Original Article

Myocardial Infarction in Patients with and without COVID-19: Comparisons of Characteristics, Clinical Courses, and Outcomes

Hossein Sheibani, MD[1*], Mehran Gheshlaghi, MD[2], Somayeh Shah Hosseini, MSc[3], Mojgan Javedani Masroor, MD[4], Salman Daliri, MSc[5]

[1]*Clinical Research Development Unit, Imam Hossein Hospital, Shahroud University of Medical Sciences, Shahroud, Iran.*
[2]*Student Research Committee, School of Medicine, Shahroud University of Medical Sciences, Shahroud, Iran.*
[3]*Imam Hossein Center for Education, Research and Treatment, Shahroud University of Medical Sciences, Shahroud, Iran.*
[4]*Shahid Akbar Abadi Clinical Research Development Unit, Iran University of Medical Sciences, Tehran, Iran.*
[5]*Clinical Research Development Unit, Imam Hossein Hospital, Shahroud University of Medical Sciences, Shahroud, Iran.*

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Abstract

Background: *COVID-19 has rapidly become a global health emergency. This infection can cause damage to various organs. Injury to myocardial cells is one of the salient manifestations of COVID-19. The clinical course and outcome of acute coronary syndrome (ACS) are influenced by various factors, including comorbidities and concomitant diseases. One of these acute concomitant diseases is COVID-19, which can affect the clinical course and outcome of acute myocardial infarction (MI).*

Methods: *The present cross-sectional study compared the clinical course and outcome of MI and some of its practical factors between patients with and without COVID-19. The study population consisted of 180 patients (129 males and 51 females) diagnosed with acute MI. Eighty patients had COVID-19 infection concurrently.*

Results: *The mean age of the patients was 65.62 years. The frequencies of non–ST-elevation MI (vs ST-elevation MI), lower ejection fractions (<30), and arrhythmias were significantly higher in the COVID-19 group than in the non–COVID-19 group (P=0.006, 0.003, and P<0.001, respectively). The single-vessel disease was the most frequent angiographic result in the COVID-19 group, while the double-vessel disease was the most frequent angiographic result in the non–COVID-19 group (P<0.001).*

Conclusion: *It appears that patients with ACS who are co-infected with COVID-19 infection need essential care.*

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Keywords: *Myocardial infarction; COVID-19; Cardiac arrhythmia; Pneumonia; SARS-CoV-2*

[*]**Corresponding Author:** ***Hossein Sheibani,*** *Associate Professor of Cardiology, Shahroud University of Medical Sciences, Head of the Vice-Chancellery of Treatment, the Clinical Research Development Unit, Imam Hossein Hospital, Imam Avenue, Shahroud, Iran. 3616911151. Tel: +98 23 32342000. Fax: +98 23 32392820. E-mail: sheybani@shmu.ac.ir.*

Introduction

The coronavirus COVID-19 pneumonia started in December 2019 and swiftly became a pandemic. COVID-19 affects the lungs and causes damage to various body organs, especially the cardiovascular system.[1] COVID-19 shows its effects through various inflammatory cytokines and thrombogenic factors.[2,3] Acute myocardial infarction (MI) is a cardiovascular emergency with different types: with elevated ST-segment (STEMI) and without elevated ST-segment (NSTEMI).

Unfortunately, COVID-19 is a highly contagious pneumonia that affects the diagnosis and treatment of patients with MI significantly.[4] Nevertheless, studies in Europe and the United States have shown that hospital admissions for MI and cardiac interventions have decreased by more than 50%.[5] Various reasons can justify this finding, and one of the reasons is the fear of being in an infected environment.[5]

Coronary heart disease can exacerbate the clinical course and outcome of patients with COVID-19.[6,7] The coexistence of these 2 acute diseases can affect the outcome of MI in several ways, including delays in referral due to the fear of COVID-19 infection and delays in diagnosis due to the similarity of laboratory findings and symptoms (eg, shortness of breath, chest pain, positive troponin, and electrocardiographic [ECG] changes).[8-10] Recent studies have indicated that although the most significant emphasis on the COVID-19 disease is on pulmonary complications, physicians need to be aware of cardiovascular complications.[11] MI with COVID-19 increases in-hospital mortality, necessitating the faster identification of these patients.[12]

The question, however, remains whether the comorbidity of MI and COVID-19 could affect a patient's clinical situation. If the answer is yes, what clinical and paraclinical data differ between COVID-19 and non–COVID-19 groups? By focusing on the difference between the 2 groups, we can effectively manage the complications of MI/COVID-19.

