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Original Research

Microvascular Dysfunction in COVID-19 Patients with Acute Coronary Syndrome

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ABSTRACT

Objective: Coronavirus disease 2019 (COVID-19) is considered to deteriorate endothelial function through hyperinflammation. We aimed to investigate microvascular dysfunction using the angiographic parameters thrombolysis in myocardial infarction frame count (TFC) and myocardial blush grade (MBG), in COVID-19 patients with acute coronary syndrome (ACS).

Methods: One hundred and sixty-five patients presented with ACS (62.4% ST elevated myocardial infarction) and underwent percutaneous coronary intervention between March 1 and June 30, 2020, were enrolled in the study. The polymerase chain reaction test was performed in case of suggestive symptoms or typical computerized tomography findings.

Results: Twenty-six patients (15.7%) were tested positive for COVID-19. Significantly higher values were observed in TFC in patients with COVID-19 ($p<0.001$), whereas COVID-19 patients had significantly lower MBGs (Grade 0 and 1) ($p<0.001$). Peak troponin-I value was also higher in the COVID-19 group (27335 vs. 15959 ng/dL, $p=0.006$). Mortality risk was higher in COVID-19 patients (38.4% vs. 7.2%, $p<0.001$). TFC and ejection fraction may predict in-hospital mortality among COVID-19 patients with ACS according to logistic regression results. In correlation analysis, TFC correlated positively with C-reactive protein ($r=0.340$, $p<0.001$) and peak troponin-I value ($r=0.369$, $p<0.001$).

Conclusion: COVID-19 is associated with slow coronary flow and microvascular impairment in ACS.

Keywords: Acute myocardial infarction, COVID-19, Myocardial blush grade, Thrombolysis in myocardial infarction flow grade, Thrombolysis in myocardial infarction frame count

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Coronavirus disease 2019 (COVID-19) affects multiple organs as well as the respiratory system. Endothelial dysfunction is considered to have a crucial role in the pathogenesis and progression of the disease.[1] Pro-inflammatory mediators, such as IL-1, IL-6, and TNF-a, cause a state called cytokine storm that especially seen at advanced stages, and leads to vascular endothelial damage, deterioration of endothelial cell functions including thrombotic regulation.[2,3] This process accounts for arterial and venous thrombosis and impairment of microvasculature that can be seen during the disease period.

Thrombolysis in myocardial infarction (TIMI) frame count (TFC) method was developed as a continuous index of coronary blood flow to enable objective and reproducible assessment and thus to overcome limitations of the con-

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ventional TIMI flow grading system.[4] TFC and its clinical value have been evaluated in several studies and higher TFC, which reflects slower coronary flow rate, has been found to be related to poor prognosis in acute coronary syndrome (ACS).[5,6] Although TFC method is also an indirect measure of microvascular impairment and has been demonstrated to have a significant correlation with several inflammatory and vascular biomarkers that reflect endothelial dysfunction, myocardial blush grade (MBG) was developed as a more direct way of assessing myocardial perfusion and found to be a useful parameter in predicting mortality after primary percutaneous coronary intervention (PCI).[7] It has been shown that COVID-19 is related to arterial and venous complications. However, data on the impacts of the disease on coronary microcirculation are scarce. In this study, we aimed to investigate microvascular impairment following primary PCI among COVID-19 patients presenting with ACS using angiographic measures corrected TFC (cTFC) and MBG.

Methods

Study Population

A total of 177 consecutive patients, who presented to our emergency department with ACS, between March 1 and June 30, 2020, were evaluated retrospectively. Eight patients were managed with medical treatment. Coronary artery bypass surgery was performed in four patients. Remaining 165 patients, in whom PCI was performed, enrolled in the study. All patients underwent chest computerized tomography (CT) before coronary angiography. SARS-CoV-2-RT-polymerase chain reaction (PCR) testing was performed if patients had symptoms suggestive of COVID-19 and/or typical CT findings of the disease. Diagnosis of ACS was made according to the current guidelines.[8-10] Information was obtained from the hospital records to determine clinical characteristics. The study was approved by Ethics Committee of our institution (acceptance number and date 1546, 30.06.2020, respectively).

