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ORIGINAL ARTICLE

Clinical characteristics and predictors of in-hospital mortality of patients hospitalized with myocardial infarction before and during COVID-19 pandemic

Short title: MI during COVID-19

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Abstract

Introduction: The COVID-19 pandemic has impacted many acute coronary syndrome (ACS) care aspects. The aim was to compare the patient profile, ACS characteristics, and the outcomes in patients referred to the invasive cardiology department before (March 2019 – February 2020) and during the COVID-19 pandemic (March 2020 – February 2021).

Material and methods: Clinical and demographic features, comorbidities, laboratory parameters at admission, and periprocedural data were recorded. The relationship of these parameters with in-hospital mortality was assessed.

Results: Before the COVID-19 pandemic, 664 patients were admitted due to ACS (mean age 67.16 ± 11.94 years, females 32.1%), and during the COVID-19 pandemic 545 ACS patients were recorded [mean age 66.02 ± 12.02 years ($p = 0.463$), females 31% ($p = 0.706$)]. A 17.8% decrease in the ACS rate was observed. During the pandemic, there were more STEMI patients (44.3% vs. 52.1%, $p < 0.001$) and fewer patients treated conservatively (24.9% vs. 8%, $p < 0.001$). Most lesions were located in the left anterior descending artery (53.4% vs. 54.7%), but post-percutaneous coronary intervention TIMI 3 was observed more frequently before the pandemic (83.9% vs. 75.1%, $p < 0.001$). Periprocedural complication rates did not differ between the groups. In-hospital outcomes did not differ between analyzed periods regarding all-cause death nor cardiac death rates, 5.3% vs. 4.6% ($p = 0.598$) and 4.5% vs. 3.7% ($p = 0.473$), respectively.

Conclusions: Based on the analysis of 1209 patients, a decrease in ACS patients admitted during the pandemic was recorded, but in-hospital mortality remained similar.

Keywords: acute coronary syndrome; coronary artery disease; SARS-CoV-2

Introduction

Cardiovascular events are one of the most common causes leading to death. Acute coronary syndrome (ACS), particularly with ST-elevation, presents a significant health and life threat for patients, and such patients should present to invasive cardiology departments as soon as possible [1]. The COVID-19 pandemic has impacted many aspects of ACS care. Two main adverse effects can be highlighted. The prognosis of patients with COVID-19-positive acute myocardial infarction (MI) is significantly worse than COVID-19-negative patients [2–4]. Unfortunately, COVID-19 cases burdened the healthcare system, and physicians observed fewer ACS patients presenting to hospitals during the pandemic [5–7].

Although it is known that COVID-19 infection raises the risk of thrombosis, some authors observed an ST-elevation myocardial infarction (STEMI) paradox during the pandemic. In a Spanish study involving 73 cardiac centers, researchers showed a 40% decrease in STEMI cases [7]. Similarly, Garcia et al. observed a 38% drop in coronary angiography procedures due to STEMI [8]. And finally, Italian authors showed a reduction in STEMI and NSTEMI cases by 26.5% and 65.1%, respectively [9].

Some possible explanations can be provided for the ACS rate drop. During the pandemic, patients were less eager to search for medical care. They might fear being admitted to a hospital. Apart from patient anxiety, healthcare providers were overburdened with COVID-19 patients [10, 11].

The aim herein, was to compare the patient profile, ACS characteristics, and the outcomes in patients with ACS referred to the invasive cardiology department before (March 2019 – February 2020) and during the COVID-19 pandemic (March 2020 – February 2021).

Material and methods

Study design and participants

The data were obtained retrospectively from the hospital database. All patients diagnosed with ACS, i.e., unstable angina, NSTEMI, or STEMI, before the COVID-19 pandemic (March 2019 – February 2020) and during the COVID-19 pandemic (March 2020 – February 2021) were included. Also, patients referred to invasive diagnostic and treated conservatively from the beginning were included. In the second period, both COVID-19-positive and COVID-19-negative patients were included.

In this study, various baseline demographic and clinical characteristics, laboratory data, and clinical outcomes in ACS patients admitted in these two periods were compared.

