

Post-acute organ complications within one year following COVID-19 hospitalization and related socioeconomic inequalities

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Post-acute organ complications following COVID-19 hospitalization and potential socioeconomic inequalities therein are understudied. In this case-control study, we use individual-level data from three national health and social registries in Belgium to assess whether COVID-19 hospitalization increases the risk of post-acute organ complications within one year among 59,351 hospitalized adults without preexisting conditions affecting the specific organ system under study at baseline. In addition, we identify socioeconomic patterns in the development of these organ complications. Overlap propensity score (PS)-weighted odds ratios (ORs) and adjusted odds ratios (aORs) were estimated. All analyses were stratified between severe and critical COVID-19 hospitalization, with the latter defined by intensive care unit admission and/or the onset of acute respiratory distress syndrome. We found significant cardiovascular (overlap PS-weighted OR 1.19, 95% CI 1.03–1.37) and pulmonary (overlap PS-weighted OR 2.05, 95% CI 1.80–2.34) complications within one year following severe COVID-19 hospitalization compared to non-COVID-19 hospitalization, with particularly higher odds following critical COVID-19 hospitalization. Among severe COVID-19 patients, those with low income, compared to those with high income, had higher odds of post-acute pulmonary complications (aOR 1.53, 95% CI 1.05–2.25). Long-term care should prioritize monitoring organ complications following COVID-19 hospitalization, especially after critical illness.

The COVID-19 pandemic, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has had an unprecedented impact on our healthcare system, with more than 4.8 million cases, 150,000 hospitalizations, and 34,000 deaths recorded in Belgium by 1st January 2024¹.

The virus mainly affects the upper respiratory tract and causes cold-like symptoms in most individuals, with many individuals experiencing mild to moderate illness and developing symptoms such as fever, diarrhea, headaches, coughing, and difficulty breathing. When the SARS-CoV-2 virus replicates in the lower respiratory tract,

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pulmonary alveoli are infected, which can lead to a severe form of the disease characterized by dyspnea and hypoxemia, progressively resulting in respiratory failure among critical cases^{2,3}. While the primary cause of COVID-19 is viral replication in the lungs, inflammation triggered by the virus can also lead to other acute organ failures⁴, such as acute myocardial, renal, and liver injuries requiring hospital care^{5–8}.

Beyond acute complications, some individuals may develop post-acute health issues several months following SARS-CoV-2 infection. The pathogenesis of post-acute COVID-19-related health complications is not yet fully understood. Proposed pathophysiological mechanisms include direct viral damage, inflammation, autoimmune responses, and endothelial injury caused by critical SARS-CoV-2 infection^{9,10}. These processes can affect multiple organ systems, leading to mid- or long-term sequelae, commonly known as “*Post-acute sequelae of COVID-19*” (PASC) or “*Long COVID*”. The World Health Organization (WHO) defined PASC as “*persistent or new symptoms three months after initial SARS-CoV-2 infection, lasting at least two months and with no other underlying explanation*”¹¹.

Symptoms such as fatigue, post-exertional malaise, cognitive impairment, chest pain, anxiety/depression, and headache can persist up to several months following the infection and are particularly prevalent among individuals who developed a critical form of the disease^{12,13}. Indeed, in a meta-analysis on 194 studies, the pooled prevalence of PASC among COVID-19 hospitalized survivors at 126 days was 52.6%, compared to 34.5% among non-hospitalized COVID-19 survivors¹³. Several studies suggest that, compared to SARS-CoV-2 test-negative controls, COVID-19 hospitalized patients faced increased risks of post-acute cardiovascular (CV) (e.g., dysrhythmias, inflammatory heart diseases, or thrombotic disorders) and pulmonary (e.g., decreased diffusion capacity) complications up to six months after COVID-19 exposure, even in the absence of preexisting CV or pulmonary conditions at baseline^{14–16}. However, one-year follow-up studies focusing on COVID-19 hospitalized patients remain limited, particularly those assessing post-acute renal and liver complications, which represent significant acute health complications following SARS-CoV-2 infection^{17,18}. This study is particularly relevant in Belgium, as the country was heavily impacted by COVID-19¹, has an important proportion of an aging population vulnerable to long-term complications¹⁹, and a substantial burden of CV diseases²⁰.

Although COVID-19 is a respiratory disease caused by a viral infection, the social component of the pandemic was quickly put forward by qualifying it as a syndemic, highlighting both biological and social interactions²¹. Indeed, beyond underlying health conditions and age, which are primary risk factors for COVID-19 critical outcomes²², sociodemographic (SD) and socioeconomic (SE) characteristics were identified as additional risk factors. Men, individuals of migrant descent and those experiencing SE hardship (e.g., higher poverty level, living in densely populated areas, lower education) presented higher COVID-19 mortality and hospitalization rates^{23–26}.

The latter might be at higher risk of critical COVID-19 outcomes due to higher vulnerability (e.g., pre-existing health issues may increase COVID-19 severity), susceptibility (e.g., poor living conditions and chronic stress weaken the immune system), exposure (e.g., higher rates of individuals working in essential sectors during the pandemic limiting remote work, lower rates of preventive measures adoption including vaccination), transmission (e.g., higher rates of individuals living in overcrowded households and neighborhoods), and reduced access to and quality of healthcare^{21,27}. Despite a clear social pattern identified in COVID-19 hospitalization, there is a dearth of knowledge of SE inequalities in the development of PASC among hospitalized COVID-19 survivors.

This study aims to address these gaps through the two following objectives: (1) identify whether COVID-19 hospitalization is associated with the development of one-year post-acute CV, pulmonary, renal,

and liver complications compared to non-COVID-19 hospitalized controls, both without preexisting conditions affecting the specific organ system under study at baseline; and (2) identify the social patterns of one-year post-acute CV and pulmonary complications following hospitalization among COVID-19 patients without preexisting conditions affecting the specific organ system under study at baseline. Those post-acute organ complications were chosen as these anatomical areas are well-known to be affected during the acute phase of SARS-CoV-2 infection, as previously established in the international literature^{5–8}.

Results

Baseline characteristics

We included 1839, 5024, 6040, and 6220 severe COVID-19 hospitalized patients without CV, pulmonary, renal, and liver conditions at baseline, respectively. Regarding critical COVID-19 hospitalized patients, 301, 833, 998, and 1031 without CV, pulmonary, renal, and liver conditions at baseline were included, respectively. The corresponding control groups consisted of 22,310, 45,036, 50,910, and 51,965 non-COVID-19 hospitalized controls without CV, pulmonary, renal, and liver diseases at baseline, respectively (Fig. 1).

The SD, SE, and health-related characteristics of severe and critical COVID-19 hospitalized patients and controls before weighting, along with the standardized mean differences (SMDs) assessing the effectiveness of overlap weights (OWs) in achieving covariate balance, are provided in Supplementary Data 1 to 4. The weighted SMDs showed negligible differences (i.e., <0.001) in baseline characteristics after weighting for both severe and critical COVID-19 patients and their respective controls (Supplementary Data 1 to 4).

Impact of COVID-19 hospitalization on the onset of post-acute organ complications

Table 1 shows overlap propensity score (PS)-weighted ORs and doubly robust overlap PS-weighted ORs, along with their 95% confidence intervals (CIs), estimated using binomial logistic regression models, assessing the development of post-acute organ complications within one year among severe COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls.

One-year post-acute organ complications were more common in severe COVID-19 patients than in controls, with 14.8%, 6.5%, 0.7%, and 0.21% of patients developing post-acute CV, pulmonary, renal and liver complications within one year following COVID-19 hospitalization, respectively, compared to 8.7%, 3%, 0.6%, and 0.20% of controls over the same duration.

Compared to non-COVID-19 hospitalized controls, severe COVID-19 hospitalized patients had significantly higher risks of developing post-acute CV (overlap PS-weighted OR 1.19, 95% CI 1.03–1.37) and pulmonary (overlap PS-weighted OR 2.05, 95% CI 1.80–2.34) complications within one year following hospitalization. However, the risks of developing one-year post-acute renal (overlap PS-weighted OR 0.96, 95% CI 0.68–1.35) and liver (overlap PS-weighted OR 0.84, 95% CI 0.46–1.52) complications following severe COVID-19 hospitalization were not statistically significant. Overlap PS-weighted aORs, adjusting for potential misspecification in the PS model that could bias the estimation of the exposure's effect on the outcome, yielded similar conclusions (Table 1). These overlap PS-weighted causal estimates focus on the subgroup of patients with comparable baseline characteristics who could plausibly have been hospitalized with or without COVID-19. As such, the results are generalizable to this overlap population, rather than to the entire study cohort.

