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Increased risk of acute myocardial infarction after COVID-19 recovery: A systematic review and meta-analysis

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ABSTRACT

Background: Few studies have analyzed the incidence and the risk of acute myocardial infarction (AMI) during the post-acute phase of COVID-19 infection.
Objective: To assess the incidence and risk of AMI in COVID-19 survivors after SARS-CoV-2 infection by a systematic review and meta-analysis of the available data.
Methods: Data were obtained searching MEDLINE and Scopus for all studies published at any time up to September 1, 2022 and reporting the risk of incident AMI in patients recovered from COVID-19 infection. AMI risk was evaluated using the Mantel–Haenszel random effects models with Hazard ratio (HR) as the effect measure with 95% confidence interval (CI) while heterogeneity was assessed using Higgins and Thomson I^2 statistic.
Results: Among 2765 articles obtained by our search strategy, four studies fulfilled the inclusion criteria for a total of 20,875,843 patients (mean age 56.1 years, 59.1% males). Of them, 1,244,604 had COVID-19 infection. Over a mean follow-up of 8.5 months, among COVID-19 recovered patients AMI occurred in 3.5 cases per 1.000 individuals compared to 2.02 cases per 1.000 individuals in the control cohort, defined as those who did not experience COVID-19 infection in the same period). COVID-19 patients showed an increased risk of incident AMI (HR: 1.93, 95% CI: 1.65–2.26, $p < 0.0001$, $I^2 = 83.5\%$). Meta-regression analysis demonstrated that the risk of AMI was directly associated with age ($p = 0.01$) and male gender ($p = 0.001$), while an indirect relationship was observed when the length of follow-up was utilized as moderator ($p < 0.001$).
Conclusion: COVID-19 recovered patients had an increased risk of AMI.

1. Introduction

Previous investigations have already reported that viral infections may represent a potential cause of AMI, especially when the respiratory tract is involved [1]. To this regard, recent analyses have demonstrated that COVID-19 is associated with an increased risk of acute myocardial infarction (AMI) [2]. However, recent studies examining the relationship between AMI and SARS-CoV-2 infection have mainly focused on the potential pathophysiological mechanisms underlying this relationship [3,4]. Conversely, data regarding the risk of AMI as a post-acute COVID-19 sequelae remain scant. Aim of the present manuscript is to assess the risk of incident AMI in COVID-19 recovered patients by performing a systematic review and meta-analysis of the available data.

2. Material and methods

2.1. Study design

This study followed the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) reporting guideline (Supplementary file 1) [5]. Data were obtained searching MEDLINE and Scopus for all studies published at any time up to September 1, 2022 and

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reporting the risk of incident AMI in COVID-19 recovered patients diagnosed between 4 months (minimum follow-up length of revised investigations) and a maximum of 12 months post discharge (maximum follow-up length of revised studies) after the infection. In the revised manuscripts, this group of patients were compared to contemporary cohorts, defined as subjects who did not experience the SARS-CoV-2 infection and developed an AMI in the same follow-up period.

2.2. Data extraction and quality assessment

The selection of studies included in our analysis was independently conducted by two authors (M.Z., C.B.) in a blinded fashion. Any discrepancies in study selection were resolved by consulting a third author (G.R.). The following MeSH terms were used for the search: "Acute myocardial infarction" AND "COVID-19 sequelae" OR "Acute myocardial infarction" AND "COVID-19". Moreover, we searched the bibliographies of the target studies for additional references. Specifically, inclusion criteria were: (i) studies enrolling subjects with previous confirmed COVID-19 infection (ii) providing the hazard ratio (HR) and relative 95% confidence interval (CI) for the risk of incident AMI after the infection compared to contemporary control cohorts. Conversely, case reports, review articles, abstracts, editorials/letters, and case series with <10 participants were excluded. Data extraction was independently conducted by two authors (M.Z., G.R). For all the reviewed investigations we extracted, when provided, the number of enrolled patients, the mean age, the gender, the prevalence of cardiovascular comorbidities such as arterial hypertension (HT), diabetes mellitus (DM), chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), obesity, pre-existing heart failure (HF), cerebrovascular disease and the length of follow-up. The quality of included studies was graded using the Newcastle-Ottawa quality assessment scale (NOS) [6].