Data collection

Demographic, clinical, periprocedural, and laboratory data from the hospital database were retrieved. The following comorbidities were taken into consideration: arterial hypertension, dyslipidemia, chronic heart failure, diabetes mellitus, chronic obstructive pulmonary disease, peripheral artery disease, chronic kidney disease, prior coronary artery bypass grafting (CABG), prior PCI (percutaneous coronary intervention), prior MI, COVID-19 status (if applicable) and clinical data associated with ACS: ACS type, time from symptoms onset, disease advancement, treatment strategy, and periprocedural complications. Additionally, information was gathered on echocardiographic parameters (left ventricular ejection fraction) and laboratory findings assessed at admission: alanine aminotransferase (ALT), complete

blood count with differential (WBC — white blood cells, RBC — red blood cells, Hgb — hemoglobin, PLT — platelets), creatinine, creatine kinase (CK-MB), C-reactive protein (CRP), eGFR, glucose, lipid profile, N-terminal pro-B-type natriuretic peptide (NT-proBNP), and troponin T (Tn-T). Information was also gathered on medications at discharge and in-hospital events.

Study endpoints

The primary study endpoint was to compare in-hospital cardiac death rates between two periods. The secondary endpoints included all-cause death, MI, stroke, and bleeding rates.

Statistical analysis

ACS patients were stratified into two cohorts regarding the time of admission: before the pandemic (March 2019 – February 2020) or during the pandemic (March 2020 – February 2021). Categorical variables are presented as numbers and percentages, and they were compared by applying the chi-square test or the Fisher exact test if appropriate. the normality of data distribution was verified using the Shapiro-Wilk test. Cumulative in-hospital mortality (all-cause as well as cardiac) was depicted as percentages in tables. Moreover, multivariable logistic regression analysis was conducted to identify independent factors linked with in-hospital all-cause mortality. Variables from Tables 1–3 that reached a p-value of < 0.1 in univariable analysis were incorporated into a multivariable model. he final multivariable model was obtained by applying a backward variable selection method. The level of statistical significance was $p < 0.05$ (two-tailed). Then, ROC curves were generated based on the multivariable logistic regression model [12]. All statistical analyses were performed using Prism 9 for Mac OS version 9.5.0 (GraphPad Software).

Results

Baseline characteristics

Before the COVID-19 pandemic (March 2019 – February 2020), 664 patients were admitted due to ACS (mean age 67.16 ± 11.94 years, females 32.1%), and during the COVID-19 pandemic (March 2020 – February 2021), 545 ACS patients were recorded [mean age 66.02 ± 12.02 years ($p = 0.463$), females 31% ($p = 0.706$)]. A 17.8% decrease in ACS patients admitted to the hospital was observed. Before the pandemic, more patients had arterial hypertension ($p = 0.038$), dyslipidemia ($p < 0.001$), diabetes type 2 ($p = 0.022$), peripheral artery disease ($p < 0.0001$), prior CABG ($p = 0.013$), and chronic obstructive pulmonary disease ($p = 0.009$); simultaneously, fewer patients had chronic kidney disease ($p < 0.0001$). During the pandemic, 22 (4%) patients with ACS and COVID-19 were admitted (Table 1).

Acute coronary syndrome characteristics and periprocedural details

During the pandemic, more patients presented with STEMI (44.3% vs. 52.1%, $p < 0.001$), and fewer patients were treated conservatively (24.9% vs. 8%, $p < 0.001$) (Table 2). Most lesions were located in the left anterior descending artery (53.4% vs. 54.7%), but post-PCI TIMI 3 was observed more frequently before the pandemic (83.9% vs. 75.1%, $p < 0.001$). One drug-eluting stent was usually implanted during PCI (74.7% vs. 70.2%, $p < 0.001$). Periprocedural complication rates did not differ between the groups (Table 3).

