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Cognitive and Functional Impairment After Mild-to-Moderate COVID-19 in Older Adults: A Longitudinal Study with Pre-Pandemic and Non-Infected Comparisons

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Abstract

Background: To evaluate the long-term association of COVID-19 with cognition and functionality in participants aged ≥65 years who underwent at least one pre-COVID-19 cognitive assessment.

Methods: In this longitudinal retrospective study, 62 participants with at least one pre-pandemic cognitive assessment underwent two additional in-person assessments (t1 and t2), divided into COVID-19 (n=31) and non-COVID-19 (n=31) groups.

Results: Baseline characteristics, including age, sex, and education level, were comparable between groups. Most COVID-19 cases were mild (93%), and the mean time between COVID-19 and t2 was 32 months. The COVID-19 group showed a 4.2-fold higher risk of dementia at t2 compared to the pre-COVID assessment (95% CI [1.31, 14.0], p=0.02) and a 3.4-fold increase in Katz score (p<0.01). They also had a higher risk of abnormal Clinical Dementia Rating Personal Care (78%, p=0.03) and Clinical Dementia Rating Orientation risk (89%, p=0.03). At t2, the non-COVID-19 group had a 42% lower risk of abnormal Mini-Mental State Examination (MMSE) scores (p=0.02) and showed improvements in the temporal (13%, p<0.01) and spatial (16%, p<0.01) orientation sub-items of the MMSE (p<0.01). No associations were found with clinical or demographic variables, oxygen use, or vaccination number.

Conclusions: Older adults with a history of COVID-19 experienced long-term cognitive and functional decline compared to non-infected peers.

Keywords: COVID-19; older; cognitive dysfunction; mental status and dementia testing; functionality

1. BACKGROUND

The COVID-19 pandemic has disproportionately affected older adults. In the first three years of the pandemic, approximately 75% of COVID-19 deaths occurred in people aged 65 years or older [1]. Older age has emerged as one of the main risk factors for long-term cognitive dysfunction and loss of functional capacity after COVID-19 [2,3]. However, older adults are a highly heterogeneous population, varying widely in frailty, multimorbidity, and baseline health status, all of which may influence both susceptibility to severe COVID-19

and the trajectory of recovery in this age group. Because adults older than 65 years already have the highest risk of cognitive decline and dementia [4,5], understanding the impact of the COVID-19 pandemic on cognition in this age group is essential.

The term “post-COVID-19 cognitive dysfunction” describes the association between COVID-19 and negative cognitive outcomes [6], with pooled prevalence estimates of cognitive impairment ranging from 20-52% within 12 weeks of infection to 21-65% beyond 12 weeks [7]. However, more recent work indicates that post-COVID-19 neurocognitive profiles are diverse rather than uniform [8]. A growing body of evidence points to inflammatory and vascular mechanisms — including systemic inflammation, blood-brain barrier dysfunction, and neurovascular unit dysfunction [9–12] — alongside neuroimaging findings of structural and metabolic brain alterations [13–20], and genetic and biomarker evidence suggesting that SARS-CoV-2 infection may accelerate or unmask neurodegenerative processes resembling Alzheimer’s disease [21–26]. Despite these findings, post-COVID-19 cognitive dysfunction is not yet fully understood [6]; the underlying mechanisms and recovery trajectories remain unclear [27], and early literature is highly heterogeneous in design and methodology, limiting the possibility of meta-analyses and robust conclusions [28–31].

Therefore, the aim of this study was to evaluate the long-term association of COVID-19 with cognitive function and functional capacity in adults aged 65 years and older. Specifically, we compared post-pandemic and pre-pandemic performance in the same individuals using common screening tests in geriatric practice.

2. MATERIALS AND METHODS

2.1 Study design and participants

We conducted a cohort study of older adults followed at the Geriatrics Service of Fluminense Federal University. The inclusion criteria were an age of 65 years or older and available data for at least one cognitive assessment before the COVID-19 pandemic.

We excluded individuals with a prior diagnosis of dementia based on the criteria of the Diagnostic and Statistical Manual of Mental Disorders (5th ed., text revision; DSM-5-TR) [32] who were classified as having moderate or severe dementia according to the Clinical Dementia Rating (CDR) scale (CDR 0.5 = no dementia; CDR 1 = mild dementia; CDR 2 = moderate dementia; CDR 3 = severe dementia) at the time of the pre-pandemic assessment. We also excluded individuals with clinical conditions that prevented an adequate cognitive assessment during in-person evaluations.

Participants were selected by convenience sampling. All eligible participants under follow-up were invited to participate. The Research Ethics Committee of the Fluminense Federal University Faculty of Medicine approved the study (CAAE 56346822.2.0000.5243). All participants provided written informed consent before inclusion.

2.2 Study procedures

Pre-pandemic cognitive and functional data, collected retrospectively between January 2018 and February 2020, were extracted from medical records, along with sociodemographic, clinical, and COVID-19-related information and recent laboratory results. Assessments conducted before 2018 were not considered.

