

Dexamethasone and COVID-19 Mortality: A Descriptive Secondary Analysis of Published Randomized Evidence

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Abstract

Background: COVID-19 can progress from respiratory infection to inflammatory lung injury and fatal organ dysfunction. Corticosteroid treatment illustrates why therapeutic decisions must account for disease severity rather than infection status alone.

Objective: To quantify the absolute mortality difference associated with dexamethasone in a published randomized comparison and explain its clinical interpretation.

Methods: This descriptive secondary analysis used aggregate results from the RECOVERY dexamethasone trial. Treatment allocation, sample sizes, and deaths within 28 days were extracted from the published report. Crude mortality proportions, the absolute risk difference, and the reciprocal absolute risk reduction were calculated. Published respiratory-support subgroup findings informed interpretation. No individual patient records were accessed, and no new participants were recruited.

Results: Among 2,104 participants allocated to dexamethasone, 482 died, compared with 1,110 of 4,321 allocated to usual care. Crude mortality was 22.91% versus 25.69%, yielding an absolute reduction of 2.78 percentage points and a crude risk ratio of 0.892. The corresponding number needed to treat was approximately 36 under the trial conditions. These calculations differ conceptually from the trial's age-adjusted mortality rate ratio. Published subgroup results indicated benefit among patients receiving respiratory support, but not among those receiving no oxygen.

Conclusion: Published randomized data demonstrate a clinically meaningful overall mortality reduction with dexamethasone in hospitalized COVID-19. However, the aggregate estimate should not be generalized to uncomplicated outpatient infection. This analysis provides an educational interpretation of existing evidence, not an independent clinical trial.

Keywords: COVID-19, dexamethasone, mortality, randomized trial, secondary analysis, respiratory support

1. Introduction

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), remains an important clinical and public health concern despite substantial changes in

population immunity, preventive measures, and therapeutic options. The disease has a broad clinical spectrum, ranging from asymptomatic infection and mild upper respiratory symptoms to pneumonia, respiratory failure, and multiorgan dysfunction. Although the risk of severe outcomes has changed over time, individuals with advanced age, chronic medical conditions, or impaired immune function may remain vulnerable. Understanding which patients are most likely to deteriorate is therefore relevant to clinical decision-making, particularly in settings where diagnostic resources and hospital capacity are limited.

The clinical course of COVID-19 reflects interactions among viral characteristics, host immune responses, underlying health status, and access to timely care. Infection can provoke inflammatory responses and endothelial injury that contribute to pulmonary and extrapulmonary complications. In severe cases, dysregulated inflammation, coagulation abnormalities, and impaired gas exchange may occur together, although their relative contributions differ between patients. These mechanisms provide a rationale for investigating routinely available clinical and laboratory indicators as potential markers of disease progression. However, associations between such indicators and adverse outcomes should not be interpreted as evidence of causation or assumed to be stable across different epidemiological periods.

Early recognition of patients at increased risk of deterioration may support appropriate monitoring, treatment planning, and allocation of hospital resources. Age, oxygen saturation, respiratory rate, and the presence of cardiovascular disease, diabetes, obesity, or chronic kidney disease are frequently considered during initial assessment. Laboratory measures, including lymphocyte count, C-reactive protein, serum creatinine, and D-dimer, may provide additional information about inflammatory activity, organ function, and coagulation disturbances. Nevertheless, individual measurements have important limitations. Their interpretation depends on the timing of illness, previous treatment, baseline abnormalities, and the clinical context in which testing is performed. A statistically significant association may also offer limited practical value if it does not improve risk assessment beyond basic bedside findings.

Evaluating these relationships remains necessary because much of the initial evidence on COVID-19 prognosis was generated before widespread vaccination and during periods characterized by different circulating variants and treatment practices. Vaccination, previous infection, and improvements in supportive and disease-specific care may alter both the frequency of severe outcomes and the prognostic relevance of particular risk factors. Furthermore, findings from highly resourced hospitals may not transfer directly to other healthcare environments. Differences in referral pathways, admission thresholds, laboratory availability, and delays in presentation can influence the characteristics of hospitalized populations and the outcomes observed. Locally generated evidence can therefore complement broader research, provided that its limitations and context are clearly acknowledged.

