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Association between COVID-19 Diagnosis and In-Hospital Mortality of Hospitalized patients with ST-Segment ElevationMyocardial Infarction

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ABSTRACT

Introduction: The 2019 coronavirus disease pandemic (Covid-19) is the worst global health crisis right now. The clinical course of covid-19 is often accompanied by hypercoagulation state, which means that COVID-19 patients could develop a number of cardiac conditions, such as myocarditis, stress-induced cardiomyopathy, etc. The possibility of morbidity as well as mortality is increased among individuals with COVID-19 who have ST-elevation myocardial infarction (STEMI) in comparison with patients without the virus who are the same gender and age.

Subjects and methods: We collected data from records in the form of personal history, comorbidities and laboratory studies including: complete blood picture (CBC), coagulation profile, D-Dimer, serum biochemical tests including cardiac biomarkers and ferritin, rapid test or PCR, computed tomography (CT), ECG results and echocardiography results if found. We intended to find the association among COVID-19 and STEMI outcomes on patient presented to Emergency Department at Suez Canal University.

Results: In-hospital mortality rates were 34.04% for patients with STEMI along with COVID-19 compared to 4.3% for those with STEMI without COVID-19. COVID 19 patients had leukocytosis and lymphocytopeni. Moreover, COVID 19 patients had elevated d-dimer, TLC and CRP.

Conclusion: When compared to patients who were not diagnosed with COVID-19, individuals with STEMI had significantly greater rates of in-hospital mortality when they also had a concurrent out-of-hospital or in-hospital COVID-19 infection.

Keywords: COVID-19, STEMI, Mortality.

INTRODUCTION

Over 20.9 million people have been affected and 7,46,133 have died as of August 15, 2020, due to the 2019 coronavirus disease pandemic (COVID-19), that has been triggered by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [(1)]. A portion of the 2019 global coronavirus disease pandemic (COVID-19), caused by SARS-CoV-2, was the virus that infected Egypt. On 14 February 2020, the virus was verified to have entered Egypt [(2)]. In Egypt from 3 January 2020 to April 2023, 516023 confirmed Coronavirus Cases and 24830 coronavirus deaths have been reported since the epidemic began, based on the World Health Organization (WHO) [(3)].

Wuhan, Hubei Provence, China was the site of the initial detection of this novel respiratory infectious illness in December 2019 [(4)]. Infected aerosols or droplets carrying the COVID-19 virus can be inhaled or become into direct touch with the nasal passages, mouth, or eyes, and then transmitted from an infected individual to a healthy one who is in close proximity to them. Symptoms of COVID19 can vary from person to person and may be also affected by different age groups or underlying comorbidities. The symptoms may take up to 14 days to appear after exposure and vary from asymptomatic to mild different symptoms [(5)].

Frequent pulmonary symptoms of COVID-19 include dry cough, rhinorrhea, dyspnea, and fever. Concerns about disease control on a global scale have prompted major research into finding an effective treatment for this enigmatic virus, due to the disease's high spread and rapid deterioration seen in patients of all ages. In addition to pulmonary symptoms, thrombosis caused by COVID-19 is common and can impact the body's arterial as well as venous systems [(6)]. Among the worst catastrophes that humanity has faced in the previous several decades is the devastating impact of the 2019 coronavirus disease (COVID-19) on healthcare systems and economy around the world. **Raman *et al.*** [(7)] estimate that the number of COVID-19 survivors globally now exceeds hundreds of millions.

Cardiovascular complications such as acute coronary syndromes, stress-induced cardiomyopathy, as well as myocarditis can develop in COVID-19 patients. There is an increased risk of morbidity and mortality for people with COVID-19 who have STEMI compared to patients without the virus who are age- and gender-matched [(8)]. Research groups are now interested in defining the long-term cardiovascular effects of SARS-CoV-2 infection due to the significant mortality and unfavorable outcomes linked to myocardial injury throughout acute COVID-19 infection [(7)]. Acute coronary syndromes (ACS), arrhythmias, as well as direct myocardial damage are all known to be brought on by the SARS-CoV-2 virus. The way the virus uses to enter cells is assumed to be the cause. The virus uses the angiotensin-converting enzyme 2 receptor, which is abundantly expressed in cardiovascular organs, to enter cells.