Table 2 shows overlap PS-weighted ORs and overlap PS-weighted aORs, along with their 95% CIs, estimated using binomial logistic regression models, assessing the development of one-year post-acute organ complications among critical COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls.

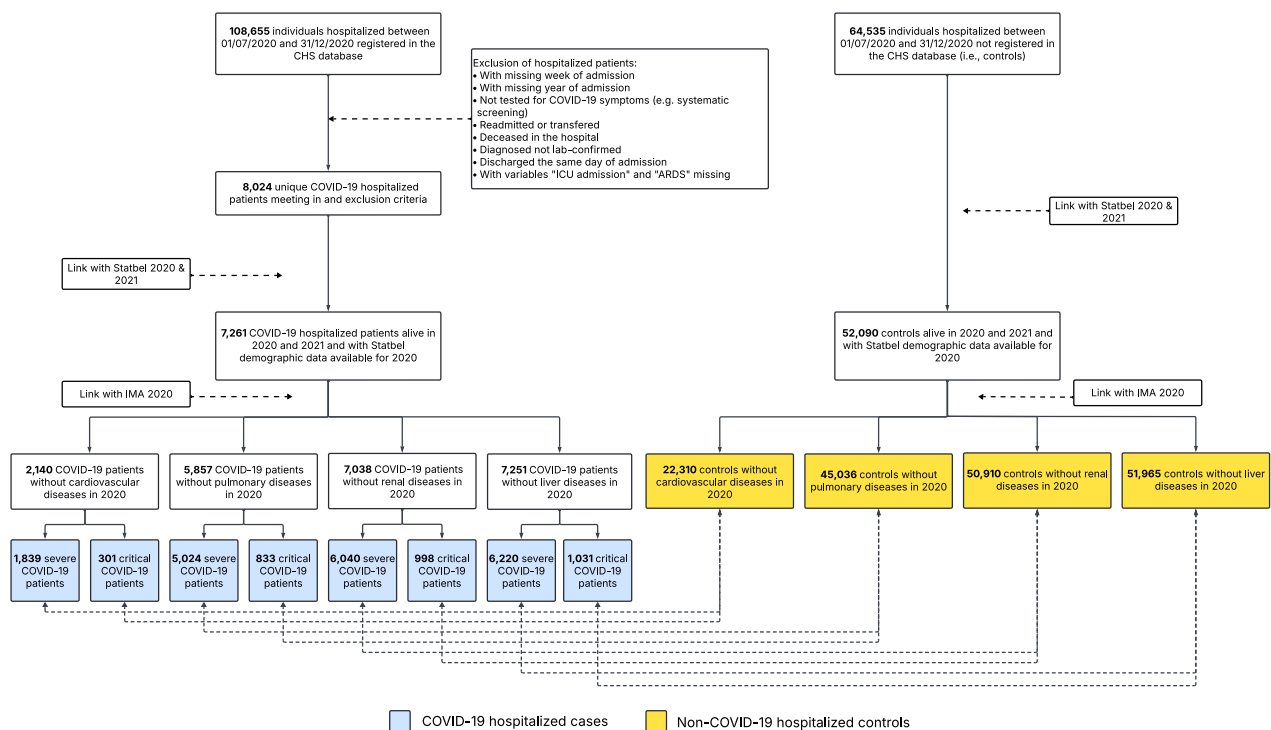


Fig. 1 | Flowchart of the study population building on the HELICON population data linkage to assess the development of post-acute organ complications within one year following COVID-19 hospitalization among severe and critical COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls, both without those organ conditions at baseline. Critical COVID-19 hospitalized patients were defined as COVID-19 patients admitted to the

intensive care unit (ICU) and/or with acute respiratory distress syndrome (ARDS). Severe COVID-19 patients were defined as COVID-19 hospitalized patients not admitted to the ICU and without ARDS. The dotted lines represent the comparison between the subcohorts of COVID-19 patients and their respective controls. Abbreviations: CHS Clinical Hospital Survey, IMA InterMutualistic Agency, Statbel Statistic Belgium.

Table 1 | Overlap propensity score (PS)-weighted odds ratios (ORs) and overlap PS-weighted adjusted ORs (aORs) estimated using binomial logistic regression models to assess the development of one-year post-acute cardiovascular, pulmonary, renal, and liver complications, respectively, among severe COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls, both without preexisting conditions affecting the specific organ system under study at baseline

Post-acute health outcomes	N severe COVID-19 patients	Severe COVID-19 patients with outcome N (%)	N controls	Controls with outcome N (%)	Overlap PS-weighted OR [95% CI]	P-value	Overlap PS-weighted aOR [95% CI]	P-value
Cardiovascular complications ^a	1839	272 (14.79)	22,310	1937 (8.68)	1.19 [1.03–1.37]	<0.001	1.20 [1.04–1.39]	0.011
Pulmonary complications ^b	5024	326 (6.49)	45,036	1332 (2.96)	2.05 [1.80–2.34]	<0.001	2.06 [1.81–2.35]	<0.001
Renal complications ^c	6040	40 (0.66)	50,910	302 (0.59)	0.96 [0.68–1.35]	0.680	0.67 [0.95–1.33]	0.756
Liver complications ^d	6220	13 (0.21)	51,965	102 (0.20)	0.84 [0.46–1.52]	0.556	0.86 [0.47–1.56]	0.615

Overlap PS-weighted aORs are adjusted for sociodemographic (age groups, sex, region of residence, living situation, migration background), socioeconomic (education, income) and health-related (cardiovascular comorbidities, pulmonary comorbidities, renal comorbidities, liver comorbidities, other comorbidities) variables.

Severe COVID-19 patients were defined as COVID-19 patients not admitted to the intensive care unit and without acute respiratory distress syndrome.

^aThe weighted logistic regression models were applied to severe COVID-19 patients and controls, both without preexisting cardiovascular conditions at baseline (N = 24,149).

^bThe weighted logistic regression models were applied to severe COVID-19 patients and controls, both without preexisting pulmonary conditions at baseline (N = 50,060).

^cThe weighted logistic regression models were applied to severe COVID-19 patients and controls, both without preexisting renal conditions at baseline (N = 56,950).

^dThe weighted logistic regression models were applied to severe COVID-19 patients and controls, both without preexisting liver conditions at baseline (N = 58,185).

Significant results (p-value < 0.05) are highlighted in bold.

Overall, most post-acute organ complications were more common among critical COVID-19 hospitalized patients than among controls, with 21.7%, 7%, 1%, and 0.1% of critical COVID-19 patients developing post-acute CV, pulmonary, renal, and liver complications, respectively, compared to 8.7%, 3%, 0.6%, and 0.2% of controls.

Compared to non-COVID-19 hospitalized controls, critical COVID-19 patients had significantly higher risks of developing post-acute CV (overlap PS-weighted OR 1.93, 95% CI 1.36–2.42) and pulmonary (overlap PS-weighted OR 2.72, 95% CI 1.98–3.79) complications within one year following hospitalization. However, the risks of developing

Table 2 | Overlap propensity score (PS)-weighted odds ratios (ORs) and overlap PS-weighted adjusted ORs (aORs), along with their 95% confidence intervals (CIs), estimated using binomial logistic regression models to assess the development of one-year post-acute cardiovascular, pulmonary, renal, and liver complications, respectively, among critical COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls, both without preexisting conditions affecting the specific organ system under study at baseline

Post-acute health outcomes	N critical COVID-19 patients	Critical COVID-19 patients with outcome N (%)	N controls	Controls with outcome N (%)	Overlap PS-weighted OR [95% CI]	P-value	Overlap PS-weighted aOR [95% CI]	P-value
Cardiovascular complications ^a	301	65 (21.69)	22,310	1937 (8.68)	1.93 [1.36–2.42]	<0.001	1.41 [1.88–2.51]	<0.001
Pulmonary complications ^b	833	58 (6.96)	45,036	1332 (2.95)	2.72 [1.98–3.79]	<0.001	2.71 [1.94–3.85]	<0.001
Renal complications ^c	998	10 (1.00)	50,910	302 (0.59)	1.54 [0.81–2.94]	0.187	1.54 [0.81–2.94]	0.185
Liver complications ^d	1031	1 (0.10)	51,965	102 (0.20)	0.41 [0.06–2.96]	0.375	0.41 [0.06–2.97]	0.377

Overlap PS-weighted aORs are adjusted for sociodemographic (age groups, sex, region of residence, living situation, migration background), socioeconomic (education, income) and health-related (cardiovascular comorbidities, pulmonary comorbidities, renal comorbidities, liver comorbidities, other comorbidities) variables.

Critical COVID-19 patients were defined as COVID-19 patients admitted to the intensive care unit and/or with acute respiratory distress syndrome.

