sciendo

Journal of Interdisciplinary Medicine

ORIGINAL RESEARCH

CARDIOLOGY // EMERGENCY MEDICINE

Myocardial Infarction in the Context of COVID-19

Adrienn Nemeth[1], Theodora Benedek[2]

[1] Emergency Department, Emergency County Hospital, Târgu Mureș, Romania
[2] Clinic of Cardiology, "George Emil Palade" University of Medicine and Pharmacy, Science and Technology, Târgu Mureș, Romania

CORRESPONDENCE

Alessandra-Aniela Cerghedi
Adrienn Nemeth
Str. Gheorghe Marinescu nr. 50
540139 Târgu Mureș, Romania
Tel: +40 746 812 890
Email: nemeth_adrienn@yahoo.com

ARTICLE HISTORY

Received: September 1, 2023
Accepted: September 7, 2023

Theodora Benedek • Str. Gheorghe Marinescu nr. 38, 540136 Târgu Mureș, Romania. Tel: +40 265 215 551, Email: theodora.benedek@gmail.com

ABSTRACT

Introduction: The COVID-19 pandemic that started in 2019 was a modern-world challenge for medical professionals. The SARS-Cov-2 virus targeted the respiratory and, later, the cardiovascular system. We aimed to identify the risk factors and particularities of acute myocardial infarction associated with SARS-Cov-2 infection. **Material and Methods:** This cross-sectional study included 92 patients admitted to the Cardiology Department of Mureș County Emergency Hospital with myocardial infarction, divided into two groups: 46 patients with COVID-19 and 46 patients without COVID-19. Demographic data, risk factors, non-communicable diseases, and laboratory findings were studied and compared. **Results:** The mean age of the patients was 65 years, and the majority were male. The identified risk factors were hypertension, body mass index >25 kg/m2, and dyslipidemia. The risk factors for poor prognosis were leukocyte count, higher neutrophil-lymphocyte ratio, higher monocyte-to-lymphocyte ratio, and higher high sensitivity troponin I levels. Left ventricular ejection fraction was significantly lower in patients with COVID-19. **Conclusions:** COVID-19 is an aggravating factor of acute myocardial infarction. This research highlights the importance of prevention against the SARS-CoV-2 virus.

Keywords: myocardial infarction, COVID-19, risk factors

INTRODUCTION

In addition to respiratory complications, the cardiovascular system is affected by the SARS-CoV-2 virus by increasing the risk of developing thrombotic incidents: myocardial infarction, pulmonary embolism, and stroke.[1–3] Several studies outlined the risk factors for COVID-19, such as cardiometabolic conditions, age, sex, and ethnicity, some of which can be superposed with the risk factors for cardiovascular diseases.[4,5]

Studies show a less favorable outcome for patients with acute coronary syndrome and COVID-19.[6–9] A concomitant diagnosis of COVID-19 was significantly associated with higher rates of in-hospital mortality compared with patients without a diagnosis of COVID-19.[10] Although acute myocardial infarction rates were lower during the early days of the pandemic, in-hospital and 1-month mortality rates were higher during the year 2020 compared to 2019.[11]

The hypothesis of this research is based on the presumption that patients with myocardial infarction and COVID-19 infection present with more severe forms of illness, cardiac pump dysfunction, and significantly decreased ejection fraction.

MATERIALS AND METHODS

This cross-sectional study included 92 patients admitted between January 2017 and February 2022 to the Department of Cardiology of Mureș County Emergency Hospital with myocardial infarction. The study population was split into two groups: the COVID group included 46 patients with simultaneous diagnosis of SARS-CoV-2 infection, revealed by typical lesions on the computed tomography (CT) scan, positive RT-PCR test, or COVID-19 antigen rapid test, and the NON-COVID group included 46 patients without evidence of SARS-CoV-2 infection.

Exclusion criteria were systemic diseases, undergoing chemotherapy, cardiotoxic treatment, cardiac devices, and trauma patients. Incomplete files were excluded.

The analyzed parameters included age, sex, body mass index (BMI), personal history of cardiovascular diseases; hypertension, diabetes mellitus, chronic smoking, dyslipidemia; level of serum lipids: cholesterol, triglycerides, routine blood test and number of lymphocytes, thrombocytes, neutrophils, cardiac enzymes: creatine kinase-myocardial band (CK-MB), high sensitivity troponin I (hsTnI), and N-terminal pro-B-type natriuretic peptide (NT-proBNP).