Interventional Procedures

All patients were given 300 mg acetylsalicylic acid, 100 IU/kg unfractionated heparin, and either 600 mg clopidogrel or 180 mg ticagrelor after arrival to the catheterization laboratory. The standard Judkins technique was used with 6F or 7F guiding catheters. Choice of stent type, size and length, pre-dilatation, and post-dilatation were at the discretion of the operator. Bare metal stents (Ephesos II, Alvimedica), everolimus-eluting stents (Xience pro, Abbott), and zotarolimus-eluting stents (Resolute integrity, Medtronic) were used. Conventional coronary angiography and PCI were performed and recorded using the Artis Zee angiography system (Siemens, Munich, Germany).

Angiographic Analysis

Coronary angiograms were analyzed to measure TIMI flow grade, cTFC, and MBG by two interventional cardiologists who were blinded to the clinical characteristics of the patients. Frame counts needed for contrast material to reach standardized distal landmarks were calculated as described by Gibson et al.[4] These landmarks were defined as distal bifurcation of the left anterior descending artery (LAD), the first branch of the posterolateral artery for the right coronary artery, and distal bifurcation with the longest total distance involving the culprit lesion for the circumflex artery. To derive corrected TIMI frame counts, TFCs for the LAD and saphenous vein graft were corrected by dividing 1.7 and 1.6, respectively. Coronary angiograms were filmed at an acquisition rate of 15 f/s; therefore, cTFC values were multiplied by 2 for adjustment. MBG was assessed according to the grading system of Van't Hof et al.[7] Grade 0 was defined as the absence of MBG 3 was defined as normal myocardial blush, similar to non-culprit arteries. Grades 1 and 2 were described as minimal and moderate blush, respectively. Post-PCI TIMI flow grades were assessed according to TIMI trial.[11] The absence of perfusion was Grade 0. If contrast, the material passed the occluded segment, but failed to opacify the distal coronary bed, classified as Grade 1. Grade 2 was defined as achievement of distal coronary bed opacification, but a slower rate of contrast material compared to non-culprit arteries. When antegrade flow or clearance rate of contrast material was similar to non-culprit arteries, classified as Grade 3.

Laboratory Analysis

Blood samples of each patient were obtained in the emergency department before coronary angiography. Troponin-I and creatinine measurements were repeated during the hospitalization period to determine peak value. Samples for complete blood count and biochemistry tests were collected in BD Vacutainer K2E (with EDTA) and BD Vacutainer SST II Advance (BD, Plymouth, UK), respectively. Biochemical tests were performed using Cobas c 501 autoanalyzer (Roche Diagnostics, Mannheim, Germany). Complete blood cell count parameters were obtained with an automated analyzer, XE-2100 (Sysmex, Kobe, Japan). Samples for SARS-CoV-2-RT-PCR test were taken using Biospeedy nasopharyngeal swab in the vNAT transfer tube. COVID-19 qPCR Detection Kit was used for the test. (Bioeksen, Istanbul, Turkey). Echocardiogram was performed using the Philips Epiq system.

Statistical Analysis

Statistical analysis was performed with SPSS Statistics 21.0. Two sample t-test was used for continuous data if followed normal distribution and variables were reported as mean and standard deviation, otherwise Mann–Whitney U-test was performed and variables were defined as median and interquartile range. Categorical data were analyzed by Chi-square test and variables were specified as percentage values. Spearman test was used to analyze the correlation between corrected TIMI frame count and troponin-I and C-reactive protein (CRP). Logistic regression was used for predictors of mortality. Variables that were found significant in the Univariate test were included in the multivariate analysis.