The present study aimed to evaluate the prevalence, clinical manifestations, angiographic results, and outcomes of STEMI and NSTEMI in 2 groups with COVID-19 and without COVID-19 in patients referred to Imam Hossein Hospital, Shahroud University of Medical Sciences.

Methods

The current cross-sectional study was performed on 180 patients admitted to the intensive care unit for COVID-19 and the coronary care unit (CCU) of Imam Hossein Hospital in Shahroud. The patients were admitted and treated with a definitive diagnosis of MI with or without acute COVID-19 infection. These patients were admitted between September 2020 and January 2021. Imam Hossein Hospital has a coronary angiography (CAG) center, admitting 700 to 900 patients per year. The follow-up period was 1 to 2 months for all cases.

In this study, the criterion for COVID-19 was a positive polymerase chain reaction test with the COVID-19 pattern on the patient's lung computed tomography (CT) scan. The criteria for MI were also associated with clinically relevant symptoms and serum-positive troponin, with the diagnosis on ECG. It should be noted that primary percutaneous coronary interventions are performed 24/7 in this center.[3,14] MI cases were divided into STEMI and NSTEMI.

Diagnoses were established when patients had symptoms of both diseases on admission to the emergency department. Additionally, when other symptoms became more pronounced during hospitalization for one of these syndromes, the patient was included in the study. The second disease was confirmed after hospitalization.

MI patients without COVID-19 were admitted to the CCU and received treatment based on updated guidelines. The primary percutaneous coronary intervention was possible in the CCU. MI patients with COVID-19 were also admitted to the intensive care unit and received treatment for both diseases based on up-to-date guidelines. CAG was conducted if necessary. CAG results were categorized as mild coronary artery disease (CAD) and significant stenosis, composed of single-vessel disease (SVD), double-vessel disease (2VD), and triple-vessel disease (3VD). Stenoses exceeding 75% were considered angiographic CAD.[15]

The following variables were assessed as the outcome of the study: CAG results (SVD, 2VD, and 3VD), ECG (arrhythmias), echocardiography (ejection fractions), blood tests, and comorbidities. All the outcomes were considered in-hospital ones.

Concerning clinical variables, the criteria were the notes of the doctor and the nurse in the file. After the hospital's laboratory released the results, laboratory variables were interpreted according to international standards. ECGs and arrhythmias were interpreted and diagnosed by a cardiologist. An expert cardiologist, via transthoracic echocardiography, measured the left ventricular ejection fraction.

Regarding the inclusion criteria, the diagnosis of MI was made according to the criteria of the latest guideline. The diagnosis of COVID-19 was based on the presence of at least one of the following criteria: the detection of the novel coronavirus nucleic acid in respiratory or blood specimens by real-time polymerase chain reaction and the existence of typical patterns in the lung CT scan for COVID-19 pneumonia. Only patients who arrived at the hospital less than 14 days from the onset of symptoms were included in the study. The patients included did not have multiple and advanced comorbidities.

Patients were excluded from the study if they had multiple

advanced comorbidities (any chronic inflammatory disease [e.g., rheumatoid arthritis]), incomplete files, and the acute phase of inflammatory diseases.

The findings were entered into the software and analyzed using the χ^2, t, and regression tests. The results were interpreted based on the significance value (P<0.05).

All clinical information was extracted from the patient's records and the COVID-19 information registration system.

Continuous variables with normal and non-normal distributions are presented based on the mean ± the standard deviation and the median, respectively. Numbers and percentages represent categorical variables. The comparison of continuous variables with normal and non-normal distributions was performed using the student *t* test and the Mann–Whitney *U* test, respectively. In addition, categorical variables were compared using the χ^2 or Fisher exact test. The categorical and continuous variables were evaluated for their ability to predict mortality, hospitalization in the intensive care unit, intubation, positive troponin rates, cardiac enzymes, and C-reactive protein (CRP) in the logistic regression model. Model building was done by dividing variables into 2 phases (yes/no and increased/decreased) so that logistic regression could be performed. Variables with P values below 0.15 and factors with known effects on the outcome were candidates for inclusion in the model.