^aThe weighted logistic regression models were applied to critical COVID-19 patients and controls, both without preexisting cardiovascular conditions at baseline ($N = 22,611$).

^bThe weighted logistic regression models were applied to critical COVID-19 patients and controls, both without preexisting pulmonary conditions at baseline ($N = 45,869$).

^cThe weighted logistic regression models were applied to critical COVID-19 patients and controls, both without preexisting renal conditions at baseline ($N = 51,908$).

^dThe weighted logistic regression models were applied to critical COVID-19 patients and controls, both without preexisting liver conditions at baseline ($N = 52,996$).

Significant results (p -value < 0.05) are highlighted in bold.

post-acute renal (overlap PS-weighted OR 1.54, 95% CI 0.81–2.94) and liver (overlap PS-weighted OR 0.41, 95% CI 0.06–2.96) complications following critical COVID-19 hospitalization were not statistically significant. Overlap PS-weighted aORs gave similar conclusions (Table 2). These overlap PS-weighted causal estimates focus on the subgroup of patients with comparable baseline characteristics who could plausibly have been hospitalized with or without COVID-19. As such, the results are most generalizable to this overlap population, rather than to the entire study cohort.

Figure 2 shows the overlap PS-weighted excess burdens per 1000 patients of one-year post-acute organ complications attributable to severe and critical COVID-19 hospitalization, respectively.

The weighted excess burden per 1000 patients of post-acute CV complications was substantially higher for critical COVID-19 hospitalization (weighted excess burden 84.83, 95% CI 36.89 – 132.77) compared to severe COVID-19 hospitalization (weighted excess burden 20.16, 95% CI 2.34 – 37.98). Similarly, a slightly higher excess burden of post-acute pulmonary complications was observed following critical COVID-19 hospitalization (weighted excess burden 38.7, 95% CI 21.35 – 56.05) compared to severe COVID-19 hospitalization (weighted excess burden 32.36, 95% CI 25.15 – 39.57). However, the excess burdens of post-acute renal complications were not statistically significant following either severe (weighted excess burden –0.28, 95% CI –2.54 – 1.98) or critical (weighted excess burden 3.52, 95% CI –2.77 – 9.81) COVID-19 hospitalizations. Similarly, the excess burdens of post-acute liver complications due to either severe (weighted excess burden –0.4, 95% CI –1.67 – 0.87) or critical (weighted excess burden –1.45, 95% CI –3.63 – 0.63) COVID-19 hospitalizations were not statistically significant (Fig. 2).

Socioeconomic patterns in the onset of post-acute organ complications

Supplementary Data 5 and 6 show the aORs and 95% CIs for the development of post-acute CV and pulmonary complications among severe and critical hospitalized COVID-19 patients according to the SE exposures of interest (i.e., education and income), respectively.

Overall, no significant SE patterns were identified in the development of post-acute CV complications among severe COVID-19 patients. However, severe COVID-19 patients aged 45 to 64 years (aOR 1.94, 95% CI 1.34–2.86) and 65 to 84 years (aOR 2.97, 95% CI 1.90–4.69), compared to those aged 18 to 44 years, as well as those with “other

comorbidities” (aOR 2.00, 95% CI 1.48–2.68), had significantly increased odds of developing post-acute CV complications. Conversely, living in Brussels compared to Flanders (aOR 0.56, 95% CI 0.32–0.96) and having a migration background (aOR 0.61, 95% CI 0.38–0.97 for second-generation migrants; aOR 0.54, 95% CI 0.30–0.93 for first-generation EU migrants; aOR 0.61, 95% CI 0.38–0.97 for first-generation non-EU migrants) were associated with decreased odds of developing post-acute CV complications (Supplementary Data 5). A SE pattern was identified in the development of post-acute pulmonary complications, with severe COVID-19 patients with low income having higher odds of developing such complications compared to those with high income (aOR 1.53, 95% CI 1.05–2.25). Other SD and health-related variables were not significantly associated with the development of post-acute pulmonary complications among severe COVID-19 patients (Supplementary Data 5).

No major social patterns were identified in the development of either post-acute CV or pulmonary complications among critical COVID-19 patients. However, having missing income, compared to high income, was associated with increased odds of developing post-acute pulmonary complications (aOR 5.25, 95% CI 1.15–23.24) (Supplementary Data 6). Supplementary Data 7, which characterizes critical COVID-19 patients with missing income by SD, SE, and health-related variables relative to the total number of critical COVID-19 patients without pulmonary diseases at baseline, showed an overrepresentation of individuals aged 85 years and over, males, first-generation non-EU migrants, and individuals with CV comorbidities, renal comorbidities, and “other comorbidities” among those with missing income.

Supplementary Data 8 and 9 present aORs and 95% CIs assessing the association between SE variables and post-acute CV and pulmonary complications among severe and critical COVID-19 patients and non-COVID-19 hospitalized controls, along with p -values for interaction. We did not identify any SE variables with a stronger association with the development of CV and pulmonary complications in either severe COVID-19 hospitalized patients compared to controls, or critical COVID-19 patients compared to controls.

Discussion

This study investigated the development of post-acute organ complications within one year following COVID-19 hospitalization and related SE inequalities in Belgium using a unique population data linkage within the HELICON project.

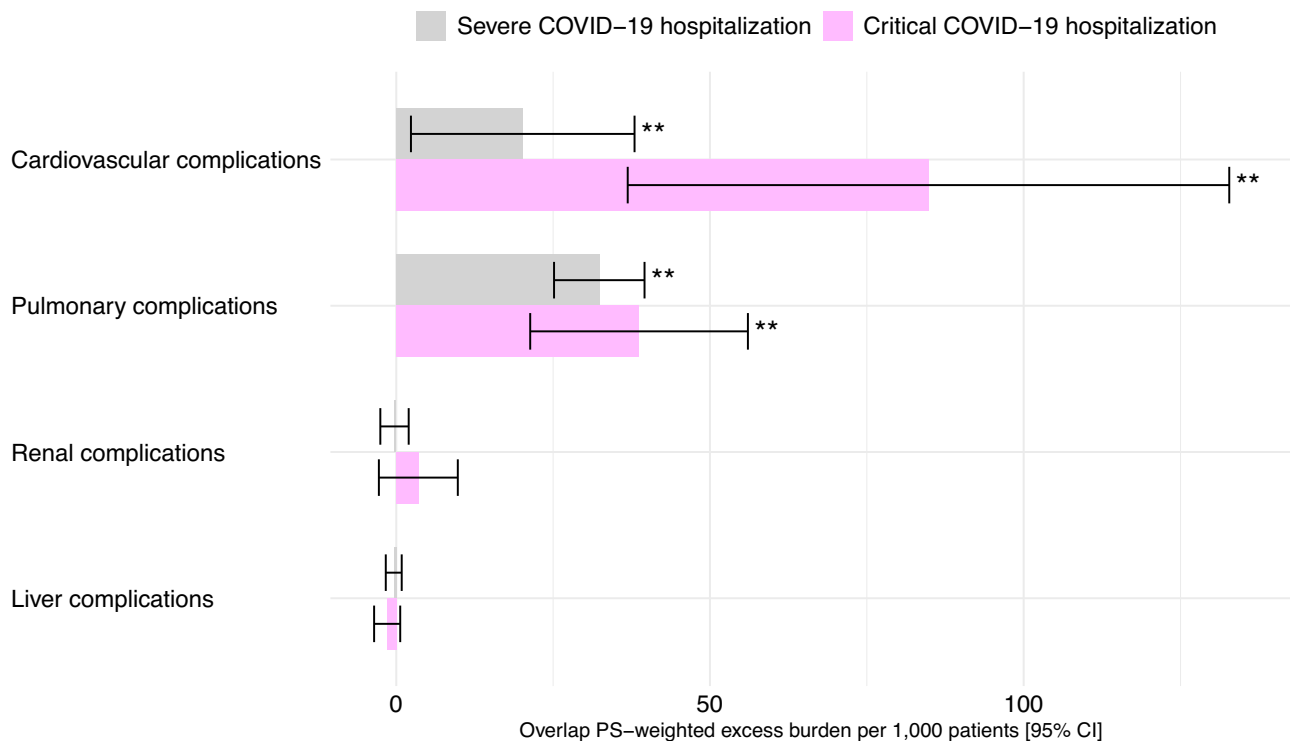


Fig. 2 | Overlap propensity score (PS)-weighted point estimates of excess burdens per 1000 patients and 95% confidence intervals (CIs) of one-year post-acute cardiovascular, pulmonary, renal, and liver complications among severe and critical COVID-19 patients compared to non-COVID-19 hospitalized controls, both without preexisting conditions affecting the specific organ system under study at baseline. The central estimate represents the overlap propensity

score (PS)-weighted excess burden per 1000 patients, and error bars indicate 95% confidence intervals. Critical COVID-19 patients were defined as COVID-19 hospitalized patients admitted to the intensive care unit (ICU) and/or with acute respiratory distress syndrome (ARDS). Severe COVID-19 patients were defined as COVID-19 hospitalized patients not admitted to the ICU and without ARDS. ** p -value < 0.05. The numerical values of Fig. 2 are available in Supplementary Table 1.

