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Research Paper

Admission phenotype and 180-day mortality in older adults hospitalized with SARS-CoV-2 infection

Jakub Kisiel[1], Michał Chojnicki[2], Arleta Kowala-Piaskowska[3], Katarzyna Wieczorowska-Tobis[4], Sławomir Tobis[5], Agnieszka Monika Neumann-Podczaska[4,6]

[1] *Multispecialist Municipal Hospital, J. Struś Memorial Hospital, Poznań, Poland*
[2] *Department of Immunobiology, Poznan University of Medical Sciences, Poznań, Poland*
[3] *Department and Clinic of Infectious Diseases, Hepatology and Acquired Immunodeficiencies, Poznan University of Medical Sciences, Poznań, Poland*
[4] *Department of Palliative Medicine, Poznan University of Medical Sciences, Poznań, Poland*
[5] *Department of Occupational Therapy, Poznan University of Medical Sciences, Poznań, Poland*
[6] *Senior Institute, Department of Medical and Health Sciences, Vizja University, Warsaw, Poland*

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Abstract

Introduction: In older inpatients, SARS-CoV-2 positivity does not always indicate that COVID-19 is the cause of admission.

Aim: To compare clinical profiles and 180-day mortality between older adults admitted for typical COVID-19 and those with incidental SARS-CoV-2 infection.

Material and methods: This retrospective cohort study included 306 hospitalized patients aged ≥60 years with SARS-CoV-2 infection. Admission phenotype was manually adjudicated as typical COVID-19 respiratory disease or incidental SARS-CoV-2 infection. Baseline characteristics, multimorbidity, CRP, IL-6, D-dimer, SpO2, and 180-day mortality were analyzed. Mortality was assessed using Kaplan–Meier curves and multivariable logistic regression.

Results: The cohort included 266 typical and 40 incidental admissions. Multimorbidity was common in both groups (79.2% vs 89.7%; $p = 0.122$). Typical admissions had lower SpO2 and higher CRP at admission (75.2 vs 34.8 mg/L; $p = 0.007$), whereas incidental admissions had higher D-dimer concentrations (2.2 vs 1.1 ng/mL; $p = 0.016$). Crude 180-day mortality was higher in the typical COVID-19 group (42.9% vs 22.5%; $p = 0.014$). After adjustment for age and multimorbidity, incidental phenotype was independently associated with lower mortality odds (OR = 0.40; 95% CI: 0.18–0.89; $p = 0.027$).

Conclusions: Typical COVID-19 and incidental SARS-CoV-2 infection are distinct geriatric admission phenotypes. Typical COVID-19 admissions are associated with higher 180-day mortality, independent of baseline multimorbidity.

Corresponding author: Michał Chojnicki; Department of Immunobiology, Poznan University of Medical Sciences, Rokietnicka 8, 60-806, Poznań, Poland.
E-mail address: michalc@ump.edu.pl

1. INTRODUCTION

During the COVID-19 pandemic, hospital triage and reporting often relied on SARS-CoV-2 test positivity rather than on the acute condition that prompted admission. As a result, many hospital cohorts combined patients admitted because of COVID-19 respiratory disease with patients admitted for other acute conditions in whom SARS-CoV-2 infection was detected incidentally. These groups may share virological status, but they do not necessarily represent the same clinical entity. Failure to distinguish between admission for COVID-19 and admission with SARS-CoV-2 can therefore bias estimates of inflammatory burden, treatment requirements, resource use, and mortality, particularly when classification is based only on administrative data or discharge coding.[1–6]

This distinction is especially relevant in older adults. In this population, acute illness often presents non-specifically, while frailty, multimorbidity, and reduced functional reserve make attribution more difficult. Delirium, falls, weakness, anorexia, or functional decline may occur in COVID-19, but may also reflect competing acute illness or baseline vulnerability. Likewise, inflammatory and prothrombotic biomarkers may capture not only SARS-CoV-2 –related disease severity but also other acute or chronic processes. In older inpatients, test positivity alone is therefore an unreliable proxy for the principal reason for hospitalization, and clinically meaningful differences between respiratory COVID-19 and incidental infection may carry important prognostic implications.[7–10]

