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ORIGINAL ARTICLE

Predictors of Worse Outcome in Covid 19 Patients Presenting with Acute Coronary Syndrome

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ABSTRACT

Background: Acute coronary syndrome (ACS) in COVID-19 patients is thought to arise from atherosclerotic plaque rupture due to endothelial damage, exacerbated by cytokine storms and inflammatory responses. This work aimed to detect the role of different demographic, clinical, functional, angiographic and laboratory data in prediction of ACS occurrence and worsening in patients with Covid 19.

Methods: This case-control study involved 120 patients, with over the age of 18 years, both genders, with confirmed COVID-19 diagnosis and suffering from ACS. Three categories of patients were identified: Group A: with COVID 19 and ACS, Group B: without COVID 19 and presenting with ACS and Group C: control group with COVID 19 and no ACS.

Results: CK-MB (AUC 0.634, p=0.036, cutoff ≤60 U/L) was a significant predictor of adverse events with 62.07% sensitivity, 50.55% specificity, 28.6% PPV, and 80.7% NPV. EF >50% (AUC 0.690, p<0.001) also predicted adverse events with 75.86% sensitivity, 40.66% specificity, 28.9% PPV, and 84.1% NPV. Univariate and multivariate analyses identified significant predictors of mortality: age, ARDS, D-dimer, CRP (admission/peak), ferritin, AST, CK-MB, EF, Killip class, GRACE score, final TIMI flow, and COVID-19. However, multivariate analysis found GRACE score and COVID-19 as independent mortality predictors.

Conclusions: COVID-19 is a reliable indicator of mortality in ACS patients, with the GRACE score being an essential tool for risk assessment.

Keywords: Acute Coronary Syndrome; Covid 19; Global Registry of Acute Coronary Events; Mortality

INTRODUCTION

In the early phases of the 2020 COVID-19 pandemic, concerns were voiced over the possible involvement of the cardiovascular system. Later, this was evident by the occurrence of a lot of cardiovascular complications that were the leading cause of both clinical deterioration and even death in some cases [1]. Reported cardiovascular involvements were such as pulmonary thromboembolism, acute coronary syndrome (ACS), myocarditis, arrhythmias, cardiomyopathy [2].

The nature of the virus and how it gets into cells was the first clue for suspicion of cardiovascular complication. Given the evidence that myocytes internalize the coronavirus through an interaction with angiotensin converting enzyme 2 receptors on the cell membrane. This carries the suspicion of a direct toxic effect of the virus on cells [3].

It is important also to emphasize other postulated mechanisms for cardiovascular affection by corona virus such as a supply-demand mismatch represented in decreased oxygen supply owing to hypoxia resulting from severe respiratory affection and increased tissue demands due to sepsis [4].

Lastly, it was discovered that in serious cases of Covid-19 systemic elevates of several cytokines

which cause the well-known. Cytokine storm leads to coronary plaque instability and rupture with initiation of the coronary thrombus formation and altered vascular permeability which leads to acute respiratory distress syndrome (ARDS) and non-cardiogenic pulmonary edema [5].

We hypothesized that there are predictors (demographic, clinical, laboratory, angiographic and functional) that can predict occurrence and worsening of ACS in Covid 19 patients.

This work aimed to detect the role of different demographic, clinical, functional, angiographic and laboratory data in prediction of ACS occurrence and worsening in patients with Covid 19.

MATERIAL

This case-control study involved 120 patients, aged 18 years or older, of both genders, who had a definitive diagnosis of COVID-19 and were experiencing ACS [ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), or unstable angina].

The study was performed from March 2021 to October 2021 with the approval of the Ethical Committee of Zagazig University Hospitals in Zagazig, Egypt (Approval No: 9019-12-10-20221). The participants or their relatives provided informed written assent.

Patients with a history of ACS, MI accompanied by LV dysfunction or pre-existing structural heart disease, serious co-morbidities like advanced cancer, end-stage renal disease, or severe chronic obstructive pulmonary disease, infections other than COVID-19, patients undergoing thrombolytic treatment, pregnant women, and those in the postpartum period were excluded from the study.

Three categories of patients were identified: Group A: with COVID 19 and ACS, Group B: without COVID 19 and presenting with ACS and Group C: control group with COVID 19 and no ACS.

All patients underwent a comprehensive history-taking, thorough clinical examination, Killip classification, global registry of acute coronary events (GRACE) score, laboratory investigations [complete blood count, lymphocyte counts, inflammatory markers (IL-6, TNF-α), D-dimer (g/L), serum ferritin (ng/mL), serum lactate dehydrogenase (LDH) (U/L), fasting glucose level, creatinine phosphokinase (CPK), electrolyte Pannel, liver function tests, N-terminal pro-brain natriuretic peptide (NT-ProBNP) (ng/L), lipid profile, C-reactive protein (CRP), kidney function tests, creatine kinase-myocardial band (CK-MB) and troponin] and radiological investigations [electrocardiogram (ECG), baseline ECG, echocardiography, coronary angiography, TIMI flow grade and computed tomography (CT)].

Diagnosis of COVID 19

One diagnostic tool is RT-PCR, which uses samples from bronchoalveolar lavage (BAL), tracheal aspirate, or nasal swabs. Sampling the nasal and oral cavities using nasopharyngeal and oropharyngeal swabs is the gold standard and main diagnostic method. The danger that the produced aerosol presents to both patients and healthcare workers makes bronchoscopy an unsuitable diagnostic tool for COVID-19 [6]. Here are the many types of COVID-19 cases: Respiratory rate is deemed moderate when symptoms are modest, including an upper respiratory tract infection and fever, but there is no hypoxia or shortness of breath. Severe symptoms are indicated by a respiratory rate of 24–30 breaths per minute and a SpO_2 level of 90–93% on room air.

