

Assessment of COVID-19-Positive Patients with ST-Segment Elevation Myocardial Infarction During the COVID-19 Pandemic

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ABSTRACT

Objective: The global impact of the coronavirus disease 2019 (COVID-19) pandemic has been shown to adversely affect the cardiovascular system. Investigating the clinical course of patients with ST-segment elevation myocardial infarction (STEMI) associated with COVID-19 infection is crucial to elucidate the mechanisms linking COVID-19 and STEMI. Assessing these patients from both cardiovascular and infection control perspectives is essential for improving clinical outcomes and reducing the burden on healthcare systems. This study aims to provide a comprehensive evaluation of the clinical course of COVID-19-positive STEMI patients.

Methods: This single-center, cross-sectional study analyzed 80 STEMI patients who underwent coronary angiography, comprising 38 COVID-19-positive individuals and 42 COVID-19-negative individuals. We assessed admission times, biochemical parameters, door-to-balloon times, angiographic data, length of hospital stay, and in-hospital mortality.

Results: The mean age in the COVID-19-positive group was 56.6±10.8 years, whereas that in the COVID-19-negative group was 53.6±10.8 years ($P = .238$). No significant differences in demographic characteristics were observed between the groups. However, C-reactive protein (CRP), white blood cell (WBC), and baseline troponin levels were significantly greater in the COVID-19-positive group ($P < .001$, $P = .005$, and $P = .037$, respectively). Echocardiographic evaluation revealed a lower ejection fraction (EF) in COVID-19-positive patients than in COVID-19-negative patients ($P = .023$). Angiographic evaluation revealed a greater thrombus burden and longer pain-to-balloon time in the COVID-19-positive group ($P = .001$ and $P < .001$, respectively). The length of hospital stay was also longer in the COVID-19-positive group ($P < .001$).

Conclusion: The findings of increased thrombus burden, prolonged pain-to-balloon time, and extended hospital stays among COVID-19-positive patients underscore the adverse cardiovascular effects of COVID-19. Delayed hospital presentation of patients with cardiovascular symptoms during the pandemic poses a challenge for timely diagnosis and management of critical cardiovascular conditions.

Keywords: COVID-19, STEMI, myocardial infarction, pandemic

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INTRODUCTION

The COVID-19 pandemic has placed immense pressure on global healthcare systems, reshaping clinical practice and leading to the near-collapse of healthcare infrastructures in many countries.¹ As the pandemic has progressed, the cardiovascular implications of COVID-19 have become particularly noteworthy. In particular, ST-segment elevation in COVID-19-positive patients has been associated with severe cardiovascular events such as acute myocardial infarction.² This clinical phenomenon has prompted healthcare professionals and researchers to seek a deeper understanding of the pathophysiological effects of COVID-19 on the cardiovascular system and to develop appropriate clinical approaches.³ However, the precise clinical characteristics, pathophysiology, and management of myocardial infarction associated with ST-segment elevation in COVID-19-positive patients remain unclear.⁴ By exploring the clinical characteristics and outcomes of COVID-19-positive STEMI patients, this study seeks to provide insights that may inform future approaches to cardiovascular care and infection control. The findings aim to support ongoing efforts to improve the management of this particularly vulnerable patient population. This study aims to comprehensively evaluate the clinical features, diagnostic methods, treatment options, and outcomes of myocardial infarction in COVID-19-positive patients presenting with ST-segment elevation.

METHODS

This cross-sectional study included all consecutive STEMI patients who presented to Recep Tayyip Erdoğan University Training and Research Hospital and underwent coronary angiography between May 1, 2020, and August 31, 2020. COVID-19 tests were conducted on patients in the emergency department, but they were admitted to the angiography unit without waiting for the results. Once the test results were available, patients were divided into two groups: positive and negative for COVID-19. Patients who presented with STEMI but did not undergo coronary angiography were excluded from the study. The following variables were assessed: the time of admission, initial troponin I level, number of door-to-wire visits, angiographic findings, length of hospital stay, and in-hospital mortality. Ethical approval was obtained from the local authority, and the study was approved by the Recep Tayyip Erdoğan University Non-Interventional Clinical Research Ethics Committee (Date: January 7, 2021 Number: 2021/E-40465587-050.01.04-9).

