ORIGINAL ARTICLE

Incidence and course of acute coronary syndrome cases after the first wave of the COVID-19 pandemic

Justyna Jankowska-Sanetra[1], Krzysztof Sanetra[2, 3], Marta Konopko[4], Monika Kutowicz[4], Magdalena Synak[4], Paweł Kaźmierczak[5], Krzysztof Milewski[1, 6, 7], Łukasz Kołtowski[8], Piotr Paweł Buszman[1, 4, 7]

[1]Department of Cardiology, American Heart of Poland, Bielsko-Biała, Poland
[2]Clinic of Cardiovascular Surgery, Andrzej Frycz Modrzewski Krakow University, Kraków, Poland
[3]Department of Cardiac Surgery, American Heart of Poland, Bielsko-Biała, Poland
[4]Department of Cardiology, Andrzej Frycz Modrzewski Krakow University, Kraków, Poland
[5]American Heart of Poland, Katowice, Poland
[6]Faculty of Medicine, University of Technology, Katowice, Poland
[7]Center for Cardiovascular Research and Development, American Heart of Poland, Katowice, Poland
[8]1st Chair and Department of Cardiology, Warsaw Medical University, Warszawa, Poland

Editorial
by Stefanini et al.

Correspondence to:
Krzysztof Sanetra, MD,
Clinic of Cardiovascular Surgery,
Andrzej Frycz Modrzewski
Krakow University,
Armii Krajowej 101,
43–316 Bielsko-Biała, Poland,
phone: +48 692 030 003,
e-mail: krzyssan@poczta.onet.pl

DOI: 10.33963/KP.a2022.0250

Received:
February 2, 2022

Accepted:
November 5, 2022

Early publication date:
November 7, 2022

ABSTRACT

Background: The collateral damage caused by the COVID-19 pandemic affected cardiovascular disease patients, mainly acute coronary syndrome (ACS) cases. Additionally, lockdown caused treatment-related concerns and reluctance to seek medical help, factors that can delay treatment.

Aim: We aimed to analyze the incidence and course of ACS after the first COVID-19 wave.

Methods: The report is based on a multi-institutional registry of 10 interventional cardiology departments. ACS patient data were gathered from June to October 2020, i.e. in the period following the first lockdown in Poland (March 30–May 31, 2020) and compared with the corresponding 2019 timeframe.

Results: Patients (2801 and 2620) hospitalized for ACS in 2019 and 2020 (June–October) represented 52.8% and 57.9% of coronary artery disease admissions, respectively. In 2020 vs. 2019, more cases of arterial hypertension (80.2% vs. 71.5%; P <0.001), diabetes (32.7% vs. 28.2%; P <0.001) hyperlipidemia (53.2% vs. 49.8%; P = 0.01), and smoking history (29.5% vs. 25.8%; P = 0.003) were detected. Median troponin and cholesterol values, as well as glycemia, were higher in 2020. Patients were more likely to undergo percutaneous treatment (91.2% vs. 87.5%; P <0.001) and were less often referred for surgery (3.7% vs. 4.9%; P = 0.03). No differences in deaths from repeat myocardial infarction, stroke, and/or composite endpoint (major adverse cardiac and cerebrovascular events [MACCE]) were noted. However, suffering from ACS in 2020 (June–October) was a risk factor for mortality based on multivariable analysis.

Conclusions: The COVID-19 pandemic affected ACS patient profile, course of treatment, and increased risk for mortality.

Key words: acute coronary syndrome, coronavirus, COVID-19, lockdown, myocardial infarction

INTRODUCTION

Acute coronary syndrome (ACS), particularly with ST-segment elevation, presents a major health risk for patients, and patients should be referred for immediate medical attention. According to the European Society of Cardiology (ESC) clinical guidelines, those patients should present to invasive cardiology departments as soon as possible [1, 2]. Any delay in treatment may be associated with adverse consequences, including mortality. However, during the COVID-19 pandemic, which was caused by SARS-CoV-2, a significant decrease in the number of ACS cases referred

WHAT'S NEW?

Reports referring to the COVID-19 pandemic rarely focus on its first stage. The period immediately following lockdown was characterized by limited healthcare access. We analyzed the time interval separating the two waves of the pandemic in Poland to determine the impact of the first lockdown on acute coronary syndrome (ACS) incidence and its treatments and outcomes. Our study showed that only the first wave of the pandemic significantly affected coronary artery disease patients. Higher numbers of both unstable angina and ST-segment elevation infarctions, when compared with the corresponding period of the previous year, were found. A higher frequency of non-communicable diseases was noted, indicating that inadequate treatment might have triggered ACS. These patients were more often treated percutaneously and less often referred for surgery. Furthermore, suffering from acute coronary syndrome right after the lockdown was a risk factor for mortality when compared with the corresponding timeframe of the previous year.

to health facilities and a significant delay in their treatment were reported worldwide [3–10]. This delay may have led to huge consequences in both hospital outcomes and out-patient mortalities. The Polish National Primary Statistical Department reported over 67 000 more deaths in 2020 than in 2019, which greatly exceeds mortality caused by the coronavirus infection [11]. Those numbers appeared greater when the following calendar years were compared. Furthermore, the lack of proper medical care and treatment for both stable angina and non-communicable diseases raised concerns regarding the incidence and severity of ACS cases following the lockdown. To address this issue we investigated the impact of only the first lockdown on the incidence of ACS, patient profiles, and clinical outcomes.

METHODS

The multi-institutional registry

All information gathered for the report was sourced from the database network, which connects 10 invasive cardiology departments in Poland. The database includes hospitalization parameters from patients admitted due to acute coronary syndrome (defined as ST-segment elevation myocardial infarction [STEMI], non-ST-segment elevation myocardial infarction [NSTEMI], and unstable angina) from June to October 2020 and the corresponding timeframe in 2019.