Study endpoint

Cardiovascular death, recurrent acute myocardial infarction (AMI), repeat revascularization (including bypass graft) and stent thrombosis are all considered major adverse cardiovascular events (MACEs) in the setting of percutaneous coronary procedures (PCI).

Statistical analysis

We utilized SPSS v26, which is developed by IBM and located in Chicago, IL, USA, to conduct statistical study. We employed analysis of variance (ANOVA) with post hoc tests

(Tukey) to compare the quantitative variables across the three groups. Displayed were the means and standard deviations (SDs). The Chi-square test was employed to assess the qualitative variables, which were presented as percentages and frequencies. The diagnostic performance was evaluated by the Roc curve, which assessed the sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV). The use of univariate regression allowed for the estimation of the relationship between the dependent and the one independent variable. We additionally employed multivariate regression to assess the relationship between our dependent variable and a plethora of independent factors. The significance level was determined using a two-tailed P value that was less than 0.05.

RESULTS

Group A had significantly higher rates of HTN, DM, prior stroke, and PCI than group B (P<0.05). Baseline characteristics, other medical history (hyperlipidaemia, ARDS, COPD, malignancy, and prior MI) and the groups didn't exhibit a significant difference in EF. In comparison to B, groups A and C showed significantly greater CK-MB levels (P<0.05). On the other hand, groups A and C did not differ significantly. Compared to groups B and C, group A had much greater levels of HS-Troponin-I and Troponin- T (P<0.05) and were significantly elevated in group B in comparison with group C (P<0.05). Group B had considerably higher troponin-I levels compared to groups A and C (P<0.05), but groups A and B did not vary significantly. **Table 1**

A considerably higher GRACE score was seen in group A when compared to group C (P<0.001), and in group B when compared to groups A and C (P<0.05). A considerably higher GRACE score was seen in group A when compared to group C (P<0.001), and in group B when compared to groups A and C (P<0.05). Groups A and B did not differ significantly with respect to Killip class. Both group A and B had substantially different MR values (P<0.05). Other findings were comparable among both groups. There were notable differences in ECG between the groups that were tested (P<0.05). **Table 2**

There was a substantial difference in mortality and mechanical ventilation across the groups (P<0.05), with group A having the highest rates. Ischemic stroke and myocardial re-infarction were insignificantly different in the groups. **Table 3**

CK-MB was a significant predictor for the occurrence of adverse events with AUC of 0.634 and P value of 0.036, at cutoff value ≤60 U/L with 62.07% sensitivity, 50.55% specificity, 28.6% PPV and 80.7% NPV. EF was a significant indicator for occurrence of adverse events with AUC of 0.690 and P value of <0.001, at cutoff value >50 % with 75.86% sensitivity, 40.66% specificity, 28.9% PPV and 84.1% NPV. Whereas D-dimer, CRP, ferritin, HS-Troponin-I, troponin- T and troponin-I were insignificant indicators for the incidence of adverse events. **Figure 1**

The univariate logistic regression analysis revealed that age, ARDS, D-dimer, CRP (at admission and peak), ferritin, AST, CK-MB, EF, Killip class, GRACE, final TIMI flow and COVID-19 were significant risk factors and predictors for mortality. When it came to mortality risk factors and predictors, multivariate logistic regression analysis only revealed GRACE and COVID-19 as significant. **Table 4**

Case 1: A male patient with 73-year-old who was presented to emergency unit by typical angina chest pain that started two hours before presentation and was associated with progressive dyspnea and diaphoresis. He was a non-smoker, hypertensive, non-diabetic with history of COPD, non-hyperlipidemic, no CKD, no malignancy, no previous stroke, no previous MI or PCI. On examination, his height is 169 cm, weight is 57 kg, BMI was 19.9, his heart rate was 92b/m, blood pressure was 130/80mmhg, his Killip classification at the time of admission was class II. ECG at the time of admission showed extensive anterior STEMI

in all anterior and lateral leads with regular sinus rhythm. The Echo was done and showed hypokinesia in anterior, apical, apico septal and apicolateral regions with EF=50%. Laboratory investigations showed: CK-MB : 29 IU/L, HS troponin I : 2036 pg/ml, troponin T :652ng/L, troponin I : 1830 ng/L, urea =54mg/dl, creatinine : 0.9 mg/dl, K: 4.4mmol/L, Na : 145mmol /L, AST : 47 U/L, ALT :29 U/L, cholesterol : 154 mg/dl, LDL=157 mg/dl, HDL: 48 mg/dl, TG:158 mg/dl, HB : 12.2 gm/dL, PLT : 193000, WBCs : 7.2 X109/L. In order to PCI, the patient was admitted to the catheterization laboratory. On angiography Coronaries showed three vessel disease with totally occluded LAD from its proximal part (GRACE score was 120) TIMI flow was 0. Two stents were implanted and TIMI flow at the end was TIMI II. CRP at admission was 12.07 mg/dl and reached a peak of 182.73mg/dl after confirming Covid 19 infection by positive swab, serum ferritin was 1191 ng/ml and D-dimer after 2 days reached 3.1 gm. There was no stroke or reinfarction. Deterioration of respiratory function occurred after two days of admission and the patient developed ARDs and mortality occurred. **Figure 2**