2.3. Data synthesis and analysis

Continuous variables were expressed as mean while categorical variables were presented as numbers and relative percentages. The cumulative incidence of incident AMI (n/N), defined as the ratio between patients experiencing the event during the follow-up period (n) and the number of patients enrolled in each study (N), were pooled using a random effects model and presented with the corresponding 95% confidence interval (CI). Conversely for the estimation of AMI risk within one year from the infection, the hazard ratio (HR) with the related 95% confidence interval (CI) was pooled using a random- effect. Predefined sensitivity analyses (leave-one-out analysis) were performed removing one study at the time, to evaluate the stability of our results. Statistical

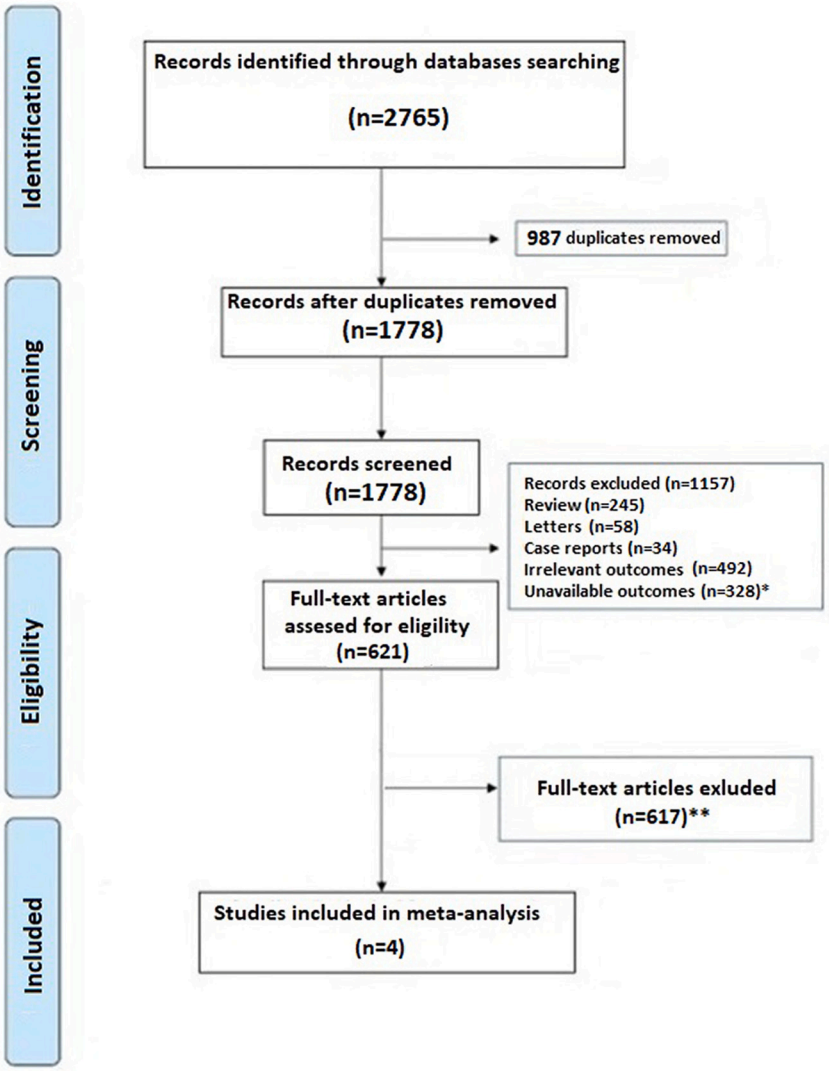

Fig. 1. PRISMA flowchart. * Articles excluded because not provided data on acute myocardial infarction events; ** Articles excluded because not provided Hazard ratio for acute myocardial infarction.

heterogeneity between groups was measured using the Higgins I^2 statistic. The presence of potential publication bias was verified by visual inspection of the funnel plot. Due to the low number of the included studies (<10), small-study bias was not examined as our analysis was underpowered to detect such bias. To further appraise the impact of potential baseline confounders, a meta-regression analysis was also performed. All meta-analyses were conducted using Comprehensive Meta-Analysis software, version 3 (Biostat, USA).