Diabetes mellitus was defined as patients having fasting plasma glucose ≥126 mg/dL and/or post-prandial plasma glucose ≥200 mg/dL and/or A1c ≥6.5% or a history of diabetes and/or taking medication for diabetes. Overweight was defined as a BMI >25 kg/m^2 and <30 kg/m^2, whereas obesity was defined as a BMI ≥30 kg/m^2 calculated using Quetlet's formula. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg and/or on antihypertensive treatment. Chronic kidney disease was defined as a glomerular filtration rate (GFR) of <90 mL/min or serum creatinine >1.2 mg/dL. Pulmonary diseases were defined as previously diagnosed by a physician and/or chronic treatment and/or CT scan lesions.

Statistical analysis

Statistical analyses were performed using the demo version of GraphPad and Microsoft Excel. Continuous variables were expressed as mean ± standard deviation (SD). Categorical variables were given as numbers or percentages. The comparison between groups was done using the Mann–Whitney U test for continuous variables and the chi-squared test or Fisher's exact test for categorical variables. A p value of <0.05 was considered statistically significant.

Ethics

The study was approved by the ethics committee of the "George Emil Palade" University of Medicine, Pharmacy, Science and Technology from Târgu Mureș (no. 1659/14.03.2022) and the ethics committee of Târgu Mureș Emergency County Hospital (no. 33208/04.02.2022).

RESULTS

Patients with myocardial infarction and COVID-19 infection were aged between 46 and 76 years, with a mean age of 64.4 years. Of the 46 patients, 33 were female and 13 were male, and 74% survived the illness. Regarding BMI, 83% had a BMI over 25 kg/m^2, and 50% had a BMI over 30 kg/m^2.

Hypertension was observed in 93.47 % (n = 43), 26.08% (n = 12) suffered from diabetes mellitus, and 32.6 % (n = 15) presented with chronic kidney disease. Chronic pulmonary illness affected 48% of patients, and 37% were active smokers at hospital admission. Only 15% of patients presented a history of myocardial infarction. Risk factors are presented in Figure 1.

The mean hospitalization time was 9 days. Most admissions lasted over a week: 8 days for 16 patients (35%) and 11 days for 13 patients (28%). Nine patients (20%) needed more than 2 weeks of hospitalization, and seven were discharged less than a week after admission.

The damage to myocardial tissue is reflected in ejection fraction depression. The study revealed that 58% of the patients who suffered a myocardial infarction and tested positive for COVID-19 had a severe decrease in left ventricular ejection fraction (LVEF) (less than 40%), 32% had an intermediate decrease (between 40% and 49%), and 9% had average ejection fraction.

A total of 38 patients (83%) underwent successful percutaneous transluminal coronary angioplasty (PTCA), and 8 patients (17%) received medical therapy in the form of thrombolysis. A total of 34 patients survived, and 12 patients deceased. Therefore, the survival rate was 74%.

We compared survivor and deceased patients with regards to risk factors, but we found no significant differences (Table 1). However, hospitalization was significantly shorter in the deceased group (p = 0.0056),