Our findings showed a 1.19 times higher odds and 20.16 excess burden per 1000 patients of post-acute CV complications among severe COVID-19 patients compared to non-COVID-19 hospitalized controls, both without CV diseases at baseline, further increasing when considering critical COVID-19 patients. These results are in line with those from international studies^{14,15,28,29}. In the United States (U.S.), COVID-19 hospitalization was associated with significant burden differences per 1000 patients at 12 months, ranging from 5.84 for dysrhythmia to 80.86 for inflammatory heart diseases. These burden differences were reported to increase with ICU admission. A subgroup analysis of non-hospitalized infected cases without preexisting CV conditions, compared to non-infected cases, yielded similar findings¹⁴. In England, Ayoubkhani et al.²⁹ observed a 1.5 times higher rate of major adverse CV events (a composite of non-fatal stroke, non-fatal myocardial infarction, and CV death) among COVID-19 patients compared to matched population controls. A similar, although slightly smaller, effect size was observed when considering COVID-19 patients without preexisting CV conditions at baseline.

Post-acute pulmonary complications, including pulmonary fibrosis and reduced lung function, have been recognized in several international studies as significant PASC^{16,29–32}. This is in line with our results showing a 2.05 and 2.72 times higher odds and a 32.36 and 38.7 excess burdens per 1000 patients of post-acute pulmonary complications among severe and critical COVID-19 patients compared to controls, both without pulmonary diseases at baseline, respectively. In England, compared to a matched control group, COVID-19-related post-acute respiratory complications were reported at rates six times higher in COVID-19 patients with preexisting pulmonary conditions, and 27 times higher in those without preexisting pulmonary conditions²⁹. Our results, suggesting higher odds and excess burdens of post-acute pulmonary complications according to disease severity, align with

some studies showing that long-term pulmonary sequelae increase with the severity of COVID-19^{16,30}.

We did not identify any significant odds or excess burden of post-acute renal complications following either severe or critical COVID-19 hospitalization, compared to non-COVID-19 hospitalization. This finding contrasts with results from other international studies^{32,33}, and is likely partly attributable to the very small number of renal cases identified in the IMA database. Similarly, we did not identify any significant association between either severe or critical COVID-19 hospitalization and the development of post-acute liver complications. Evidence on renal and especially liver function impairment during the post-acute COVID-19 phase remains limited. COVID-19-associated acute kidney injury (AKI), a common acute complication in COVID-19 hospitalized patients, has been linked to post-AKI kidney function impairment and elevated serum creatinine^{34–36}. A 6-month follow-up cohort study reported a 1.36 increased risk and an 25.24 excess burden per 1000 patients of chronic kidney disease (CKD) following COVID-19 hospitalization, further increasing with ICU admission³³. Regarding post-acute liver complications, findings generally indicate no significant liver function impairment post-COVID-19 hospitalization³⁷, although critical COVID-19 cases may exhibit elevated liver enzymes and markers of inflammation¹⁷.

Research on SE inequalities in PASC remains limited, especially among hospitalized COVID-19 survivors, with inconsistent findings and the use of varying definitions of PASC, making it difficult to identify clear socioeconomic patterns therein³⁸. Several population-based studies from the UK showed that the least deprived individuals, compared to the less deprived, present higher odds of COVID-19-related persistent symptoms at least four weeks following COVID-19 diagnosis^{39–42}. In the U.S., Hirschtick et al.⁴³ identified a 1.5 times higher prevalence of 30-day COVID-19-related symptoms following

SARS-CoV-2 infection among individuals with lower income levels. However, several population-based studies, of smaller sample sizes, found no significant association between PASC and SE factors such as income or education levels^{44–46}.

Although a SE gradient has been observed in COVID-19 hospitalization, established as one of the strongest predictors of PASC⁴⁷, long-term follow-up studies on SE inequalities in post-acute complications following COVID-19 hospitalization are scarce. Huang et al.¹⁶ did not identify any significant association between education level and diffusion impairment or percentage change in CT score among COVID-19 patients discharged from the hospital. Unexpectedly, a study conducted in China showed higher odds of lung diffusion impairment at two years among COVID-19 hospitalized survivors with higher levels of education, compared to those with lower levels of education³¹. While we did not identify any association between education and post-acute CV complications, our results indicate a 1.48 times higher odds of post-acute pulmonary complications among severe COVID-19 patients with low income, compared to those with high income. However, among critical COVID-19 patients, post-acute pulmonary complications show no SE patterns.

Similar unclear social patterns are observed in international studies on racial and ethnic disparities in PASC, with some research reporting a lower risk of PASC following infection among Black or Asian individuals compared to White individuals^{39,40}, and some showing no significant association between ethnic minority status and PASC^{44,48}. In our study, compared to individuals born in Belgium, individuals with a migration background presented lower odds of developing post-acute CV complications following severe COVID-19 hospitalization. As the study outcomes were based on drug prescriptions, these results may be explained by the lower healthcare access and utilization among migrants in Europe, which is often influenced by factors such as language and communication barriers, lack of insurance, lower socioeconomic status (SES), limited GP consultation, and high out-of-pocket healthcare costs^{49,50}.

Despite scarce and mixed findings regarding SE patterns of post-acute health complications, especially among COVID-19 hospitalized patients, factors such as being a female, older age, smoking, and preexisting comorbidities (i.e., high BMI, anxiety/depression, chronic obstructive pulmonary disease, diabetes, immunosuppression, and ischemic heart disease) have been associated with a higher likelihood of developing PASC in a meta-analysis of 41 studies conducted by Tsampasian et al.⁴⁷. Preexisting comorbidities were identified in our results as the strongest risk factor for post-acute CV complications following severe COVID-19 hospitalization. Older age was identified in our study as increasing the odds of developing post-acute CV complications among severe COVID-19 patients. We did not identify any gender disparities in the development of post-acute organ complications, similarly to other studies^{13,43,45}. Smoking rates are generally higher in SE disadvantaged groups, and smoking-related social health inequalities are widening in many countries⁵¹. Although we could not adjust for smoking as a confounder in our model, it may have contributed to the higher risk of post-acute pulmonary complications observed in our study among severe COVID-19 patients with low income compared to those with high income.

Although clinical factors, such as disease severity and preexisting health conditions, seem to play a dominant role in long-term outcomes following COVID-19 hospitalization, SE inequalities in those outcomes remain present. SE disparities in PASC can be attributed to several factors⁵²: (1) SE inequalities in preexisting health conditions, shown to be a strong risk factor for PASC⁴⁷; (2) SE inequalities in COVID-19 infection and disease severity leading to an increased risk of PASC, as well as SE inequalities in vaccination, which reduces the risk of PASC^{47,53–55}; and (3) lower access to and quality of healthcare among deprived SE groups⁵⁶.