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Therefore, a direct cytotoxic impact of the virus on cardiomyocytes is supported by direct viral engagement through these receptors [(9)].

The precise causes of the extra mortality from ischemic heart disease throughout the pandemic are still unknown, despite the identification of numerous likely and probable factors. For example, an abundance of information already exists demonstrating how patients with newly diagnosed or pre-existing cardiovascular conditions have encountered gaps and delays in treatment, particularly during COVID-19 peak periods [(10)]. Furthermore, an increasing amount of evidence indicates that some COVID-19-affected patients have an elevated risk of thrombogenic events, such as acute coronary events, during as well as after the acute infection phase [(11)]. Another possibility is that the increased cardiovascular risks seen in the early stages of the pandemic were linked to COVID-19 infections that were typically more severe and were brought on by more virulent strains of SARS-CoV-2 [(10)].

The goal of the research was to identify and comprehend the relationship among COVID-19 and STEMI outcomes.

STUDY DESIGN AND METHODS

This study was carried out as a retrospective cohort study at Suez CanalUniversity Hospital, Ismailia City, Egypt.

Population and sample: All patients 18 years or older confirmed with STEMI presented at Emergency Department at Suez Canal University Hospital with COVID-19 or patient admitted with COVID-19 developed STEMI with the following inclusion and exclusion criteria.

Inclusion criteria: Patients with cardiac chest pain who were hospitalized with STEMI at apercutaneous coronary intervention and had or not COVID-19. Age 18 years or older. Both males and females.

Exclusion criteria: Patients less than 18 years old. After cardiac arrest. Patients who refused to share the study and patients who had STEMI in the past 30 days

Data collection:

- Patients admitted by reviewing the patients' retrospective data on records from January 2020 to June 2022.
- Patients were distributed into 2 groups: group 1 (STEMI patients with COVID-19) and group 2 (STEMI patients without COVID-19).
- ***Data collection included:***
  1. **Patient demographics:** Age, gender, ethnicity, height and weight.
  2. **Comorbidities:** History of chronic illness (chronic asthma, diabetes, hypertension, chronic liver and kidney disease, thyroid disease, malignancy, bleeding disorders and cardiac arrhythmia or congenital heart)
  3. **Drug history.**
  4. **Laboratory tests** (Complete blood count-coagulation profile and D-Dimer- CRP- CK, CK-MB, Troponin I, LDH, serum ferritin, serum sodium, serum potassium, serum creatinine and rapid test or PCR).
  5. **Radiological investigations:** CT chest
  6. **ECG results and echocardiography results if found.**
  7. **Management.**
  - In-hospital medicine and diagnosis; therapies (invasive mechanical ventilation, fibrinolytic therapy, PCI, vasopressor utilization), and outcomes (length of stay, effectiveness of revascularization, reinfarction while hospitalized, and mortality).
- Based on institutional standards, the treating interventional cardiologist at every site had discretion over the first reperfusion technique, which might have been fibrinolytic treatment or primary PCI.
- Following 6 months of follow-up, patients were either sent home or returned for invasive revascularization if the procedure was successful.
- The confidentiality of data was protected by de-anonymization of records and limiting access to data to the principal investigator only.

Ethical approval:

The study was approved from ethical committee of Emergency Medicine Department, faculty of Medicine, Suez Canal University. An informed written consent was obtained from all patients.