Laboratory findings at admission and medications at discharge

During the pandemic, patients characterized higher cardiac necrosis markers (both troponin and CK-MB), but higher NT-proBNP levels (3781.6 ± 9552.1 pg/mL vs. 2317.3 ± 4980.6 pg/mL, $p = 0.001$) were observed before the pandemic (Table 4). Moreover, during the pandemic, more patients received aspirin (77.8% vs. 96.3%, $p < 0.001$), new antiplatelets

(ticagrelor: 33.8% vs. 51.2%, $p < 0.001$), ACE inhibitors (69.8% vs. 77.8%, $p = 0.002$), beta-blockers (75.4% vs. 86.2%, $p < 0.001$), and statins (79.2% vs. 97.2%, $p < 0.001$) at discharge (Table 5).

In-hospital outcomes

In-hospital outcomes did not differ between analyzed periods regarding all-cause death or cardiac death rates, 5.3% vs. 4.6%, $p = 0.598$ and 4.5% vs. 3.7%, $p = 0.473$, respectively. No differences were observed if patients were analyzed with STEMI, patients undergoing PCI within the left main, or patients with TIMI 0 flow at baseline coronary angiography (Table 6). Additionally, the outcomes of patients were analyzed with COVID-19 and without COVID-19. Patients with COVID-19 and ACS had a statistically significant higher risk of all-cause death (18.2% vs. 4.0%, $p = 0.014$) but not cardiac death (9.1% vs. 3.4%, $p = 0.191$).

Risk factors for cardiac death

Taking into account previous variables, the multivariable models for cardiac death in those two periods are presented in Table 7, and ROC curves are in Figure 1. The same variables were entered into both models, i.e., age, STEMI, SYNTAX value, and chronic kidney disease.

Discussion

There was a 17.8% decrease in ACS patients admitted to the hospital during the COVID-19 pandemic. During the pandemic, more patients presented with STEMI (44.3% vs. 52.1%, $p < 0.001$), and fewer patients were treated conservatively (24.9% vs. 8%, $p < 0.001$). Most lesions were located in the left anterior descending artery (53.4% vs. 54.7%), but post-PCI TIMI 3 was observed more frequently before the pandemic (83.9% vs. 75.1%, $p < 0.001$). Periprocedural complication rates did not differ between the groups. In-hospital outcomes did

not differ between analyzed periods regarding all-cause death nor cardiac death rates, 5.3% vs. 4.6%, p = 0.598, and 4.5% vs. 3.7%, p = 0.473, respectively.

The database conducted by the Jagiellonian University Medical College and endorsed by the Association of Cardiovascular Interventions of the Polish Cardiac Society disclosed that the COVID-19 pandemic exerted a significant effect on interventional cardiology in Poland. A significant drop in the number of coronary angiography and PCI procedures was noted, as well as the use of modern imaging and physiologic assessment techniques. In comparison to 2019, a significant 25% drop in the total number of coronary angiography (172 521 vs. 130 662) as well as PCI procedures were recorded (101 716 vs. 82 349) [13–15]. Similar trends were also noted in other countries where COVID-19 torpedoed planned and unplanned hospitalization. Wang et al. disclosed a substantial decrease in hospitalization rates during the COVID-19 pandemic: total (–182 per 100 000) and unscheduled one (–39 per 100 000) caused by stroke (–1.51 per 100 000), acute MI (–1.32 per 100 000), or heart failure (–8.7 per 100 000) [16]. the following underlying mechanisms can be mentioned: patient anxiety about COVID-19 contraction, overburden of pre-pandemic hospitalizations, or introducing pandemic mitigation actions, e.g., rescheduling non-urgent diagnostic procedures or surgeries [17]. In the present study, more patients were treated conservatively before the pandemic, and more STEMI patients were recorded during the pandemic. This might suggest that during the pandemic, more commonly, patients with severe and persisting symptoms, as in acute MI with total vessel occlusion, decided to present to Emergency Departments (ED). Before the pandemic, more patients with chest pain presented to ED, and in further diagnostic, no obstructive coronary artery disease was confirmed. Other findings also confirm this observation. Pre-PCI TIMI 0 was more frequent during the pandemic (55.6% vs. 30.8%, p < 0.001) (more patients with STEMI and fresh thrombus during the pandemic), and post-PCI TIMI 3 was more frequent before the pandemic (83.9% vs. 75.1%, p < 0.001). The upsurge of