Results

Baseline clinical and laboratory values are reported in Table 1. Twenty-six patients (15.7%) were tested positive for COVID-19. There were no significant differences between the two groups for age, the prevalence of hypertension, diabetes mellitus, smoking, and chronic kidney disease. However, dyslipidemia was more common among the

Table 1. Baseline clinical characteristics and laboratory parameters of the study population

Parameters	COVID-positive (n=26)	COVID-negative (n=139)	P
Age (years)	62.8±13.8	57.6±12.0	0.051
Male gender, n (%)	14 (53.8)	116 (83.5)	0.001
Hypertension, n (%)	16 (61.5)	67 (48.2)	0.21
Diabetes mellitus, n (%)	10 (38.5)	38 (27.3)	0.25
Dyslipidemia, n (%)	5 (19.2)	57 (41)	0.035
Smoking, n (%)	9 (34.6)	70 (50.4)	0.14
Chronic kidney disease, n (%)	3 (11.5)	11 (7.9)	0.46
Previous history of CAD, n (%)	10 (38.5)	31 (22.3)	0.08
Presentation			0.56
STEMI, n (%)	16 (61.6)	87 (62.6)	
Anterior, n (%)	12 (46.2)	42 (30.2)	
Inferior, n (%)	4 (15.4)	34 (24.5)	
Lateral, n (%)	0	6 (4.3)	
Posterior, n (%)	0	5 (3.6)	
NSTE-ACS, n (%)	10 (38.5)	52 (37.4)	
Atrial fibrillation, n (%)	2 (7.7)	6 (4.3)	0.61
Ejection fraction (%)	37.5±1.8	50.6±0.8	<0.001
Prior CABG, n (%)	4 (15.4)	10 (7.3)	0.24
In-hospital mortality, n (%)	10 (38.4)	10 (7.2)	<0.001
Contrast-induced nephropathy, n (%)	6 (23.1)	11 (8.1)	0.035
Length of hospitalization (day)*	6 (3–12)	3 (2–5)	0.002
CRP (mg/L)*	78.1 (39–222)	36.6 (15.1–88)	0.001
Peak troponin-I (ng/dL)*	27335 (12,038–52,808)	15,959 (2813–27,619)	0.006
Creatinine (mg/dL)*	0.91 (0.79–1.27)	0.86 (0.74–1.03)	0.11
eGFR (mL/min/1.73 m^2)*	68.5 (49.5–93.5)	93.0 (73–104)	0.003
Hemoglobin (g/dL)	12.7 ± 2.3	14.4 ± 1.7	<0.001
White blood cell (10^3/mL)	15.4 ± 5.9	11.5 ± 3.6	0.003
Neutrophil (10^3/mL)	11.8 ± 5.9	8.82 ± 1.17	0.20
Lymphocyte (10^3/mL)	2.52 ± 2.2	2.67 ± 1.3	0.64
Platelet (10^3/mm^3)	256.9 ± 80	246.8 ± 73.7	0.52
Mean platelet volume (fL)	9.85 ± 1.33	9.57 ± 1.07	0.24

Data were given as mean ± standard deviation or percentage. *: Median (interquartile range). CAD: Coronary artery disease; STEMI: ST-segment elevation myocardial infarction; NSTE-ACS: Non-ST-elevation acute coronary syndrome; CABG: Coronary artery bypass graft; CRP: C-reactive protein; eGFR: Estimated glomerular filtration rate.

COVID-19-positive group. The previous history of coronary artery disease and atrial fibrillation rates was similar. The most common presentation was ST elevated myocardial infarction in both groups (61.6% of the positive group and 62.6% of the negative group). Patients with COVID-19 were more likely to have lower left ventricular ejection fraction values (37.5% vs. 50.6%, p<0.001). COVID-19 patients significantly had longer hospitalization periods (median 6 days vs. 3 days). In-hospital mortality was also higher in patients with COVID-19 (38.4% vs. 7.2%, p<0.001). While white blood cell counts, CRP, and peak troponin-I values were significantly higher in the COVID-19-positive group, hemoglobin levels and glomerular filtration rates were lower compared to the negative group. After exclusion of patients, which already on dialysis treatment, the development of contrast-induced nephropathy was more often in COVID-19 patients (23.1% vs. 8.1%, p=0.035).