Statistical analysis

Data tested for normality using Shapiro-Wilk test. Continuous, normally distributed variables were presented as mean ± SD depending on their distribution and non- normally distributed variables as median and interquartile range. Statistical differences in variables were compared using a one-way ANOVA for variables of normal distribution followed by the Bonferroni post hoc test. Categorical variables are presented as frequencies and or percentages, and inter- group comparisons were analyzed using the chi-square test. Multivariate regression analysis was used to estimate the relationships between dependent variables and two or more independent variables (predictor variable), which incorporated demographic, clinical, and facility characteristics used to develop a propensity score on which those without COVID-19 were matched to those with COVID-19. Pearson correlation analysis was used to examine the correlations of biochemical markers and the

outcome of the infection with COVID-19 in myocardial infarction patients. Statistical significance was accepted if $P \leq 0.05$. All statistical analyses were performed using the statistical package for social science (SPSS) program version 25.0.0. 0. Only the main effects entered in the models.

RESULTS

This study was a retrospective cohort study that was conducted on 94 patients presented to Emergency Department Suez Canal University Hospital through reviewing the patients' retrospective data on records from January 2020 to June 2022. Patients were diagnosed with STEMI with or without COVID-19 infection and fulfilled the inclusion criteria. Table (1) described the demographic characteristics of the study group. Both groups were matched in age and sex. The mean age of the MI patients was 54.44 ± 13.18 years, while the mean age of the MI patients infected with COVID-19 was 57.21 ± 12.16 years. The MI group involved 36 males (76.6%) and 11 females (23.4%), whereas the MI patients infected with COVID-19 involved 32 males (68.1%) as well as 15 females (31.9%) that revealed male predominance in both groups.

Table (1): Demographic data of the study groups

All patients (n=94)		MI patients (n=47)	MI patients infected with COVID-19 (n=47)	*P* value
Mean age ± SD (years)		54.44±13.18	57.21 ± 12.16	0.287*
Gender Number (%)	**Male**	36 (76.6 %)	32 (68.1 %)	0.256^
	Female	11 (23.4%)	15 (31.9 %)	

*Student's T test. ^Chi^2 test, SD: standard deviation. The threshold of significance was if $p \leq 0.05$.

Table (2) demonstrated the symptoms reported by the MI patients, chest pain was found in 30 (63.8%) of cases, dyspnea in 26 (55.3%), dizziness in 25 (53.19%), palpitations in 15 (31.9%), fainting in 11 (23.4%), fever in 10 (21.27%), headache in10 (21.27%) and cough in 5 (10.6%).

Table (2): Symptoms of the MI group (n=47)

Symptoms	**Frequency**	**Percentage**
Chest pain	30	63.8%
Dyspnea	26	55.3%
Palpitations	15	31.9%
Fever	10	21.27%
Cough	5	10.6%
Headache	10	21.27%
Fainting	11	23.4%
Dizziness	25	53.19%

Data were expressed as percentage and frequency.

Table (3) demonstrated the symptoms reported by the MI patients infected with COVID-19, dyspnea was found in 38 (80.8%), fever in 32 (68.08%), bone aches in 30 (63.82%), headaches in 29 (61.7%), chest pain in 27 (57.44%), fatigue in 25(53.19%), fainting in 22 (46.8%), palpitations in 21 (44.68%), cough in 20 (42.55%), sore throat in 15 (31.91%), diarrhea in 11 (23.4%) and dizziness in 8 (17.02%).

Table (3): Symptoms of the STEMI with COVID-19 group (n=47)

Symptoms	**Frequency**	**Percentage**
Chest pain	27	57.44%
Dyspnea	38	80.8%
Palpitations	21	44.68%
Fever	32	68.08%
Cough	20	42.55%
Headache	29	61.7%
Bone Aches	30	63.82%
Fainting	22	46.8%
Dizziness	8	17.02%
Sore throat	15	31.91%
Fatigue	25	53.19%
Diarrhea	11	23.4%

Data were expressed as percentage and frequency.

All of the cardiac indicators utilized to diagnose MI were not significantly different between the two groups (Table 4).