Analyzed parameters

Data were anonymous, and only the patient unique number was assigned by the computer system. We included in the analysis: date of admission and discharge, hospitalization department, discharge characteristics, data regarding diagnosis (primary diagnosis and diagnosis after the hospitalization), SARS-CoV-2 infection status, comorbidities, procedure characteristics, anamnesis, pharmacotherapy, laboratory tests, echocardiography, hospitalization course and complications (such as death, repeat infarction, bleeding, stroke, any type of vascular complications), discharge to an intensive care unit and the composite endpoint of major adverse cardiac and cerebrovascular events (MACCE) defined as death, and/or myocardial infarction (MI; repeat MI in patients presenting with MI on admission or MI in patients presenting with unstable angina on admission) and/or stroke.

Routine COVID-19 testing

In 2020, all the patients underwent routine PCR (polymerase chain reaction) tests upon their admission to the hospital. In emergent cases, antigen tests were also performed to avoid any delay in diagnosis and treatment. Further COVID-19 testing depended on the patient's symptoms or contact with infected patients or personnel.

Research ethics board approval

The approval of the research ethics board was not mandatory for the analysis as the report was fully retrospective and contained datasets with anonymized information. No additional intervention was administered to any of the study patients. Following the National Code on Clinical Research, research ethics board consent is not obligatory for real retrospective studies.

Statistical analysis

Categorical data are shown as numbers (percentages). Continuous data are presented as mean (standard deviation) or median (interquartile range [IQR]). The normal distribution of analyzed parameters was verified with the Saphiro-Wilk test. Normal distribution datasets were compared using Student's t-test while non-normally distributed data were compared using the Mann-Whitney U test. The χ^2 test was used to analyze categorical data.

To address the impact of continuous and binary predictors (including hospitalization timeframe) on outcomes, the Cox proportional-hazards regression model was used. Potential predictors of mortality and composite endpoint (MACCE) were searched. The observation was conducted during hospitalization; censoring was made in cases that did not reach the event. The variables included in the model were admission timeframe (June–October 2020 or June–October 2019), myocardial infarction at baseline, Canadian Cardiovascular Society (CCS) class IV for angina, glycemia at baseline, cholesterol value at baseline, troponin values on admission, baseline ejection fraction, male sex, and age.

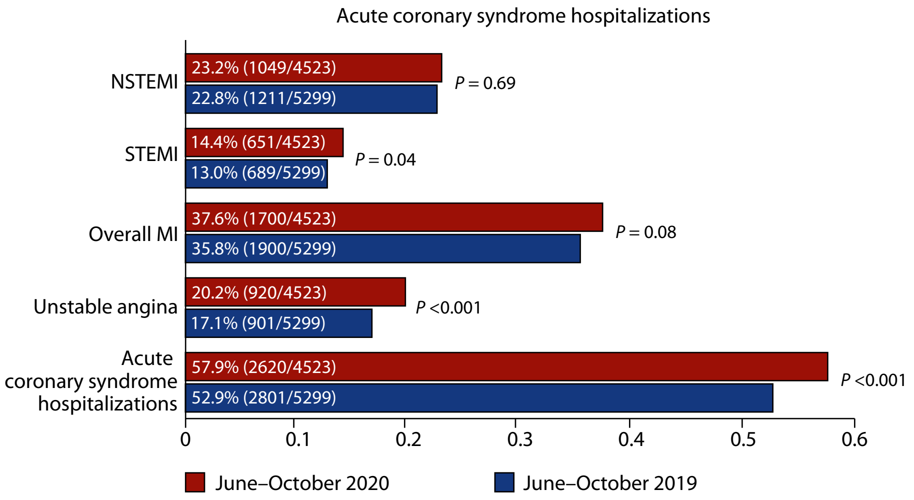

Figure 1. Hospitalizations for acute coronary syndrome with relation to the total number of hospitalizations for coronary artery disease. Data are presented as percentages (numbers)

Abbreviations: MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI; ST-segment elevation myocardial infarction

A stepwise variable selection procedure was used (variables were entered if *P* <0.3). MedCalc v.18.5 software (MedCalc Software, Ostend, Belgium) was used for data processing. A *P*-value of ≤0.05 was considered statistically significant.

Data presentation

The results from the current report are presented in three sections. The first includes data from patients' admission records and gives the baseline characteristics and their comparison in the evaluated time intervals. The second part compares the treatment characteristics in the same time intervals. Finally, the third section refers to hospitalization outcomes and potential complications as described on a timeline from admission date.

RESULTS

Overall, there were 5299 patients hospitalized in June–October 2019 (2801 acute coronary syndrome cases — 52.9%; 2498 elective cases — 47.1%) and 4523 patients hospitalized in June–October 2020 (2620 acute coronary syndrome cases — 57.9%; 1903 elective cases — 42.1%). These data reflect a significantly higher number of ACS hospitalizations related to overall hospitalizations for coronary artery disease following lockdown when compared with the corresponding time interval of the previous calendar year (Figure 1). The main reason for this observation was an increase in the incidence of unstable angina and STEMI with a similar number of NSTEMI cases (Figure 1).

The patients were similar in terms of age, sex, obesity, and symptoms when compared to June–October 2019. However, they presented more frequently with arterial hypertension, hyperlipidemia, diabetes, and were active smokers (Table 1). They had greater baseline values of high-sensitivity troponin T, cholesterol, and higher glycemia. The myocardial contractility, presented as ejection fraction, was similar in both timeframes. There were 15 (0.6% patients) SARS-CoV-2 infections in June–October 2020. Please note that none of the analyzed departments was a dedicated COVID-19 facility.

The pandemic affected not only the patient profile but also the course of treatment. The patients mostly underwent percutaneous revascularization. In this group, one-stage treatment of two arteries was more frequent than two-stage treatment during one hospitalization, which also contributed to relatively shorter hospitalization (Tables 2 and 3). A lower number of patients was referred for coronary artery bypass grafting procedures (Table 2). Patients who were not qualified for angioplasty or bypass grafting and received pharmacotherapy as dedicated treatment were included in the "non-invasive treatment group" (Table 2).