Our study has several limitations. First, to isolate the effect of COVID-19 on post-acute organ complications, we limited our study population to hospitalized patients with and without COVID-19. While this approach allowed us to control for the effect of hospitalization itself on the development of post-acute organ complications, it may introduce selection bias in the interpretation of both COVID-19 outcomes and social inequalities, potentially leading to paradoxical findings⁵⁷. For example, individuals with higher SES may only be hospitalized when severely ill, whereas those with lower SES might be hospitalized even with moderate illness. As a result, hospitalized patients with higher SES could appear sicker on average, creating an observed “survival advantage” for patients with lower SES⁵⁷. However, we controlled for baseline health characteristics and applied overlap weighting to achieve covariate balance, strengthening confidence in our findings. Nonetheless, because our analysis included only hospitalized patients, our conclusions regarding social inequalities in post-acute organ complications are limited to this subgroup and may not reflect broader disparities among all COVID-19 infections. Second, we lacked data on key mediators of SE disparities in COVID-19 critical outcomes, such as the type of occupation, the adoption of preventive measures (e.g., mask wearing, vaccination) and healthcare access, which would have better captured underlying pathways of SE inequalities in post-acute organ complications. Future research on the population in Belgium should consider the roles of these factors in the causal pathway between SE position and PASC to better capture its SE patterns. Third, the CHS database covers about 50% of hospitalized COVID-19 cases in Belgium, likely leading to an underestimation of our results, as some non-COVID-19 hospitalized controls could have been COVID-19 cases not registered in the CHS database. Fourth, the IMA pseudopathology groups, based on medication consumption, did not allow for identifying specific post-acute complications. As a result, individuals with preexisting conditions at baseline were excluded from our analyses. Moreover, while the IMA pseudopathology groups provide an acceptable proxy for baseline chronic diseases, these indicators have been shown to have a high rate of false negatives, resulting in low sensitivity. This underestimation may stem from two main factors: the stringent case definition (≥ 90 DDD per year) and the fact that some medications lack specificity to a single pseudopathology. To improve accuracy in estimating the prevalence of pseudopathologies, IMA data should ideally be supplemented with clinical or diagnostic data⁵⁸. Additionally, IMA lacks data on several well-documented PASC commonly observed in hospitalized patients, such as fatigue, brain fog, and depression¹³. As a result, the analyses are restricted to broader organ-related complications, preventing a comprehensive assessment of post-acute health outcomes following severe COVID-19. Fifth, because each pseudopathology group provided by IMA included only a very small number of severe and especially critical COVID-19 patients, we were not able to investigate the SE patterns of post-acute renal and liver complications, and we had to merge a set of comorbidities into one single variable to allow estimates to converge properly. Future studies with larger sample sizes are needed to explore the SE patterns of post-acute renal and liver complications, as well as to identify the impact of comorbidities on PASC following COVID-19 hospitalization in more detail. Finally, our study period was primarily influenced by the Alpha variant. Future research should therefore consider the impact of other variants, vaccination (which began in Belgium on 28 December 2020, near the end of our study period), and reinfection on PASC.

In conclusion, our study identified significant risks of one-year post-acute cardiovascular and pulmonary complications following COVID-19 hospitalization, increasing with disease severity, among patients without preexisting health conditions in these organ systems at baseline. Having underlying health conditions at baseline was the strongest risk factor for the development of post-acute cardiovascular complications among COVID-19 hospitalized patients. While no

socioeconomic patterns were observed for post-acute cardiovascular complications, post-acute pulmonary complications were significantly more frequent among severe COVID-19 patients with lower income levels. These findings highlight the need for long-term follow-up care for patients discharged after COVID-19 hospitalization, particularly those who experienced critical illness and have underlying health conditions, while also considering socioeconomic inequalities.

Methods

Study design

This study relies on data from the HELICON project, a population data linkage set up by Sciensano, the National Institute for Health in Belgium, to study the long-term health impact of the COVID-19 crisis in Belgium⁵⁹. The HELICON project contains selected variables from three national health and social registries linked at the individual level using the pseudonymized Belgian social security number.

To build the HELICON cohort, Statistics Belgium (Statbel) provided SD and SE data for a 10% random sample of the adult population legally residing in Belgium as of 1 January 2020. Next, a link was made with the Clinical Hospital Survey (CHS) database, containing patient-level data on approximately 50% of the patients admitted with a confirmed COVID-19 diagnosis across all 103 hospitals in Belgium as of 1 July 2020. Statbel then completed the sample by adding SD and SE data for patients registered in the CHS database but not included in the initial 10% random sample. Finally, data from the InterMutualistic Agency (IMA) were linked for both the random population sample and the hospitalized patients. IMA provided two datasets: one including information on reimbursed healthcare services and medications dispensed in public pharmacies over a year, and one recording all hospitalization stays per patient. As the health insurance system is compulsory in Belgium, IMA covers more than 99% of the total population⁶⁰. Within the HELICON project, IMA datasets were available for the reference years 2020 and 2021. The protocol of the HELICON population data linkage has been published elsewhere⁵⁹.

Study population and study period

Figure 1 shows the flowchart of the study population within the HELICON project. Between 1 July 2020 (marking the start of COVID-19 hospital case identification) and 31 December 2020, a total of 108,655 individuals were hospitalized with a confirmed COVID-19 diagnosis, as recorded in the CHS database. Based on IMA data, individuals hospitalized during the same period but not recorded in the CHS database were considered as controls ($N = 64,535$). This time frame was selected to enable the assessment of one-year post-acute organ complications using IMA data for the reference year 2021. Specifically, we selected hospitalized individuals without COVID-19 as the control group to isolate the effect of COVID-19 itself on the development of post-acute organ complications, independent of the impact of hospitalization. Selecting community controls from Statbel could have resulted in attributing post-acute complications to the hospitalization itself, rather than isolating the specific effects of COVID-19 on our outcomes of interest.

Among patients hospitalized with a confirmed COVID-19 diagnosis, we excluded those with missing week and year of admission, those not tested for COVID-19 symptoms (e.g., systematic screening), those readmitted or transferred, those who died in the hospital, those whose diagnosis was not confirmed by a positive PCR or antigen test, those discharged the same day as admission, and those with missing values for the admission to the intensive care unit (ICU) and the onset of acute respiratory distress syndrome (ARDS). This resulted in 8024 unique hospitalized COVID-19 survivors meeting the inclusion and exclusion criteria.

Next, both COVID-19 hospitalized patients and non-COVID-19 hospitalized controls were linked to Statbel data to obtain SD and SE information for the reference year 2020. In addition, only COVID-19

hospitalized patients and controls who were still alive in 2021 were included to avoid misclassifying death as the absence of post-acute organ complications in 2021 following hospitalization in 2020.

To accurately assess the onset of post-acute organ complications within one year following hospitalization, we created subcohorts of both COVID-19 hospitalized patients and non-COVID-19 hospitalized controls without preexisting CV, pulmonary, renal, or liver conditions at baseline, respectively, using IMA data for the reference year 2020.

Given the association between disease severity and the onset of post-acute organ complications, each subcohort of COVID-19 hospitalized patients was stratified into:

- Critical COVID-19 patients: defined as COVID-19 hospitalized patients admitted to the ICU and/or with ARDS;
- Severe COVID-19 patients: defined as COVID-19 hospitalized patients not admitted to the ICU and without ARDS.

Outcomes

The study outcomes consisted of the onset of post-acute CV, pulmonary, renal, and liver complications, as recorded in the IMA database for 2021, among COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls. For each organ system analyzed, hospitalized patients with preexisting conditions affecting that specific system at baseline (as recorded in the IMA database for 2020) were excluded, while preexisting conditions in other organ systems were retained.

The post-acute organ complications considered in this study were based on pseudopathology groups defined by the National Institute for Health and Disability Insurance (RIZIV-INAMI). These groups are created on the basis of Anatomical Therapeutic Chemical (ATC) codes established by the WHO⁶¹. An individual is assigned to a pseudopathology group when the total number of defined daily doses (estimated quantity of active ingredient for a 70 kg adult per day of a drug prescribed for its main action) for all ATC codes in this group is greater than or equal to 90.

A detailed description of the pseudopathologies, along with the related ATC codes and substances used to define the four post-acute organ complications considered in this study is available in Supplementary Data 10. The methodology used to build the pseudopathology groups has been described by IMA elsewhere⁶².

Covariates

SD covariates included age group, sex, region of residence, migration background, and living situation. The Region of residence was distinguished between the Brussels Capital Region, the Flemish Region, and the Walloon Region and was included as a confounder in the analyses because of the difference in hospitalization coverage by regions in the CHS database, as well as the known association between SE status and Regions in Belgium^{63,64}. Migration background was based on a combination of the first nationality and the parents' country of origin and distinguished between "Born in Belgium", "Second-generation migrants", "First-generation European (EU) migrants", and "First-generation non-EU migrants". The living situation gave a partial picture of the social environment and distinguished between "Couples with children", "Couples without children", "Single parents", "Single-persons household", "Collective households" (e.g., nursing homes, prisons), and "Other" (e.g., adult children still living with their parents, other co-residents).

SE covariates included education and income levels. Education level was classified into five categories based on the International Standard Classification of Education (ISCED): "Primary or less" (ISCED 0 to ISCED 1), "Lower secondary" (ISCED 2), "Upper secondary" (ISCED 3 to ISCED 4), "Higher education" (ISCED 5 to ISCED 8), and "Missing". Income data was available as the deciles of the yearly net taxable income per person and grouped as: "Low income" (deciles 1 to 4),

“Middle income” (deciles 5 to 7), “High income” (deciles 8 to 10), and “Missing”.

Health-related covariates consisted of comorbidities at baseline, defined based on the pseudopathology groups available in the IMA database for the reference year 2020, as described above. The following comorbidities were considered: “Cardiovascular comorbidities”, “Pulmonary comorbidities”, “Renal comorbidities”, “Liver comorbidities”, and “Other comorbidities”. The latter category included the following pseudopathologies: mucoviscidosis, morbid obesity, organ transplantation, immunodeficiency (HIV and no HIV), neurological disorders (e.g., Parkinson, multiple sclerosis, Alzheimer), psychosis, diabetes, and inflammatory and autoimmune disorders (e.g., rheumatoid arthritis, Crohn’s disease).