3. Results

3.1. Search results and included studies

A total of 2765 articles were obtained using our search strategy. After excluding duplicates and preliminary screening, 621 full-text articles were assessed for eligibility, 617 studies were excluded for not meeting the inclusion criteria, leaving 4 investigations fulfilling the inclusion criteria (Fig. 1) [7–10].

3.2. Characteristics of the population and quality assessment

Overall, 20,875,843 patients (mean age 56.1 years, 59.1% males were included in this analysis [7–10].). Among them 1,245,157 had confirmed COVID-19 infection. The general characteristics of the included studies are showed in Table 1. Although the demographic characteristics and the concomitant comorbidities were not systematically recorded in all the investigations, the cohorts mainly consisted of middle-aged patients. The mean length of follow-up was 8.5 months ranging between 4 and 12 months. The reviewed investigations identified the occurrence of AMI by screening the medical records of enrolled patients using the International Classification of Diseases 10th Revision (ICD-10) codes I21 and I22 [7–10]. Quality assessment showed that all studies were of moderate-high quality according to the NOS scale [6].

3.3. Pooled post-discharged incidence of acute myocardial infarction

The cumulative post-discharge rate of incident AMI in recovered COVID-19 patients ranged between 0.1 and 1.1% among the reviewed studies [7–10]. A random effect model revealed a pooled incidence of post COVID-19 AMI in 0.5% of cases (95% CI:0.3–0.0.8, I^2: 99.8%) (Fig. 2, Panel A). Sensitivity analysis showed a combined incidence rate, which remained statistically significant, across a range from 0.3% of cases (95% CI:0.1–0.5, I^2:99.8%) to 0.7% of cases (95% CI: 0.2 to 1.2; I^2:99.3%), suggesting that no single investigation had an undue impact on the study outcome. The visual inspection of the funnel plot is presented in Supplementary file 2, Panel A); however, the visual assessment of the funnel plot cannot reassure about the presence of an asymmetry due to the limited number of studies included into the analysis. Conversely, always a random-effect model showed a pooled incidence of AMI among contemporary controls in 0.2 of cases (95% CI: 0.18–0.23, I2: 99.7%) (Supplementary file 3, Panels A and B for the forest and the funnel plots, respectively). Also in this case, sensitivity analysis confirmed yielded results.

3.4. Long-term risk of acute myocardial infarction

After a mean follow-up of 8.5 months, recovered COVID-19 patients presented a higher risk of incident AMI (HR: 1.93, 95% CI: 1.65—2.26, $p < 0.0001$, $I^2 = 83.5\%$) (Fig. 2, Panel B). Also in this case, the visual assessment of the funnel plot cannot reassure about the presence of an asymmetry due to the limited number of studies included into the analysis accordingly to the inclusion criteria (Supplementary file 4) while the sensitivity analysis confirmed the yielded results reporting an HR ranging between 1.88 (95% CI: 1.60–2.21, $p < 0.0001$) and 2.00 (95% CI; 1.87–2.13, $p < 0.0001$), implying that the obtained results were not driven by any single study. A meta-regression analysis showed

Table 1
General characteristics of the population reviewed. AMI: Acuter myocardial infarction; HT: Arterial Hypertension; DM: Diabetes Mellitus; COPD: chronic obstructive pulmonary disease; CKD: Chronic Kidney disease; HF: Heart failure; FW: Follow-up; NR: Not reported. NOS: Newcastle-Ottawa quality assessment scale. *Defined as Chronic Pulmonary disease; **Only DM type 2; ***Data not reported for the unmatched cohort.