The maximal observation time was 24.0 days in 2019 and 27 days in 2020. Median observation time was 3.63 (1.6–4.9) days and 3.2 (1.5–4.6) days, respectively. The comparative analysis of hospitalization outcomes did not show significant differences in mortality, incidence of myocardial infarction or stroke, or the composite endpoint of MACCE. However, a trend toward greater mortality was visible (Table 3). More frequent hematomas of access sites were reported in 2019 (Table 3).

As mentioned, the hospitalization period was longer in the analyzed timeframe of 2019 when compared to 2020 (median [IQR], 3.63 [1.58–4.92] days vs. 3.16 [1.51–4.58] days; *P* <0.001).

In a multivariable analysis, suffering from ACS in 2020 was a risk factor for mortality (Figures 2, 3). Other significant risk factors for mortality included myocardial infarction at baseline and advanced age. Greater ejection fraction at baseline was a factor that decreased the risk

Table 1. Patient baseline characteristics

	2019 (June–October) 2801 patients	2020 (June–October) 2620 patients	*P*-value
Age, years, median (IQR)	67.0 (60.0–74.0)	67.0 (61.0–74.0)	0.85
Male sex, n (%)	1798 (64.2)	1637 (62.5)	0.19
Arterial hypertension, n (%)	2004 (71.5)	2102 (80.2)	<0.001
Hyperlipidemia, n (%)	1395 (49.8)	1393 (53.2)	0.01
Cholesterol at baseline, mg/dl, median (IQR)	167.0 (130.0–206.0)	183.0 (147.0–221.0)	<0.001
Low-density lipoprotein cholesterol, mg/dl, median (IQR)	107.2 (71.6–146.3)	111.5 (79.0–145.6)	0.13
High-density lipoprotein cholesterol, mg/dl, median (IQR)	42.0 (36.0–49.0)	43.0 (36.0–53.0)	0.19
Triglycerides, mg/dl, median (IQR)	115.0 (82.0–156.2)	114.0 (78.0–165.0)	0.99
Diabetes, n (%)	790 (28.2)	856 (32.7)	<0.001
Glycemia at baseline, mg/dl, median (IQR)	102.0 (87.0–123.5)	109.0 (100.0–143.5)	<0.001
Chronic kidney disease, n (%)	312 (11.1)	326 (12.4)	0.14
Creatinine, mg/dl, median (IQR)	0.9 (0.7–1.1)	0.9 (0.8–1.1)	0.004
COPD/asthma, n (%)	199 (7.1)	188 (7.2)	0.92
Malignancy, n (%)	44 (1.6)	28 (1.1)	0.11
Obesity, n (%)	864 (30.8)	858 (32.7)	0.13
Weight, kg, median (IQR)	80.0 (70.0–90.0)	81.0 (72.0–93.0)	0.01
BMI, kg/m^2, median (IQR)	28.0 (25.0–31.0)	28.0 (25.0–32.0)	0.10
Active smoking, n (%)	724 (25.8)	773 (29.5)	0.003
CCS class for angina, n (%)			
CCS II	120 (4.4)	89 (3.4)	0.21
CCS III	1651 (58.9)	1543 (58.9)	
CCS IV	1030 (36.7)	988 (37.7)	
Baseline echocardiography			
LVEF %, median (IQR)	50.0 (43.0–55.0)	50.0 (42.0–55.0)	0.71
LV-end systolic diameter, mm, median (IQR)	35.0 (30.0–40.0)	35.0 (30.0–40.0)	0.67
LV-end diastolic diameter, mm, median (IQR)	51.0 (47.0–55.0)	51.0 (47.0–55.0)	0.85
Left atrium, mm, median (IQR)	40.0 (36.0–43.0)	40.0 (36.0–44.0)	0.91
Right ventricle, mm, median (IQR)	29.0 (26.0–32.0)	29.0 (26.0–32.0)	0.26
Intraventricular septum, mm, median (IQR)	11.0 (10.0–12.0)	11.0 (10.0–12.0)	0.62
Posterior wall, mm, median (IQR)	11.0 (10.0–12.0)	11.0 (10.0–12.0)	0.47
Mitral valve regurgitation, n (%)	213 (7.6)	193 (7.4)	0.74
Aortic valve regurgitation, n (%)	26 (0.9)	26 (1.0)	0.81
Aortic valve stenosis, n (%)	81 (2.9)	76 (2.9)	0.98
Symptomatic HF, n (%)	1034 (36.9)	971 (37.1)	0.91
NYHA II class, n (%)	651 (62.9)	614 (63.2)	
NYHA III class, n (%)	300 (29.0)	244 (25.1)	0.009
NYHA IV class, n (%)	83 (8.1)	113 (11.7)	
HFrEF, n (%)	549 (53.1)	495 (50.9)	
HFmrEF, n (%)	409 (39.6)	445 (45.9)	<0.001
HFpEF, n (%)	76 (7.3)	31 (3.2)	
Blood cell count parameters, median (IQR)			
Hemoglobin, g/dl	13.1 (11.8–14.3)	13.3 (11.9–14.5)	0.12
Red blood cell count, $\times 10^{12}$/l	4.32 (3.9–4.7)	4.22 (3.8–4.6)	<0.001
White blood cell count, $\times 10^9$/l	7.5 (5.3–9.6)	7.4 (5.1–9.8)	0.20
Platelet count, $\times 10^9$/l	205.0 (166.0–245.0)	206.0 (170.0–250.0)	0.31
Hs-troponin T at baseline, pg/ml, median (IQR)	206.5 (14.0–2790.0)	725.0 (79.5–6505.0)	<0.001

Data are presented as median (interquartile range) and number (percentage)

Abbreviations: BMI, body mass index; CCS, Canadian Cardiovascular Society class for angina; COPD, chronic obstructive pulmonary disease; HF, heart failure; HFmrEF, heart failure with mid-range ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; Hs-troponin T, high sensitivity troponin T; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association class for heart failure

for mortality (Figure 2). The stepwise analysis method removed cholesterol and troponin concentrations from the model (data were entered if $P <0.3$).