Case 2: 41-year-old female patient who was presented to Mahalla cardiac center emergency unit by Typical anginal chest pain that started 8 hours before presentation and was associated with progressive dyspnea. She was a non-smoker, non-hypertensive, non-diabetic with no history of COPD, non-hyperlipidemic, no CKD, no malignancy, no previous stroke, no previous MI or PCI. On examination, her height is 168 cm, weight is 76 kg, BMI was 26.93, her heart rate was 87b/m, blood pressure was 130/80 mmHg, her Killip classification at the time of admission was class I. ECG at the time of admission was normal with regular sinus rhythm. The Echo was done and showed hypokinesia in inferior, posterior and apicolateral regions with EF=51% with mild MR. Laboratory investigations showed: CK-MB : 60IU/L, Hs troponin I : 1817pg/mL, troponin T :287 ng/L, troponin I : 1817ng/L, urea =22mg/dl, creatinine : 0.7 mg/dl, K: 4.1mmol/l, Na : 139 mmol/l, AST : 51U/L, ALT :47 U/L, cholesterol: 194 mg/dl, LDL=153 mg/dl, HDL: 57 mg/dl *TG:190 mg/dl, HB: 11.2gm/dl, PLT: 222000, WBCs: 11X109/L. Catheterization Laboratory admission was made for main PCI. On angiography Coronaries showed 2 vessel disease with totally occluding LCX (GRACE score was 143) TIMI flow was I. Two stents were implanted and TIMI flow at the end was TIMI III. CRP at admission was 24.4 mg/dl and declined to reach 11.27mg/dl. Serum ferritin was 80 ng/ml. D dimer after 2 days reached 0.47 gm. There was no stroke or reinfarction and no mortality. **Figure 3**

Table 1: Baseline characteristics, medical history of the studied groups

		Group A (n=40)	Group B (n=40)	Group C (n=40)	P
Baseline characteristics					
Age (years)		58.55±11.2	55.9±9.15	56.4±10.58	0.482
Sex	**Male**	24(60.0%)	26(65.0%)	30(75.0%)	0.350
	Female	16(40.0%)	14(35.0%)	10(25.0%)	
Weight (Kg)		69.55±8.26	69.58±9.2	69.95±9.32	0.975
Height (m)		1.64±0.05	1.63±0.05	1.62±0.05	0.970
BMI (Kg/m^2)		25.9 ±3.64	26.18±3.82	26.7±3.73	0.677
Smoking		16 (40.0%)	11(27.5%)	8(20.0%)	0.139
Medical history					
HTN		25(62.5%)	19(47.5%)	13(32.5%)	**0.027***
DM		22(55.0%)	13(32.5%)	9(22.5%)	**0.008***
Hyperlipidemia		14(35.0%)	10(25.0%)	5(12.5%)	0.062
CKD		14(35.0%)	6(15.0%)	4(10.0%)	**0.013***
ARDS		7(17.5%)	3(7.5%)	5(12.5%)	0.401
COPD		9(22.5%)	5(12.5%)	3(7.5%)	0.147
Malignancy		4(10.0%)	3(7.5%)	1(2.5%)	0.392
Prior stroke		7(17.5%)	5(12.5%)	0(0.0%)	**0.027***
Prior MI		10(25.0%)	6(15.0%)	3(7.5%)	0.099
Prior PCI		11(27.5%)	6(15.0%)	0(0.0%)	**0.002***
Cardiac biomarkers					
CK-MB (U/L)		68.4±22.8	56.3±5.04	63.4±21.95	**0.016***
P1=0.002*, P2= 0.325, **P3=0.048***					
HS-Troponin-I (ng/L)		2039.9±885.2	2957.6±1004.5	235.8±98.3	**0.002***
P1<0.001*, P2<0.001*, P3<0.001*					
Troponin- T (ng/L)		588.4±118.1	180.45±109.05	94.88±51.5	**<0.001***
P1<0.001*, P2<0.001*, P3<0.001*					
Troponin-I (ng/L)		1755.3±790.2	1858.4±894.14	55.8±15.37	**<0.001***
P1=0.586, **P2<0.001*, P3<0.001***					
EF (%)		51.78±3.22	52.65±2.72	51.9±3.15	0.382

Data is presented as mean ± SD or frequency (%). * Significant P value < 0.05. P1: P value between groups A and B, P2: P value between groups A and C, P3: P value between groups B and C, BMI: Body mass index. HTN: Hypertension, DM: Diabetes mellitus, CKD: Chronic kidney disease, ARDS: Acute respiratory distress syndrome, COPD: chronic obstructive pulmonary disease, MI: Myocardial infarction, PCI: Percutaneous Coronary Intervention, CK-MB: creatine kinase-myocardial band, HS: high sensitivity, EF: Ejection fraction.

Table 2: Clinical scores, coronary angiography findings and ECG of the studied groups

		Group A (n=40)	Group B (n=40)	Group C (n=40)	P
Clinical scores					
Killip class	**I**	20(50.0%)	18(45.0%)	---	0.920
	II	9(22.5%)	11(27.5%)	---	
	III	7(17.5%)	6(15.0%)	---	
	IV	4(10.0%)	5(12.5%)	---	
GRACE		105.03±14.3	124.9±14.1	60.4±6.17	<0.001*
P1<0.001*, P2<0.001*, P3<0.001*					
Coronary angiography findings					
Extent of CAD	**1-vessel**	14(35.0%)	12(30.0%)	0(0.0%)	0.288
	2- vessels	12(30.0%)	15(37.5%)	0(0.0%)	
	3- vessels	8(20.0%)	3(7.5%)	0(0.0%)	
	No lesion	6(15.0%)	10(25.0%)	40(100.0%)	
Culprit vessel	**LM**	2(5.0%)	1(2.5%)	----	0.870
	LAD	18(45.0%)	16(40.0%)	----	
	LCX	12(30.0%)	13(32.5%)	----	
	RCA	8(20.0%)	10(25.0%)	----	
TIMI flow before	**0**	10(25.0%)	11(27.5%)	----	0.750
	1	14(35.0%)	13(32.5%)	----	
	2	9(22.5%)	6(15.0%)	----	
	3	7(17.5%)	10(25.0%)	----	
Final TIMI flow	**0**	0(0.0%)	0(0.0%)	----	0.715
	1	2(5.0%)	1(2.5%)	----	
	2	1(2.5%)	2(5.0%)	----	
	3	35(87.5%)	37(92.5%)	----	
Stent number		2.13±1	1.85±1.15	---	0.258
MR	**Mild**	16(40.0%)	31(77.5%)	---	0.002*
	Moderate	22(55.0%)	9(22.5%)	---	
	Severe	2(5.0%)	0(0.0%)	---	
ACS	**STEMI**	29(72.5%)	24(60.0%)	---	0.103
	NSTEMI	11(27.5%)	16(40.0%)	---	
ECG					
Normal		10(25.0%)	5(12.5%)	39(97.5%)	<0.001*
ST-segment depression		8(20.0%)	18(45.0%)	1(2.5%)	
ST-segment elevation		22(55.0%)	17(42.5%)	0(0.0%)	