In-person assessments were conducted between February 2023 and April 2024. The first assessment (t1) took place from February to September 2023, and the second (t2) from October 2023 to April 2024. For each participant, the interval between t1 and t2 was at least six months. All assessments were performed by physicians trained in geriatrics. At t1, participants underwent a series of cognitive and functional tests and completed screening tests for depression and anxiety. All participants were invited for a second assessment (t2), at least six months after t1, using the same screening tests.

Participants were divided into two groups. The COVID-19 group included older adults with a confirmed SARS-CoV-2 infection (RT-PCR or antigen test) between the date of the pre-pandemic cognitive assessment and the in-person assessments. The non-COVID-19 group included those who reported no history of SARS-CoV-2 infection and had no positive test during this period.

2.3 Outcomes and assessments

The primary outcomes were cognitive impairment and functional decline. Cognition was assessed using the MMSE and CDR. Functional status was assessed using the Katz Index of Independence in Activities of Daily Living and the Lawton Instrumental Activities of Daily Living Scale. The MMSE assesses cognitive function through simple questions and tasks that explore orientation (in time and space), attention, memory, language, and visuoconstructive abilities, producing a total score that indicates the degree of cognitive impairment. This study used the Brazilian version of the MMSE proposed by Brucki et al. [33], developed to standardise administration of the instrument in Brazil; classification as normal or abnormal followed the schooling-adjusted cut-offs from that reference: 20 points

for illiterate participants, 25 for one to four years of schooling, 26 for five to eight years, 28 for nine to eleven years, and 29 for more than eleven years. The CDR combines information from the patient and an informant to rate the degree of impairment across six domains (memory, orientation, judgment and problem solving, community affairs, home and hobbies, and personal care) to classify the degree of dementia. The Katz scale measures functional status in basic activities of daily living, such as bathing, dressing, using the toilet, mobility, continence, and eating, whereas the Lawton scale evaluates more complex activities, such as using the telephone, shopping, preparing meals, managing finances, and handling medications, indicating the degree of independence in daily life.

The patient interview for CDR classification was unstructured, and the CDR Global Score was obtained from the website of the National Alzheimer's Coordinating Center using the CDR® Dementia Staging Instrument calculator (available free of charge at https://naccdata.org/data-collection/tools-calculators/cdr). The Geriatric Depression Scale and the Geriatric Anxiety Inventory were used to screen for depression and anxiety, respectively. The total scores and final classification regarding normality/abnormality for all tests were calculated according to previous research reporting the validation and cross-cultural adaptation of these tests for the Brazilian geriatric population, in which the scores and results have been adjusted for schooling level [33,34,35,36,37,38,39,40].

2.4 Statistical analysis

Categorical variables are presented as absolute counts and percentages. Continuous variables are presented as the mean ± standard deviation when normally distributed, or the median and interquartile range otherwise. We used the Kolmogorov-Smirnov test to

evaluate data distribution. To compare groups (COVID-19 vs. non-COVID-19) and time points (pre-pandemic, t1, t2) for quantitative MMSE, Katz, and Lawton scores, we used repeated-measures regression models with a negative binomial distribution and a log link. The estimated effect of COVID-19 on mean test scores was derived from these models. For binary outcomes (normal vs. abnormal), MMSE and CDR results were dichotomized, and the relative risk of abnormality was estimated using Poisson regression models with robust variance. Analysis of covariance was used to compare the absolute change in MMSE scores between time points across groups. Poisson regression with robust variance was also used to compare the proportion of participants whose MMSE and CDR total and subscale scores deteriorated or remained abnormal over time.

In addition to the main analyses, we conducted a sensitivity analysis excluding participants in the COVID-19 group who had required oxygen therapy, to explore whether the findings were biased by more severe cases. This sensitivity analysis yielded materially unchanged estimates, indicating that the observed associations were not driven by the most severe COVID-19 cases. All models were adjusted for age, sex, and income. Given the statistically significant difference in the time interval between pre-pandemic and t2 assessments between groups, the time elapsed between these assessments (in months) was additionally included as a covariate in the regression models. This adjustment was imposed to account for the potential confounding effects of differences in follow-up duration and aging exposure between COVID-19 and non-COVID-19 groups. Sensitivity analyses including this variable confirmed the robustness of the observed associations. We considered $p < 0.05$ to be statistically significant for all comparisons. Graphs were created in RStudio (version 2023.12.0.369) and statistical analyses were performed with SAS 9.4.

3. RESULTS

A total of 562 participants were evaluated. Among these, 228 participants who did not have a pre-pandemic cognitive assessment and 197 with incomplete medical record data were excluded. Another 66 participants were excluded because they had moderate to severe dementia, and nine participants were lost to follow-up during the study. Ultimately, 62 older adults were included in the study (31 each in COVID-19 and non-COVID-19 groups) (Figure 1).