On the basis of the Fourth Universal Definition of Myocardial Infarction, patients with ST-segment elevation in two or more consecutive leads on electrocardiogram accompanied by chest pain were considered to have STEMI.⁵ Patients who experienced

cardiac arrest at the time of admission were excluded from the study. Additionally, patients treated more than 12 hours after the onset of chest pain were not included. The treatment protocol followed the ESC-STEMI guidelines. Primary percutaneous coronary intervention (PCI) was performed in all patients. After primary PCI, angiographic images were reviewed, and thrombolysis in myocardial infarction (TIMI) flow scores and angiographic modified thrombus grades were assessed, ranging from Grade 0 (no thrombus present) to Grade 5 (complete occlusion of the vessel by thrombus).⁶

All patients' COVID-19 diagnoses were confirmed by PCR and thoracic CT. COVID-19-positive patients were treated according to the guidelines of the Turkish Ministry of Health. Deaths related to COVID-19, including those caused by acute respiratory distress syndrome, septic shock, and respiratory failure, were recorded separately.⁷

Statistical Analysis

Statistical analyses were performed via SPSS version 22.0 (IBM SPSS Corp., Armonk, NY, USA). Continuous variables are expressed as the means \pm standard deviations or as minimum and maximum values, whereas categorical variables are presented as percentages. The distribution of continuous variables was assessed via the Kolmogorov–Smirnov test. Continuous variables were compared via Student's *t* test or the Mann–Whitney *U* test, as appropriate. Categorical variables were compared via the chi-square test. A *p* value of $<.05$ was considered statistically significant.

RESULTS

A total of 80 STEMI patients who met the inclusion criteria were enrolled in the study, comprising 38 COVID-19-positive patients and 42 COVID-19-negative patients. The mean age in the COVID-19-positive group was 56.6 ± 10.8 years, whereas the mean age in the COVID-19-negative group was 53.6 ± 10.8 years ($P = .238$). There was no significant difference between the two groups in terms of demographic characteristics such as sex, diabetes status, or family history (Table 1). When laboratory data were compared between the groups, the CRP, WBC, and baseline troponin levels were significantly greater in the COVID-19-positive group [$1.11 (.80-2.8)$ vs. $.80 (.60-2.5)$, $P < .001$; 11.2 ± 3.6 vs. 9.2 ± 2.6 , $P = .005$; $865 (100-7000)$ vs. $625 (160-3000)$, $P = .037$]. No statistically significant differences were observed in the other laboratory parameters.

According to the echocardiographic assessment, the ejection fraction (EF) of COVID-19-positive patients was significantly lower than that of COVID-19-negative patients (49 ± 9 vs. 54 ± 7.9 , $P = .023$). In the angiographic evaluation of the patients, the

thrombus burden and pain-to-wire time were significantly greater in the COVID-19-positive group than in the COVID-19-

negative group [55 vs. 19, $P = .001$; 294 ± 206 vs. 131 ± 142 , $P < .001$].

Table 1. Baseline Clinical and Laboratory Characteristics According to COVID-19 Diagnosis

Variable Description	COVID Positive (n= 38)	COVID Negative (n= 42)	P
Age Years	56.6 \pm 10.8	53.6 \pm 10.8	.238
Gender	59.5	55.3	.437
Diabetes %	19	18.4	.586
Hypertension %	19	21	.521
Smoking Status	38	29	.480
Dyslipidemia %	41.8	42	.560
Family History of CAD	15.8	16.7	.884
SBP, mmHg	132 \pm 17	133 \pm 24	.943
CRP mg/dL	1.11 (0.80-2.8)	.80 (.60-2.5)	<.01
Hemoglobin g/dL	12.8 \pm 1.3	12.5 \pm 1.3	.345
WBC Count $\times 10^3/\mu\text{L}$	11.2 \pm 3.6	9.2 \pm 2.6	.005
Baseline Troponin I Ng/L	865 (100-7000)	625 (160-3000)	.037
LDL Cholesterol	150 \pm 15	146 \pm 14	.176
HDL Cholesterol mg/dL	30 \pm 13	30 \pm 10	.935
Triglycerides mg/dL	205 \pm 71	192 \pm 69	.702
GFR	60.8 \pm 18	63 \pm 17	.460
BMI	23 (16-31)	24 (16.9-36)	.206
EF %	49 \pm 9.4	54 \pm 7.9	.023
Thrombus Burden / High %	55	19	.001
Pain to Wire Time Minutes	294 \pm 206	131 \pm 142	<.001

Abbreviations: SBP: Systolic Blood Pressure, CRP: C-reactive protein; EF: ejection fraction; LDL: low-density lipoprotein; HDL: high-density lipoprotein; GFR: glomerular filtration rate; BMI: body mass index. The normal threshold value for troponin in our laboratory is <100 Ng/L.