Data is presented as mean ± SD or frequency (%). * Significant P value < 0.05. P1: P value between groups A and B, P2: P value between groups A and C, P3: P value between groups B and C, GRACE: global registry of acute coronary events, CAD: coronary artery disease, LM: left main coronary artery, LCX: left circumflex coronary artery, LAD: left anterior descending coronary artery, RCA: right coronary artery, STEMI: ST-segment elevation myocardial infarction, NSTEMI: non ST-segment elevation myocardial infarction, TIMI: thrombolysis in myocardial infarction, MR: Mitral regurgitation, ECG: electrocardiogram.

Table 3: Outcome and adverse events of the studied groups

	Group A (n=40)	Group B (n=40)	Group C (n=40)	P
Ischemic stroke	2(5.0%)	1(2.5%)	3(7.5%)	0.590
Mortality	12(30.0%)	5(12.5%)	4(10.0%)	**0.037***
Myocardial re-infarction	6(15.0%)	3(7.5%)	4(10.0%)	0.547
Mechanical ventilation	8(20.0%)	0(0.0%)	5(12.5%)	**0.015***

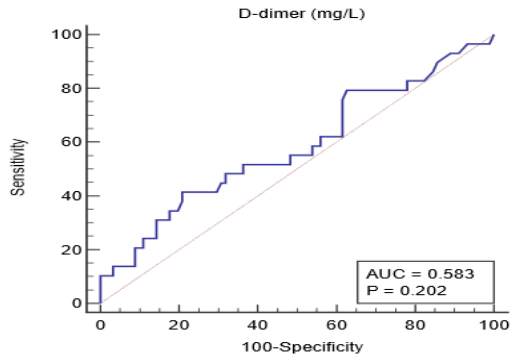
Data is presented as frequency (%). * Significant P value < 0.05.