Authors	Sample size	COVID-19 patients	Age (years)	Males	Previous AMI	HT	DM	COPD	CKD	Obesity	HF	Cancer	Cerebrovascular disease	FW-length (months)	NOS
Cohen et al. [7]	2.895.943	133,366	75.7	1.227.545 (42.0)	110.805 (3.8)	2.081.772 (72.0)	938.043 (32.7)	578.650 (20)*	528.314 (14.0)	478.902 (17.0)	334,654 (12)	418.700 (14.4)	364.782 (13.0)	4	8
Wang et al. [8]	2.940.988	691,455	43.8	1.241.483 (42.2)	NR	440.998 (14.9)	188.488 (6.4)**	51,592 (1.7)	59,177 (2.0)	286,338 (9.7)**	NR	NR	NR	12	7
Xie et al. [9]	5.791.407	153,760	62.5	5.228.431 (90.2)	NR	1.525.944 (26.3)	1.321.907 (22.8)	633.000 (10.9)	970.057 (16.7)	2.462.44 (42.5)	NR	357.192 (6.1)	NR	12	8
Daugherty et al. [10]	9.247.505	266,586	42.4	4.640.393 (50.2)	1499 (0.6)	NR	521.699 (5.6)	NR	NR	NR	NR	NR	NR	6	6

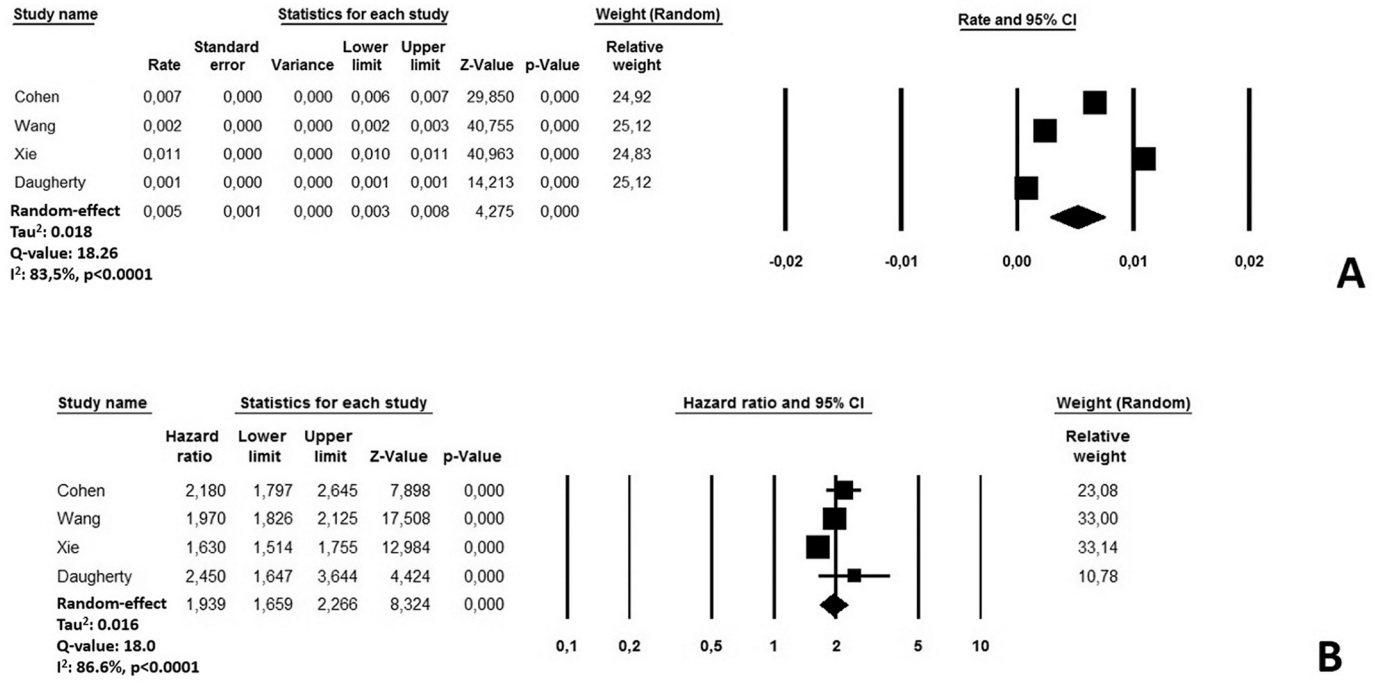

Fig. 2. **(A)** Forest plots investigating the pooled incidence of acute myocardial infarction after 8.5 months from COVID-19 infection. (B) Forest plots investigating the risk of acute myocardial infarction after 8.5 months from COVID-19 infection.

