of SARS-CoV-2 infection of multiple organ systems. Broad tissue tropism ofSARS-CoV-2could be explained by instability of S protein of SARS-CoV-2due to existence of novel furin cleavage site.

In present research we found that there had been high variation among studied groups as regard (EF, chest pain, Development of stroke, re-infarction, Heart failure, MR, and regional wall motion abnormalities).

In agreement with our research, Imazio *et al.*[15] stated that COVID-19 can cause acute myocardial injury because of myocardial ischemia and non-ischemic process. In up to 23% of studied cases, injury is linked to more severe disease conditions, like advancement of HF. nine According to researches conducted in China, up to seventeen percent of COVID studied cases had increased troponin levels.

Chen *et al.*,[16] reported that Heart failure was 1 of most commonly detected COVID-19 problems, with described incidence of 24% in all studied cases and 49% in studied cases who died.

Guan *et al.*[17] showed that In research of 1099 studied cases withCOVID-19 from mainland China, 24% had any comorbidity (39% were critically ill), fifteen percent had hypertension (24% were critically ill), 7% had diabetes (16% were critically ill), and 3% had coronary heart disease (6% were critically ill).

4.1. Conclusion

Patients with Acute coronary syndrome are related to in-hospital mortality, and indicator of worse prognosis in studied cases infected with SARS-CoV-2. Risk stratification and pretest probability evaluation are critical for improving diagnostic accuracy of COVID-19 cardiovascular problems, with as accurate differences of myocarditis from acute coronary syndrome as possible, careful sign of invasive diagnostic tests, and subsequent implementation of appropriate treatments.

Disclosure

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Conflicts of interest

The authors declared that there were NO conflicts of Interest.

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