Observational studies using routinely collected hospital data offer a feasible approach to examining these questions in everyday practice. Such studies can characterize patient profiles at admission and assess associations between baseline findings and subsequent clinical outcomes. Their interpretation requires attention to potential confounding, incomplete documentation, selective laboratory testing, and changes in clinical management during the study period. Clear definitions of disease severity and outcomes are especially important, as intensive care admission and respiratory support may reflect resource availability as well as physiological need.

Against this background, the present study aims to evaluate the association of admission demographic characteristics, pre-existing conditions, and routinely available clinical and laboratory parameters with subsequent in-hospital deterioration among adults hospitalized with laboratory-confirmed COVID-19. The analysis focuses on information available at initial assessment to distinguish potential prognostic indicators from consequences of later disease progression. By examining these factors within a defined clinical setting and study period, the study seeks to provide context-specific evidence that may inform early risk assessment and identify priorities for further investigation, rather than establish universally applicable prediction rules.

2. Materials and Methods

A retrospective observational cohort study will be conducted at two tertiary hospitals to evaluate the association between the admission neutrophil-to-lymphocyte ratio (NLR) and 28-day in-hospital mortality among adults hospitalized with coronavirus disease 2019 (COVID-19). The study will use routinely collected electronic medical records for admissions between January 1, 2022, and December 31, 2023. The protocol and statistical analysis plan will be finalized before outcome-linked analyses begin. This design is intended to assess prognostic associations rather than establish a causal effect of inflammatory markers on clinical outcomes.

Eligible patients will be aged 18 years or older and admitted primarily for acute COVID-19, with severe acute respiratory syndrome coronavirus 2 infection confirmed by a nucleic acid amplification test within seven days before or 48 hours after admission. Admission diagnoses and clinical notes will be reviewed to distinguish symptomatic COVID-19 from incidental infection. Patients will be excluded if they have an active hematological malignancy, received cytotoxic chemotherapy during the preceding 30 days, or lack an admission differential blood count. Patients transferred from another hospital after more than 24 hours of inpatient treatment will also be excluded because their initial laboratory measurements may reflect substantial prior treatment. Only the first eligible admission per patient will be analyzed.

The index time will be hospital admission. Admission NLR will be calculated by dividing the absolute neutrophil count by the absolute lymphocyte count from the earliest eligible blood sample collected within 24 hours. Samples obtained before systemic corticosteroid administration will be preferred; sampling and medication timestamps will be recorded. Counts will be expressed in identical units before calculation. Values reported as zero or below an assay's quantification limit will undergo laboratory-record verification and will not be replaced with arbitrary constants. Unresolvable values will be treated as missing. Laboratory platforms and reference intervals will be documented for each hospital.

The primary outcome will be all-cause death occurring in hospital within 28 days of admission. Discharge alive before day 28 will be treated as a competing event rather than interpreted as confirmation of survival through day 28 outside hospital. Patients remaining hospitalized will be administratively censored at day 28. Transfers will be followed through linked receiving-hospital records where available; otherwise, follow-up will end at transfer. Secondary outcomes will include invasive mechanical ventilation, intensive care admission, and hospital length of stay. These outcomes will be interpreted cautiously because clinical eligibility, bed availability, and discharge practices may influence their occurrence.

Prespecified covariates will include age, sex recorded in the medical record, body mass index, smoking status, diabetes mellitus, hypertension, chronic cardiovascular disease, chronic lung disease, chronic kidney disease, immunosuppressive conditions, and documented COVID-19 vaccination status. Vaccination will be classified according to doses received at least 14 days before admission, with undocumented status retained as unknown. Additional variables will include symptom duration, respiratory rate, room-air oxygen saturation where available, oxygen-support category, serum creatinine, C-reactive protein, and calendar quarter. Oxygen saturation measured during supplemental oxygen administration will not be treated as interchangeable with room-air measurements. Antiviral, corticosteroid, and immunomodulatory treatments will be described but not automatically included as baseline confounders when initiated after admission.

Trained reviewers will extract data using a standardized dictionary and pilot-tested collection form. A randomly selected 10% of records will undergo independent duplicate review. Disagreements concerning eligibility, exposure timing, or outcomes will be resolved by consensus with a senior clinician. Automated checks will identify duplicate encounters, implausible laboratory results, inconsistent timestamps, and missing outcome information. Identifiers will be replaced with study codes, and the linkage file will be stored separately on access-controlled institutional systems.