Table (4): Cardiac markers of both studied groups (n=94) according to presence of MI

Cardiac markers	**MI patients (n=47)**	**MI patients infected with COVID-19 (n=47)**	**P value**
CK (U/L)	1459.1 ± 173.17	1510.54 ± 160.19	.138* ^
CK-MB (U/L)	151.56 ± 17.21	153.54 ± 19. 41	.6*^
Troponin I (U/L)	23.32 ± 5.2	25.40 ± 5.10	.053* ^
LDH (U/L)	560.60 ± 33.99	571.79 ± 31.65	.10*^

Data were expressed as mean and standard deviation, median, range. *One way ANOVA test. ^Kruskall-Wallis test.SD: standard deviation. The threshold of significance was if $p<0.05$. CK: creatinine kinase. LDH: lactate dehydrogenase.

Table (5) explained the hematological parameters among the study groups, there were a statistically significant difference in total leukocytes count, absolute lymphocytic count, D-dimer and CRP among the study

groups with a p value < 0.01 and there were non-statistically significance in Hb, Platelets, PT and INR.

Table (5): Hematological tests among the study groups (n=94)

Tests Mean ± SD	MI patients (n=47)	MI patients infected with COVID-19 (n=47)	P value
Hemoglobin(gm/dl)	13.8560 ± 1.76435	13.5938 ± 2.25033	0.527*
Platelets*10^3/ul	238.5625 ± 9.74999	229.0625 ± 8.25915	0.602*
TLC *10^3/ul	7.49 ±1.58	16.61 ± 2.36	<0.01*
Absolute lymphocytes	2612.36 ± 452.53	530.5625 ± 55.4489	**<0.01***
D-dimer (µg/ml)	0.2608 ± 0.157	2.3490 ± 0.679	**<0.01***
CRP (mg/L)	3.47 ± 0.34	21.2917 ± 2.22825	**<0.01***
PT	13.33 ± 1.3	13.38 ± 1.27	0.543
INR	1.17 ± 0.7	1.22 ± 0.30	0.145
Data is expressed as mean and standard deviation. P is significant when < 0.05.			

*One way ANOVA test. SD: standard deviation
The threshold of significance was if p<0.05. TLC: total leucocytes count CRP: C - reactive protein.

Table (6) demonstrated confirmatory diagnostic tools for COVID-19 infection. PCR only was done in 4 (8.51%) and combined PCR + CT –chest was done in 43 (91.48%).

Table (6): Confirmatory tests for COVID-19 Infection (n=47)

confirmatory tests	Frequency	Percentage
PCR	**4**	**8.51%**
PCR+CT	**43**	**91.48%**

Data were expressed as percentage and frequency.PCR: polymerase chain reaction CT: computed tomography.

Figure (1) showed that in COVID-19 patients, 28 patients (59.57%) were CORAD 4 while only 19 patients (40.4%) were CORAD 5.

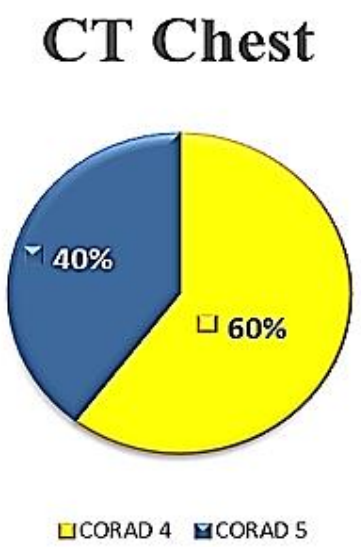

Figure (1): CT findings of the chest.

Table (7) illustrated that in MI patients without COVID-19 infection, 45 patients (95.7%) are discharged and only 2 patients (4.3%) were died in comparison with the MI patients infected with COVID-19 with 31 patients (65.9%) were discharged and 16 (34.1%) patients were died, which revealed that COVID-19 infection in MI associated with increased mortality rate among MI patients presented to the hospital.

Table (7): Fate of the myocardial infarction patients in relation to the infection with COVID-19

All patients (n=94)		MI patients (n=47)	MI patients infected with COVID-19 (n=47)
Patient fate (%)	**Discharged**	45 (95.7%)	31 (65.9 %)
	Died	2 (4.3%)	16 (34.04 %)

Data was expressed as percentage and frequency.