Risk factors for MACCE included myocardial infarction at baseline and advanced age. However, a trend for significance of ACS timeframe as a risk factor was apparent (Figures 4, 5). The stepwise analysis method removed CCS class IV for angina, baseline troponin and cholesterol concentrations, glycemia, ejection fraction, and male sex from the model (data were entered if $P <0.3$).

DISCUSSION

The first report describing the impact of the pandemic on ACS cases in Poland was already published by Hawranek et al. [12]. The authors presented valuable data showing that the patients in 2020 were younger than in previous years, more often transferred from another hospital, were rarely referred for coronary artery bypass graft (CABG) and waited for longer periods from admission to coronarography. They also demonstrated a trend toward a higher incidence of STEMI, but no statistical significance was ob-

Table 2. Acute coronary syndrome treatment in time intervals

	2019 (June–October) 2801 patients	2020 (June–October) 2620 patients	*P*-value
Non-invasive treatment (angiography, pharmacotherapy)	214 (7.6)	134 (5.1)	<0.001
Percutaneous revascularization	2450 (87.5)	2390 (91.2)	<0.001
Infarct-related artery distribution (STEMI cases)			
LAD	272 (39.5)[a]	283 (43.5)[a]	0.33
Cx	190 (27.6)[a]	166 (25.5)[a]	
RCA	227 (32.9)[a]	202 (31.0)[a]	
Number of arteries treated (initial procedure)			
Single artery	2184 (89.1)[b]	1969 (82.4)[b]	<0.001
Two arteries	227 (9.3)[b]	389 (16.3)[b]	
Three arteries	39 (1.6)[b]	32 (1.3)[b]	
Two stages of treatment during hospitalization	534 (19.1)	263 (10.0)	<0.001
Intraprocedural glycoprotein IIb/IIIa inhibitor (intravenous)	483 (17.2)	413 (15.8)	0.14
Referred for CABG	137 (4.9)	96 (3.7)	0.03
Detailed pharmacotheraphy			
Statins	2537 (90.6)	2358 (90.0)	0.47
atorvastatin	1341 (52.8)	1228 (52.1)	0.76
rosuvastatin	1154 (45.5)	1086 (46.1)	
simvastatin	42 (1.7)	44 (1.8)	
Acetylsalicylic acid	2496 (89.1)	2361 (90.1)	0.23
P2Y12 receptor inhibitors	2564 (91.5)	2365 (90.3)	0.10
clopidogrel	1121 (43.7)	997 (42.2)	0.26
prasugrel	773 (30.1)	764 (32.3)	
ticagrelor	670 (26.2)	604 (25.5)	
Anticoagulation therapy:	340 (12.1)	354 (13.5)	0.13
VKA	31 (9.1)	31 (8.7)	0.87
NOAC	309 (90.9)	323 (91.3)	
Complete revascularization at discharge	2158 (77)	1970 (75.2)	0.11

Data are presented as numbers (percentage)
[a]Percentage of STEMI cases. [b]Percentage of patients treated invasively

Abbreviations: CABG, coronary artery bypass grafting; Cx, circumflex artery; LAD, left anterior descending artery; NOAC, non-vitamin K antagonist oral anticoagulants; RCA, right coronary artery; STEMI, ST-segment elevation myocardial infarction; VKA, vitamin K antagonists

Table 3. Hospitalization outcomes

	2019 (June–October) 2801 patients	2020 (June–October) 2620 patients	*P*-value
Mortality, n (%)	50 (1.9)	70 (2.7)	0.08
Myocardial infarction, n (%)	26 (0.9)	28 (1.1)	0.60
Stroke, n (%)	7 (0.2)	3 (0.1)	0.25
MACCE, n (%)	87 (3.1)	101 (3.8)	0.13
Minor bleeding, n (%)	11 (0.3)	5 (0.2)	0.17
Major bleeding, n (%)	2 (0.07)	3 (0.1)	0.60
Vascular complications — false aneurysm, n (%)	6 (0.2)	10 (0.4)	0.26
Stent thrombosis, n (%)	9 (0.3)	8 (0.3)	0.92
Hematoma — access site, n (%)	36 (1.3)	14 (0.5)	0.004
Duration of hospitalization, median (IQR)	3.63 (1.6– 4.9)	3.2 (1.5– 4.6)	<0.001

Abbreviations: MACCE, major adverse cardiac and cerebrovascular events (mortality, myocardial infarction, stroke)

served. The results from our study are similar with respect to corresponding endpoints; we observed a lower number of CABG cases and a higher number of STEMI cases, both of which reached statistical significance in our report. We did not observe differences in patient ages between studies. However, some differences in study design were noted, which may be the reason for the differences. One difference included the analyzed timeframe, as our study included patients from June to October, which is the interval separating the two major surges in the pandemic and mainly reflects the population's health situation following the first lockdown. In this case, healthcare availability was improved when compared to the lockdown itself, which may have contributed to different observations regarding both patient admission and effects of treatment.

Another report described hospitalizations and interventional procedures in Poland in the region inhabited by 2.5 million people during the SARS-CoV-2 pandemic. The authors noticed a lack of significant decrease in the number of STEMI patients, significant reduction of interventional revascularization procedures in NSTEMI patients, and significant decrease in the total number of hospitalizations unrelated to coronary interventions [13].