STEMI patients during the pandemic was also observed in other studies. Yendrapali et al. reported an increase from 15–18% to 32% [18]. This contrasts with the earlier mentioned STEMI paradox showed in other studies. In a Spanish study involving 73 cardiac centers, researchers showed a 40% decrease in STEMI cases [7]. Similarly, Garcia et al. observed a 38% drop in coronary angiography procedures due to STEMI [8]. And finally, Italian authors showed a reduction in STEMI and NSTEMI cases by 26.5% and 65.1%, respectively [9]. Milovancev et al. showed decreased ED visits and hospitalizations not just in outbreaks but throughout the whole COVID-19 year. This highlights the risk of continuous delay of required healthcare for emergency life-threatening cardiovascular diseases [19].

Other authors observed increased comorbidity rates during the pandemic [1]. However, in our study, the opposite was recorded. Before the pandemic, more patients had arterial hypertension ($p = 0.038$), dyslipidemia ($p < 0.001$), diabetes type 2 ($p = 0.022$), peripheral artery disease ($p < 0.0001$), prior CABG ($p = 0.013$), and chronic obstructive pulmonary disease ($p = 0.009$); simultaneously, fewer patients had chronic kidney disease ($p < 0.0001$). This might be difficult to explain, especially bearing in mind the widespread problems with access to healthcare facilities during the pandemic.

Several studies also indicated elevated death and complication rates related to acute MI and stroke during the pandemic [20–22]. Therefore, increasing in-hospital death rates for non-COVID-19 urgent diseases such as acute MI or stroke were expected. Unfortunately, no significant changes in the in-hospital mortality rates as compared to the pre-pandemic period were observed. This might partially be explained by the fact that only 4% of these patients were COVID-19-positive. No MI mechanical complications were observed, which can be associated with acute ischemia [23].

The treated population is associated with the differences in medications at discharge. During the pandemic, more patients received aspirin (77.8% vs. 96.3%, $p < 0.001$), new antiplatelets

(ticagrelor: 33.8% vs. 51.2%, $p < 0.001$), ACE inhibitors (69.8% vs. 77.8%, $p = 0.002$), beta-blockers (75.4% vs. 86.2%, $p < 0.001$), and statins (79.2% vs. 97.2%, $p < 0.001$). This can be explained by the fact that during the pandemic, there were more STEMI patients with TIMI 0 at baseline. Such patients were treated more aggressively with statins and more potent antiplatelet drugs.

Study limitations

This study has several limitations. First, this was a retrospective study; therefore, residual confounding factors may exist. Second, not all laboratory parameters were collected in all patients. Third, only a small percentage of patients were COVID-19-positive. And finally, only in-hospital outcomes are presented.

Conclusions

Based on the analysis of 1209 patients, a decrease in ACS patient admission during the pandemic was recorded, but in-hospital mortality remained similar.

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Statement of competing interests:

The authors declare no conflict of interest.

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Figure 1. ROC curves based on the multivariable model. NPP — negative predictive power; PPP — positive predictive power