Despite this, few studies in hospitalized older adults have used patient-level clinical adjudication to determine whether SARS-CoV-2 infection was the primary reason for admission. Yet this distinction is precisely what makes comparison between these groups meaningful: if they differ in presentation, inflammatory response, and outcomes, then analyzing them together obscures clinically relevant heterogeneity; if they do not, that assumption should be demonstrated rather than presumed. Recent work suggests that outcome estimates may change substantially when primary COVID-19 is separated from incidental SARS-CoV-2 infection.[1,3,6] In addition, most previous studies have focused on short-term outcomes, whereas in geriatric patients, a longer-term prognosis may better reflect the interaction among the index illness, frailty, and competing comorbidities.

We therefore compared older adults admitted for typical COVID-19 respiratory disease with those admitted for non-respiratory acute conditions and found to have incidental SARS-CoV-2 infection. Using manual physician-led adjudication of the admission indication, we examined whether these groups differed in inflammatory and prothrombotic trajectories and whether the admission phenotype was associated with a 180-day mortality.

2. AIM

To compare clinical and biomarker profiles and the 180-day mortality between older adults admitted for COVID-19 respiratory disease and those with incidental SARS-CoV-2 infection, accounting for multimorbidity.

3. MATERIAL AND METHODS

3.1. Study design and population

This was a retrospective, single-center cohort study conducted in a temporary COVID-19 referral hospital in Poland. During the study period, the hospital operated as a dedicated center for patients with confirmed SARS-CoV-2 infection who required oxygen therapy or more advanced inpatient care and received both direct emergency admissions and transfers from other hospitals. Consecutive patients aged 60 years or older with laboratory-confirmed SARS-CoV-2 infection admitted between April and December 2021 were eligible for inclusion. Data were obtained from electronic medical records. Patients were excluded if the admission records did not allow a reliable determination of the primary reason for hospitalization or if long-term vital status could not be established.

3.2. Data sources and variables

Clinical data were collected retrospectively by a trained physician team using a predefined case report form comprising 93 clinical, laboratory, imaging, and treatment variables per patient. The dataset included geriatric measures, such as baseline functional status assessed with Performance Status and the presence of cognitive disturbance at admission, comorbidities including multimorbidity, presenting symptoms, laboratory findings at admission and during hospitalization, and major treatment modalities, including oxygen therapy, ventilatory support, corticosteroids, antivirals, and immunomodulatory treatment. This level of detail allowed us to distinguish baseline vulnerability from acute disease-related abnormalities and to examine changes during hospitalization rather than relying solely on admission data. Multimorbidity was defined a priori as the presence of at least two chronic conditions unrelated to COVID-19 and was analyzed as a binary variable.

3.3. Phenotype classification

Because SARS-CoV-2 positivity alone does not establish the reason for hospitalization in older adults, admission phenotype was determined by expert-led manual adjudication rather than ICD-10 coding, discharge diagnoses, or automated database rules. The objective was to identify the principal acute process responsible for hospital admission and to minimize misclassification typical of administrative datasets.

Phenotype assignment followed a 2-step review process, with initial assessment by a general practitioner and independent verification by an infectious disease specialist. Uncertain cases were resolved by interdisciplinary diagnostic consensus.

Two a priori clinical phenotypes were defined. Typical COVID-19 referred to admission primarily for acute COVID-19 respiratory disease, defined by hypoxaemia and/or radiologically confirmed pneumonia in the presence of compatible

respiratory symptoms. Incidental SARS-CoV-2 infection referred to admission primarily for another acute medical or surgical condition, with SARS-CoV-2 infection detected on routine testing but not considered the main cause of hospitalization. Thus, the comparison was between 2 clinically distinct real-world phenotypes: admission due to COVID-19 respiratory disease and admission for a non-COVID acute condition with coincidental SARS-CoV-2 infection.