The Cox proportional-hazards regression model was designed to evaluate the strong indicators of adverse outcomes. The impact of the timeframe for admission on

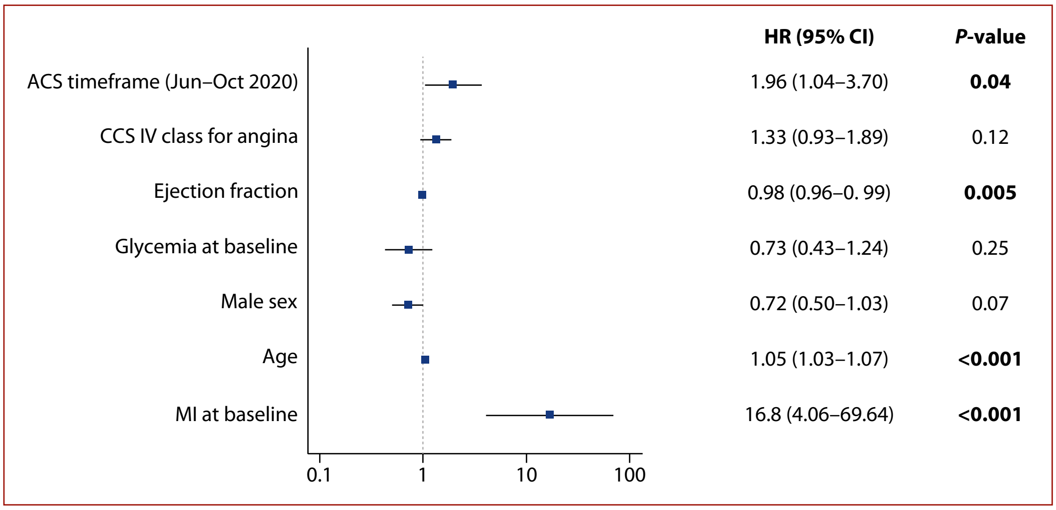

Figure 2. Forest plot of hazard ratios for mortality (Cox proportional hazards regression model). Markers represent point estimates of hazard ratios. Horizontal bars indicate 95% confidence intervals

Abbreviations: ACS, acute coronary syndrome; MI, myocardial infarction; other — see Table 2

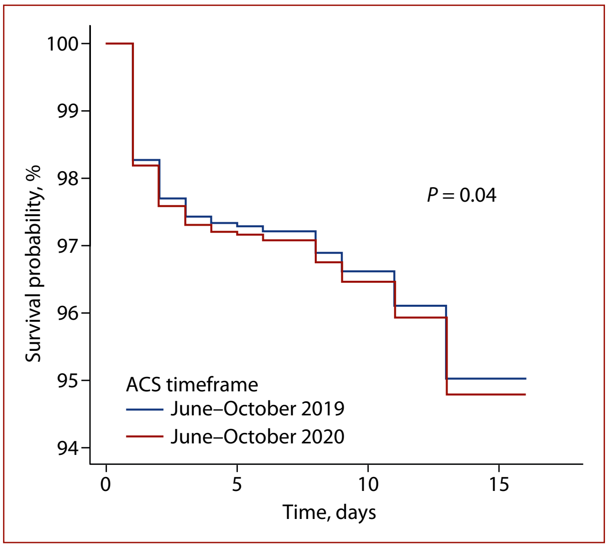

Figure 3. Cox proportional hazards cumulative survival curves with respect to different acute coronary syndrome timeframes adjusted for myocardial infarction at baseline, CCS class IV for angina, glycemia at baseline, baseline ejection fraction, male sex, and age

Abbreviations: CCS, Canadian Cardiovascular Society Class for angina; other — see Figure 2

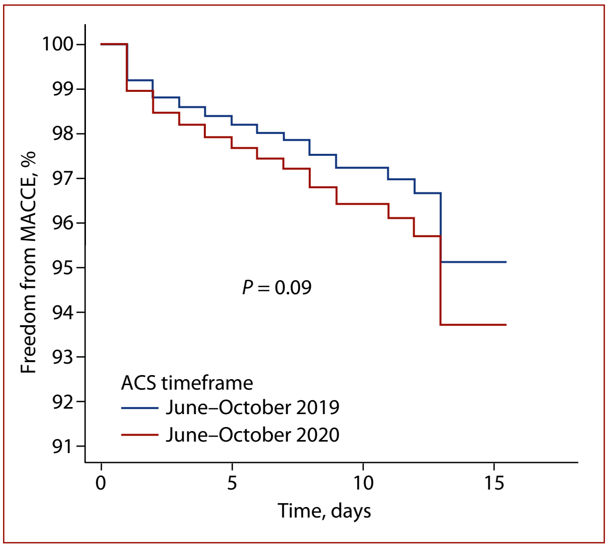

Figure 5. Cox proportional hazards freedom from MACCE curves with respect to different acute coronary syndrome timeframes adjusted for myocardial infarction at baseline and age

Abbreviations: see Figures 2, 4

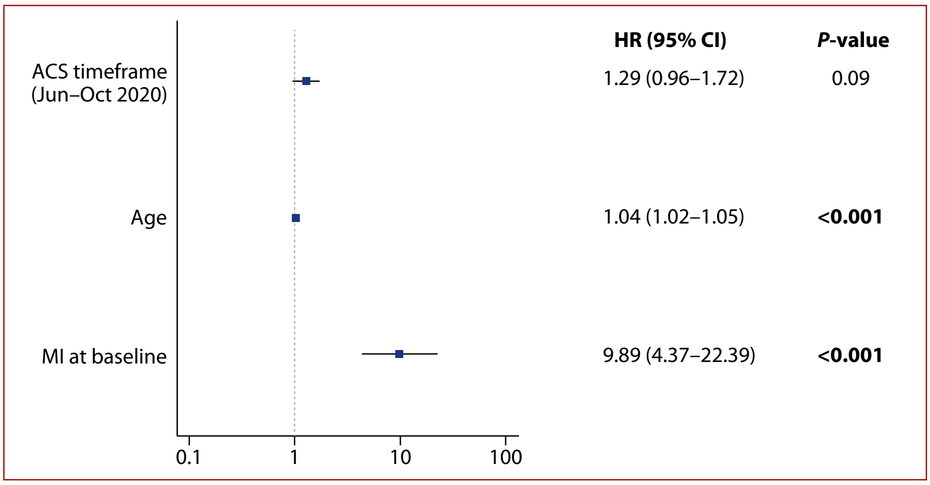

Figure 4. Forest plot of hazard ratios for MACCE (Cox proportional hazards regression model). Markers represent point estimates of hazard ratios. Horizontal bars indicate 95% confidence intervals