a significant direct relationship between the risk of incident AMI and age ($p = 0.01$) and male gender as moderators, while an indirect association was observed when the follow-up length ($p < 0.001$) was adopted as moderating variables (Table 2).

4. Discussion

Our findings, based on a large population of >20 million subjects, demonstrated that AMI occurred in about 0.5% of COVID-19 recovered patients over the follow-up period. Furthermore, after COVID-19 recovery, survivors had an approximately 93% excess in the risk of AMI, which was inversely related with the length of the follow-up. Notably, the incidence and the risk of AMI in COVID-19 recovered patients resulted higher compared to controls over a mean follow-up of 8.5 months. Conversely, the risk of AMI after hospital discharge was lower compared to that observed after other inflammatory conditions of the respiratory tract, such as pneumonia [11,12]. However, we cannot exclude that missed AMI events may have contributed to the high risk observed in our analysis, being the AMI cases identified by the revision of ICD codes in the population analyzed. Furthermore, this uncertainty is reflected by the observed high statistical heterogeneity. Meta-regression analysis contributed to define this high heterogeneity showing that the risk of AMI events directly increased with aging, as previously reported in subjects from the general population, as well as in men. However, it is also true that the comparison of rates between gender strictly depends by the age range of the population under study. Furthermore, when the death rate is high from causes other than the disease of interest, the incidence rates of the illness are generally overestimated in traditional Kaplan–Meier survival analysis due to existence of competing risks. Moreover, our results evidenced that the AMI risk was is not just limited to the acute phase of COVID-19 but also during the early phase after recovery. Unfortunately, the revised studies did not systematically report data regarding potential risk factors for AMI, as well as their clinical presentation and type (i.e., with ST-segment elevation or not). Moreover, no data were provided regarding the AMI-related mortality rate either in recovered COVID-19 subjects as well as for controls. The observed AMI incidence in COVID-19 recovered patients was about 3.5 AMI cases per 1000 people. However, making a real comparison with the current epidemiological records derived from the general population is difficult because those data are generally based on 1000 persons per year while our results cannot be referred to the same period. However, a previous investigation reporting the incidence of recognized and unrecognized AMI in subjects aged >55 years, demonstrated that during a mean follow-up of 6.4 years, the incidence rate of this event was 5.0 per 1000 person years. This result appears higher compared to that observed in our cohort of COVID-19 recovered patients which perhaps had similar demographical features [13]. Nevertheless, it should be noted that the study by Torbal et al. was based on a longer follow-up period and performed into the 1990s, when several cardioprotective agents were different and differently influenced the cardiovascular protection [13]. Probably, we have underestimated the incidence of AMI both in COVID-19 recovered patients as well as in the control groups. Indeed, previous investigations have widely reported a lower incidence of acute coronary syndromes during the COVID-19 pandemic; moreover, we cannot exclude that some AMIs have been underdiagnosed or misinterpreted as cardiovascular injuries related to the acute infection [14–17].

Before the COVID-19 pandemic, different analyses pointed out the risk of AMI after an infection [18]. To this regard, it has been described that the risk of AMI returns to baseline within few months after resolution of the infection and the risk of acute events seems to be more pronounced among patients with a more severe disease [14–16]. Unfortunately, data on the severity of the SARS-CoV-2 infection were not

Table 2
Meta regression analysis for the risk of acute myocardial infarction after COVID-19 infection. CI: Confidence interval.