3.4. Data collection and outcomes

Baseline variables included age, sex, and multimorbidity, defined as the presence of at least two chronic conditions unrelated to COVID-19. Variables reflecting geriatric vulnerability, including functional and cognitive status, were extracted from routine admission documentation. To assess inflammatory and prothrombotic burden, C-reactive protein (CRP), interleukin-6 (IL-6), and D-dimer levels were recorded both at admission and at the highest value observed during hospitalization. Oxygen saturation (SpO2) was recorded at admission and at its lowest level during the hospital stay. The primary outcome was all-cause mortality within 180 days of admission. Long-term vital status was determined by registry-based follow-up available for the parent cohort.

3.5. Statistical analysis

Continuous variables are presented as medians with interquartile ranges (IQR) and were compared using the Mann–Whitney U test. Categorical variables are presented as counts and percentages and were compared using the chi-square test or Fisher's exact test, as appropriate. Survival up to 180 days was assessed using Kaplan–Meier estimates, with between-group differences evaluated by the log-rank test. The association between admission phenotype and 180-day mortality was examined in a multivariable logistic regression model including phenotype as the main exposure and age and multimorbidity as prespecified confounders. Variables reflecting acute disease severity during hospitalization were not included because they were considered part of the phenotype rather than baseline confounders. The regression model was kept simple, including only age and multimorbidity as baseline confounders. Respiratory variables, inflammatory markers, and in-hospital factors were not included because they were closely related to the admission phenotype itself and to the severity of the acute illness, rather than representing independent baseline confounders. The small size of the incidental SARS-CoV-2 group was another reason to avoid a more extensive model. Results are reported as odds ratios (OR) with 95% confidence intervals (CI). All tests were two-sided, and p-values below 0.05 were considered statistically significant.

4. RESULTS

The study included 306 patients, of whom 266 were classified as having typical COVID-19 and 40 as having incidental SARS-CoV-2 infection. Baseline characteristics are shown in Table 1. Both groups had a high prevalence of multimorbidity, affecting 79.2% of patients with typical COVID-19 and 89.7% with incidental SARS-CoV-2 infection ($p = 0.122$). The distribution of most chronic conditions was similar across groups. In contrast, a history of stroke/TIA (33.3% vs 17.7%; $p = 0.022$) and chronic liver disease (20.5% vs 4.5%; $p = 0.001$) was more frequent in patients with incidental infection.

Table 1. Baseline characteristics according to admission phenotype.

Variable	Total (n = 306)	Typical COVID-19 (n = 266)	Incidental SARS-CoV-2 (n = 40)	P-value
Demographics				
Age, years	74 [67–81] (n = 305)	74 [67–82] (n = 266)	74 [67–78.5] (n = 39)	0.591
Male sex	143/305 (46.9%)	126/266 (47.4%)	17/39 (43.6%)	0.659
Baseline geriatric profile				
Severe functional limitation (Performance Status 3)	58/303 (19.1%)	50/264 (18.9%)	8/39 (20.5%)	0.816
Cognitive disturbance at admission	95/305 (31.1%)	85/266 (32.0%)	10/39 (25.6%)	0.427
Multimorbidity and comorbidities				
Multimorbidity (≥2 chronic conditions)	245/304 (80.6%)	210/265 (79.2%)	35/39 (89.7%)	0.122
Hypertension	203/304 (66.8%)	176/265 (66.4%)	27/39 (69.2%)	0.727
Diabetes	99/304 (32.6%)	84/265 (31.7%)	15/39 (38.5%)	0.400
Cardiovascular disease (non-hypertension)	186/304 (61.2%)	160/265 (60.4%)	26/39 (66.7%)	0.452
Kidney disease	41/303 (13.5%)	32/264 (12.1%)	9/39 (23.1%)	0.062
Liver disease	20/304 (6.6%)	12/265 (4.5%)	8/39 (20.5%)	0.001
Malignancy (active or history)	59/304 (19.4%)	49/265 (18.5%)	10/39 (25.6%)	0.292
Previous stroke/TIA/cerebrovascular disease	60/304 (19.7%)	47/265 (17.7%)	13/39 (33.3%)	0.022
Pulmonary disease	36/304 (11.8%)	32/265 (12.1%)	4/39 (10.3%)	1.000

Data are presented as median [IQR] (n) or n/N (%). P-values were calculated using the Mann–Whitney U test for age and the chi-square test or Fisher's exact test, as appropriate, for categorical variables. Cognitive disturbance was defined as any documented abnormal cognitive status at admission (score ≥1 on the admission scale). Multimorbidity was defined as ≥2 chronic conditions unrelated to COVID-19. Malignancy includes active cancer or a history of cancer.