All eligible admissions will be included without outcome-based sampling. Continuous variables will be summarized using means and standard deviations or medians and interquartile ranges, as appropriate; categorical variables will be reported as frequencies and percentages. NLR will be

analyzed continuously after logarithmic transformation, with restricted cubic splines used to assess nonlinearity if the number of deaths permits. Cumulative incidence functions will describe mortality with discharge alive as a competing event. Adjusted associations will be estimated using cause-specific Cox regression, stratified by hospital, and reported with 95% confidence intervals. Model complexity will be constrained by the observed event count; no automated stepwise selection will be used.

Missing covariate data will be characterized by variable and hospital. Multiple imputation will be considered when its assumptions are clinically plausible, alongside complete-case sensitivity analyses. Further sensitivity analyses will exclude samples obtained after corticosteroid administration and assess the influence of unlinked transfers. Proportional hazards assumptions will be checked using residual diagnostics. Secondary analyses will be considered exploratory. Institutional ethics approval and a waiver of individual consent will be requested before data access, subject to local requirements for retrospective minimal-risk research. Findings will be reported with explicit acknowledgment of residual confounding, selection bias, and the absence of systematic post-discharge mortality ascertainment.

3. Results

The following results describe a simulated retrospective cohort constructed to illustrate the reporting of a COVID-19 hospital study; they are not findings from an actual patient dataset. The cohort comprised adults admitted with laboratory-confirmed SARS-CoV-2 infection during a hypothetical 12-month observation period. Severe respiratory disease was defined as a requirement for oxygen at a flow rate of at least 6 L/min, high-flow nasal oxygen, noninvasive ventilation, or invasive mechanical ventilation at any point during hospitalization. Comparisons were made between patients who met this definition and those who did not.

Of 526 admission records assessed, 46 were excluded: 19 represented repeat admissions, 15 lacked laboratory confirmation, and 12 involved incidental infection without a COVID-19-related reason for admission. The final analytical population included 480 patients. All included patients had a documented discharge disposition, and each individual contributed only the first eligible admission. The median age was 59 years, with an interquartile range of 45–71 years; 278 patients (57.9%) were male. At least one recorded chronic condition was present in 312 patients (65.0%). Hypertension was the most frequently documented condition, followed by diabetes mellitus and obesity.

Severe respiratory disease occurred in 180 patients (37.5%), while 300 patients (62.5%) remained below the prespecified respiratory-support threshold. Patients with severe disease were older and more frequently had diabetes, obesity, or chronic kidney disease. The proportion of male patients was also higher in the severe group, although the absolute difference was comparatively modest. Completion of a primary COVID-19 vaccination series before admission was documented in 180 patients (37.5%). This proportion was lower among patients with severe disease than among those without severe disease. Baseline characteristics and selected hospital outcomes are summarized in Table 1.

Table 1. Characteristics and outcomes of the simulated hospitalized COVID-19 cohort

Characteristic	Overall (n = 480)	Severe disease (n = 180)	Nonsevere disease (n = 300)
Age, years, median (IQR)	59 (45–71)	67 (55–76)	54 (41–66)
Male sex, n (%)	278 (57.9)	112 (62.2)	166 (55.3)
Hypertension, n (%)	216 (45.0)	104 (57.8)	112 (37.3)
Diabetes mellitus, n (%)	144 (30.0)	76 (42.2)	68 (22.7)
Obesity, n (%)	120 (25.0)	60 (33.3)	60 (20.0)
Chronic kidney disease, n (%)	48 (10.0)	28 (15.6)	20 (6.7)
Completed primary vaccination series, n (%)	180 (37.5)	42 (23.3)	138 (46.0)

Admission oxygen saturation on room air, %, median (IQR)	93 (88–96)	87 (82–91)	95 (93–97)
Intensive care admission, n (%)	60 (12.5)	54 (30.0)	6 (2.0)
Invasive mechanical ventilation, n (%)	32 (6.7)	32 (17.8)	0 (0.0)
Hospital stay, days, median (IQR)	8 (5–12)	12 (8–17)	6 (4–9)
In-hospital death, n (%)	36 (7.5)	30 (16.7)	6 (2.0)

The median interval from symptom onset to hospital admission was 6 days (interquartile range, 4–9). Patients with severe disease presented a median of 7 days after symptom onset, compared with 5 days among patients without severe disease. Fever was recorded in 374 patients (77.9%), cough in 355 (74.0%), dyspnea in 268 (55.8%), and fatigue in 302 (62.9%). Gastrointestinal symptoms were documented in 74 patients (15.4%). Dyspnea showed the largest difference between respiratory-severity groups, occurring in 146 patients (81.1%) with severe disease and 122 patients (40.7%) without severe disease.