Items	N° of interactions	Coeff.	95% CI	p
Age (years)	4	0.008	0.06 to 0.013	0.01
Males (%)	4	0.004	0.001 to 0.007	0.001
Diabetes melliuts (%)	4	−0.002	−0.020 to 0.015	0.79
FW lenght (months)	4	−0.029	−0.071 to −0.012	<0.001

systematically collected by the reviewed studies, making impossible a specific sub-analysis. From a pathophysiological point of view, it has been reported that COVID-19 patients may exhibit prolonged SARS-CoV-2 Ribonucleic Acid (RNA) shedding for up to 83 days in the upper respiratory tract, associated with high viral RNA loads [19,20]. Furthermore, in infected as well as in recovered patients, a persistent hypercoagulable state may further increase the risk of coronary thrombosis at the sites of plaque disruption [21,22]. Doubtless, other concomitant factors may contribute to coronary thrombosis, such as the production of neutrophil extracellular traps (NETs) from intraplaque and circulating neutrophils, increased platelet activity, impaired fibrinolysis and overall decreased anticoagulant function of the endothelium [23–27]. Although further dedicated studies are needed to exactly determine the incidence of AMI after COVID-19 infection, our data may be useful for minimizing the risk of AMI after in COVID-19 survivors, although our results must be considered preliminary and cannot be directly translated into clinical practice as recommendations regarding the type and regimen of antiplatelet therapies or thrombophylactic strategies.

4.1. Limitations

Our study has several limitations related to the observational nature of the reviewed studies with all their inherited biases. Potential underestimation of the AMI incidence could derive from the absence of a specific and dedicated follow-up; indeed, the larger part of the articles reviewed identified the occurrence of incident AMIs from larger medical records dataset using the relative ICD-10 codes and it is known that this methodological approach has modest sensitivity to diagnose AMI in the general population [28]. Furthermore, few studies have analyzed the risk of AMI in patients recovered from COVID-19 infection, limiting the number of the observations included in the meta-analysis; however, the number of patients enrolled mitigates, at least partially, this limit. We cannot exclude that sampling bias by the competing risk of death may also have led to the underestimation of the real cumulative incidence of AMIs. Similarly, no data regarding the type and number of vaccinations against SARS-CoV-2 as well as the patients experiencing AMI during the acute phase of the infection were systematically reported, making impossible any type of sub-analysis. At the same manner, the absence of information regarding the different COVID-19 variants has further limited our conclusions. Finally, the reviewed data may have underestimated the real impact of AMI after COVID-19 especially during the early phase of the pandemic, both due to the presence of undiagnosed cases and patients lost during the follow-up period. Despite these limitations, to the best of our knowledge, this study is the first systematic review and meta-analysis providing a clear estimation on the incidence and the risk of AMI in patients who recovered from COVID-19.

5. Conclusions

AMI represents a relatively rare but potential post-acute COVID-19 sequelae in the long-term period that might benefit from aggressive prevention strategies and appropriate follow-up of COVID-19 patients.

Data availability statement

The data that support the findings of this study are available from the reviewed studies.

Author statement

Marco Zuin: Conceptualization, Data Curation, Methodology, Writing- Original draft, Formal analysis; Statistical analysis. Gianluca Rigatelli: Investigation, Data curation, Writing- Original draft preparation. Valentina Battisti: Investigation, data collection. Giulia Costola: Investigation, data collection. Loris Roncon: Reviewing and Editing. Claudio Bilato: Validation, Supervision, Reviewing and Editing.

Declaration of Competing Interest

None of the authors have conflicts of interest to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2022.12.032.