Patients admitted for typical COVID-19 had lower oxygen saturation at admission than those with incidental SARS-CoV-2 infection and were more likely to present with dyspnoea and radiological pneumonia. CRP concentrations at admission were higher in the typical COVID-19 group, while D-dimer concentrations were higher in the incidental group. IL-6 concentrations at admission were similar between groups (Table 2).

Data are shown as median (IQR) or n/N (%). Continuous variables were compared with the Mann–Whitney U test and categorical variables with the chi-square or Fisher exact test, as appropriate.

Differences observed at admission were also reflected in the subsequent course of hospitalization. Table 3 summarizes the use of respiratory support and COVID-19-directed treatment, as well as the highest inflammatory marker values and the lowest recorded oxygen saturation during the hospital stay.

Table 2. Admission clinical and laboratory findings according to admission phenotype.

Variable	Total (n = 306)	Typical COVID-19 (n = 266)	Incidental SARS-CoV-2 (n = 40)	P-value
Clinical findings at admission				
SpO2 at admission, median (IQR)	94.0 (90.0–96.0)	93.0 (90.0–96.0)	95.0 (93.0–97.0)	0.005
Dyspnoea, n (%)	180/305 (59.0)	171/266 (64.3)	9/39 (23.1)	<0.001
Radiologically confirmed pneumonia, n (%)	235/299 (78.6)	213/260 (81.9)	22/39 (56.4)	<0.001
Laboratory findings at admission				
CRP, mg/L, median (IQR)	67.0 (28.9–127.6)	75.2 (33.3–135.1)	34.8 (16.4–93.4)	0.007
IL-6, pg/mL, median (IQR)	43.2 (17.1–90.8)	43.6 (17.3–95.3)	26.9 (13.4–62.4)	0.325
D-dimer, ng/mL, median (IQR)	1.2 (0.7–2.2)	1.1 (0.7–2.0)	2.2 (1.0–5.5)	0.016

Table 3. In-hospital course, treatment, and laboratory extremes according to admission phenotype.

Variable	Total (n = 306)	Typical COVID-19 (n = 266)	Incidental SARS-CoV-2 (n = 40)	P-value
Respiratory course				
Lowest SpO2 during hospitalization, median (IQR)	87.0 (80.8–91.0)	86.0 (80.0–91.0)	91.0 (87.0–93.0)	<0.001
Any oxygen therapy during hospital stay, n (%)	248/305 (81.3)	224/266 (84.2)	24/39 (61.5)	<0.001
Invasive mechanical ventilation, n (%)	52/305 (17.0)	46/266 (17.3)	6/39 (15.4)	0.767
Inflammatory / thrombotic peaks				
Peak CRP, mg/L, median (IQR)	110.5 (52.5–185.8)	113.1 (52.1–188.3)	95.3 (56.5–167.2)	0.830
Peak IL-6, pg/mL, median (IQR)	58.5 (23.6–157.6)	58.0 (25.5–157.0)	59.3 (18.0–172.2)	0.946
Peak D-dimer, ng/mL, median (IQR)	1.9 (1.1–6.2)	1.8 (1.0–6.2)	2.3 (1.2–6.2)	0.308
Treatment during hospitalisation				
Corticosteroids, n (%)	228/305 (74.8)	213/266 (80.1)	15/39 (38.5)	<0.001
Antivirals (remdesivir), n (%)	153/305 (50.2)	142/266 (53.4)	11/39 (28.2)	0.003
Immunomodulators (tocilizumab or baricitinib), n (%)	51/306 (16.7)	51/266 (19.2)	0/40 (0.0)	<0.001
Hospital course				
Length of stay, days, median (IQR)	12.0 (9.0–16.0)	12.0 (9.0–15.8)	16.0 (11.5–25.5)	<0.001

Table 4A. 180-day all-cause mortality by admission phenotype.