Abbreviations: MACCE, major adverse cardiac and cerebrovascular events (death, repeat infarction, stroke); other — see Figure 2 adverse outcomes may be associated with limited access to healthcare and fear of COVID-19 as the reasons why patients did not present for medical care when experiencing various symptoms. Another reason could be a possible delay in directing patients to an invasive cardiology department.

Regarding the analyzed time interval, two of referenced studies reported very similar observations. There was a decrease in admissions for myocardial infarction during the first wave of the pandemic, but a significant reversal in this decline in April and May 2020, following the national lockdown [7, 8].

Importantly, the number of both post-lockdown and overall pandemic deaths caused by ACS may be heavily underestimated. Some reports pointed out an increase in the number of out-of-hospital deaths and cardiac arrests when compared to the period before the pandemic [14].

The same study reported that in-hospital survival after out-of-hospital cardiac arrest was 64% lower than before the pandemic [14]. Other reports show an increase in hospital deaths following the lockdown, which ranged from 4.1% to 9.6% when compared with the corresponding time interval before the pandemic [7, 15–17]. This finding may be caused by both limited access to healthcare facilities and also by delay in patients' reaction to symptoms. This important aspect showing the fear of COVID-19 as a reason for patients not presenting for medical care when experiencing various symptoms, including chest pain, has already been reported by multiple studies [18–21].

The issue of the number of non-communicable diseases in the analyzed time intervals should not be avoided. The global population was forced to modify social behavior for both epidemiological and economic reasons. In most cases, modifications were associated with changes in diet and avoidance of physical exercise.

It is not feasible that the lockdown alone could have resulted in such a rapid development of the disease itself. However, the lack of proper treatment for non-communicable diseases surely produced an impact on the incidence of ACS cases by triggering adverse events. Both systolic and diastolic hypertension independently predicted adverse outcomes, including myocardial infarction [22]. A linear increase in the risk of MI with an increase in blood pressure has been reported [23]. Poor glycemic control in diabetic patients increases inflammatory responses, induces apoptosis, causes endothelial dysfunction, and stimulates platelet aggregation and accumulation [24–28], and as such, may significantly contribute to worsening the frequency and prognosis in ACS patients. Notably, several patients in both groups were not in CCS class IV for angina, which may be related to both a great incidence of CCS III unstable angina and high incidence of uncontrolled diabetes, which affects the symptoms significantly [29, 30]. Smoking is obviously one of major risk factors for coronary artery disease, and the risk of acute myocardial infarction increases with the number of cigarettes smoked per day [31–33]. Notably, it has been proven that during the pandemic people smoked more, driven by COVID-19-related stress, more time spent at home, and boredom [34]. These stressful situations probably became aggravated when the lockdown ended and new challenges in daily routine, still affected by the pandemic, emerged.

The association between populational health and mental and social issues only worsened the situation [35–40]. From this perspective, further increases, not only in incidence of acute coronary syndromes but also in occurrence of more complex cases can be expected.

In-hospital treatment for ACS also differed when compared to the corresponding timeframe of the previous calendar year. First, a higher number of patients qualified for percutaneous revascularization. This finding is expected as a higher incidence of STEMI could be one of the reasons for performing salvage percutaneous coronary intervention (PCI) instead of qualifying patients for surgical treatment by a heart team [1, 2]. However, some other negative effects of the pandemic can be observed. From patients' perspectives, multiple hospitalizations for diagnosis, further preparation for surgery, transfer from one hospital to another, long therapeutic processes, and rehabilitation are particularly dangerous in epidemiological terms. As such, most heart teams probably favored shorter therapeutic processes and qualified borderline cases for percutaneous treatment. Second, it was obvious that patients with lower peri-operative immunity are especially prone to infection, which may have significantly changed the mortality and morbidity rates. From a surgical perspective, surgical procedures during the pandemic should focus on the most urgent cases that cannot be postponed and cannot be treated percutaneously. This observation is supported by international reports, which also present a decrease in the number of surgically treated patients [41].

Study limitations

The design of the study (retrospective dataset analysis) has the limitations of such reports. Moreover, it was difficult to assess true long-term survival and complication incidence in those patients, as they were admitted to the hospital with a much higher occurrence of comorbidities when compared to the pre-pandemic period. This aspect may affect the incidence of repeat ACS cases, morbidity, and mortality in upcoming years.

In conclusion, it should be emphasized that the COVID-19 pandemic affects the ACS patient profile, course of treatment, and increases the risk for mortality. This effect already became apparent after the first wave of the pandemic in Poland. Further progression of this effect can be expected.

Article information

Conflict of interest: None declared.

Funding: None.