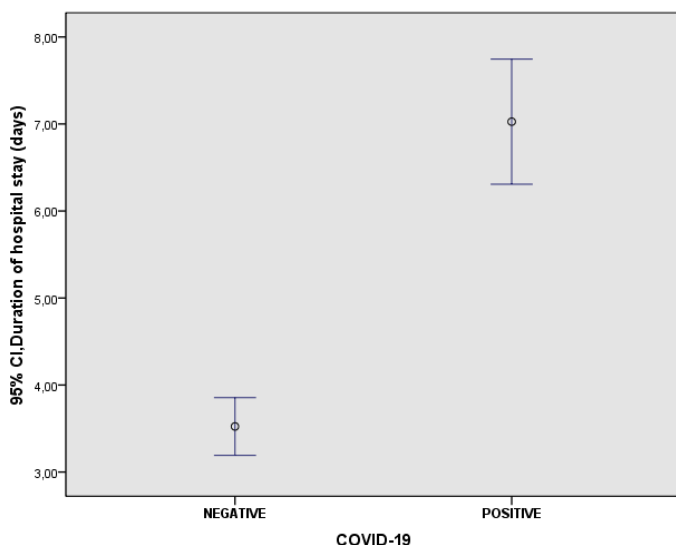


Figure 1. The length of hospital stay among participants

In terms of the length of hospital stay, COVID-19-positive patients had significantly longer hospital stays than did COVID-19-negative patients (7.02 ± 2.1 vs. 3.5 ± 1 , $P < .001$) (Figure 1). In the angiographic evaluation, three COVID-19-positive patients were

found to have less than 50% stenosis in the coronary arteries, whereas all COVID-19-negative patients had a responsible lesion causing significant stenosis in the coronary arteries. Furthermore, 16.7% of the COVID-19-positive patients were identified as having multivessel disease, whereas 18.4% of the COVID-19-negative patients were identified, with no significant difference between the groups ($P = .837$). In the COVID-19 group, one patient died in the hospital due to cardiovascular collapse, whereas there were no in-hospital deaths in the negative group.

DISCUSSION

In this study, the demographic characteristics, laboratory findings, and echocardiographic and angiographic assessments of STEMI patients in the COVID-19-positive and -negative groups were examined. While there were no statistically significant differences in demographic characteristics between the COVID-19-positive and -negative groups, elevated biochemical parameters indicating inflammation, a lower ejection fraction, a greater thrombus burden, and increased hospital stays were observed in COVID-19-positive patients. These findings may suggest that the risk factors associated with COVID-19 independently contribute to the occurrence of STEMI.

According to the literature, the excessive and uncontrolled release of cytokine signaling molecules, which are crucial in the immune system's response to infection, can lead to a condition known as a "cytokine storm".⁸ COVID-19, characterized as a virus that triggers inflammatory responses, is particularly associated with increased cytokine release.^{9,10} In line with the literature, our laboratory findings revealed that CRP, WBC, and baseline troponin levels were significantly higher in the COVID-19-positive group. These findings support the notion that COVID-19 infection may adversely affect the cardiovascular system by enhancing the inflammatory response.^{11,12} In COVID-19 patients, there has been an increase in endothelial damage and the risk of thrombosis in the vascular wall. Platelets, depending on their mean platelet volume and function, can mediate clotting and inflammation, potentially reducing plaque stability and increasing the tendency for atherosclerotic plaques to rupture. This condition may increase the risk of acute coronary syndrome.¹³ Furthermore, studies suggest that the inflammatory response may have direct toxic effects on the myocardium and could lead to myocyte damage.¹⁴ This situation suggests that COVID-19 may increase the risk of cardiovascular complications such as heart failure, arrhythmias, and myocarditis.

In a study examining the echocardiographic results of 447 COVID-19 patients, a decrease in ejection fraction (EF) values and impaired wall motion were observed. When angiographic findings were evaluated, multivessel disease was reported in these patients.¹⁵ Similarly, in our study, echocardiographic assessments revealed that EF in COVID-19-positive patients was significantly lower than that in COVID-19-negative patients. These findings support the notion that COVID-19 may directly affect the myocardium and adversely impact cardiac function.

The widespread distribution of angiotensin-converting enzyme II receptors in the myocardium facilitates the entry of COVID-19 into myocardial cells.¹⁶ In this context, the myocardium may become a direct target for the virus, leading to inflammation and pathological changes in cardiac tissue. Myocardial damage and immune response mechanisms can trigger the development of myocarditis. As a result of myocarditis, a decrease in myocyte contractility and electrolyte imbalances can lead to severe cardiovascular complications such as heart failure, arrhythmias, and valvular heart disease.¹⁷ This situation supports the idea that COVID-19 may cause transient or permanent functional impairments in the myocardium.