Table 4: Logistic regression analysis of the risk factors predicting mortality

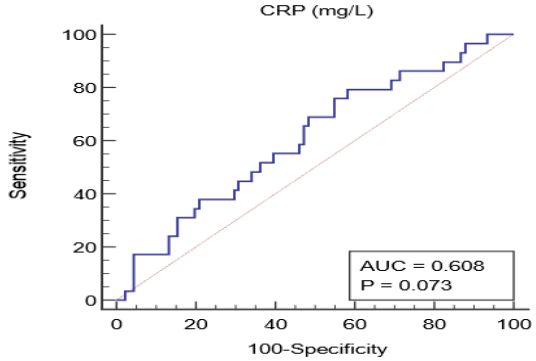
	Univariate			Multivariate		
	OR	**95%CI**	**P**	**OR**	**95%CI**	**P**
Age (years)	1.0489	1.0096 to 1.0897	**0.014***	0.9734	0.8736 to 1.0848	0.626
Sex	0.2793	0.0770 to 1.0128	0.052	0.3289	0.0887 to 1.2192	0.096
BMI (Kg/m^2)	0.9675	0.8623 to 1.0855	0.574	0.9799	0.8823 to 1.0882	0.704
Smoking	0.2526	0.0521 to 1.2259	0.088	0.6461	0.1264 to 3.3042	0.599
HTN	0.123	0.0384 to 0.3979	0.054	1.8481	0.5951 to 5.7396	0.288
DM	0.5754	0.2222 to 1.4901	0.255	4.9110	0.7300 to 33.036	0.101
Hyperlipidemia	1.0010	0.9933 to 1.0088	0.794	0.4925	0.1665 to 1.4567	0.200
CKD	1.0176	0.9751 to 1.0619	0.423	1.1350	0.5081 to 2.5352	0.757
ARDS	0.1236	0.0384 to 0.3979	**<0.001***	0.2205	0.0467 to 1.0408	0.056
COPD	1.1609	0.9912 to 1.3598	0.064	1.2455	0.5917 to 2.6218	0.563
Malignancy	1.2416	0.8140 to 1.8939	0.315	0.4481	0.0363 to 5.5316	0.531
Prior stroke	1.0049	0.9889 to 1.0212	0.550	0.3108	0.0558 to 1.7317	0.182
Prior MI	1.1725	0.8101 to 1.6970	0.398	1.8320	0.913 to 3.6745	0.088
Prior PCI	0.4414	0.1368 to 1.4244	0.171	11.2976	0.7347 to 173.72	0.082
HR (beat/min)	1.0140	0.9674 to 1.0629	0.562	0.9997	0.9472 to 1.0551	0.992
SBP (mmHg)	1.0161	0.9549 to 1.0812	0.614	1.0498	0.9745 to 1.1310	0.200
DBP (mmHg)	1.0204	0.9558 to 1.0893	0.545	1.0346	0.9599 to 1.1151	0.373
Hb (g/dL)	1.3538	0.5946 to 3.0822	0.471	1.5701	0.6035 to 4.0849	0.355
PLT (*109/L)	0.9974	0.9894 to 1.0055	0.528	0.9979	0.9894 to 1.0064	0.626
WBCs (*109/L)	1.0579	0.8084 to 1.3845	0.682	1.3624	0.9326 to 1.9903	0.110
D-dimer (mg/L)	0.1595	0.0500 to 0.5093	**0.002***	1.4025	0.9227 to 2.1318	0.113
CRP at admission (mg/L)	0.2613	0.0969 to 0.7043	**0.008***	0.9955	0.9861 to 1.0049	0.344
CRP peak (mg/L)	1.0057	1.0004 to 1.0111	**0.036***	1.006	0.9987 to 1.0133	0.108
Ferritin (ng/mL)	1.0030	1.0010 to 1.0050	**0.003***	1.0004	0.9987 to 1.0022	0.633
ALT (U/L)	0.9672	0.9218 to 1.0148	0.173	0.9791	0.9283 to 1.0327	0.438
AST (U/L)	0.1034	0.0360 to 0.2969	**<0.001***	1.017	0.9678 to 1.0688	0.504
Creatinine (mg/dL)	8.6947	0.9774 to 77.3430	0.052	11.8936	0.398 to 355.141	0.153
Urea (mg/dL)	0.9811	0.9393 to 1.0247	0.389	0.9501	0.8986 to 1.0046	0.072
K^+ (mEq/L)	0.4842	0.0730 to 3.2108	0.452	0.3507	0.0310 to 3.9636	0.397
Na^+ (mEq/L)	0.9876	0.8390 to 1.1626	0.881	0.9765	0.7893 to 1.2081	0.827
Cholesterol (mg/dL)	1.0076	0.9952 to 1.0201	0.229	1.0015	0.9819 to 1.0215	0.881
TG(mg/dL)	1.0011	0.9878 to 1.0145	0.875	0.9818	0.9599 to 1.0043	0.112
HDL (mg/dL)	0.9638	0.8987 to 1.0335	0.301	0.9206	0.8369 to 1.0128	0.089
LDL (mg/dL)	0.9924	0.9734 to 1.0117	0.435	0.9794	0.9520 to 1.0076	0.151
CK-MB (U/L)	0.9711	0.9442 to 0.9988	**0.041***	0.9717	0.9441 to 1.0002	0.051
HS-Troponin-I (ng/L)	1.0002	0.9998 to 1.0005	0.301	1.0001	0.9997 to 1.0006	0.541
Troponin- T (ng/L)	1.0008	0.9997 to 1.0019	0.164	0.9999	0.9994 to 1.0008	0.731
Troponin-I (ng/L)	1.0002	0.9998 to 1.0006	0.348	0.9998	0.9992 to 1.0004	0.564
EF (%)	0.0587	0.0179 to 0.1927	**<0.001***	1.1528	0.9810 to 1.3546	0.084
Killip class	0.0515	0.0095 to 0.2794	**0.001***	1.2410	0.7910 to 1.9470	0.347
GRACE	0.2435	0.0688 to 0.8622	**0.028***	1.0492	1.0098 to 1.0902	**0.014***
Extent of CAD	1.7500	0.5911 to 5.1814	0.312	1.5393	0.8208 to 2.8867	0.179
Culprit vessel	2.4286	0.7586 to 7.7753	0.135	11.0757	0.3579 to 342.75	0.169
Final TIMI flow	0.2759	0.0928 to 0.8203	**0.020***	0.4436	0.1473 to 1.3353	0.148
Stent number	1.0934	0.6391 to 1.8705	0.745	0.5219	0.1857 to 1.4664	0.217
TR	1.0796	0.6053 to 1.9255	0.795	0.9669	0.3990 to 2.3430	0.941
STEMI/NSTEMI	1.7513	0.9141 to 3.3554	0.091	0.5121	0.2074 to 1.2644	0.147
COVID-19	1.5642	1.0084 to 2.4261	**0.045***	1.0463	1.0068 to 1.0874	**0.021***

BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, CKD: chronic kidney disease, ARDS: acute respiratory distress syndrome, COPD: chronic obstructive pulmonary disease, MI: myocardial infarction, PCI: percutaneous coronary intervention, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: haemoglobin, PLT: platelet, WBCs: white blood cells, CRP: C-reactive protein, ALT: alanine

aminotransferase, AST: aspartate aminotransferase, K: potassium, Na: sodium, HDL: high density lipoprotein, LDL: low density lipoprotein, CK-MB: creatine kinase-myocardial band, HS: high sensitivity, EF: ejection fraction, GRACE: global registry of acute coronary events, STEMI: ST-segment elevation myocardial infarction, NSTEMI: non ST-segment elevation myocardial infarction, TIMI: thrombolysis in myocardial infarction, TR: tricuspid regurgitation, OR: odds ratio, CI: confidence interval, *: statistically significant as P value <0.05.