Outcome	Typical COVID-19 (n = 266)	Incidental SARS-CoV-2 (n = 40)	P-value
Deaths within 180 days, n (%)	114/266 (42.9)	9/40 (22.5)	0.014

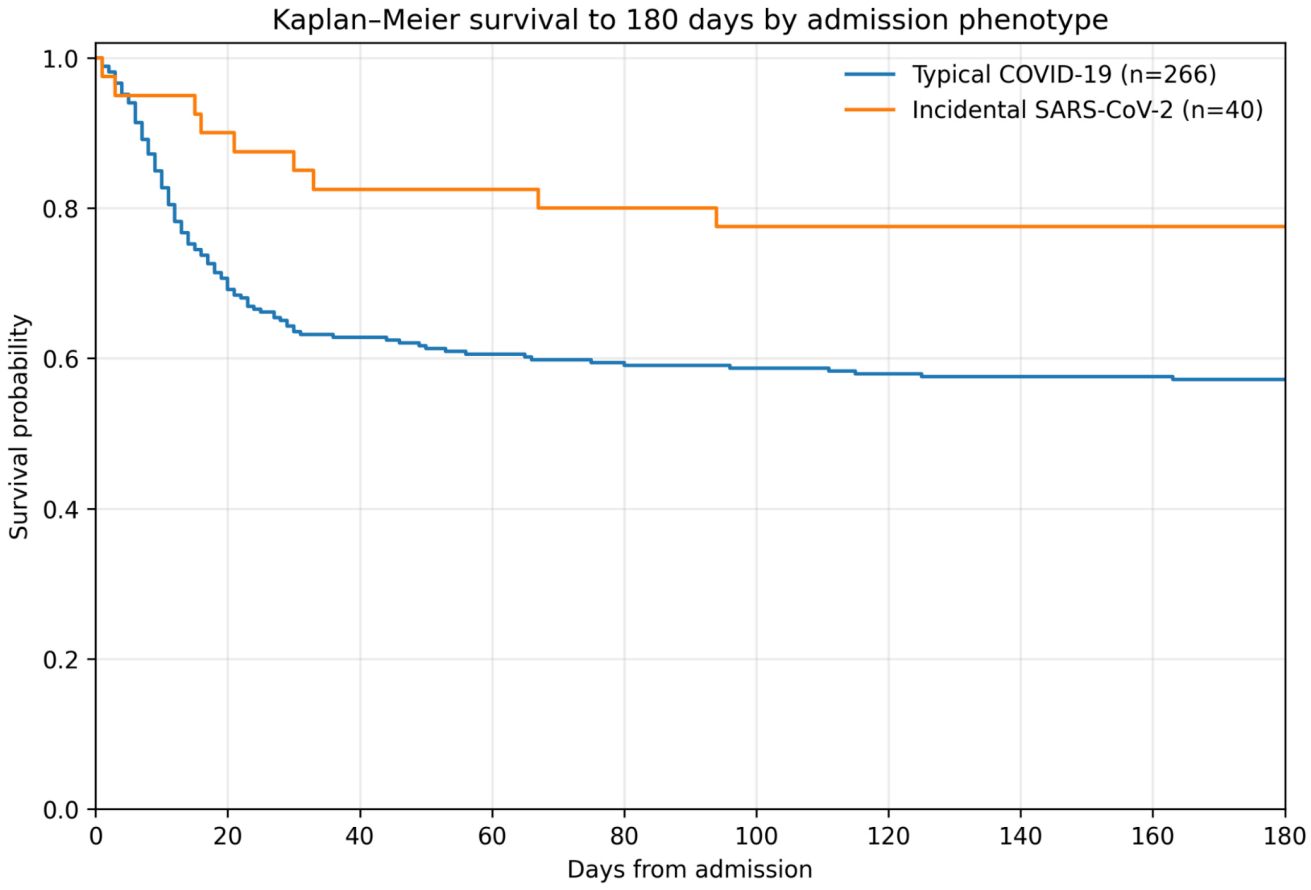

Figure 1. Kaplan–Meier curves for 180-day all-cause mortality according to admission phenotype in patients aged 60 years or older with SARS-CoV-2 infection.

Table 4B. Multivariable logistic regression for 180-day all-cause mortality.