Observations indicate that during the pandemic, there has been an increase in STEMI presentations, with patients presenting later and having shorter hospital stays.¹⁸ Angiographic evaluations revealed that the thrombus burden and pain-to-wire time were significantly greater in COVID-19-positive patients than in COVID-19-negative patients, and the length of hospital

stay was significantly greater. While ST elevation is a rare and dramatic complication of coronary stenting, it necessitates urgent revascularization to prevent long-term cardiac dysfunction. It has been suggested that increased platelet aggregation in infected patients may lead to a greater risk of ST elevation.¹⁹ Therefore, COVID-19 may increase the risk of complications in STEMI patients and prolong the treatment process.

Thromboembolic events are increasingly recognized as a complication associated with COVID-19.^{15,20} Although increased thrombotic risk is linked primarily to systemic inflammation and endothelial damage, studies suggest that it may also stem from the effects of COVID-19 on coagulation mechanisms. Fizzah A. et al. presented strong evidence of increased thrombus burden, prolonged hospital stays, and poorer prognosis in patients with STEMI and concomitant COVID-19 infection.²¹ Similarly, we observed that the thrombus burden and pain-to-wire time significantly increased in COVID-19-positive patients, along with extended hospital stays. In our study, we emphasize the necessity for careful monitoring of potential cardiovascular complications by addressing the increased risk of thromboembolic events associated with COVID-19. A study examining 214 patients with acute coronary syndrome (ACS) revealed that the COVID-19 pandemic is not an independent primary risk factor for the development of ACS, with the leading risk factors being hypertension and elevated LDL levels. This finding also indicates that smoking and the increased stress associated with living alone have a more pronounced effect on ACS.²² However, in contrast to this viewpoint, some studies suggest that COVID-19 symptoms may complicate the identification of critical cardiovascular symptoms in patients, leading those experiencing ACS to confuse classic symptoms such as chest pain or shortness of breath with COVID-19 symptoms.²³ This situation may result in delayed hospital presentations for COVID-19 patients with ACS, consequently delaying the diagnosis and treatment of critical cardiovascular events.¹

Delayed intervention can prolong the duration of pain and increase the risk of permanent myocardial damage, heart failure, and even death. In our study, we associated the increase in pain-to-wire time with prolonged time to emergency medical intervention in COVID-19-positive patients. Similarly, in a study conducted by Aktaş et al., the door-to-balloon time was longer in COVID-19 patients, and those with COVID-19 symptoms, particularly when preoccupied with signs such as shortness of breath, may not receive timely diagnosis and treatment for critical conditions such as myocardial infarction.¹⁸ Therefore, we emphasize the critical importance of patients with COVID-19 symptoms recognizing cardiovascular emergency signs such as ACS and seeking timely medical assistance.

Limitations of the Study

The primary limitation of our study is the limited number of cases due to its single-center design. Additionally, only patients with ST-segment elevation myocardial infarction who underwent coronary angiography (CAG) were included in our study. Other acute coronary syndrome patients were not included, which prevents generalization to all acute coronary syndrome patients. The status of patients who did not undergo CAG, who presented with cardiopulmonary arrest, who received thrombolytic treatment, and who were followed up with medical therapy remains uncertain.

CONCLUSION

During the COVID-19 pandemic, the delayed presentation of patients with acute coronary syndrome (ACS) poses a significant issue that may hinder timely diagnosis and treatment of critical cardiovascular conditions. It is crucial for patients to consider cardiovascular symptoms alongside COVID-19 symptoms and take the necessary steps to seek emergency medical assistance. The increased thrombus burden, prolonged pain-to-wire time,

and extended hospital stays in COVID-19-positive patients reflect the adverse effects of infection on the cardiovascular system. These findings are important for understanding the cardiovascular complications of COVID-19 and managing patients appropriately. However, these findings need to be validated through further research, and advanced studies are needed to better understand the mechanisms of the relationship between COVID-19 and STEMI.

Ethics Committee Approval: Recep Tayyip Erdoğan University Noninterventional Clinical Research Ethics Committee approved the study with decision number 2021/08 on January 7, 2021.

Informed Consent: Written informed consent was obtained from all participants.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – HD, HD; Design – HD, HD; Supervision – HD; Materials – HD, HD; Data Collection and/or Processing – HD; Analysis – HD; Literature Review – HD; Writing – HD, HD; Critical Review – HD, HD.