Admission measurements showed greater physiological disturbance in the severe group. Median respiratory rate was 28 breaths/min in patients with severe disease and 20 breaths/min in those without severe disease. Median room-air oxygen saturation was 87% and 95%, respectively. Of the 180 patients meeting the severe-disease definition, 118 already met the respiratory-support threshold during the initial hospital assessment. The remaining 62 developed qualifying respiratory deterioration after admission, at a median of 2 days (interquartile range, 1–4). Thus, the severe group included both patients presenting with advanced respiratory compromise and patients whose oxygen requirements subsequently increased.

Admission laboratory findings were available for most, but not all, participants. C-reactive protein was recorded in 456 patients, D-dimer in 432, and absolute lymphocyte count in 470. Among patients with available measurements, median C-reactive protein was 96 mg/L in the severe group and 38 mg/L in the nonsevere group. Corresponding median D-dimer concentrations were 1.24 and 0.62 mg/L fibrinogen-equivalent units. Median absolute lymphocyte counts were 0.78 and $1.12 \times 10^9/L$, respectively. These comparisons used available observations without imputing missing laboratory values. Laboratory differences were descriptive and did not establish whether inflammatory abnormalities preceded respiratory deterioration.

Supplemental oxygen of any intensity was administered to 302 patients (62.9%), including all 180 patients classified as having severe disease and 122 receiving lower-flow oxygen without crossing the severity threshold. Among severe cases, the highest level of respiratory support was conventional oxygen at or above 6 L/min in 88 patients, high-flow nasal oxygen in 42, noninvasive ventilation in 18, and invasive mechanical ventilation in 32. Intensive care admission occurred in 60 patients. The six intensive care admissions in the nonsevere group were for nonrespiratory indications, including hemodynamic instability or cardiac monitoring, without qualifying oxygen requirements.

In a prespecified multivariable logistic regression model containing age, sex, hypertension, diabetes, obesity, chronic kidney disease, and vaccination status, older age was associated with greater odds of severe respiratory disease. The adjusted odds ratio per 10-year increase was 1.38 (95% confidence interval, 1.19–1.61). Diabetes and obesity were also associated with higher odds, with adjusted odds ratios of 1.67 (1.06–2.64) and 1.72 (1.07–2.77), respectively. Completed primary vaccination was associated with lower odds, with an adjusted odds ratio of 0.46 (0.29–0.73). Confidence intervals for sex, hypertension, and chronic kidney disease included unity. These estimates describe associations within the simulated hospitalized population rather than causal effects or population-level vaccine effectiveness.

Overall, 444 patients (92.5%) survived to hospital discharge and 36 (7.5%) died during admission. Mortality was 16.7% in the severe group and 2.0% in the nonsevere group, an absolute difference of

14.7 percentage points. Among the 32 patients receiving invasive mechanical ventilation, 18 died. Median hospital stay was longer in the severe group, although duration was influenced by both discharge and death. No post-discharge outcomes were available. Consequently, the reported mortality, respiratory-support use, and length-of-stay findings were restricted to the index hospitalization.

4. Discussion

The clinical relevance of COVID-19 extends beyond the initial respiratory infection to the interaction between viral injury, host inflammatory responses, and pre-existing vulnerability. In interpreting clinical findings, disease severity should therefore be considered a multidimensional outcome rather than a direct consequence of infection alone. Differences in age, underlying health, immune protection, and access to timely treatment can substantially influence presentation and recovery. This framework is particularly important for observational research, in which associations between patient characteristics and adverse outcomes may reflect several overlapping biological and healthcare-related pathways.

Age is a central consideration in the interpretation of COVID-19 outcomes. Older adults commonly have reduced physiological reserve, altered immune responses, and a higher burden of chronic disease, all of which may contribute to clinical deterioration. However, chronological age should not be treated as interchangeable with frailty or functional dependence. Younger patients with substantial comorbidity may also experience severe illness, while some older individuals recover without major complications. If an association between age and unfavorable outcomes is identified, its magnitude should be interpreted alongside functional status, comorbidity burden, and differences in clinical management rather than assumed to represent an isolated age effect.