A 2-sided α of less than 0.05 was considered statistically significant. The data were analyzed using SPSS 21.0 for Windows.

Results

The mean age of the COVID-19 and non–COVID-19 groups was 69.44 years and 62.56 years, respectively. Totally, 180 patients were admitted and treated with a definitive diagnosis of MI between the March 2020 crisis and January 2021. Additionally, 80 patients also had COVID-19 infection concurrently. The prevalence of NSTEMI was 70% in the COVID-19 group and 49% in the non–COVID-19 group; the difference was statistically significant (P=0.006). Anterior and inferior MIs were the most common forms of involvement in both groups, but anterior MI was significantly more frequent in the COVID-19 group than in the non–COVID-19 group (P =0.005) (Table 1).

The prevalence of kidney injury (creatinine >1.5 mg) was 16.1% overall. Whereas only 2% of the non–COVID-19 group had high creatinine levels, 33.75% of the COVID-19 group had elevated levels (P<0.001) (Table 1).

Leukocytosis was more frequent in the COVID-19 group than in the non–COVID-19 group (51.25% vs 10%). The leukocyte count was more likely normal in the non–COVID-19 group, with the difference between the 2 groups constituting statistical significance (P<0.001) (Table 1). The prevalence of a positive CRP test was 73.75% in the COVID-19 group and 12% in the non–COVID-19 group. The frequency of a high erythrocyte sedimentation rate was 80% in the COVID-19 group and 4% in the non–COVID-19 group (P=0.001 in both cases) (Table 1).

The rate of a reduced ejection fraction (<30%) after MI was significantly different between the 2 groups, and the rate of reduction in the group with COVID-19 was higher (P=0.003). The difference in the mean ejection fraction between the 2 groups (42.38 vs 46.20) was significant (P=0.020) (Table 2). The prevalence of cardiac arrhythmias was significantly different, too, in that the COVID-19 group experienced more cardiac arrhythmias than did the non–COVID-19 group (P<0.001) (Table 1). The type of arrhythmia was different between the 2 groups insofar as the incidence of atrial fibrillation rhythms, complete atrioventricular blocks, monomorphic ventricular tachycardia, and complete right bundle branch blocks was more common in the group with COVID-19, while complete left bundle branch blocks and atrioventricular blocks (except for atrioventricular blocks grade III) were more frequent in the group without COVID-19. The incidence of other arrhythmias was not significantly different between the 2 groups (Table 1).

The need to perform cardiopulmonary resuscitation (CPR) was more in the COVID-19 group than in the non–COVID-19 group, and the difference was statistically significant (P<0.001). None of the patients in the non–COVID-19 group required intubation, while 16.25% of the patients in the group with COVID-19 required intubation (P=0.001). No in-hospital deaths occurred in the group without COVID-19, whereas 18.75% of those with COVID-19 died in the hospital (P=0.001) (Table 1). None of the patients with COVID-19 who underwent coronary catheterization had mild CAD, and all had significant coronary stenosis, while 4.1% of the COVID-19 group had mild CAD. The most prevalent CAG results were SVD and 2VD in the COVID-19 and non–COVID-19 groups, respectively; the difference was statistically significant (P<0.001) (Table 1).

For the adjustment of confounding variables, such as diabetes, body mass index, age, sex, and occupation, in the logistic regression model, they were compared between the 2 groups of MI patients with and without COVID-19 (Table 3). In general, the association between COVID-19 and acute MI increased the incidence of electrical and mechanical complications. It also predicted a worse clinical course of the disease with the need for intubation and CPR, as well as increased in-hospital deaths. However, some confounding factors, such as body mass index, did not differ significantly between the 2 groups, while others, such as age, sex, occupation, and diabetes, when adjusted by the regression model, confirmed the previous results.