Angiographic parameters are presented in Table 2. Rates of reduced post-PCI TIMI flow grade (Grades 0, 1, and 2) were similar between two groups (13.6% vs. 12%, p=0.56), whereas COVID-19 patients had significantly higher corrected TIMI frame counts (39 vs. 22, p<0.001) and lower MBG (Grades 0 and 1) (57.7% vs. 3.8%, p<0.001). There were no significant differences for procedural characteristics such as pre-dilatation and post-dilatation rates, implanted stent lengths, and diameters. LAD culprit lesions predominated in both groups.

Corrected TIMI frame count (OR: 1.130 CI 1.051–1.214, p=0.001) and ejection fraction (OR: 0.889 CI 0.794–0.996, p=0.042) were predictors of mortality in COVID-19-positive group according to logistic regression test results (Tables 3 and 4). In Spearman test, cTFC correlated positively with CRP (r=0.340, p<0.001) and peak troponin-I value (r=0.369, p<0.001) (Fig. 1a and 1b).

Table 2. Angiographic parameters of the study population

Parameters	COVID-positive (n=26)	COVID-negative (n=139)	p
Post-PCI TIMI flow grade <3, n (%)	3 (12)	18 (13.6)	0.56
Corrected TIMI frame count	39 (30–52)	22 (18–26)	<0.001
Myocardial blush grade <2, n (%)	15 (57.7)	5 (3.8)	<0.001
Multivessel disease, n (%)	13 (50)	86 (60.4)	0.79
Culprit lesion, n (%)			0.31
LAD	13 (50)	65 (46.7)	
RCA	8 (30.8)	41 (29.5)	
Cx	3 (11.5)	30 (21.6)	
Saphenous vein graft	2 (7.7)	3 (2.2)	
Stent diameter (mm)	3 (2.75–3)	3 (2.75–3)	0.15
Stent length (mm)	23 (18–36.7)	26 (18–34)	0.78
Pre-dilatation, n (%)	21 (80.8)	124 (89.2)	0.32
Post-dilatation, n (%)	16 (61.5)	98 (70.5)	0.45

Data were given as median (interquartile range) or percentage. PCI: Percutaneous coronary intervention; TIMI: Thrombolysis in myocardial infarction; LAD: Left anterior descending artery; RCA: Right coronary artery; Cx: Circumflex artery.

Table 3. Univariate logistic regression analysis for independent predictors of in-hospital mortality

Variables	Univariate logistic regression analysis	
	OR (%95 CI)	p
Corrected TIMI frame count	1.168 (1.090–1.252)	<0.001
Ejection fraction	0.841 (0.766–0.923)	<0.001
Myocardial blush grade	0.678 (0.380–1.209)	0.188
Age	1.028 (0.978–1.081)	0.272
CRP (mg/L)	1.006 (1.001–1.012)	0.025
Hemoglobin (g/dL)	0.808 (0.642–1.016)	0.069

TIMI: Thrombolysis in myocardial infarction; CRP: C-reactive protein.

Table 4. Multivariate logistic regression model for independent predictors of in-hospital mortality

Variables	Multivariate logistic regression analysis	
	OR (%95 CI)	p
Corrected TIMI frame count	1.130 (1.051–1.214)	0.001
Ejection fraction	0.889 (0.794–0.996)	0.042

TIMI: Thrombolysis in myocardial infarction.