References

[1] V.F. Corrales-Medina, M. Madjid, D.M. Musher, Role of acute infection in triggering acute coronary syndromes, Lancet Infect. Dis. 10 (2010) 83–92.
[2] D. Modin, B. Claggett, C. Sindet-Pedersen, M.C.H. Lassen, K.G. Skaarup, J.U. S. Jensen, M. Fralick, M. Schou, M. Lamberts, T. Gerds, E.L. Fosbøl, M. Phelps, K. H. Kragholm, M.P. Andersen, L. Køber, C. Torp-Pedersen, S.D. Solomon, G. Gislason, T. Biering-Sørensen, Acute COVID-19 and the incidence of ischemic stroke and acute myocardial infarction, Circulation. 142 (2020) 2080–2082.
[3] F. Kwenandar, K.V. Japar, V. Damay, T.I. Hariyanto, M. Tanaka, N.P.H. Lugito, A. Kurniawan, Coronavirus disease 2019 and cardiovascular system: a narrative review, Int. J. Cardiol. Heart Vasc. 29 (2020), 100557.
[4] T. Guo, Y. Fan, M. Chen, X. Wu, L. Zhang, T. He, H. Wang, J. Wan, X. Wang, Z. Lu, Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19), JAMA Cardiol. 5 (2020) 811–818.
[5] D. Moher, A. Liberati, J. Tetzlaff, D.G. Altman, PRISMA Group, Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement, PLoS Med. 6 (2009), e1000097.
[6] G.A. Wells, B. Shea, D. O'Connell, J. Peterson, V. Welch, M. Losos, P. Tugwell, The Newcastle-Ottawa Scale (NOS) for Assessing the Quality if Nonrandomized Studies in meta-Analyses. http://wwwohrica/programs/clinical_epidemiology/oxfordasp, 2012.
[7] K. Cohen, S. Ren, K. Heath, M.C. Dasmariñas, K.G. Jubilo, Y. Guo, M. Lipsitch, S. E. Daugherty, Risk of persistent and new clinical sequelae among adults aged 65 years and older during the post-acute phase of SARS-CoV-2 infection: retrospective cohort study, BMJ. 376 (2022), e068414.
[8] W. Wang, C.Y. Wang, S.I. Wang, J.C. Wei, Long-term cardiovascular outcomes in COVID-19 survivors among non-vaccinated population: a retrospective cohort study from the TriNetX US collaborative networks, EClinicalMedicine. 53 (2022), 101619.
[9] Y. Xie, E. Xu, B. Bowe, Z. Al-Aly, Long-term cardiovascular outcomes of COVID-19, Nat. Med. 28 (2022) 583–590.
[10] S.E. Daugherty, Y. Guo, K. Heath, M.C. Dasmariñas, K.G. Jubilo, J. Samranvedhya, M. Lipsitch, K. Cohen, Risk of clinical sequelae after the acute phase of SARS-CoV-2 infection: retrospective cohort study, BMJ. 373 (2021), n1098.
[11] J.A. Davidson, A. Banerjee, L. Smeeth, H.I. McDonald, D. Grint, E. Herrett, H. Forbes, R. Pebody, C. Warren-Gash, Risk of acute respiratory infection and acute cardiovascular events following acute respiratory infection among adults with increased cardiovascular risk in England between 2008 and 2018: a retrospective, population-based cohort study, Lancet Digit. Health. 3 (2021) e773–e783.
[12] V.F. Corrales-Medina, K.N. Alvarez, L.A. Weissfeld, D.C. Angus, J.A. Chirinos, C. C. Chang, A. Newman, L. Loehr, A.R. Folsom, M.S. Elkind, M.F. Lyles, R. A. Kronmal, S. Yende, Association between hospitalization for pneumonia and subsequent risk of cardiovascular disease, JAMA. 313 (2015) 264–274.
[13] A. de Torbal, E. Boersma, J.A. Kors, G. van Herpen, J.W. Deckers, D.A. van der Kuip, B.H. Stricker, A. Hofman, J.C. Witteman, Incidence of recognized and unrecognized myocardial infarction in men and women aged 55 and older: the Rotterdam Study, Eur. Heart J. 27 (2006) 729–736.
[14] N. Sutherland, N.H. Dayawansa, B. Filipopoulos, S. Vasanthakumar, O. Narayan, F. A. Ponnuthurai, W. van Gaal, Acute coronary syndrome in the COVID-19 pandemic: reduced cases and increased ischaemic time, Heart Lung Circ. 31 (2022) 69–76.
[15] M. Zuin, G. Mugnai, A. Zamboni, E. Zakja, R. Valle, G. Turiano, S. Themistoclakis, D. Scarpa, S. Saccà, L. Roncon, F. Rizzetto, P. Purita, A. Polo, I. Pantano, A. Mugnolo, G. Molon, S. Meneghin, D. Mancuso, M. Lia, G. Grassi, A. Cutolo, F. Chirillo, P. Bozzini, S. Bonapace, M. Anselmi, G. Rigatelli, C. Bilato, Decline of admission for acute coronary syndromes and acute cardiovascular conditions during COVID-19 pandemic in Veneto region, Viruses. 14 (2022) 1925.
[16] S. De Rosa, C. Spaccarotella, C. Basso, M.P. Calabrò, A. Curcio, P.P. Filardi, M. Mancone, G. Mercuro, S. Muscoli, S. Nodari, R. Pedrinelli, G. Sinagra, C. Indolfi, Società Italiana di Cardiologia and the CCU academy investigators group. Reduction of hospitalizations for myocardial infarction in Italy in the COVID-19 era, Eur. Heart J. 41 (2020) 2083–2088.
[17] M. Zuin, G. Rigatelli, G. Zuliani, C. Bilato, P. Zonzin, L. Roncon, Incidence and mortality risk in coronavirus disease 2019 patients complicated by acute cardiac injury: systematic review and meta-analysis, J. Cardiovasc. Med. (Hagerstown) 21 (2020) 759–764.
[18] V.F. Corrales-Medina, M. Madjid, D.M. Musher, Role of acute infection in triggering acute coronary syndromes, Lancet Infect. Dis. 10 (2010) 83–92.