Table 1. Comparisons of categorical variables between groups of MI with and without COVID-19*

	MI With COVID19 (n=80)	MI Without COVID19 (n=100)	*P*
Age (y)			0.006
30-59	17 (21.2)	41 (41)	
≥60	63 (78.7)	59 (59)	
Sex			1.000
Male	57 (71.2)	72 (72)	
Female	23 (28.7)	28 (28)	
BMI			0.282
20-24.9	19(23.7)	33 (33)	
25-29.9	50 (62.5)	51 (51)	
≥30	11 (13.7)	16 (16)	
MI Type			0.006
STEMI	24 (30)	51 (51)	
NSTEMI	56 (70)	49 (49)	
STEMI Location			0.005
Anterior	21 (87.5)	34 (66.6)	
Inferior	3 (12.5)	17 (33.4)	
DM (Yes)	36 (45)	55 (55)	1.000
Anemia (Yes)	62 (77.5)	66 (66)	1.000
Hgb			1.000
<14	62(48.4)	66 (51.6)	
>14	18(34.6)	34 (45.4)	
Serum Urea >45	33 (41.2)	16 (16)	<0.001
Cr >1.5	53 (66.2)	98 (98)	<0.001
WBC			<0.001
<5000	12 (15)	13 (13)	
5000-10000	27 (33.7)	77 (77)	
>10000	41 (51.2)	10 (10)	
PLT			0.329
<150000	10 (13)	8 (8)	
150000-450000	69 (86.2)	83 (83)	
>450000	5 (6.2)	4 (4)	
CRP (Positive)	59 (73.7)	12 (12)	<0.001
ESR >20 (%)	64 (80)	4 (4)	<0.001
EF <55 (%)	63 (78.7)	70 (70)	0.232
45-55	29 (46)	40 (57.1)	0.044
30-45	22 (34.9)	24 (34.3)	0.342
<30	12 (19)	6 (8.6)	0.003
Cardiac Arrhythmias (Yes)	43 (53.7)	33 (33)	<0.001
Acute MR (Yes)	5 (6.2)	3 (3)	<0.001
CPR (Yes)	13 (16.2)	2 (2)	<0.001
CAG Results			<0.001
No CAG	39 (48.7)	2 (2)	
Mild CAD	0 (0)	6 (4.1)	
Significant Stenosis	41 (100)	92 (93.9)	
SVD	27 (65.8)	25 (25.5)	
2VD	7 (17)	42 (42.8)	
3VD	7 (17)	25 (25.5)	
Intubation (Yes)	13 (16.2)	0 (0)	<0.001
Death (Yes)	15 (18.7)	0 (0)	<0.001

*Data are presented as n (%).

MI, Myocardial infarction; Cr, Creatinine; PLT, Platelet; ESR, Erythrocyte sedimentation rate; WBC, White blood cell; LYMPH, Lymphocyte; ICU, Intensive care unit; CRP, C-Reactive protein; EF, Ejection fraction; Acute MR, Acute mitral regurgitation; CAG, Coronary angiography; CAD, Coronary artery disease; SVD, Single-vessel disease; 2VD, Double-vessel disease; 3VD, Triple-vessel disease; STEMI, ST-segment-elevation myocardial infarction; NSTEMI, Non–ST-segment-elevation myocardial infarction; Hgb, Hemoglobin; BMI, Body mass index; Death, In-hospital mortality

Table 2. Comparisons of continuous variables between groups of MI with and without COVID-19*, **

	Normal Range	MI With COVID19 (n=80)	MI Without COVID19 (n=100)	*P*
Age	(y)	69.44±13.77	62.56±13.22	0.001
BMI	(kg/m^2)	26.56±3.45	26.31±3.91	0.665
Hgb	(g/dL)	12.20±2.33	13.26±1.64	0.001
Men	13.2-16.6 (g/dL).			
Women	11.6-15 (g/dL)			
FBS	<99 (mg/dL)	134.81±70.15	129.33±56.97	0.500
WBC	4500-11000 (per µL)	10797.50±5974.03	7272.03±2150.06	0.001
PLT	150.000-400.000 (per µL)	231489.23±83887.21	251293.11±105293.12	0.239
Serum Urea	5-20 (mg/dL)	55.94±37.26	36.59±11.55	0.001
Cr		1.36±0.70	1.067±0.20	0.001
Men	0.7 to 1.3 (mg/dL)			
Women	0.6 to 1.1 (mg/dL)			
EF	> 55 (%)	42.38±12.72	46.20±9.18	0.020
ESR		42.73±31.02	5.90±14.16	0.001
Men	1–13 (mm/h)			
Women	1–20 (mm/h)			
CRP	<10 (mg/L)	19.45±12.13	11.22±3.21	0.001
TG	<150 (mg/L)	104.06±50.33	117.65±45.18	0.058
TC	<200 (mg/L)	168.06±57.34	144.76±38.67	0.002
LDL	<100 (mg/L)	93.82±28.35	83.25±32.00	0.020
HDL	>60 (mg/L)	45.20±11.63	51.47±16.06	0.004