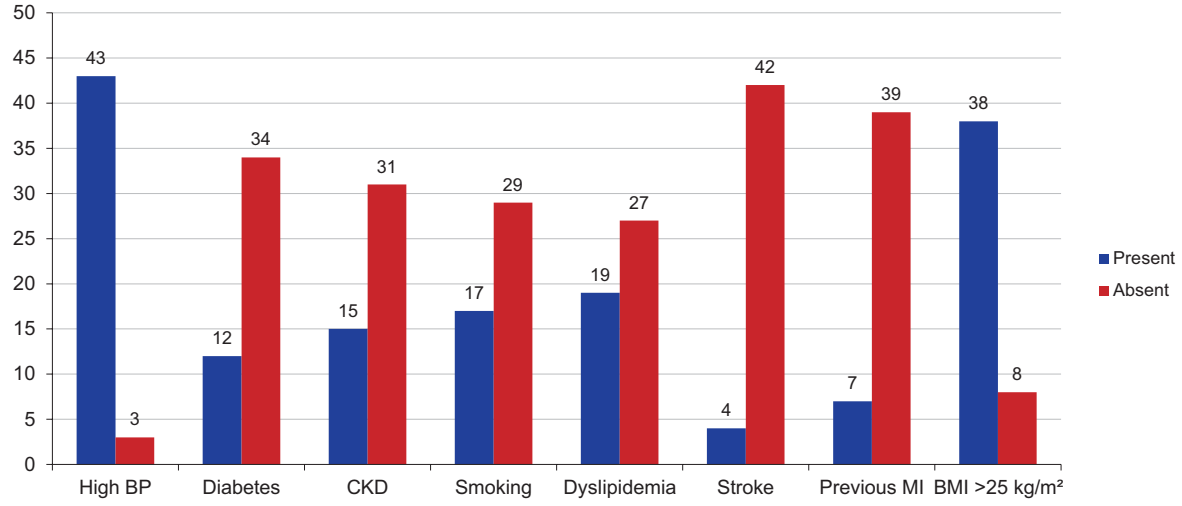

FIGURE 1. Risk factors in myocardial infarction and COVID-19. BP, blood pressure; CKD, chronic kidney disease, MI, myocardial infarction

with a mean of 6 days. The mean BMI was 32.54 kg/m² in the deceased group, higher than in the survivor group (p = 0.0447). hsTnI levels were significantly higher (p = 0.043), while the leukocyte count was significantly lower (p <0.0001) in the survivor group. The neutrophil-to-lymphocyte ratio (NLR) revealed a mild stress level in the survivor group and severe stress in the deceased group (p = 0.0154).

Comparing the two groups, we found significant differences regarding BMI – infected patients had increased body weight, and their heart function was more affected by myocardial infarction – LVEF had a lower value.

TABLE 1. Comparison between deceased and survivors with myocardial infarction and COVID-19

Variable	Survivors	Deceased	p value
Age (years)	63.08 ± 11.77	68.16 ± 14.88	0.2373
Sex			
Female	7	6	0.0699
Male	27	6	
Days of hospitalization	10 ± 4	6 ± 3	0.0056
BMI	29.93 ± 6.72	32.54 ± 3.34	0.0447
Leukocyte count (/mm³)	11,102 ± 3,684	17,284 ± 4,845	<0.0001
NLR	7.39 ± 6.3	14.16 ± 10.5	0.0154
PLR	198.34 ± 133.64	292.37 ± 162.79	0.0541
MLR	0.7424 ± 0.36	1.16 ± 0.82	0.0289
Triglycerides (mg/dL)	153.23 ± 54.7	159.75 ± 45.4	0.72
Colesterol (mg/dL)	143.85 ± 80.16	160.83 ± 50	0.497
NT-proBNP (pg/mL)	6782.11 ± 8129	11524.33 ± 9457.2	0.19
CK-MB (ng/mL)	177.19 ± 162.28	134.91 ± 43.12	0.9354
hsTnI (ng/mL)	9770.476 ± 7816.9	4131.41 ± 2211.7	0.0433
Pulmonary disease			
Yes	19	5	0.5012
No	14	7	
LVEF (%)	38.40 ± 7.488	35.5 ± 5.633	0.2492
Treatment (n)			
PTCA	30	8	0.1778
Medical	4	4	

PLR, platelet-to-lymphocyte ratio

DISCUSSION

This research studied patients suffering from myocardial infarction and COVID-19 infection and found correlations between risk factors, laboratory findings, and myocardial damage following acute coronary events.

COVID-19 infection causes respiratory failure and heart tissue damage; research from the last 3 years outlines the virus' tropism for myocardial tissue.[12,13] LVEF was significantly lower in patients with COVID-19, and there were no significant differences between survivors and deceased patients ($p = 0.2492$), meaning that myocardial damage was present regardless of the clinical outcome.

Increased BMI was a risk factor that presented statistical significance in both comparisons ($p = 0.03$ and $p = 0.04$), suggesting that patients with a higher body weight are more vulnerable to infection, and that an increased BMI is a poor prognostic factor in both myocardial infarction and COVID-19. Increased leukocyte count, NLR, monocyte-to-lymphocyte ratio (MLR), and hsTnI levels were also factors predicting a poor prognosis.