Cardiometabolic and respiratory disorders provide another important context for understanding variation in clinical course. Diabetes, obesity, cardiovascular disease, and chronic pulmonary conditions may influence immune regulation, endothelial function, and the ability to tolerate hypoxemia. Nevertheless, these conditions frequently coexist and are associated with social and behavioral determinants of health. An observed relationship between a particular comorbidity and disease severity would therefore not, by itself, establish causality. Analyses that distinguish individual diagnoses from cumulative disease burden may offer greater clinical value, although residual confounding can remain even after statistical adjustment.

Laboratory abnormalities can support risk assessment but require similarly cautious interpretation. Elevated inflammatory markers, lymphopenia, and disturbances in coagulation may accompany more severe COVID-19, yet none is sufficiently specific to explain an individual patient's trajectory in isolation. Their significance depends on the timing of measurement, baseline health, concurrent infection, and treatment exposure. Measurements obtained after deterioration may primarily reflect established disease rather than predict its development. Consequently, any relationship between admission biomarkers and subsequent outcomes is most informative when temporal ordering is clear and when the biomarkers are evaluated alongside routinely available clinical indicators, including oxygen saturation and respiratory rate.

The timing of presentation and treatment may further shape observed outcomes. Delayed evaluation can coincide with more advanced respiratory compromise, but the interval from symptom onset to admission is influenced by symptom recognition, healthcare availability, and referral practices. Hospital-based populations may consequently overrepresent patients with severe disease or limited access to early care. Differences in admission thresholds can also complicate comparisons between institutions. These considerations make it important to interpret hospitalization, intensive care admission, and mortality as related but distinct endpoints, each affected by clinical need and local service capacity.

Vaccination, previous infection, circulating variants, and changing treatment practices are additional determinants of applicability. Findings generated during one phase of the pandemic may not transfer directly to another. Immune protection can modify the probability of severe disease without eliminating infection, while antiviral and anti-inflammatory treatment can alter subsequent progression in eligible patients. Where information on these factors is incomplete, conclusions should

remain restricted to the population and period represented by the study.

Taken together, these considerations support an integrated approach to interpreting COVID-19 research. Clinical characteristics and laboratory findings are best understood as complementary signals rather than independent explanations of severity. Their practical importance depends on whether they improve recognition of patients requiring closer observation or timely intervention beyond standard clinical assessment. Any proposed risk-stratification approach should therefore prioritize reproducibility, temporal validity, and usefulness across relevant patient groups.

The clinical implications of these findings should be considered within the broader context of COVID-19 as a heterogeneous disease rather than a single, uniform respiratory illness. Differences in presentation and recovery may reflect interactions among host vulnerability, immune history, viral characteristics, and access to timely care. Accordingly, clinical assessment should integrate symptoms, physiological measurements, comorbid conditions, and the trajectory of illness. Reliance on any isolated feature may underestimate risk in patients whose condition initially appears stable but subsequently deteriorates.

A practical implication is the importance of risk-stratified follow-up. Older adults and patients with substantial cardiometabolic, pulmonary, or immunocompromising conditions may benefit from closer monitoring, particularly when symptoms persist or functional capacity declines. However, risk stratification should not be interpreted as a substitute for individualized assessment, because clinically important illness can also occur in younger patients without documented comorbidity. Where home monitoring is used, patients require clear instructions on device limitations, warning symptoms, and when to seek urgent evaluation. Monitoring strategies must also account for unequal access to reliable equipment and clinical advice.

Persistent symptoms after the acute phase warrant particular attention. Fatigue, breathlessness, cognitive difficulties, and reduced exercise tolerance may arise through several overlapping pathways, including residual organ injury, deconditioning, sleep disturbance, and psychological distress. These symptoms should neither be automatically attributed to ongoing viral activity nor dismissed when routine investigations are unrevealing. A structured assessment can help identify treatable alternative diagnoses and guide appropriate rehabilitation. Rehabilitation should be individualized, with attention to symptom exacerbation after exertion rather than a uniform expectation of progressively increasing activity.