*Data are presented as mean±SD or n (%).
**The measurement scale for the mean: Adding up the scores and dividing the total by the number of scores
The measurement scale for the median: The middle position score after ordering the data
MI, Myocardial infarction; BMI, Body mass index; Hgb, Hemoglobin; FBS, Fasting blood sugar; WBC, White blood cell; PLT, Platelet; Cr, Creatinine; EF, Ejection fraction; ESR, Erythrocyte sedimentation rate; CRP, C-reactive protein; TG, Triglyceride; TC, Total cholesterol; LDL, Low-density lipoprotein; HDL, High-density lipoprotein

Table 3. Relationship between a categorical dependent variable and independent variables (Logistic Regression analysis results for effect of COVID19 infection on predictive categorical variables of MI course and outcome after control effect of DM, Sex, Age, Anemia and Job)

Variable for the COVID-19-Positive Group	Odds Ratio (95% CI)	*P*
MI Type		
STEMI	0.41 (0.22-0.76)	0.005
NSTEMI	2.42 (1.31-4.51)	0.004
STEMI Location		
Anterior	4.36 (2.79-8.43)	0.045
Inferior	0.77 (0.32-0.97)	0.014
Anemia		
Hgb <14	0.56 (0.29-1.09)	0.093
Uremia		
Serum urea >45	3.69 (1.84-7.39)	<0.001
Kidney Injury		
Cr >1.5	24.96 (5.71-109.08)	<0.001
Leukocyte Change		
Leukocytosis (WBC>10000)	11.69 (5.90-34.27)	<0.001
Leukopenia (WBC<5000)	2.63 (0.65-4.02)	0.035
PLT Change		
Thrombocytosis (PLT>450000)	3.08 (0.58-16.36)	0.580
Thrombocytopenia (PLT<150000)	0.57 (0.20-1.57)	0.277
CRP		
Positive	3.88 (1.35-8.76)	0.002
ESR		
Increased (>20)	82.67 (26.71-255.78)	<0.001
HF		
EF<55 (%)	0.63 (0.32-1.25)	0.186
Cardiac Arrhythmias	2.99 (1.61-5.55)	<0.001
CPR Positive	9.51 (2.08-43.50)	0.004
New Acute MR	9.25 (4.16-20.57)	<0.001
Intubation	24.11 (13.20-56.34)	0.008
Death (In-Hospital Mortality)	2.90 (2.09-5.86)	0.006
CAG Result		
No CAG	1.34 (1.11-4.57)	0.033
Mild CAD	0.15 (0.05-0.40)	<0.001
Significant Stenosis	0.01 (0.01-0.04)	<0.001
SVD	18.06 (3.94-82.68)	0.782
2VD	0.26 (0.09-0.41)	0.008
3VD	0.12 (0.01-0.57)	0.009
High TC		
TC >200 (mg/dL)	0.32 (0.13-0.74)	0.008
Low HDL		
HDL <45 (mg/dL)	0.60 (0.04-0.87)	0.095
High LDL		
LDL >130 (mg/dL)	1.883 (0.333-1.092)	0.032

MI, Myocardial infarction; Cr, Creatinine; PLT, Platelet; ESR, Erythrocyte sedimentation rate; WBC, White blood cell; LYMPH, Lymphocyte; ICU, Intensive care unit; CRP, C-Reactive protein; EF, Ejection fraction; HF, Heart failure; Acute MR, Acute mitral regurgitation; CAG, Coronary angiography; CAD, Coronary artery disease; SVD, Single-vessel disease; 2VD, Double-vessel disease; 3VD, Triple-vessel disease; STEMI, ST-segment-elevation myocardial infarction; NSTEMI, Non–ST-segment-elevation myocardial infarction; Hgb, Hemoglobin; TG, Triglyceride; TC, Total cholesterol; LDL, Low-density lipoprotein; HDL, High-density lipoprotein