Variable	OR	95% CI	P-value
Incidental SARS-CoV-2 phenotype	0.40	0.18–0.89	0.027
Age, per year	1.06	1.03–1.09	<0.001
Multimorbidity	1.80	0.93–3.48	0.079

Immunomodulators were defined as tocilizumab and/or baricitinib. Variables unavailable in the source dataset were not included.

P-value calculated using the chi-square test or Fisher's exact test, as appropriate.

In multivariable logistic regression adjusted for age and multimorbidity, incidental SARS-CoV-2 infection was associated with lower odds of 180-day mortality than typical COVID-19 (Table 4B).

5. DISCUSSION

The admission phenotype was associated with long-term risk in this cohort of older adults with SARS-CoV-2 infection. Patients admitted for typical COVID-19 respiratory disease had higher 180-day mortality than those with incidental SARS-CoV-2 infection, despite a less adverse chronic disease profile. This suggests that the prognosis of older SARS-CoV-2-positive inpatients may not be explained solely by baseline

comorbidity and that the acute admission phenotype may carry additional prognostic information. This interpretation is consistent with previous studies showing that SARS-CoV-2-positive hospitalizations are clinically heterogeneous and that separating admission for typical COVID-19 respiratory disease from incidental SARS-CoV-2 infection is methodologically important.[1,3,4]

The clinical and laboratory findings were in keeping with this interpretation. Typical COVID-19 was associated with lower oxygen saturation and higher CRP concentrations, consistent with acute hypoxaemic respiratory disease and a stronger inflammatory response. In contrast, incidental SARS-CoV-2 infection was associated with higher D-dimer levels, which may reflect the acute non-respiratory conditions leading to admission rather than the viral infection itself. Taken together, these findings support the view that SARS-CoV-2-positive admissions should not automatically be treated as a uniform clinical group. Such heterogeneity is also consistent with seroepidemiological observations from Poland indicating that many SARS-CoV-2 infections may have remained previously unrecognized.[11]

The observed difference persisted after adjustment for age and multimorbidity. This suggests that the classification was not fully explained by baseline vulnerability alone. At the same time, this finding should be interpreted with caution, as frailty is known to be associated with adverse COVID-19 outcomes in older adults and was not fully captured in our analysis.[7] The regression model was kept simple and included only age and multimorbidity as baseline confounders. Respiratory variables, inflammatory markers, and in-hospital factors were not included because they were closely related to the admission phenotype itself and to the severity of the acute illness, rather than representing independent baseline confounders. The small size of the incidental SARS-CoV-2 group further limited the extent of adjustment. Residual confounding, including frailty, treatment variation, and vaccination status, cannot be excluded.

These findings are also relevant to the interpretation of hospital-based COVID-19 cohorts. Analyses that combine all SARS-CoV-2-positive admissions into a single category may misattribute outcomes, especially in settings where infection status influenced placement of care but not necessarily the main reason for hospitalization. Mixing patients admitted because of COVID-19 with those admitted for other acute conditions may affect mortality estimates, biological interpretation, and comparability across studies. This concern has also been raised in previous EHR-based and adjudicated studies.[1,3,6]

The same distinction has practical implications for the organization of care, because patients with incidental infection often require management determined primarily by the admitting pathology rather than by the infection itself.

This study has several limitations. It was retrospective, single-center, and included a relatively small incidental group, which limited the precision of comparisons and the extent of multivariable adjustment. Differences in testing intensity during hospitalization may have affected peak laboratory values, particularly D-dimer and IL-6. Residual confounding cannot be excluded, including that related to frailty, treatment variation, and vaccination status. Multimorbidity was captured as a binary variable rather than with a weighted comorbidity index. The main strengths of the study were patient-level adjudication of the reason for admission, an independent 2-physician review with consensus resolution, and ascertainment of the 180-day mortality in an older inpatient cohort.

6. CONCLUSION

Typical COVID-19 respiratory disease and incidental SARS-CoV-2 infection appear to represent distinct clinical phenotypes in older inpatients and may not be best analyzed as a single SARS-CoV-2-positive cohort. In this study, admission for typical COVID-19 was associated with higher 180-day mortality than incidental SARS-CoV-2 infection, even after adjustment for age and multimorbidity. These findings suggest that pathogen positivity alone may be an insufficient proxy for clinical triage or outcome attribution in older adults. They also support the value of careful clinical adjudication of the primary reason for admission in epidemiological studies and in the organization of care.