REFERENCES

1. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021; 42(14): 1289–1367, doi: 10.1093/eurheartj/ehaa575, indexed in Pubmed: 32860058.
2. Neumann FJ, Sousa-Uva M, Ahlsson A, et al. 2018 ESC/EACTS Guidelines on myocardial revascularization. Eur Heart J. 2019; 40(2): 87–165, doi: 10.1093/eurheartj/ehy394, indexed in Pubmed: 30165437.
3. Perrin N, Iglesias JF, Rey F, et al. Impact of the COVID-19 pandemic on acute coronary syndromes. Swiss Med Wkly. 2020; 150: w20448, doi: 10.4414/smw.2020.20448, indexed in Pubmed: 33382905.
4. Secco GG, Zocchi C, Parisi R, et al. Decrease and delay in hospitalization for acute coronary syndromes during the 2020 SARS-CoV-2 pandemic. Can J Cardiol. 2020; 36(7): 1152–1155, doi: 10.1016/j.cjca.2020.05.023, indexed in Pubmed: 32447060.
5. Showkathali R, Yalamanchi R, Sankeerthana MP, et al. Acute Coronary Syndrome admissions and outcome during COVID-19 Pandemic-Report from large tertiary centre in India. Indian Heart J. 2020; 72(6): 599–602, doi: 10.1016/j.ihj.2020.09.005, indexed in Pubmed: 33357652.
6. De Filippo O, D'Ascenzo F, Angelini F, et al. Reduced rate of hospital admissions for ACS during COVID-19 outbreak in Northern Italy. N Engl J Med. 2020; 383(1): 88–89, doi: 10.1056/NEJMc2009166, indexed in Pubmed: 32343497.
7. Gluckman TyJ, Wilson MA, Chiu ST, et al. Case rates, treatment approaches, and outcomes in acute myocardial infarction during the coronavirus disease 2019 pandemic. JAMA Cardiol. 2020; 5(12): 1419–1424, doi: 10.1001/jamacardio.2020.3629, indexed in Pubmed: 32766756.
8. Mafham MM, Spata E, Goldacre R, et al. COVID-19 pandemic and admission rates for and management of acute coronary syndromes in England. Lancet. 2020; 396(10248): 381–389, doi: 10.1016/S0140-6736(20)31356-8, indexed in Pubmed: 32679111.
9. Kiss P, Carcel C, Hockham C, et al. The impact of the COVID-19 pandemic on the care and management of patients with acute cardiovascular disease: a systematic review. Eur Heart J Qual Care Clin Outcomes. 2021; 7(1): 18–27, doi: 10.1093/ehjqcco/qcaa084, indexed in Pubmed: 33151274.
10. Kwok CS, Gale CP, Curzen N, et al. Impact of the COVID-19 pandemic on percutaneous coronary intervention in England: insights from the British Cardiovascular Intervention Society PCI database cohort. Circ Cardiovasc Interv. 2020; 13(11): e009654, doi: 10.1161/CIRCINTERVENTIONS.120.009654, indexed in Pubmed: 33138626.
11. Statistics related to covid-19 infection, Polish Primary Statistical Department. Available at: https://stat.gov.pl/.
12. Hawranek M, Grygier M, Bujak K, et al. Characteristics of patients from the Polish Registry of Acute Coronary Syndromes during the COVID-19 pandemic: the first report. Kardiol Pol. 2021; 79(2): 192–195, doi: 10.33963/KP.15756, indexed in Pubmed: 33463992.
13. Drożdż J, Piotrowski G, Zielińska M, et al. Hospitalizations and interventional procedures in cardiology departments in the region of 2.5 million inhabitants during the SARS-CoV-2 pandemic. Kardiol Pol. 2021; 79(5): 572–574, doi: 10.33963/KP.15984, indexed in Pubmed: 34125933.
14. Marijon E, Karam N, Jost D, et al. Out-of-hospital cardiac arrest during the COVID-19 pandemic in Paris, France: a population-based, observational study. Lancet Public Health. 2020; 5(8): e437–e443, doi: 10.1016/S2468-2667(20)30117-1, indexed in Pubmed: 32473113.
15. De Rosa S, Spaccarotella C, Basso C, et al. Reduction of hospitalizations for myocardial infarction in Italy in the COVID-19 era. Eur Heart J. 2020; 41(22): 2083–2088, doi: 10.1093/eurheartj/ehaa409, indexed in Pubmed: 32412631.
16. Popovic B, Varlot J, Metzdorf PA, et al. Changes in characteristics and management among patients with ST-elevation myocardial infarction due to COVID-19 infection. Catheter Cardiovasc Interv. 2021; 97(3): E319–E326, doi: 10.1002/ccd.29114, indexed in Pubmed: 32667726.
17. Tam CCF, Cheung KS, Lam S, et al. Impact of coronavirus disease 2019 (COVID-19) outbreak on outcome of myocardial infarction in Hong Kong, China. Catheter Cardiovasc Interv. 2021; 97(2): E194–E197, doi: 10.1002/ccd.28943, indexed in Pubmed: 32367683.
18. Lazzerini M, Barbi E, Apicella A, et al. Delayed access or provision of care in Italy resulting from fear of COVID-19. Lancet Child Adolesc Health. 2020; 4(5): e10–e11, doi: 10.1016/S2352-4642(20)30108-5, indexed in Pubmed: 32278365.
19. Marín-Jiménez I, Zabana Y, Rodríguez-Lago I, et al. COVID-19 and inflammatory bowel disease: questions arising from patient care and follow-up during the initial phase of the pandemic (February-April 2020). Gastroenterol Hepatol. 2020; 43(7): 408–413, doi: 10.1016/j.gastrohep.2020.05.003, indexed in Pubmed: 32419715.
20. Hammad TA, Parikh M, Tashtish N, et al. Impact of COVID-19 pandemic on ST-elevation myocardial infarction in a non-COVID-19 epicenter. Catheter Cardiovasc Interv. 2021; 97(2): 208–214, doi: 10.1002/ccd.28997, indexed in Pubmed: 32478961.
21. Pessoa-Amorim G, Camm CF, Gajendragadkar P, et al. Admission of patients with STEMI since the outbreak of the COVID-19 pandemic: a survey by the European Society of Cardiology. Eur Heart J Qual Care Clin Outcomes. 2020; 6(3): 210–216, doi: 10.1093/ehjqcco/qcaa046, indexed in Pubmed: 32467968.
22. Flint AC, Conell C, Ren X, et al. Effect of systolic and diastolic blood pressure on cardiovascular outcomes. N Engl J Med. 2019; 381(3): 243–251, doi: 10.1056/NEJMoa1803180, indexed in Pubmed: 31314968.
23. Ali I, Akman D, Bruun NE, et al. Importance of a history of hypertension for the prognosis after acute myocardial infarction--for the Bucindolol Evaluation in Acute myocardial infarction Trial (BEAT) study group. Clin Cardiol. 2004; 27(5): 265–269, doi: 10.1002/clc.4960270504, indexed in Pubmed: 15188939.
24. Esposito K, Nappo F, Marfella R, et al. Inflammatory cytokine concentrations are acutely increased by hyperglycemia in humans: role of oxidative stress. Circulation. 2002; 106(16): 2067–2072, doi: 10.1161/01.cir.0000034509.14906.ae, indexed in Pubmed: 12379575.
25. Risso A, Mercuri F, Quagliaro L, et al. Intermittent high glucose enhances apoptosis in human umbilical vein endothelial cells in culture. Am J Physiol Endocrinol Metab. 2001; 281(5): E924–E930, doi: 10.1152/ajpendo.2001.281.5.E924, indexed in Pubmed: 11595647.
26. Williams SB, Goldfine AB, Timimi FK, et al. Acute hyperglycemia attenuates endothelium-dependent vasodilation in humans in vivo. Circulation. 1998; 97(17): 1695–1701, doi: 10.1161/01.cir.97.17.1695, indexed in Pubmed: 9591763.
27. Stegenga ME, van der Crabben SN, Levi M, et al. Hyperglycemia stimulates coagulation, whereas hyperinsulinemia impairs fibrinolysis in healthy humans. Diabetes. 2006; 55(6): 1807–1812, doi: 10.2337/db05-1543, indexed in Pubmed: 16731846.
28. Sakamoto T, Ogawa H, Kawano H, et al. Rapid change of platelet aggregability in acute hyperglycemia. Detection by a novel laser-light scattering method. Thromb Haemost. 2000; 83(3): 475–479, indexed in Pubmed: 10744156.
29. Elliott MD, Heitner JF, Kim H, et al. Prevalence and prognosis of unrecognized myocardial infarction in asymptomatic patients with diabetes: a two-center study with up to 5 years of follow-up. Diabetes Care. 2019; 42(7): 1290–1296, doi: 10.2337/dc18-2266, indexed in Pubmed: 31010876.
30. MacDonald MR, Petrie MC, Home PD, et al. Incidence and prevalence of unrecognized myocardial infarction in people with diabetes: a substudy of the Rosiglitazone Evaluated for Cardiac Outcomes and Regulation of Glycemia in Diabetes (RECORD) study. Diabetes Care. 2011; 34(6): 1394–1396, doi: 10.2337/dc10-2398, indexed in Pubmed: 21562320.
31. Friedman GD, Petitti DB, Bawol RD, et al. Mortality in cigarette smokers and quitters. Effect of base-line differences. N Engl J Med. 1981; 304(23): 1407–1410, doi: 10.1056/NEJM198106043042308, indexed in Pubmed: 7231464.