[19] M. Dalager-Pedersen, M. Søgaard, H.C. Schønheyder, H. Nielsen, R.W. Thomsen, Risk for myocardial infarction and stroke after community-acquired bacteremia: a 20-year population-based cohort study, Circulation 129 (2014) 1387–1396.
[20] C. Bergh, K. Fall, R. Udumyan, H. Sjöqvist, O. Fröbert, S. Montgomery, Severe infections and subsequent delayed cardiovascular disease, Eur. J. Prev. Cardiol. 24 (2017) 1958–1966.
[21] D.M. Musher, M.S. Abers, V.F. Corrales-Medina, Acute infection and myocardial infarction, N. Engl. J. Med. 380 (2019) 171–176.
[22] M. Cevik, M. Tate, O. Lloyd, A.E. Maraolo, J. Schafers, A. Ho, SARS-CoV-2, SARS-CoV, and MERS-CoV viral load dynamics, duration of viral shedding, and infectiousness: a systematic review and meta-analysis, Lancet Microbe. 2 (2021) e13–e22.
[23] B. Zhou, J. She, Y. Wang, X. Ma, Duration of viral shedding of discharged patients with severe COVID-19, Clin. Infect. Dis. 71 (2020) 2240–2242.
[24] T. Alnima, M.M.G. Mulder, B.C.T. van Bussel, H. Ten Cate, COVID-19 coagulopathy: from pathogenesis to treatment, Acta Haematol. 145 (2022) 282–296.
[25] F.A. von Meijenfeldt, S. Havervall, J. Adelmeijer, A. Lundström, M. Magnusson, N. Mackman, C. Thalin, T. Lisman, Sustained prothrombotic changes in COVID-19 patients 4 months after hospital discharge, Blood Adv. 5 (2021) 756–759.
[26] L. Townsend, H. Fogarty, A. Dyer, et al., Prolonged elevation of D-dimer levels in convalescent COVID-19 patients is independent of the acute phase response, J. Thromb. Haemost. 19 (2021) 1064–1070.
[27] F.A. von Meijenfeldt, C. Thålin, T. Lisman, Sustained prothrombotic changes in convalescent patients with COVID-19, Lancet Haematol. 8 (2021), e475.
[28] L. Nedkoff, D. Lopez, J. Hung, M. Knuiman, T.G. Briffa, K. Murray, E. Davis, S. Aria, K. Robinson, J. Beilby, M.S.T. Hobbs, F.M. Sanfilippo, Validation of ICD-10-AM coding for myocardial infarction subtype in hospitalisation data, Heart Lung Circ. 31 (2022) 849–858.