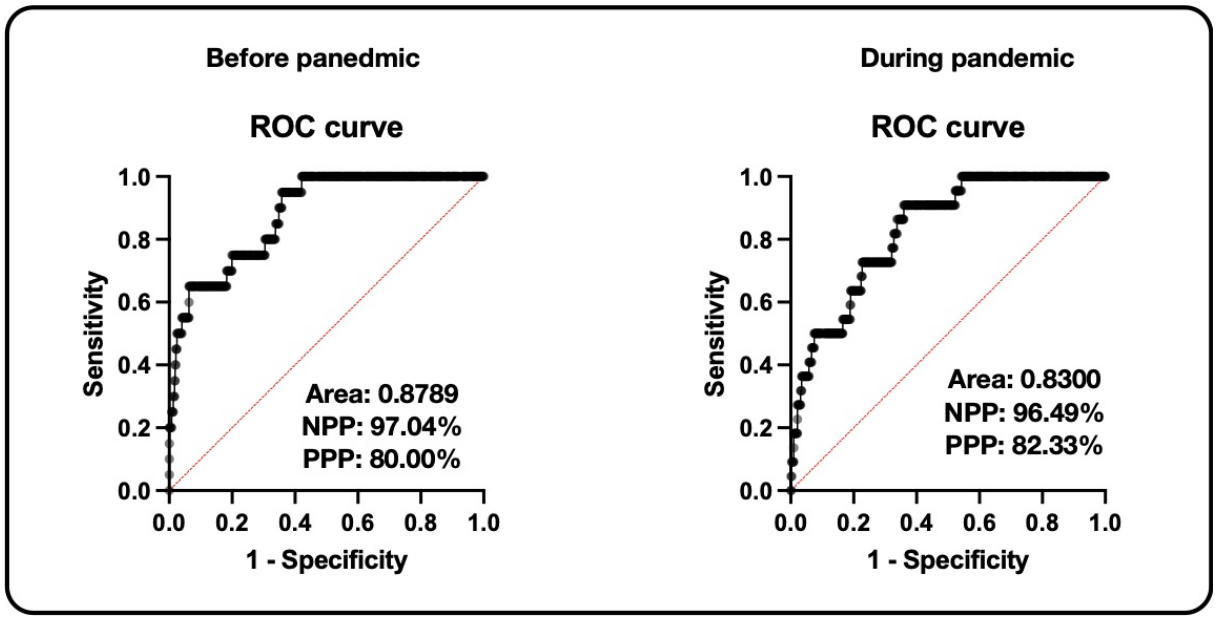

Table 1. Baseline characteristics

Parameter	**Mar 2019 – Feb 2020** **N = 664**	**Mar 2020 – Feb 2021** **N = 545**	**P-value**
Age [years]	67.16 ± 11.94	66.02 ± 12.02	0.463
Females	213 (32.1)	169 (31.0)	0.706
Arterial hypertension	454 (68.4)	341 (62.6)	0.038
Dyslipidemia	514 (77.4)	353 (64.8)	< 0.001
Diabetes type 2	229 (34.5)	154 (28.3)	0.022
Peripheral artery disease	89 (13.4)	24 (4.4)	< 0.001
Chronic kidney disease	58 (8.7)	76 (13.9)	< 0.001
Carotid artery disease	12 (1.8)	11 (2.0)	0.516
Chronic obstructive pulmonary disease	39 (5.9)	12 (2.2)	0.009
Heart failure	291 (43.8)	211 (38.7)	0.079
Prior CABG	53 (8.0)	25 (4.6)	0.013
Prior PCI	149 (22.4)	97 (17.8)	0.178
Prior MI	145 (21.8)	90 (16.5)	0.234
COVID-19	–	22 (4.0)	< 0.001
Left ventricular ejection fraction	48.6 ± 11.2	46.7 ± 12.4	0.324

CABG — coronary artery bypass grafting; PCI — percutaneous coronary intervention; MI — myocardial infarction

Table 2. Acute coronary syndrome characteristics

Parameter	**Mar 2019 – Feb 2020** **N = 664**	**Mar 2020 – Feb 2021** **N = 545**	**P-value**
Type			
UA	89 (13.4)	63 (11.6)	< 0.001
NSTEMI	281 (42.3)	198 (36.3)	
STEMI	294 (44.3)	284 (52.1)	
Time from symptoms onset [h]	20.6 ± 27.13	17.9 ± 22.9	0.442
Disease advancement			
Coronary angiography	626 (94.3)	543 (99.6)	< 0.001
1VD	262 (41.9)	227 (41.8)	0.001
2VD	180 (28.8)	167 (30.8)	
3VD	136 (21.7)	130 (23.9)	
3VD+LM	48 (7.7)	19 (3.5)	
CTO	43 (6.9)	17 (3.1)	0.113
Treatment strategy			
Conservative treatment	165 (24.9)	44 (8.0)	< 0.001
Revascularization	499 (75.1)	501 (92.0)	
PCI	438 (87.8)	433 (86.4)	0.572
CABG	61 (13.2)	68 (13.6)	