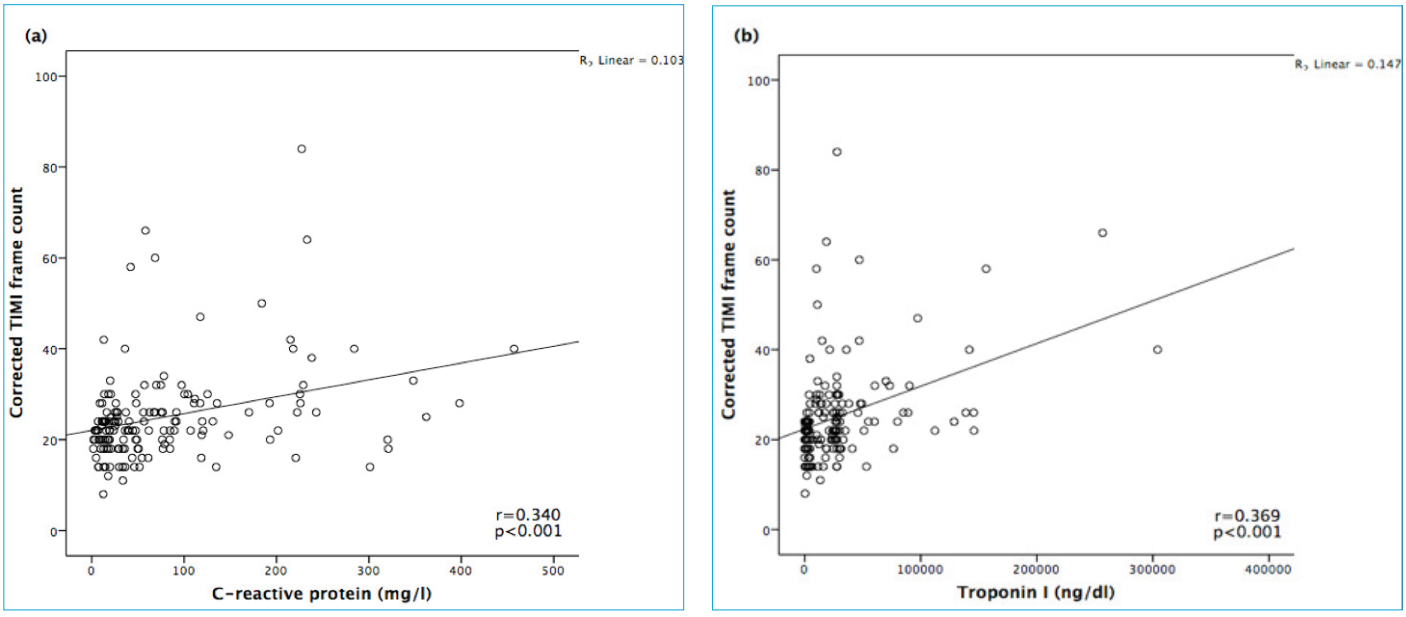

Figure 1. Correlation of corrected TIMI frame count with C-reactive protein (a) and troponin I (b).

Discussion

In our study, we demonstrated that COVID-19 patients with ACS had significantly higher cTFC and lower MBG values which may reflect impairment of epicardial flow and microvasculature. Furthermore, cTFC and ejection fraction were related to mortality risk among COVID-19-positive group.

Endothelial dysfunction is considered to have a major role in the pathophysiology of COVID-19 and accounts for different clinical presentations such as pulmonary thromboembolism, multiorgan infarctions, and failures during disease period. It has been hypothesized that Sars-Cov-2 targets endothelial cells, which are expressing angiotensin-converting enzyme 2, and causes damage and impairs their functions; this impairment leads to hyperinflammation, hypercoagulable state, and eventually microvascular thrombosis through triggering complement, platelet, and leukocyte activation.[1]

Although cTFC is considered to reflect only epicardial blood flow, it may be also a parameter of microvascular function.[12,13] Several inflammatory mediators and endothelial markers of injury were demonstrated to be associated with slow coronary flow.[14-16] Hyperinflammation and endothelial cell damage seen in COVID-19 may account for higher cTFC values observed in our study.