A description of the data holders and time frame for all SD, SE, and health-related variables considered in this study is available in Supplementary Table 2.

Exposures

In the first objective, which aimed to determine whether COVID-19 hospitalization led to the onset of post-acute organ complications compared to non-COVID-19 hospitalization, the exposure was defined as hospitalization with a confirmed COVID-19 diagnosis based on a positive PCR or antigen test between 1 July 2020 and 31 December 2020 in Belgium.

In the second objective, aiming to identify SE inequalities in the development of post-acute organ complications among COVID-19 patients hospitalized during the same period, education and income

were considered as the SE exposures of interest. In both objectives, COVID-19 hospitalization was stratified between severe and critical COVID-19 hospitalization, as defined above.

Figure 3 presents a schematic overview of the outcome, exposure, covariates, and study population according to each objective considered in this study.

Statistical analyses

In the first objective, the aim was to determine whether COVID-19 hospitalization led to the onset of post-acute organ complications compared to non-COVID-19 hospitalization. Within this objective, first, severe and critical COVID-19 hospitalized patients, along with non-COVID-19 hospitalized controls, were described with the frequency rates and percentages for categorical variables. Second, propensity scores (PS) with overlap weights (OWs) were applied to address imbalances in the characteristics between exposed and unexposed individuals. For each individual, the PS model was estimated as follows:

$$\text{logit}P(A, |, U_{SD}, U_{SE}, U_{Health}) = \beta_0 + \beta_{U_{SD}} U_{SD} + \beta_{U_{SE}} U_{SE} + \beta_{U_{Health}} U_{Health} \quad (1)$$

where A is COVID-19 hospitalization, and U_{SD} , U_{SE} and U_{Health} denote the SD, SE, and health-related covariates, respectively.

In our study population, the distribution of PS among COVID-19 hospitalized patients and non-COVID-19 hospitalized controls showed evidence of extreme values across all patient subcohorts (see

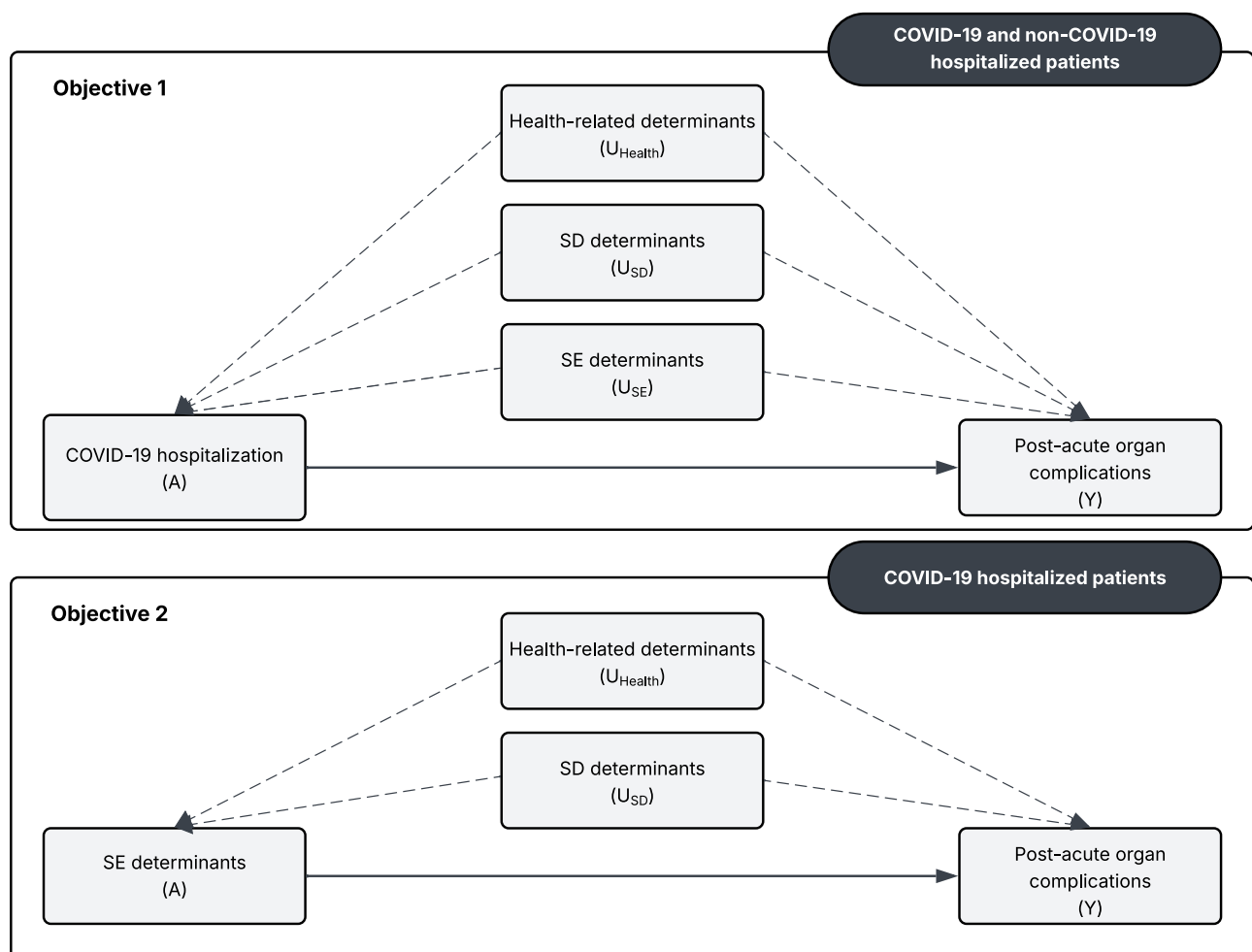


Fig. 3 | Schematic overview of the outcome (Y), exposure (A), covariates (U), and study population considered in each objective. Abbreviation: SD socio-demographic, SE socioeconomic.

Supplementary Figs. 1 to 4 for patient subcohorts without CV, pulmonary, renal, and liver diseases at baseline, respectively).

This is a common limitation of balancing weight methods based on PS, as extreme values can inflate variance and bias estimates when using the standard inverse probability of treatment weighting method^{65–67}. To address this issue, OWs were applied, which are known to provide an accurate estimate of the exposure effect and its variance even in the presence of extreme PS values^{65–67}. OW, denoted ow_i , are defined as $(1 - PS)$ for exposed individuals and PS for unexposed individuals. This approach down-weights observations in the tails of the PS distribution and assigns greater weight to those in regions of overlap, creating a weighted pseudo-population where confounders are balanced^{65,66}. The effectiveness of OW in achieving covariate balance was evaluated using the weighted standardized mean difference (SMD)⁶⁸. Importantly, OWs focus the causal estimates on the overlap population, meaning the results primarily apply to individuals who are sufficiently similar to plausibly belong to either treatment group, rather than to the entire study population⁶⁹.

Overlap PS-weighted odds ratios (ORs) and 95% confidence intervals (CIs) were then estimated using multivariable binary logistic regression models. The outcome model was defined as follows:

$$\text{logit } P(Y_i|A) = \beta_{0i} + \beta_{Ai}A, i \in \{CV, \text{pulmonary}, \text{renal}, \text{liver}\} \quad (2)$$

where Y_i denotes the occurrence of a specific post-acute organ complication ($i=CV$, pulmonary, renal, or liver), and each observation was weighted by ow_i .

To address potential PS model misspecification, we conducted an adjusted analysis on the weighted sample (Eq. (3)), which literature suggests can enhance robustness beyond weighting alone as long as either the outcome model or the PS model was correctly specified⁷⁰.

$$\begin{aligned} \text{logit } P(Y_i|A, U_{SD}, U_{SE}, U_{Health}) = & \beta_{0i} + \beta_{Ai}A + \beta_{U_{SD}i}U_{SD} + \beta_{U_{SE}i}U_{SE} \\ & + \beta_{U_{Health}i}U_{Health}, i \in \{CV, \text{pulmonary}, \text{renal}, \text{liver}\} \end{aligned} \quad (3)$$

with each observation weighted by ow_i . However, this approach does not guarantee doubly robust estimation, which requires specific methods, such as fitting a canonical link GLM fit via maximum likelihood with weighted score equations, followed by regression standardization⁷¹. Variance estimation was performed using robust sandwich variance estimates, shown to produce valid estimates even in the case of model misspecification⁷².

Third, overlap PS-weighted excess burdens of one-year post-acute organ complications per 1000 patients due to COVID-19 hospitalization were computed by estimating the difference in incidence rates of post-acute organ complications between COVID-19 hospitalized patients and non-COVID-19 hospitalized controls.