CABG — coronary artery bypass grafting; PCI — percutaneous coronary intervention; UA — unstable angina; NSTEMI — non-ST-elevation myocardial infarction; STEMI — ST-elevation myocardial infarction; VD — vessel disease; LM — left main; CTO — chronic total occlusion

Table 3. Periprocedural characteristics

Parameter	**Mar 2019 – Feb 2020** **N = 438**	**Mar 2020 – Feb 2021** **N = 433**	**P-value**
SYNTAX	15.34 ± 9.68	11.84 ± 8.31	0.032
SYNTAX 2	34.36 ± 12.88	31.59 ± 11.71	0.029

Lesion location	N = 438	N = 433	
LM	16 (3.7)	31 (7.2)	
LAD	234 (53.4)	237 (54.7)	< 0.001
LCx	96 (21.9)	37 (8.5)	
RCA	84 (19.2)	113 (26.1)	
Bypass	8 (1.8)	15 (3.5)	
TIMI pre	N = 626	N = 543	
0	193 (30.8)	302 (55.6)	
1	104 (16.6)	21 (3.9)	< 0.001
2	189 (30.2)	188 (34.6)	
3	140 (22.4)	32 (5.9)	
TIMI post	N = 626	N = 543	
0	29 (4.6)	113 (20.8)	
1	16 (2.6)	8 (1.5)	< 0.001
2	56 (8.9)	14 (2.6)	
3	525 (83.9)	408 (75.1)	
Bifurcation	47 (10.7)	26 (6.0)	0.014
Thrombectomy	11 (2.5)	8 (1.8)	0.644
GP IIb/IIIa inhibitor	48 (10.9)	54 (12.5)	0.528
Stents No			
1	327 (74.7)	304 (70.2)	
2	90 (20.5)	92 (21.2)	< 0.001
3	21 (4.8)	28 (6.5)	
4	0	9 (2.1)	
UFH	373 (85.1)	50 (11.5)	
LMWH	62 (14.2)	382 (88.2)	
Bivalirudin	3 (0.7)	1 (0.2)	< 0.001
Stent type	438	433	
SES	195 (44.5)	176 (40.6)	
EES	173 (39.5)	186 (42.9)	0.303
ZES	70 (16.0)	71 (16.4)	
Periprocedural complications			
Dissection	12 (2.7)	5 (1.2)	0.139

Distal embolization	18 (4.1)	10 (2.3)	0.178
No reflow	13 (2.9)	7 (1.6)	0.258
Perforation	0	1 (0.2)	0.451

LM — left main; LAD — left anterior descending artery; LCx — left circumflex artery; RCA — right coronary artery; TIMI — thrombolysis in myocardial infarction; GP — glycoprotein; SES — sirolimus-eluting stent; EES — everolimus-eluting stent; ZES — zotarolimus-eluting stent

Table 4. Laboratory findings at admission

Parameter	**Mar 2019 – Feb 2020** **N = 664**	**Mar 2020 – Feb 2021** **N = 545**	**P-value**
Leucocytes [10^3/μL]	14.53 ± 9.1	16.72 ± 9.5	0.0001
Red blood cells [10^6/μL]	4.67 ± 2.23	5.12 ± 1.3	0.0001
Hemoglobin [g/dL]	13.71 ± 2.1	15.96 ± 5.6	0.0001
Platelets [10^3/μL]	252.06 ± 163.63	248.8 ± 80.4	0.601
eGFR [mL/min]	87.19 ± 36.4	71.45 ± 26.02	0.0001
Glucose [mg/dL]	152 ± 72.6	152.01 ± 82.3	0.998
NT-proBNP [pg/mL]	3781.6 ± 9552.1	2317.3 ± 4980.6	0.001
Max. troponin T [μg/L]	1.57 ± 2.32	3.17 ± 21.52	0.057
Max. CK-MB [IU/L]	56.4 ± 100.5	72.88 ± 158.20	0.028
ALT [IU/L]	55.4 ± 23.7	45.08 ± 59.63	0.0001
Total cholesterol [mg/dL]	182.8 ± 97.4	182.96 ± 61.91	0.974
LDL [mg/dL]	129.4 ± 79.1	124.86 ± 52.73	0.205
HDL [mg/dL]	47.9 ± 15.2	46.86 ± 16.77	0.233
Triglycerides [mg/dL]	142.9 ± 111.1	146.98 ± 131.85	0.656
C-reactive protein [mg/L]	8.35 ± 132.6	4.52 ± 33.18	0.509