As demonstrated in table (8), Pearson correlation analysis revealed that COVID-19 infection in MI was negatively correlated with the outcome (r =-0.577) (p≤0.01), while it was positively correlated to TLC (r=0.917), absolute lymphocytic count (r=0.671), CRP (r=0.718) and D- dimer (r=0.662) with a p < 0.01. While, there was no statistically significant correlation to CK, CK-MB, Troponin I or LDH.

Table (8): Correlation between the biochemical markers and the outcome to the infection with COVID-19 In myocardial infarction patients

	COVID 19 in MI patients	
	Correlation coefficient	P value
CK	.060	.560*
CK-MB	.074	.476*
Troponin I	.043	.678*
LDH	.072	.487*
TLC	**0.917**	**.001***
Absolute lymphocytic count	**0.671**	**.001***
CRP	**0.718**	**.001***
D-dimer	**0.662**	**.001***
Outcome	**-0.577**	**.001***

*Pearson correlation The threshold of significance was if p<0.05. CK: creatinine kinase LDH: lactate dehydrogenase TLC: total leucocytes count CRP: c-reactive protein.

Table (9) showed the predictors for the existence of COVID-19 infection in the STEMI group, the results revealed that increase in TLC, D-dimer and CRP are risk factors to the presence of COVID-19 infection in MI patients.

Table (9): Multivariate regression analysis of COVID-19 infection

Independent variables	B	Std. Error	Sig.	95% Confidence Interval	
				Lower Bound	Upper Bound
TLC	9.094	.414	**.000**	8.272	9.917
D dimer	2.089	.247	**.000**	1.599	2.578
CRP	17.851	1.794	**.000**	14.288	21.414
Dependent factor: infection with COVID 19 in MI patients					

Statistical significance was accepted if $p \leq 0.05$. TLC: total leucocytes count CRP: C - reactive protein.

DISCUSSION

The new SARS-CoV-2 is the primary cause of the 2019 coronavirus disease pandemic, which is causing a great deal of morbidity and mortality around the world. Biomarker indications of myocardial injury is evident in a large percentage of hospitalized patients with COVID-19 infection. This finding is related with a higher probability of in-hospital morbidity as well as mortality [12]. According to **Vogel *et al.*** [13], STEMI is among the most dangerous cardiovascular emergencies since it is the most immediate sign of ischemic heart disease.

Enhanced hypercoagulability in patients having STEMI and COVID-19 can lead to a heavy thrombus burden, which in turn can cause unsatisfactory outcomes during PPCI owing to slow flow or no reflow, necessitating new methods of treatment [14]. Before epidemics, like SARS triggered by SARS-CoV-1 as well as Middle East Respiratory Syndrome, previously proposed the possibility of a link among respiratory diseases along with myocardial infarction (MI), as the infected had a greater probability of adverse cardiovascular complications, such as MI and death [15].

The mean age of the participants in the current study was 57.21 ± 12.16 years for the COVID-19 group and 54.44 ± 13.18 years for the non-COVID-19 group, however there was no statistically significant difference among the two groups (p=0.287). There was no significant difference among the two groups, according to **Markson *et al.*** [16], which indicated that the average age of hospitalizations was 65.7 ± 0.8 years for COVID-19 patients and 66.5 ± 0.1 years for patients without the virus. There was also male gender predominance in both groups (p=0.256) without a statistically significant difference between both groups. **Akhtar *et al.*** [17] also reported male predominance among patients with or without infection.

In this study regarding the medical history and risk factors comparing STEMI patients having Covid-19 infection, there were a greater medical history of hypertension, diabetes mellitus, chronic liver disease and chronic renal disease than the non-infected group and had a lesser frequency of dyslipidemia, smoking and obesity than in the non-infected group. The frequency of cardiovascular disease was similar in both groups. **Markson *et al.*** [16] found that individuals with MI who did not have COVID-19 were less likely to have dyslipidemia (64.6 % vs. 70.4%, p < 0.0001), smoking (23.5 % vs. 28.2%, p < 0.0001), and hypertension (37.1 % vs. 40.1%, p = 0.004). Both AMI patients with and without concurrent COVID-19 had similar levels of obesity as well as chronic liver disease.