(A)



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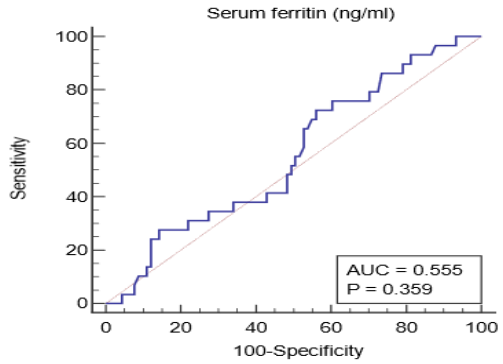

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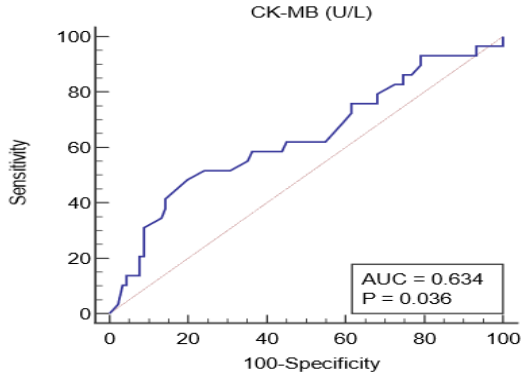

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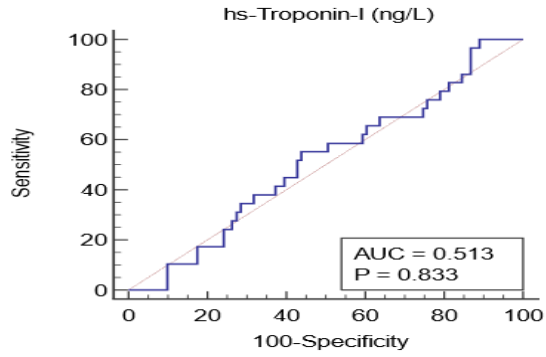

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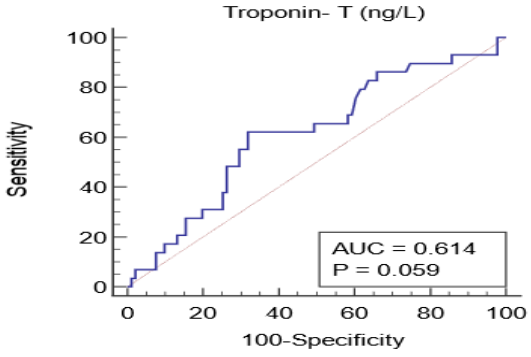

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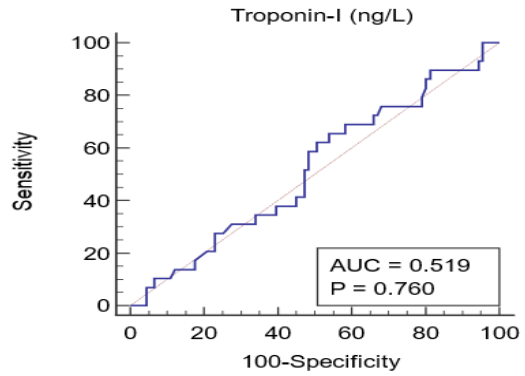

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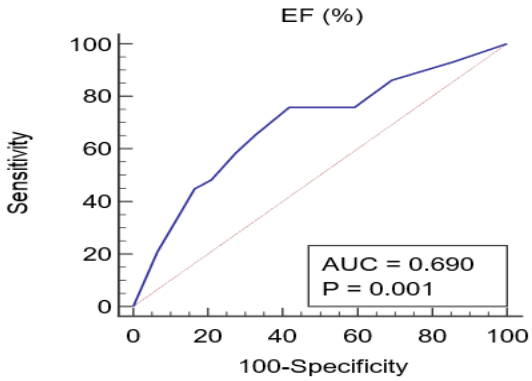
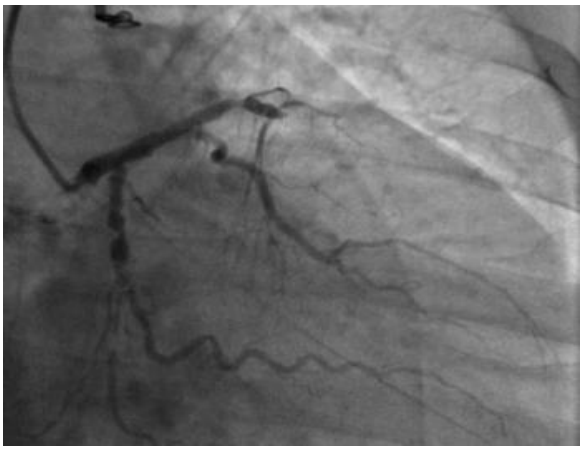


(H)

Figure 1: ROC curve analysis of (A) d-dimer, (B) C-reactive protein, (C) ferritin, (D) creatine kinase-myocardial band, (E) high sensitivity-Troponin-I, (F) troponin-T, (G) troponin-I and (H) ejection fraction for the prediction of the adverse events



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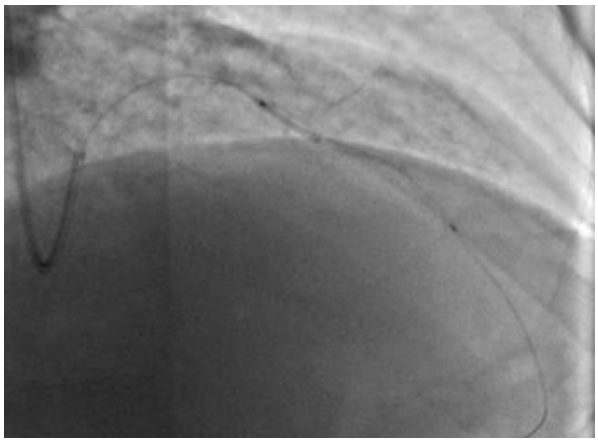