All analyses described above were applied separately to severe COVID-19 patients and their controls, and to critical COVID-19 patients and their controls.

The second objective aimed to identify SE patterns in the development of post-acute CV and pulmonary complications among COVID-19 hospitalized patients. Post-acute renal and liver complications were excluded due to insufficient event rates in certain covariate and exposure categories, preventing reliable convergence of estimates when limiting the analysis to COVID-19 patients. Adjusted odds ratios (aORs) and 95% CIs were estimated using multivariable binomial logistic regression models to assess the association between post-acute CV and pulmonary complications and the SE exposures of interest (i.e., education, income) among COVID-19 hospitalized patients, respectively. The analyses described above were stratified between severe and critical COVID-19 patients.

As sensitivity analyses, we aimed to identify whether the impact of SE determinants on the development of post-acute CV and pulmonary

complications differed among COVID-19 hospitalized patients compared to non-COVID-19 hospitalized controls, respectively. First, COVID-19 patients and controls were stratified and aORs and 95% CIs were estimated using multivariable binary logistic regression models. Next, multivariable logistic regression models were applied to the full study population, including both COVID-19 patients and controls. In these models, an interaction term between COVID-19 hospitalization and the SD, SE, and health-related covariates was introduced. This allowed to extract the p-value for interaction testing H_0 that the effect of covariates on the outcomes is similar in both groups. These regression models with an interaction term were applied separately to severe COVID-19 patients and controls, as well as to critical COVID-19 patients and controls.

Analytical approach and tools

All analyses were performed in R (version 4.3.2)⁷³. In our analyses, p-values were derived from two-sided Wald tests. A significance level of $\alpha = 0.05$ was used. The package “tableone” was used to calculate the SMD⁶⁸. The package “sandwich” was used to compute robust variance estimates⁷⁴.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The pseudonymized individual health data used in this study have been deposited in the Healthdata.be database: <https://healthdata.sciensano.be/>. The pseudonymized individual health data are available under restricted access due to General Data Protection Regulation (GDPR) legislation in Belgium; access can be obtained by submitting a request to the Information Security Committee (ISC) of Healthdata.be. The raw pseudonymized individual health data are protected and are not publicly available due to data privacy laws.

References

1. Sciensano. Belgium COVID-19 Dashboard. <http://datastudio.google.com/reporting/c14a5cfc-cab7-4812848c0369173148ab/page/tpRKB?feature=opengraph> (2025).
2. Xu, Z., Li, S., Tian, S., Li, H. & Kong, L. Full spectrum of COVID-19 severity still being depicted. *Lancet* **395**, 947–948 (2020).
3. Centers for Disease Control and Prevention. Symptoms of COVID-19 <https://www.cdc.gov/covid/signssymptoms/index.html> (2024).
4. Lamers, M. M. & Haagmans, B. L. SARS-CoV-2 pathogenesis. *Nat. Rev. Microbiol.* **20**, 270–284 (2022).
5. Berlin, D. A., Gulick, R. M. & Martinez, F. J. Severe Covid-19. *N. Engl. J. Med.* **383**, 2451–2460 (2020).
6. Qiu, H., Li, J., Li, J., Li, H. & Xin, Y. COVID-19 and acute cardiac injury: clinical manifestations, biomarkers, mechanisms, diagnosis, and treatment. *Curr. Cardiol. Rep.* **25**, 817–829 (2023).
7. Tazarghi, A. et al. Liver injury in COVID-19: an insight into pathobiology and roles of risk factors. *Viol. J.* **21**, 65 (2024).
8. Sabaghian, T. et al. COVID-19 and acute kidney injury: a systematic review. *Front. Med.* **9**, 705908 (2022).
9. Castanares-Zapatero, D. et al. Pathophysiology and mechanism of long COVID: a comprehensive review. *Ann. Med.* **54**, 1473–1487 (2022).
10. Shirbhate, E. et al. Understanding the role of ACE-2 receptor in the pathogenesis of COVID-19 disease: a potential approach for therapeutic intervention. *Pharmacol. Rep.* **73**, 1539–1550 (2021).
11. World Health Organization. Post COVID-19 condition (Long COVID). <https://www.who.int/europe/news-room/factsheets/item/post-covid-19-condition> (2022).

12. Chen, C. et al. Global prevalence of post-coronavirus disease 2019 (covid-19) condition or long covid: a meta-analysis and systematic review. *J. Infect. Dis.* **226**, 1593–1607 (2022).
13. O'Mahoney, L. L. et al. The prevalence and long-term health effects of Long Covid among hospitalised and non-hospitalised populations: a systematic review and meta-analysis. *eClinicalMedicine* **55**, 101762 (2023).
14. Xie, Y., Xu, E., Bowe, B. & Al-Aly, Z. Long-term cardiovascular outcomes of COVID-19. *Nat. Med.* <https://doi.org/10.1038/s41591-022-01689-3> (2022).
15. Lim, J. T. et al. Long-term cardiovascular, cerebrovascular, and other thrombotic complications in COVID-19 survivors: a retrospective cohort study. *Clin. Infect. Dis.* **78**, 70–79 (2024).
16. Huang, C. et al. 6-month consequences of COVID-19 in patients discharged from hospital: a cohort study. *Lancet* **401**, e21–e33 (2023).
17. Wong, Y. J. et al. A systematic review and meta-analysis of the COVID-19-associated liver injury. *Ann. Hepatol.* **19**, 627–634 (2020).
18. Silver, S. A. et al. The prevalence of acute kidney injury in patients hospitalized with covid-19 infection: a systematic review and meta-analysis. *Kidney. Med.* **3**, 83–98.e1 (2021).
19. Statbel. Age. <https://statbel.fgov.be/fr/themes/census/population/age#panel-14>.
20. Sciensano. Belgian National Burden of Disease Study (BeBOD). <https://burden.sciensano.be/shiny/mortality/> (2023).
21. Bambra, C., Lynch, J. & Smith, K. E. *The Unequal Pandemic: COVID-19 and Health Inequalities*. (Policy Press, 2021).
22. Du, P. et al. A systematic review and meta-analysis of risk factors associated with severity and death in COVID-19 patients. *Can. J. Infect. Dis. Med. Microbiol.* **2021**, 6660930 (2021).
23. Gustafsson, P. E., Sebastian, M. S., Fonseca-Rodriguez, O. & Connolly, A.-M. F. Inequitable impact of infection: social gradients in severe COVID-19 outcomes among all confirmed SARS-CoV-2 cases during the first pandemic wave in Sweden. *J. Epidemiol. Community Health* **76**, 261–267 (2022).
24. Soares, R., de, C. M., Mattos, L. R. & Raposo, L. M. Risk factors for hospitalization and mortality due to COVID-19 in Espírito Santo State, Brazil. *Am. J. Trop. Med. Hyg.* **103**, 1184–1190 (2020).
25. Irizar, P. et al. Ethnic inequalities in COVID-19 infection, hospitalisation, intensive care admission, and death: a global systematic review and meta-analysis of over 200 million study participants. *eClinicalMedicine* **57**, 101877 (2023).
26. Cavillot, L. et al. Unravelling demographic and socioeconomic patterns of COVID-19 death and other causes of death: results of an individual-level analysis of exhaustive cause of death data in Belgium, 2020. *Arch. Public Health* **82**, 209 (2024).
27. Abel, Z. D. V., Roope, L. S. J., Duch, R. & Clarke, P. M. Access to healthcare services during the COVID-19 pandemic: a cross-sectional analysis of income and user-access across 16 economically diverse countries. *BMC Public Health* **24**, 2678 (2024).
28. Negreira-Caamaño, M. et al. Cardiovascular events after COVID-19 hospitalization: long-term follow-up. *Rev. Esp. Cardiol.* **75**, 100–102 (2022).
29. Ayoubkhani, D. et al. Post-COVID-19 syndrome in individuals admitted to hospital with COVID-19: retrospective cohort study. *BMJ* **372**, n693 (2021).
30. Han, X. et al. Six-month Follow-up Chest CT Findings after Severe COVID-19 Pneumonia. *Radiology* **299**, E177–E186 (2021).
31. Huang, L. et al. Health outcomes in people 2 years after surviving hospitalisation with COVID-19: a longitudinal cohort study. *Lancet Respir. Med.* **10**, 863–876 (2022).
32. Raman, B. et al. Multiorgan MRI findings after hospitalisation with COVID-19 in the UK (CMORE): a prospective, multicentre, observational cohort study. *Lancet Respir. Med.* **11**, 1003–1019 (2023).
33. Al-Aly, Z., Xie, Y. & Bowe, B. High-dimensional characterization of post-acute sequelae of COVID-19. *Nature* **594**, 259–264 (2021).
34. Fabrizi, F., Alfieri, C. M., Cerutti, R., Lunghi, G. & Messa, P. COVID-19 and acute kidney injury: a systematic review and meta-analysis. *Pathogens* **9**, 1052 (2020).
35. Tan, B. W. L. et al. Long-term kidney function recovery and mortality after COVID-19-associated acute kidney injury: an international multi-centre observational cohort study. *eClinicalMedicine* **55**, 101724 (2023).
36. Nugent, J. et al. Assessment of acute kidney injury and longitudinal kidney function after hospital discharge among patients with and without COVID-19. *JAMA Netw. Open* **4**, e211095 (2021).
37. Zhang, Y. et al. Liver impairment in COVID-19 patients: a retrospective analysis of 115 cases from a single centre in Wuhan city, China. *Liver Int.* **40**, 2095–2103 (2020).
38. Lammers, N., Beese, F., Hoebel, J., Poethko-Müller, C. & Wachtler, B. Social inequalities in long-term health effects after COVID-19—a scoping review. *Int. J. Public Health* **69**, 1606739 (2024).
39. Thompson, E. J. et al. Long COVID burden and risk factors in 10 UK longitudinal studies and electronic health records. *Nat. Commun.* **13**, 3528 (2022).
40. Whitaker, M. et al. Persistent COVID-19 symptoms in a community study of 606,434 people in England. *Nat. Commun.* **13**, 1957 (2022).
41. Subramanian, A. et al. Symptoms and risk factors for long COVID in non-hospitalized adults. *Nat. Med.* **28**, 1706–1714 (2022).
42. Shabnam, S. et al. Socioeconomic inequalities of Long COVID: a retrospective population-based cohort study in the United Kingdom. *J. R. Soc. Med.* **116**, 263–273 (2023).
43. Hirschtick, J. L. et al. Population-based estimates of post-acute sequelae of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (PASC) prevalence and characteristics. *Clin. Infect. Dis.* **73**, 2055–2064 (2021).
44. Wu, Q., Ailshire, J. A. & Crimmins, E. M. Long COVID and symptom trajectory in a representative sample of Americans in the first year of the pandemic. *Sci. Rep.* **12**, 11647 (2022).
45. Westerlind, E., Palstam, A., Sunnerhagen, K. S. & Persson, H. C. Patterns and predictors of sick leave after Covid-19 and long Covid in a national Swedish cohort. *BMC Public Health* **21**, 1023 (2021).
46. Ogungbe, O. et al. Cardiac postacute sequelae symptoms of SARS-CoV-2 in community-dwelling adults: cross-sectional study. *Open Heart* **9**, e002084 (2022).
47. Tsampasian, V. et al. Risk factors associated with post-COVID-19 condition: a systematic review and meta-analysis. *JAMA Intern. Med.* **183**, 566–580 (2023).
48. Tenforde, M. W. Symptom duration and risk factors for delayed return to usual health among outpatients with COVID-19 in a multistate health care systems network—United States, March–June 2020. *MMWR Morb. Mortal Wkly Rep.* **69**, 993–998 (2020).
49. Galanis, P. et al. Healthcare services access, use, and barriers among migrants in Europe: a systematic review. Preprint at <https://doi.org/10.1101/2022.02.24.22271449> (2022).
50. Lebano, A. et al. Migrants' and refugees' health status and health-care in Europe: a scoping literature review. *BMC Public Health* **20**, 1039 (2020).
51. Hitchman, S. C. et al. Socioeconomic Status and Smokers' Number of Smoking Friends: findings from the International Tobacco Control (ITC) Four Country Survey. *Drug Alcohol Depend.* **143**, 158–166 (2014).
52. Banerjee, A. Disparities by social determinants of health: links between long covid and cardiovascular disease. *Can. J. Cardiol.* **40**, 1123–1134 (2024).
53. Khanijahani, A., Iezadi, S., Gholipour, K., Azami-Aghdash, S. & Naghibi, D. A systematic review of racial/ethnic and socioeconomic disparities in COVID-19. *Int. J. Equity Health* **20**, 248 (2021).