Previously, O. Rodriguez-Leor et al.[17] reported increased rates of stent thrombosis, in-hospital mortality, and major adverse cardiovascular events in COVID-19 patients with ST-segment elevation myocardial infarction (STEMI). Furthermore, Choudry et al.[18] showed lower MBGs and higher rates of stent thrombosis in COVID-19 and STEMI patients. Although we did not report any stent thrombosis in both groups, similarly, we also demonstrated lower MBGs in COVID-19-positive group that may be due to microvascular thrombosis and/or thrombus embolization to distal coronary microcirculation after PCI. We found significantly lower ejection fraction and higher peak troponin-I values among COVID-19 patients compared with the negative group. These findings may provide more evidence of greater impairment of tissue-level perfusion in COVID-19. Nevertheless, higher peak troponin-I values and lower ejection fraction of COVID-19 patients may be due to the higher rate of anterior MI in this group. In addition, a previous history of coronary artery disease that

seen more common in COVID-19 group may cause lower ejection fraction as well.

In the present study, we observed 2 times longer hospitalization period in COVID-19 patients compared to COVID-19-negative patients. It is reasonable for a disease affecting multiple organ systems as well as severe cardiac involvement. In a postmortem case series of 26 patients, electron microscopic findings demonstrated coronavirus-like particles in the renal tubular epithelium.[19]

We found that estimated glomerular filtration rate was significantly lower on admission and contrast-induced nephropathy was more often in COVID-19 patients, which may reflect concomitant renal injury due to direct infection of the tubular epithelium.

Predictive and prognostic values of cTFC and MBG in patients with ACS were well-known from previous studies. Furthermore, we showed that cTFC and ejection fraction may predict mortality among COVID-19-positive group. The main goal of reperfusion therapies is to provide myocardial reperfusion. As previously said, COVID-19 is considered as a disease of microvascular dysfunction. Developing therapies for COVID-19 based on the microvasculature may improve cardiovascular outcomes of the disease as well.

This is a single-center trial and the main limitation of the study is a small sample size. The number of events is low. Furthermore, we did not take samples for SARS-CoV-2-RT-PCR test from patients without any suggestive symptoms or CT findings. Furthermore, it has a retrospective design, and follow-up data of patients are absent.

Conclusion

COVID-19 is associated with slow coronary flow and microvascular impairment, reflected as higher cTFC and lower MBG, in patients with ACS. Corrected TIMI frame count and ejection fraction may predict mortality among COVID-19-postive group.

Disclosures

Ethics Committee Approval: The study was approved by the Ethics Committee of University of Health Sciences Türkiye, Sisli Hamidiye Etfal Training and Research Hospital (No: 1546, dated 30.06.2020).

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Conflict of Interest: None declared.

Authorship Contributions: Concept – O.A., E.K.; Design – S.S., E.K.; Supervision – O.A., K.K., S.S.; Fundings – K.K., O.S.S., G.M.D.; Materials – G.M.D., M.C.S.; Data collection and/or processing – G.M.D., M.C.S.; Analysis and/or interpretation – K.K., S.S., M.C.S.; Literature review – O.S.S.; Writing – E.K., O.A.; Critical review – O.S.S., E.K., O.A., M.C.S., G.M.D., S.S., K.K.