eGFR — estimated glomerular filtration rate; NT-proBNP — N-terminal pro-brain natriuretic peptide; CK-MB — creatine kinase MB; ALT — alanine aminotransferase; LDL — low-density lipoprotein; HDL — high-density lipoprotein

Table 5. Medications at discharge

Parameter	**Mar 2019 – Feb 2020** **N = 664**	**Mar 2020 – Feb 2021** **N = 545**	**P-value**
Aspirin	517 (77.8)	525 (96.3)	< 0.001
Clopidogrel	282 (42.5)	211 (38.7)	
Prasugrel	2 (0.3)	9 (1.7)	
Ticagrelor	224 (33.8)	279 (51.2)	< 0.001
ACE inhibitor	463 (69.8)	424 (77.8)	
ARB	23 (3.5)	21 (3.9)	0.002
ARNI	2 (0.3)	2 (0.4)	
Betablocker	500 (75.4)	470 (86.2)	< 0.001
Statin	525 (79.2)	530 (97.2)	< 0.001
MRA	126 (19)	123 (22.6)	0.134
Diuretics	243 (36.7)	196 (36)	0.810
Ezetimibe	27 (4.1)	29 (5.3)	0.337
Fibrate	5 (0.8)	0	0.068
Ca-blocker	118 (17.8)	104 (19.1)	0.601
Flozins	15 (2.3)	11 (2.0)	0.844
Vitamin K antagonists	10 (1.5)	5 (0.9)	0.145
Rivaroxaban	17 (2.6)	23 (4.2)	
Dabigatran	13 (2.0)	18 (3.3)	
Apixaban	10 (1.5)	6 (1.1)	

ACE — angiotensin-converting enzyme; ARB — angiotensin II receptor blocker; ARNI — angiotensin receptor-neprilysin inhibitor; MRA — aldosterone receptor antagonist

Table 6. In-hospitals outcomes

Parameter	**Mar 2019 – Feb 2020**	**Mar 2020 – Feb 2021**	**P-value**
Whole study population	**N = 664**	**N = 545**	
Death	35 (5.3)	25 (4.6)	0.598
Cardiac death	30 (4.5)	20 (3.7)	0.473
Stroke	1 (0.15)	0	1
STEMI	**N = 294**	**N = 284**	
Death	22 (7.5)	20 (7.0)	0.874
Cardiac death	21 (7.1)	17 (5.9)	0.6173
Stroke	0	0	1
Left main	**N = 16**	**N = 31**	
Death	5 (31.3)	2 (6.5)	0.036
Cardiac death	5 (31.3)	2 (6.5)	0.036
Stroke	0	0	1
TIMI 0 at baseline	**N = 193**	**N = 302**	
Death	1 (0.5)	7 (2.3)	0.158
Cardiac death	1 (0.5)	7 (2.3)	0.158
Stroke	0	0	1

STEMI — ST-elevation myocardial infarction; TIMI — thrombolysis in myocardial infarction

Table 7. Multivariable analysis

Variable	**Mar 2019 – Feb 2020**		**Mar 2020 – Feb 2021**	
	OR	**95% CI**	**OR**	**95% CI**
Age [years]	1.065	1.022–1.115	1.057	1.014–1.104
STEMI	7.465	2.270–34.450	7.556	2.742–24.99
SYNTAX	1.070	1.025–1.117	1.062	1.019–1.106
CKD	6.859	2.432–19.79	2.596	0.714–8.490

OR — odds ratio; CI — confidence interval; STEMI — ST-elevation myocardial infarction;

CKD — chronic kidney disease