54. Cavillot, L. et al. Sociodemographic and socioeconomic disparities in COVID-19 vaccine uptake in Belgium: a nationwide record linkage study. *J. Epidemiol. Community Health* **77**, jech-2023-220751 (2023).
55. Fernández-Martínez, N. F. et al. Socioeconomic differences in COVID-19 infection, hospitalisation and mortality in urban areas in a region in the South of Europe. *BMC Public Health* **22**, 2316 (2022).
56. Nunes, B. P., Thumé, E., Tomasi, E., Duro, S. M. S. & Facchini, L. A. Socioeconomic inequalities in the access to and quality of health care services. *Rev. Saude Publica* **48**, 968–976 (2014).
57. Banack, H. R. & Kaufman, J. S. The “obesity paradox” explained. *Epidemiology* **24**, 461–462 (2013).
58. Sciensano. Résultats de l'Enquête de Santé 2023-2024. <https://www.sciensano.be/fr/resultats-de-lenquete-de-sante-2023-2024> (2025).
59. De Pauw, R. et al. Social inequalities and long-term health impact of COVID-19 in Belgium: protocol of the HELICON population data linkage. *BMJ Open* **13**, e069355 (2023).
60. Gerkens, S. et al. Performance du système de santé belge: rapport 2024. Centre fédéral d'Expertise des Soins de Santé (KCE), KCE Reports 376B (2024).
61. Norwegian Institute of Public Health. ATCDDD - ATC/DDD Index. https://atcddd.fhi.no/atc_ddd_index/ (2024).
62. Agence Intercommunale. Échantillon Permanente Steekproef EPS R13 – FLAGS Release 20190201. https://imaaim.be/IMG/pdf/eps_r13_-_flags_release_20190201_nl_-_vs2.pdf (2023).
63. Statbel. Revenus fiscaux. <https://statbel.fgov.be/fr/themes/menages/revenus-fiscaux> (2023).
64. Statbel. Niveau d'instruction. <https://statbel.fgov.be/fr/themes/census/education/niveau-d-instruction> (2024).
65. Thomas, L. E., Li, F. & Pencina, M. J. Overlap Weighting: a propensity score method that mimics attributes of a randomized clinical trial. *JAMA* **323**, 2417–2418 (2020).
66. Li, F., Thomas, L. E. & Li, F. Addressing extreme propensity scores via the overlap weights. *Am. J. Epidemiol.* **188**, 250–257 (2019).
67. Zajichek, A. M. & Grunkemeier, G. L. Propensity scores used as overlap weights provide exact covariate balance. *Eur. J. Cardio-Thorac. Surg.* **66**, ezae318 (2024).
68. Yoshida, K. Standardized mean difference. <https://cran.r-project.org/web/packages/tableone/vignettes/smd.html> (2022).
69. Vansteelandt, S. & Daniel, R. M. On regression adjustment for the propensity score. *Stat. Med.* **33**, 4053–4072 (2014).
70. Funk, M. J. et al. Doubly robust estimation of causal effects. *Am. J. Epidemiol.* **173**, 761–767 (2011).
71. Gabriel, E. E. et al. Inverse probability of treatment weighting with generalized linear outcome models for doubly robust estimation. *Stat. Med.* **43**, 534–547 (2024).
72. Shu, D., Young, J. G., Toh, S. & Wang, R. Variance estimation in inverse probability weighted Cox models. *Biometrics* **77**, 1101–1117 (2021).
73. R Core Team. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing. <https://www.R-project.org/> (2020).
74. Zeileis, A., Lumley, T., Graham, N. & Koell, S. sandwich: Robust Covariance Matrix Estimators. <https://cran.r-project.org/web/packages/sandwich/sandwich.pdf> (2025).

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Author contributions

L.C. reviewed the literature; L.C., N.S., L.V.d.B., D.D.S., K.V., S.G., R.D.P., and B.D. Conceived the study; L.C., N.S., L.V.d.B., R.D.P. and B.D. selected the study population; L.C., N.S., L.V.d.B., R.D.P. and B.D. designed the statistical methodology; L.C. conducted the statistical analyses; L.C., N.S., and B.D. interpret the findings; L.C. wrote the first draft of the paper. L.C., L.V.D.B., J.G., E.B., J.A.F.L., P.H., P.S., D.D.S., K.V., S.G., R.D.P., N.S. and B.D. Revised the text. L.C., L.V.D.B., J.G., E.B., J.A.F.L., P.H., P.S., D.D.S., K.V., S.G., R.D.P., N.S. and B.D. approved the final version of this manuscript and accepted responsibility for its submission for publication.

Competing interests

The authors declare no competing interests.

Additional information

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