The interpretation of observational associations requires considerable caution. Even when analyses account for measured demographic and clinical characteristics, residual confounding may remain. Disease severity at presentation, prior infection, vaccination history, treatment timing, socioeconomic circumstances, and health-seeking behavior can influence both recorded exposures and subsequent outcomes. Associations therefore should not be interpreted as evidence that a particular characteristic directly causes deterioration or delayed recovery. Similarly, treatment-related associations may reflect confounding by indication, because patients with more severe illness are more likely to receive intensive interventions.

Several methodological considerations limit the certainty and generalizability of the conclusions. Selection into testing, clinical assessment, or follow-up may preferentially include symptomatic individuals and those with better access to healthcare. Conversely, patients facing substantial barriers to care may be underrepresented despite having greater clinical needs. Incomplete documentation and self-reported information can introduce misclassification, while loss to follow-up may bias recovery estimates if participation differs according to symptom burden. Changes in circulating variants, population immunity, and clinical management also mean that findings from one pandemic period may not transfer directly to another. These limitations should inform interpretation rather than be treated as reasons to disregard potentially useful clinical patterns.

Future research should prioritize prospective, multicenter designs with standardized definitions of acute severity, recovery, and persistent symptoms. Repeated assessments would better distinguish transient complaints from sustained impairment and clarify how clinical trajectories vary across patient groups. Integrating patient-reported outcomes with objective functional measures could improve the relevance of follow-up. Analyses should also examine whether observed differences

remain after accounting for vaccination, reinfection, treatment access, and social disadvantage. Where feasible, intervention studies are needed to determine whether targeted monitoring or rehabilitation improves outcomes rather than merely identifying patients at higher risk.

Overall, the findings are best understood as supporting a cautious, patient-centered approach to COVID-19 care rather than establishing definitive causal pathways. Their principal value lies in informing clinical vigilance and identifying questions for more rigorous investigation. Continued attention to both acute complications and longer-term functional recovery remains important, while recommendations should be updated as population immunity, viral evolution, and the evidence base change.

5. Conclusion

COVID-19 remains a clinically important disease whose consequences extend beyond the acute phase of infection. The central conclusion of this investigation is that its clinical burden should be considered within a broader framework encompassing individual vulnerability, timely access to care, and recovery after acute illness. A positive diagnostic result alone does not adequately characterize disease impact, and the absence of hospitalization should not be interpreted as evidence of complete or uncomplicated recovery.

The clinical interpretation of COVID-19 outcomes requires particular attention to age, underlying medical conditions, immune status, and prior exposure through infection or vaccination. These factors may influence both the likelihood of severe illness and the subsequent need for medical support. However, observed associations should not be treated as proof of causation, especially when differences in healthcare access, treatment availability, and health-seeking behavior may also contribute. Risk assessment is therefore most useful when it supports individualized clinical judgment rather than replacing it with a single demographic or laboratory indicator.

The practical implications favor a coordinated approach that connects prevention, early recognition of deterioration, evidence-based acute management, and appropriate follow-up. Vaccination should remain part of prevention strategies, particularly for populations at increased risk, while clinical decisions should reflect current recommendations and patient-specific circumstances. Patients reporting persistent fatigue, breathlessness, cognitive difficulties, or reduced functional capacity after infection warrant careful assessment. Such symptoms should neither be dismissed nor automatically attributed to COVID-19 without consideration of alternative diagnoses and pre-existing conditions.

Interpretation must nevertheless remain cautious. Without detailed information on sampling, follow-up completeness, and adjustment for confounding, the precision and generalizability of conclusions cannot be firmly established. Changes in circulating variants, population immunity, testing practices, and treatment pathways may further limit the transferability of observations across settings and time periods. In particular, short follow-up cannot establish the long-term course of post-infectious symptoms, and routinely collected clinical measures may fail to capture limitations important to patients.

Future investigations should use clearly defined outcomes, adequately characterized comparison groups, and longitudinal assessments that incorporate both clinical events and patient-reported recovery. Greater inclusion of underserved populations would help clarify whether apparent differences in outcomes reflect biological vulnerability, unequal access to care, or both. Overall, the most defensible implication is the need for proportionate, risk-informed COVID-19 care that addresses immediate complications while recognizing uncertainty about longer-term effects. Continued research and adaptable health services are necessary to translate evolving evidence into meaningful improvements in patient outcomes without overstating the certainty of available findings.

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