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REFERENCES

1. Driggin E, Madhavan MV, Bikdeli B, et al. Cardiovascular considerations for patients, health care workers, and health systems during the COVID-19 pandemic. *J Am Coll Cardiol*. 2020;75(18):2352–2371.
2. Del Prete A, Conway F, Della Rocca DG, et al. COVID-19, acute myocardial injury, and infarction. *Card Electrophysiol Clin*. 2022;14(1):29–39.
3. Ghasemzadeh N, Kim N, Amlani S, et al. A review of ST-elevation myocardial infarction in patients with COVID-19. *Cardiol Clin*. 2022;40(3):321–328.
4. Wańha W, Wybraniec M, Kapłon-Cieślicka A, et al. Myocardial infarction in the shadow of COVID-19. *Cardiol J*. 2020;27(5):478–480.
5. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction. *Glob Heart*. 2018;13(4):305–338.
6. Sianos G, Papafaklis MI, Daemen J, et al. Angiographic stent thrombosis after routine use of drug-eluting stents in STEMI: the importance of thrombus burden. *J Am Coll Cardiol*. 2007;50(7):573–583.
7. Sabatine MS, Braunwald E. Thrombolysis In Myocardial Infarction (TIMI) Study Group: JACC Focus Seminar 2/8. *J Am Coll Cardiol*. 2021;77(22):2822–2845.
8. Tufan A, Avanoğlu Güler A, Matucci-Cerinic M. COVID-19, immune system response, hyperinflammation and repurposing antirheumatic drugs. *Turk J Med Sci*. 2020;50(SI-1):620–632.
9. Fajgenbaum DC, June CH. Cytokine storm. *N Engl J Med*. 2020;383(23):2255–2273.
10. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. *JAMA Cardiol*. 2020;5(7):802–810.
11. Soy M, Keser G, Atagündüz P, et al. Cytokine storm in COVID-19: pathogenesis and overview of anti-inflammatory agents. *Clin Rheumatol*. 2020;39(7):2085–2094.
12. Baran Akpınar F, Tuncer M, Dayan A. Factors associated with admission to the intensive care unit in hospitalized COVID-19 patients. *Turk J Fam Pract*. 2020;26(3):124–130.
13. Oncul M, Karakurt C, Elkiran O, et al. Evaluation of frequency, follow-up and treatment of multisystem inflammatory syndrome related to COVID-19. *Ann Med Res*. 2022;29(11):1238–1245.
14. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential effects of coronaviruses on the cardiovascular system: a review. *JAMA Cardiol*. 2020;5(7):831–840.
15. Gharibzadeh A, Shahsanaei F, Rahimi PN. Clinical and cardiovascular characteristics of patients suffering STEMI after COVID-19: a systematic review and meta-analysis. *Curr Probl Cardiol*. 2023;48(1):101045.
16. Chung MK, Zidar DA, Bristow MR, et al. COVID-19 and cardiovascular disease: from bench to bedside. *Circ Res*. 2021;128(8):1214–1236.
17. Siripanthong B, Nazarian S, Muser D, et al. Recognizing COVID-19-related myocarditis: pathophysiology and guideline for diagnosis and management. *Heart Rhythm*. 2020;17(9):1463–1471.
18. Aktaş H, Yildirim O, Gül M, İnci S. Comparison of AMI patients undergoing coronary angiography during the COVID-19 pandemic and the pre-pandemic period. *MN Cardiol*. 2021;27(4):199.
19. Koc H, Barutcu G, Duman H. Case report of STEMI due to early-stage multiple stent thrombosis accompanied by COVID-19. *Rize Med J*. 2023;1(4):20–25.
20. Bikdeli B, Madhavan MV, Jimenez D, et al. COVID-19 and thrombotic

- or thromboembolic disease: implications for therapy and follow-up. *J Am Coll Cardiol*. 2020;75(23):2950–2973.
21. Choudry FA, Hamshire SM, Rathod KS, et al. High thrombus burden in COVID-19 patients with STEMI. *J Am Coll Cardiol*. 2020;76(10):1168–1176.
22. Ferhatbegovic L, Pojskic B. Characteristics of acute coronary syndrome during COVID-19 pandemic. *Atherosclerosis*. 2023;379:90.
23. Welt FGP, Shah PB, Aronow HD, et al. Catheterization laboratory considerations during the COVID-19 pandemic. *J Am Coll Cardiol*. 2020;75(18):2372–2385.