In the current study regarding the symptoms, STEMI patients having Covid-19 infection had presentation mainly in the form of Dyspnea, fever, bone ache and headache rather than in non- infected patients who mainly were presented with the classical symptoms of chest pain, dyspnea, dizziness and palpitations. Concerning clinical presentation, a sample of 230 patients who tested positive for COVID-19 and had a STEMI were more likely to experience unusual symptoms like dyspnea and syncope than chest pain, according to the NACMI

(North American COVID-19 Myocardial Infarction) registry. This group had been compared to a historical control group [18].

In this study, no statistically significant difference were detected among both groups regarding any of the laboratory investigations in the form of random blood sugar, serum creatinine, and serum electrolytes. There were also no statistically significant difference among both groups regarding any of the cardiac markers used for diagnosis of MI and no difference in both groups in Hemoglobin level and platelet counts, PT and INR.

In our study, a statistically significant differences were observed in total leukocytes count, absolute lymphocytic count, D-dimer and CRP among the study groups with a p value <0.01. There were also increased TLC, reduced absolute lymphocyte count and significantly improved both D- dimer as well as CRP among Covid-19 infected patients. Another study showed that 26% of the patients had leukopenia, 13.3% had leukocytosis, as well as 62.5% had lymphopenia in a study that was a systematic review and meta-analysis of 2361 SARS-CoV2 patients. The purpose of this retrospective study was to compare the findings of regular blood tests in two groups of patients referred to the Emergency Department in Italy. Those with a confirmed infection status using molecular testing (n = 102) and those without (n = 105). Upon admission, there were notable disparities (P <.05) in the levels of WBC count (6.47 ± 2.61 × 103/μL), CRP (8.71 ± 8.12 mg/dL), and LDH (388.0 ± 154.5 U/L) within the RT-PCR positive group, the majority of whom were male. These values were higher within the patients infected with COVID-19. Positive status was not associated with platelet count [19]. Our findings are consistent with those of **Spiezia *et al.*** [20] who also found that many studies compared SARS-CoV-2 infected individuals to healthy controls by analyzing the major coagulation indicators like as PT, INR, as well as D-dimer in various disease groups. A total of 22 COVID-19 patients were evaluated for coagulation disorders. When compared to healthy controls (n = 44), there was a significant rise in plasma D-dimer levels (5.3 ± 2.1 μg/mL).

Patients in critical illness had significantly greater levels of PT and D-dimer in comparison with those having mild as well as severe disease, and these levels linked positively with disease severity, according to a large retrospective study (n = 380) [21]. A separate study found that coagulation measures can accurately predict which diseases will be mild, severe, or critical (AUC) >0.8) [22]**. Chen *et al.*** [23] found that CRP and LDH levels were substantially greater in severe cases than in mild ones. While, WBC, neutrophil, CRP, as well as DH counts increased as the disease's severity or criticality increased, lymphocyte counts decreased.

In the current study among the Covid-19 infected group, 19 (40.4%) were CORAD 4 and 28 (59.6%) were CORAD 5. In the present study, COVID-19 infection in MI was negatively correlated with the outcome (r =-0.577) ($p \leq 0.01$), while it was positively correlated to TLC (r=0.917), absolute lymphocytic count (r=0.671), CRP (r=0.718) and D- dimer(r=0.662) with a $p \leq 0.01$. While there was no significant correlation between CK, CK-MB, troponin I or LDH. Also this study revealed that in MI patients without COVID-19 infection, 45 patients (95.7%) were discharged and only 2 patients (4.3%) were died in comparison with the MI patients infected with COVID-19 where 31 patients (65.9%) were discharged and 16 (34.1%) patients were died, which revealed that COVID-19 infection in MI was associated with increased mortality rate among MI patients.