Informed consent

Informed consent was not required due to the retrospective nature of the study, which was based on routinely collected medical record data and did not involve any additional patient intervention.

Ethics approval

All procedures performed in this study involving human participants were conducted in accordance with institutional ethics committee standards and with the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Conflict of interest

The authors declare no conflict of interest.

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Author contributions

Study design: JK, MC, AK-P, KW-T, ST, AMN-P
Data collection: JK, MC, AK-P, KW-T, AMN-P
Statistical analysis: JK, MC, KW-T, ST, AMN-P
Data interpretation: JK, MC, AK-P, KW-T, ST, AMN-P
Manuscript preparation: JK, MC, KW-T, AMN-P
Literature search: JK, MC, AK-P, KW-T, AMN-P

References

1. Klann JG, Strasser ZH, Hutch MR, et al. Distinguishing Admissions Specifically for COVID-19 From Incidental SARS-CoV-2 Admissions: National Retrospective Electronic Health Record Study. *J Med Internet Res*. 2022; 24(5):37931. https://doi.org/10.2196/37931.
2. Tsai J, Traub E, Aoki K, et al. Incidentally Detected SARS-CoV-2 Among Hospitalized Patients in Los Angeles County, August to October 2020. *J Hosp Med*. 2021;16(8): 480–483. https://doi.org/10.12788/jhm.3641.
3. Hohl CM, Cragg A, Purssell E, et al. Comparing methods to classify admitted patients with SARS-CoV-2 as admitted for COVID-19 versus with incidental SARS-CoV-2: A cohort study. *PLOS One*. 2023;18(9):0291580. https://doi.org/10.1371/journal.pone.0291580.
4. McAlister FA, Hau JP, Xu V. The burden of incidental SARS-CoV-2 infections in hospitalized patients across pandemic waves in Canada. *Sci Rep*. 2023;13:6635. https://doi.org/10.1038/s41598-023-33569-2.
5. Moura CS, Morrison LJ, Hohl CM, et al. Administrative data ICD-10 diagnostic codes identifies most lab-confirmed SARS-CoV-2 admissions but misses many discharged from the Emergency Department. *Sci Rep*. 2024; 14(1):6008. https://doi.org/10.1038/s41598-023-49501-7.
6. Oelsner EC, Krishnaswamy A, Rustamov R, Balte PP, Ali T, Allen NB. Classifying COVID-19 hospitalizations in epidemiology cohort studies: The C4R study. *PLOS One*. 2025; 20(2):0316198. https://doi.org/10.1371/journal.pone.0316198.
7. Hewitt J, Carter B, Vilches-Moraga A, et al. The effect of frailty on survival in patients with COVID-19 (COPE): A multicentre, European, observational cohort study. *Lancet Public Health*. 2020;5(8):444-e451. https://doi.org/10.1016/S2468-2667(20)30146-8.
8. Pan D. Frailty and mortality in patients with COVID-19. *Lancet Public Health*. 2020;5(11):581. https://doi.org/10.1016/S2468-2667(20)30229-2.
9. van Son JE, Kahn ECP, van der Bol JM, Barten DG, Blomaard LC, van Dam C. Atypical presentation of COVID-19 in older patients is associated with frailty but not with adverse outcomes. *Eur Geriatr Med*. 2023;14:285–293. https://doi.org/10.1007/s41999-022-00736-z.
10. Tavares J, Figueiredo D, Passos L, Sobrinho L, Souza E, Pedreira L. Atypical Presentation of COVID-19 in Older Adults: A Scoping Review. *Port J Public Health*. 2023;41(3): 198–217. https://doi.org/10.1159/000534250.
11. Cieślińska A, Fiedorowicz E, Rozmus D, et al. Cross-sectional assessment of a history of SARS-CoV-2 infections using IgG. *Pol Ann Med*. 2023;30(2):91–95. https://doi.org/10.29089/paom/161497.