NLR is a prognostic factor used in intensive care that reflects the physiologic stress described by Zahorec *et al.*[14] More recent research by Yang *et al*. highlights the predictive value of NLR as an independent risk factor and high body weight in the context of COVID-19 infection. NLR is a reliable prognostic score in myocardial infarction and COVID-19 infection.[15]

hsTnI levels were significantly lower in deceased patients, which contradicts some of the research indicating it is a marker of cardiac tissue damage. One possible source of error can be the reduced number of subjects included in the study, as data from the literature reflect the results from 525 medical units.[12,13] On the other hand, these numbers can be interpreted in the context that hsTnI peaks 18–24 h from onset and then decreases in the following 2 weeks. Since deceased patients presented a shorter hospitalization and earlier death, the survivors might have presented sepsis and longer, constant cardiac tissue damage, reflected in the elevation of hsTnI.[16] Another hypothesis is that deceased patients were hospitalized earlier for COVID-19, and death may have occurred before the hsTnI peak if the acute coronary syndrome happened during hospitalization. Furthermore, in the presence of multiple chronic illnesses, deceased subjects might have tolerated poorly the myocardial dysfunction and presented earlier to the emergency department compared to survivors, when their hsTnI levels were lower.

CONCLUSION

COVID-19 infection is an aggravating factor of myocardial infarction, and this research outlines the importance of preventive measures in and out of the hospital. Poor prognosis factors identified are leukocyte count, NLR, MLR, level of hsTnI, and increased body weight. Educating the population to reduce cardiovascular risk factors is essential for prevention. However, the COVID-19 pandemic was a test for humanity and forced the medical system to fight and create instruments to treat an unknown disease with yet unknown long-term effects.

CONFLICT OF INTEREST

Nothing to declare.

REFERENCES

1. Novelli G, Biancolella M, Mehrian-Shai R, et al. COVID-19 update: the first 6 months of the pandemic. *Hum Genomics*. 2020;14:48.
2. Thygesen K. 'Ten Commandments' for the Fourth Universal Definition of Myocardial Infarction 2018. *Eur Heart J*. 2019;40:226.
3. Forchette L, Sebastian W, Liu T. A Comprehensive Review of COVID-19 Virology, Vaccines, Variants, and Therapeutics. *Curr Med Sci*. 2021;41:1037–1051.
4. O'Hearn M, Liu J, Cudhea F, et al. Coronavirus Disease 2019 Hospitalizations Attributable to Cardiometabolic Conditions in the United States: A Comparative Risk Assessment Analysis. *J Am Heart Assoc*. 2021;10:e019259.
5. Caramelo F, Ferreira N, Oliveiros B. Estimation of risk factors for COVID-19 mortality – preliminary results. Preprint at medRxiv. 2020;02.24.20027268.
6. Biswas S, Thakur V, Kaur P, et al. Blood clots in COVID-19 patients: simplifying the curious mystery. *Med Hypotheses*. 2021;146:110371.
7. To KK, Sridhar S, Chiu KH, et al. Lessons learned 1 year after SARS-CoV-2 emergence leading to COVID-19 pandemic. *Emerg Microbes Infect*. 2021;10:507–535.
8. Del Prete A, Conway F, Della Rocca DG, et al. COVID-19, Acute Myocardial Injury, and Infarction. *Card Electrophysiol Clin*. 2022;14:29–39.
9. Katsoularis I, Fonseca-Rodríguez O, Farrington P et al. Risk of acute myocardial infarction and ischaemic stroke following COVID-19 in Sweden: a self-controlled case series and matched cohort study. *Lancet*. 2021;398:599–607.
10. Saad M, Kennedy KF, Imran H, et al. Association Between COVID-19 Diagnosis and In-Hospital Mortality in Patients Hospitalized With ST-Segment Elevation Myocardial Infarction. *JAMA*. 2021;326:1940–1952.
11. Mefford MT, An J, Gupta N, et al. Rates of Acute Myocardial Infarction During the COVID-19 Pandemic. *Perm J*. 2021;25:21.074.
12. Fu T, Mamaliga G, Pierce JD, Gilkeson R, Gupta A. COVID-19 infection complicated by acute ST-elevation myocardial infarction. *Clin Imaging*. 2021;78:117–120.
13. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. *JAMA Cardiol*. 2020;5:831–840.
14. Zahorec R. Neutrophil-to-lymphocyte ratio, past, present and future perspectives. *Bratisl Lek Listy*. 2021;122:474–488.
15. Yang AP, Liu JP, Tao WQ, Li HM. The diagnostic and predictive role of NLR, d-NLR, and PLR in COVID-19 patients. *Int Immunopharmacol*. 2020;84:106504.
16. Simoiu M. Importanța troponinei ca marker al leziunilor miocardice în sepsis [The importance of troponin as marker of cardiac injuries in sepsis]. *Practica Medicală*. 2012;VII:219–221.