These findings corroborated those of **Silva *et al.*** [24], who previously showed that a severe SARS-CoV-2 infection has several effects on the cardiovascular system. A higher risk for mortality is linked to myocardial damage, which is found in 25% of COVID-19 patients hospitalized. Acute myocardial infarction types I and II, which are serious cardiovascular events, are more common in those infected with SARS-CoV-2, which greatly raises the risk of cardiac damage. A poor prognosis follows from this.

Our multivariate analysis confirmed previous findings that TLC, D-dimer, as well as CRP were COVID-19 infection indicators in the STEMI group. This corresponds to a large collection of cases of COVID-19 patients from the New York City area which involved baseline measurements of D-dimer (n = 5,700). A common observation between those suffering from COVID-19, especially those with serious symptoms that is correlated with a high mortality rate, was an increase of D-dimer in the peripheral bloodstream. The median level was 438 ng/ml (IQR: 262-872 ng/ml) (Reference normal range [0-229 ng/ml]) [25]**.** While, there may be a number of factors contributing to the increased frequency and poorer clinical outcomes of AMI in patients having concurrent COVID-19, our most important finding was that in-hospital mortality, there was significantly greater in STEMI patients with COVID-19 infections. These results are associated with a cytokine storm that occurs during a COVID-19 infection. This storm starts and encourages a thrombo-inflammatory process, causing atherosclerotic plaques to become unstable or rupture, and additional coronary plaques to develop. Virus infection can also harm the myocardium directly, making hypoxia as well as oxidative stress worse. This increases the risk of cardiogenic shock, cardiac arrest, as well as death [26]**.**

Patients presenting with ACS while also infected with COVID-19 constitute a significant proportion. These cases were shown to have higher rates of negative

outcomes in the initial observations. Possible causes of the worse clinical outcomes include: 1. First, the direct pathophysiological effects of SARS-CoV-2 infection, which could enhance the likelihood of plaque, rupture as well as thrombus propagation. 2. Patient as well as system-related issues that contributed to the inappropriate administration of medical care [27, 28]. Specifically, within STEMI patients, COVID-19 was linked to a substantial rise in cardiovascular as well as non-cardiovascular mortality in the study of **Rodriguez-Leor *et al.* [29].** Individuals with both STEMI and COVID-19 were studied by a group of international researchers. 144 individuals had STEMI and 121 had NSTE-ACS. Despite the ACS subtype, the study's major conclusions held. Patients with ACS who tested positive for COVID-19 had the following characteristics compared to control cohorts collected from the BCIS National PCI Audit as well as the Myocardial Ischemia National Audit Project (MINAP) databases in the UK: 1. A higher risk of morbidity. 2. A longer period between detecting symptoms and requesting medical help, and in the event of a STEMI, a lower rate of prompt reperfusion therapy. 3. A higher number of patients admitted to the intensive care unit with the need for a ventilator and possibly hemodynamic support. 4. An increase of over 100% in cardiogenic shock and other unfavorable in-hospital clinical events. 5. The risk for mortality while hospitalized has increased fourfold **Kite *et al.* [30]**

CONCLUSION

The study found that individuals with acute myocardial infarction who also had a COVID-19 infection had a much worse prognosis, with greater rates of mortality while hospitalized and more cardiorespiratory problems.

LIMITATIONS

- Small sample size.
- A Single center study.
- Failure to follow up on course of disease and fate of the patient who were discharged.
- Limited resources in our hospital to detect coagulopathy in COVID -19 patients such as fibrinogen and anti-thrombin, protein c and protein s.

RECOMMENDATIONS

- A larger study is wanted to be conducted to validate these findings.
- Monitoring of laboratory biomarkers of COVID-19 infection in STEMI patients could help in the identification of the course and severity of manifestations, in order to enable early management and more aggressive intervention.
- Further research aimed at customized risk stratification are necessary in relation to the vast variety of symptoms as well as prognostic severity connected with COVID-19 infection.
- Further studies to elucidate the mechanisms of COVID-19–associated cardiovascular complications and to assess whether treatments targeting COVID-19 infection and its associated inflammatory response can reduce adverse events occurrence are warranted.

Conflicts of interests: All authors stated that they hade no conflicts of interest.

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