References

1. Gavriilaki E, Anyfanti P, Gavriilaki M, Lazaridis A, Douma S, Gkaliagkousi E. Endothelial dysfunction in COVID-19: lessons learned from coronaviruses. Curr Hypertens Rep 2020;22:63. [CrossRef]
2. Libby P, Lüscher T. COVID-19 is, in the end, an endothelial disease. Eur Heart J 2020;41:3038-44. [CrossRef]
3. Li M, Chen S, Xiang X, Wang Q, Liu X. Effects of SARS-CoV-2 and its functional receptor ACE2 on the cardiovascular system. Herz 2020;45:659-62. [CrossRef]
4. Gibson CM, Cannon CP, Daley WL, Dodge JT Jr, Alexander B Jr, Marble SJ, et al. TIMI frame count: a quantitative method of assessing coronary artery flow. Circulation 1996;93:879-88. [CrossRef]
5. Gibson CM, Dotani MI, Murphy SA, Marble SJ, Dauterman KW, Michaels AD, et al; RESTORE Investigators. Correlates of coronary blood flow before and after percutaneous coronary intervention and their relationship to angiographic and clinical outcomes in the RESTORE trial. Randomized Efficacy Study of Tirofiban for Outcomes and REstenosis. Am Heart J 2002;144:130-5. [CrossRef]
6. Gibson CM, Murphy SA, Rizzo MJ, Ryan KA, Marble SJ, McCabe CH, et al. Relationship between TIMI frame count and clinical outcomes after thrombolytic administration. Thrombolysis In Myocardial Infarction (TIMI) Study Group. Circulation 1999;99:1945-50. [CrossRef]
7. van 't Hof AW, Liem A, Suryapranata H, Hoorntje JC, de Boer MJ, Zijlstra F. Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Zwolle Myocardial Infarction Study Group. Circulation 1998;97:2302-6. [CrossRef]
8. Thygesen K. 'Ten Commandments' for the fourth universal definition of myocardial infarction 2018. Eur Heart J 2019;40:226. [CrossRef]
9. Collet JP, Thiele H, Barbato E, Barthélémy O, Bauersachs J, Bhatt DL, et al; ESC Scientific Document Group. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J 2021;42:1289-1367. Erratum in: Eur Heart J 2021;42:1908. Erratum in: Eur Heart J 2021;42:1925. Erratum in: Eur Heart J 2021 May 13. [CrossRef]
10. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al; ESC Scientific Document Group. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: the task force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39:119-77. [CrossRef]
11. TIMI Study Group. The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. N Engl J Med 1985;312:932-6. [CrossRef]
12. Sun H, Fukumoto Y, Ito A, Shimokawa H, Sunagawa K. Coronary microvascular dysfunction in patients with microvascular angina: analysis by TIMI frame count. J Cardiovasc Pharmacol 2005;46:622-6. [CrossRef]

13. Zalewski J, Zmudka K, Musialek P, Zajdel W, Pieniazek P, Kadzielski A, et al. Detection of microvascular injury by evaluating epicardial blood flow in early reperfusion following primary angioplasty. Int J Cardiol 2004;96:389-96. [CrossRef]
14. Li JJ, Qin XW, Li ZC, Zeng HS, Gao Z, Xu B, et al. Increased plasma C-reactive protein and interleukin-6 concentrations in patients with slow coronary flow. Clin Chim Acta 2007;385:43-7. [CrossRef]
15. Selcuk MT, Selcuk H, Temizhan A, Maden O, Ulupinar H, Baysal E, et al. Asymmetric dimethylarginine plasma concentrations and L-arginine/asymmetric dimethylarginine ratio in patients with slow coronary flow. Coron Artery Dis 2007;18:545-51. [CrossRef]
16. Turhan H, Saydam GS, Erbay AR, Ayaz S, Yasar AS, Aksoy Y, et al. Increased plasma soluble adhesion molecules; ICAM-1, VCAM-1, and E-selectin levels in patients with slow coronary flow. Int J Cardiol 2006;108:224-30. [CrossRef]
17. Rodriguez-Leor O, Alvarez ABC, de Prado AP, Rossello X, Ojeda S, Serrador A, et al. In-hospital outcomes of COVID-19 ST elevation myocardial infarction patients. EuroIntervention 2021;16:1426-33. [CrossRef]
18. Choudry FA, Hamshere SM, Rathod KS, Akhtar MM, Archbold RA, Guttmann OP, et al. High Thrombus burden in patients with COVID-19 presenting with ST-segment elevation myocardial infarction. J Am Coll Cardiol 2020;76:1168-76. [CrossRef]
19. Su H, Yang M, Wan C, Yi LX, Tang F, Zhu HY, et al. Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China. Kidney Int 2020;98:219-27. [CrossRef]