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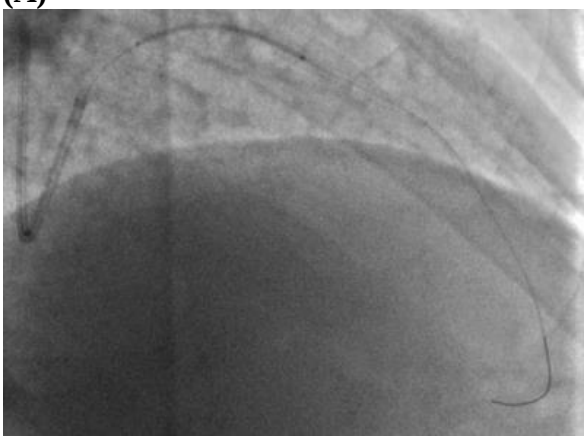

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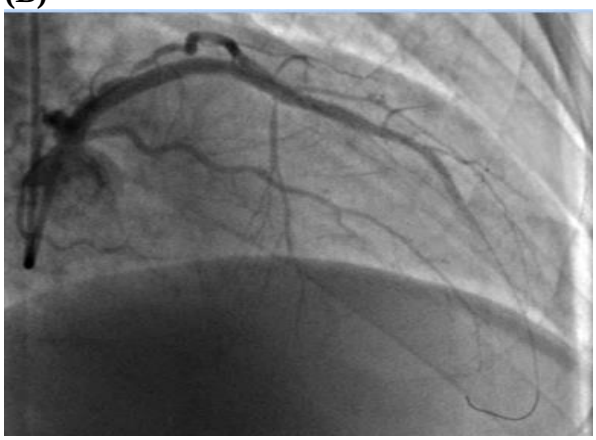

(D)

Figure 2: (A) Totally occluded left anterior descending coronary artery by a thrombus, inflation of the (B) first stent, (C) second stent and (D) final results

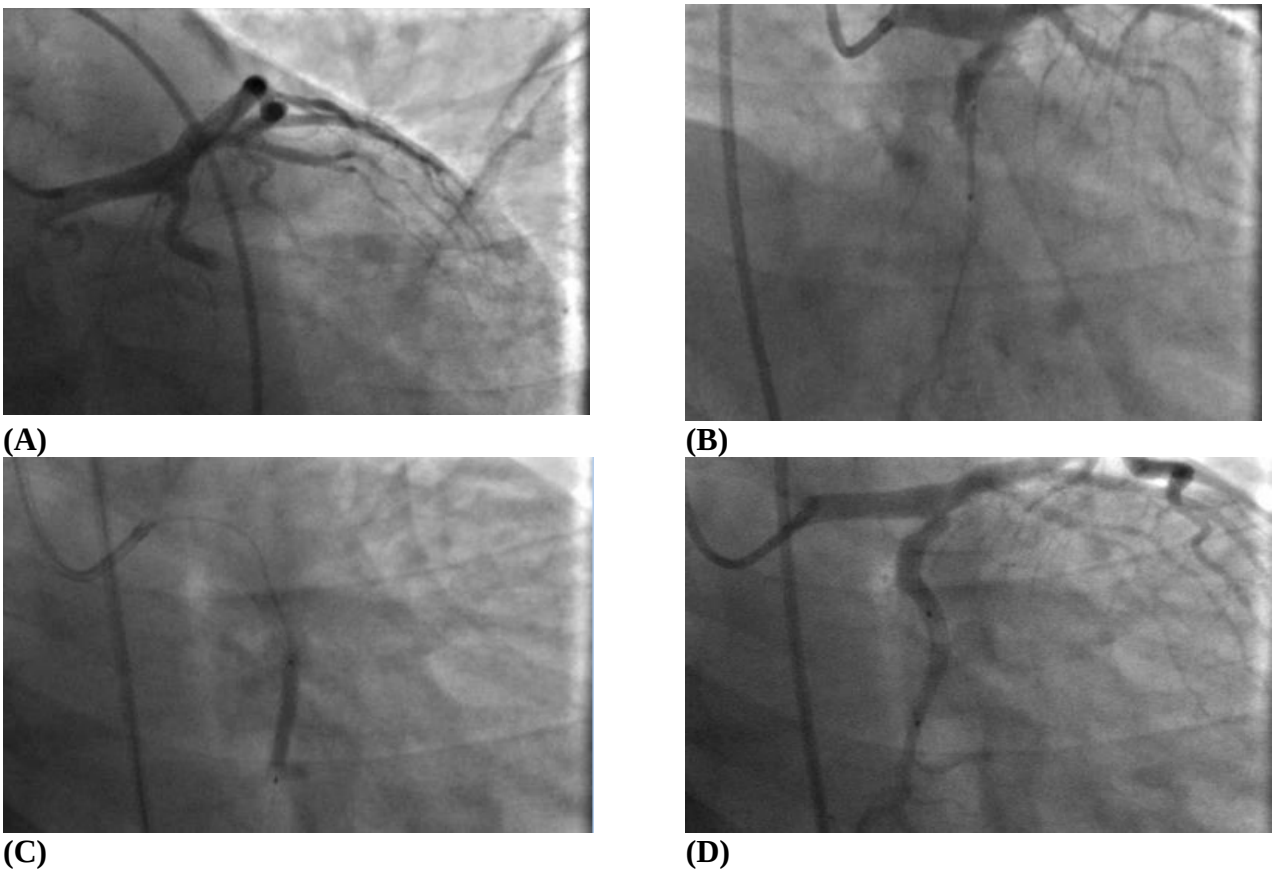

Figure 3: (A) Totally occluded left circumflex coronary artery in its proximal part, inflation of the (B) first stent, (C) second stent and (D) final result

DISCUSION

The COVID-19 pandemic, emerging at the start of 2020, quickly revealed its potential for severe cardiovascular complications [4].