32. Willett WC, Green A, Stampfer MJ, et al. Relative and absolute excess risks of coronary heart disease among women who smoke cigarettes. N Engl J Med. 1987; 317(21): 1303–1309, doi: 10.1056/NEJM198711193172102, indexed in Pubmed: 3683458.
33. Teo KK, Ounpuu S, Hawken S, et al. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. Lancet. 2006; 368(9536): 647–658, doi: 10.1016/S0140-6736(06)69249-0, indexed in Pubmed: 16920470.
34. Popova L, Henderson K, Kute N, et al. "I'm bored and I'm stressed": A qualitative study of exclusive smokers, ENDS users, and transitioning smokers or ENDS users in the time of COVID-19. Nicotine Tob Res. 2021 [Epub ahead of print], doi: 10.1093/ntr/ntab199, indexed in Pubmed: 34610133.
35. Holland D, Heald AH, Stedman M, et al. Assessment of the effect of the COVID-19 pandemic on UK HbA1c testing: implications for diabetes management and diagnosis. J Clin Pathol. 2021 [Epub ahead of print], doi: 10.1136/jclinpath-2021-207776, indexed in Pubmed: 34645702.
36. Clemmensen C, Petersen MB, Sørensen TIA. Will the COVID-19 pandemic worsen the obesity epidemic? Nat Rev Endocrinol. 2020; 16(9): 469–470, doi: 10.1038/s41574-020-0387-z, indexed in Pubmed: 32641837.
37. Pettus J, Skolnik N. Importance of diabetes management during the COVID-19 pandemic. Postgrad Med. 2021; 133(8): 912–919, doi: 10.1080/00325481.2021.1978704, indexed in Pubmed: 34602003.
38. Gopalan HS, Misra A. COVID-19 pandemic and challenges for socio-economic issues, healthcare and National Health Programs in India. Diabetes Metab Syndr. 2020; 14(5): 757–759, doi: 10.1016/j.dsx.2020.05.041, indexed in Pubmed: 32504992.
39. Banerjee M, Chakraborty S, Pal R. Diabetes self-management amid COVID-19 pandemic. Diabetes Metab Syndr. 2020; 14(4): 351–354, doi: 10.1016/j.dsx.2020.04.013, indexed in Pubmed: 32311652.
40. Lim MA, Huang I, Yonas E, et al. A wave of non-communicable diseases following the COVID-19 pandemic. Diabetes Metab Syndr. 2020; 14(5): 979–980, doi: 10.1016/j.dsx.2020.06.050, indexed in Pubmed: 32610263.
41. Gaudino M, Chikwe J, Hameed I, et al. Response of Cardiac Surgery Units to COVID-19: An Internationally-Based Quantitative Survey. Circulation. 2020; 142(3): 300–302, doi: 10.1161/CIRCULATIONAHA.120.047865, indexed in Pubmed: 32392425.