Discussion

The present study was a comprehensive investigation encompassing the effects of COVID-19, MI, and COVID-19/MI on patients. Some of the findings observed were due to COVID-19 complications and were not related to MI. Nonetheless, we must focus on the impacts of COVID-19/MI and determine which complications result from COVID-19 and MI simultaneously.

The following laboratory tests and medical interventions exhibit a rise or test positive due to COVID-19 in patients with MI: CRP, the erythrocyte sedimentation rate, leukocytosis, and intubation.[6]

The results listed below could be a consequence of concurrent COVID-19 and MI complications in patients with MI:

- The COVID-19 group was more disposed to NSTEMI than the non–COVID-19 group.
- The COVID-19 group was more disposed to a lower ejection fraction than the non–COVID-19 group.
- The COVID-19 group was more disposed to arrhythmias.
- The need for CPR was more frequent in the COVID-19 group than in the non–COVID-19 group.
- Regarding all our patients with STEMI, the COVID-19 group was more disposed to anterior STEMI than the non–COVID-19 group (compared with posterior STEMI).
- SVD was more frequent in the COVID-19 group, while 2VD was more frequent in the non–COVID-19 group.

Studies have shown that cardiovascular diseases increase the risk of a severe clinical course in patients with COVID-19.[7] The clinical course and outcome of acute MI are also influenced by acute and chronic comorbidities.[17] These findings chime with our study; therefore, when the focus is placed on the type of MI, there could be a relationship between NSTEMI and COVID-19 due to the different frequencies of the MI type between the 2 groups.

Some issues have been reported regarding the ejection fraction in patients with COVID-19 infection. According to a prior investigation, a decreased ejection fraction was significant in this group of patients,[18] which is concordant with our study. The decreased ejection fraction could result from the simultaneous effect of COVID-19 and MI, but we need further data to demonstrate that.

Arrhythmias could be a cardiovascular manifestation of COVID-19. Arrhythmias were reported in 16.7% of hospitalized patients with COVID-19 and had a strong relationship with the prognosis of patients. In other words, a higher frequency of arrhythmias was associated with a worse medical condition.[19] We also obtained the same result in the current study. We also observed an increased chance of arrhythmias in the comorbidity of COVID-19 and MI.

Recent articles have revealed that COVID-19 could be associated with MI, resulting in a poor prognosis.[20] Some studies have also reported that the need for CPR is associated with a poor prognosis in patients with COVID-19.[21] Therefore, a high frequency of CPR can be expected in the comorbidity of MI and COVID-19 than MI.

Despite the high frequency of NSTEMI compared with STEMI in our COVID-19 group, the mortality rate was higher in that group. Simultaneous complications of COVID-19 (inflammatory mediators) and MI could worsen the clinical situation of patients even if the frequency of NSTEMI was high compared with STEMI. This finding indicates that MI could be more dangerous in patients with COVID-19.

All our patients with COVID-19 and MI had significant coronary stenosis on angiography, chiefly in 1 coronary artery. This finding suggests a greater role for MI acute embolic events than atherosclerosis.

Temporary abnormalities in the lipid profile of patients with acute MI are known. This disorder was particularly pronounced in the reduced high-density lipoprotein and increased low-density lipoprotein in the COVID-19 group. This elevation could be secondary to cytokine storms and cause the activation of iatrogenic mechanisms.[22]

There were some limitations regarding the sample size due to the pandemic situation, and a retrospective/cohort study can enlighten more aspects of the comorbidity of COVID-19 and MI. In addition, systematic reviews can demonstrate and compare the cardiovascular events of COVID-19.

Conclusion

According to the findings of the present study, co-infection with COVID-19 in patients with acute MI worsens the clinical course and outcome. Although the results of this study are consistent with previous studies, more investigations with higher sample sizes are recommended due to the limited research.

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