The studied groups showed a substantial difference in terms of HTN, DM, prior stroke, and PCI. being greater in group A then group B. Other medical history (hyperlipidemia, ARDS, COPD, malignancy, and prior MI) was no notable variants between the studied groups. As Rashid et al. [7] shown, patients in the COVID-19 ACS group were more likely to experience troponin elevation, in-hospital cardiac arrest (6.3% vs. 3.0%), cardiogenic shock (9.6% vs. 3.9%), and pulmonary oedema (9.0% vs. 3.4%). Cardiovascular illness (23.7% vs. 13.4%), high blood pressure (69.4% vs. 58.3%), insulin-treated diabetes (13.6 vs. 7.5%), and heart failure (15.7% vs. 8.0%) were significantly more prevalent among them.

There was no discernible difference in the HRs of groups B and C, while it was much higher in group A. Both the SBP and the DBP were relatively unchanged when comparing the two groups. In a similar vein, Milovančev et al. [8] showed that COVID-19 ACS patients had a significantly lower median body mass index (25.7 (23.5, 28.2) vs. 27.5 (26.2, 28.8)), a faster heart rate (85 (70, 100) vs. 80 (70, 90) bpm, $p = 0.05$), and a greater probability of sudden cardiac arrest (21.7% vs. 6.6%). There was an increase in serious complications for patients in the COVID-19 ACS group while they were in the hospital.

Regarding the laboratory findings, WBCs, D-dimer, CRP at admission and peak, serum ferritin, and serum creatinine were notably distinct among the groups, while the groups showed negligible differences in other laboratory findings (Hb, PLT, ALT, AST, urea, K+, and Na+ levels). Group B had a considerably greater WBCs than groups A and C, but groups A and C did not differ considerably. Compared to group B, D-dimer

levels were substantially elevated in both groups A and C, with not a substantial variance between groups A and C. The level of CRP at admission and peak and serum ferritin were markedly elevated in group A compared to groups B and C and was substantially more in group C compared to group B. The serum creatinine level was considerably greater in group A than in groups B and C, and it was also substantially higher in group B compared to group C. The study's findings revealed that total cholesterol, TG, and LDL levels were markedly raised in both ACS patients with and without COVID-19 than those with COVID-19 but without ACS. ACS patients without COVID-19 showed greater HDL levels than those with the virus. Çınar et al. [9] showed similar results, namely that ACS participants with COVID-19 had substantially higher WBCs and neutrophil counts, creatinine levels, total globulin, C-reactive protein, and troponin I than those without the virus. When compared to group C, both A and B exhibited substantially elevated levels of total cholesterol, TG, and LDL. However, there was not a substantial variance between two groups. Levels of HDL were substantially greater in group B in comparison to group A, and there wasn't a noticeable distinction among group A and C or among group B and C.

This underscores the importance of lipid management in ACS patients and hints at a unique interaction between COVID-19 and lipid metabolism, particularly HDL, which could influence treatment strategies for ACS with relation to COVID-19.

Regarding the cardiac biomarkers, CK-MB was substantially raised in both groups A and C in comparison to group B, although groups A and C were not substantially distinct from one another. hs-Troponin-I and Troponin- T were notably elevated in group A compared to groups B and C and were markedly elevated in group B compared to group C. In comparison to groups A and C, troponin-I levels were substantially greater in group B, while there was no substantial distinction within groups A and B.

Supporting our results, Rashid et al. [7] found markedly elevated levels of peak troponin, creatinine, tachycardia, and a reduced blood pressure. It is established that in-hospital outcomes in COVID-19 cases were adversely affected by troponin elevations that exceed the upper reference limit by three times.

Group B had a substantially greater GRACE score compared to groups A and C, and group A had a greater GRACE score than group C. There was a negligible distinction among group A and group B in terms of Killip class. Similarly, Milovančev et al. [8] found that COVID-19 ACS group was with a substantially more prevalence of moderate MR (31.3%) than the general population (10.9%), a moderate TR (21.7%) compared to the general population (8.5%), and a severe TR (2.4% vs. 0%).

Group A demonstrated substantially greater rates of mortality and mechanical ventilation than the other groups. Ischemic stroke and myocardial re-infarction were not substantially distinct among the groups. Our findings were corroborated by Rashid et al. [7] who demonstrated that the COVID-19 ACS group had higher rates of death at 30 days (24.2% vs. 5.1% in the non-COVID-19 ACS group) and in the hospital itself. In-hospital death rates were 24.2% vs. 5.1% and 30-day mortality rates were 41.9% vs. 7.2% for the COVID-19 ACS group compared to the non-COVID-19 ACS group. Following adjustment for demographic, presenting, comorbidity, and pharmacological variations at baseline, in comparison to non-COVID-19 ACS patients, patients with ACS were more likely to die in the hospital (adjusted odds ratio: 3.27; 95% CI: 2.41-4.42) and within 30 days (adjusted odds ratio: 6.53; 95% CI: 5.12-8.36), according to the hierarchical multilevel logistic regression model.

The study's limited sample size and single-centered design are its limitations. The duration of patient follow-up was relatively short which limits comprehension of the chronic effects of COVID-19 on patients with ACS. Certain potential variables, that is detailed medication histories or genetic predispositions, were not accounted for, which might influence outcomes.

Conclusions:

COVID-19 patients with ACS exhibited higher

HR and high levels of markers such as CRP, serum ferritin, and cardiac injury indicators like CK-MB and high-sensitivity Troponin-I. The GRACE score is a crucial tool for risk assessment, and the study also demonstrated that COVID-19 is a reliable indicator of mortality in ACS patients. These findings emphasize the need for comprehensive monitoring and tailored management strategies in this patient group, focusing on cardiac biomarkers and systemic inflammation to improve outcomes and mitigate the risk of severe complications.

Conflicts of Interest:

The authors have no financial or proprietary interest in any material discussed in this article.

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