The general characteristics of the study population are summarized in Table 1. The sample had a mean age of 78.8 years (±7.6), 50 participants (80.6%) were women, and the mean schooling level was 6.5 years (±5). The non-COVID-19 group had a mean income of 2.5 minimum wages (1 minimum wage corresponds to 260 pounds), whereas the COVID-19 group had a mean income of 1.8, with no significant difference between groups. The most common comorbidities were hypertension (83%), dyslipidemia (64%), heart disease (25%), diabetes (24%), and hypothyroidism (22%). The mean number of regularly used medications was 6.7 (±2.6), and the mean number of central nervous system medications was 0.2.

The mean values of laboratory tests, including those directly related to cognition, were within normal limits, as shown in Table 1. Cognitive deficits were not associated with any clinical or demographic variables, oxygen use, or number of vaccines administered at the time of infection. Regarding the characteristics of infection in the COVID-19 group, most participants were not hospitalized (81%) or did not require oxygen (87%). Six participants (19%) who were hospitalized were not placed in intensive care units, and four (13%) who

required oxygen therapy received it non-invasively via a nasal catheter or face mask without positive pressure. At the time of infection, 45% of the participants were not vaccinated, whereas 51% had received two vaccine doses. At t1, 79% of participants had received at least four vaccine doses. Only one participant in the COVID-19 group reported mild reinfection. The mean interval between COVID-19 diagnosis and t2 was 32 months. The mean time between the pre-pandemic assessment and the t2 assessment was significantly shorter in the COVID-19 group (46 months) than in the non-COVID-19 group (58 months; $p < 0.01$).

3.1 Current cognitive and functional impairment in participants with COVID-19

MMSE and CDR were used to determine cognitive status, and the Lawton and Katz scales were used to determine the current functional status of participants (Table 2). For participants in the non-COVID-19 group, we observed a 42% lower risk of abnormal MMSE scores ($p = 0.02$) (Figure 2A), a 12% relative increase in the total MMSE score, and a 13% and 16% relative increase in the temporal and spatial orientation sub-items of the MMSE ($p < 0.01$) (Figure 2B), respectively, compared to the COVID-19 group. In the CDR test, on average, participants in the non-COVID-19 group had an 89% lower risk of "CDR Abnormal Orientation" and a 78% lower risk of "CDR Abnormal Personal Care" ($p = 0.03$) (Figure 2A). Participants in the non-COVID-19 group also recorded a relative increase of 13% in the Lawton scale ($p = 0.02$) but a relative reduction of 50% in the Katz scale ($p = 0.04$) (Figure 2B). Comparing the change in MMSE classification between the pre-pandemic assessment and t2, the COVID-19 group had a 77% greater risk of the result deteriorating or remaining abnormal relative to the non-COVID-19 group (relative risk 1.77, 95% CI [1.06, 2.96], p =

0.03). However, we observed no significant reduction in the risk of dementia in the non-COVID-19 group based on the total CDR (relative risk 0.26, 95% CI [0.06, 1.07]) (Figure 2A).

3.2 Longitudinal cognitive and functional impairment of participants affected by COVID-19

The results of pre-pandemic MMSE, CDR, Lawton, and Katz tests were compared with those from the t2 assessment. The COVID-19 group showed a mean reduction in the total MMSE score of 3.6 points, whereas the non-COVID-19 group showed the mean reduction of zero points ($p < 0.01$) (Table 3). On average, participants in the COVID-19 group had a 3.4-fold higher relative risk of abnormality in the total MMSE score ($p < 0.01$) (Figure 3A) and a 13% relative reduction in the total MMSE score ($p < 0.01$) (Figure 3B). Regarding the total CDR, on average, participants in the COVID-19 group had a 4.2-fold increased risk of being classed as having dementia (relative risk 4.2, 95% CI [1.31, 14.0], $p = 0.02$) (Figure 3A). Regarding the Lawton scale, participants in the COVID-19 group showed a relative reduction of 14% ($p < 0.01$) (Figure 3B) compared to a 7% reduction in the non-COVID-19 group (relative effect 0.93, 95% CI [0.90, 0.97], $p < 0.01$). Finally, we observed a 3.4-fold relative increase in the Katz scale score for the COVID-19 group ($p < 0.01$) (Figure 3B). Figure 4 shows the variation in total MMSE scores by group and time. Figure 5 compares the groups between time points and shows the percentage of participants who had cognitive impairment based on the total MMSE, the percentage of participants who had dementia based on the total CDR, and the percentage of participants with greater dependence in instrumental (Lawton < 21) and basic (Katz ≥ 3) activities of daily living.

After adjustment for potential confounding factors, these results indicated that SARS-CoV-2 infection was associated with cognitive and functional deterioration.

4. DISCUSSION

In this study, we observed an association between prior SARS-CoV-2 infection and worse cognitive and functional performance among older adults with mild-to-moderate COVID-19, assessed on average 2.5 years after the acute phase. At t2, absence of infection was associated with higher scores on the total MMSE and its temporal and spatial orientation sub-items, reflecting a relative cognitive advantage in the non-COVID-19 group. This is consistent with the longitudinal data: the non-COVID-19 group's mean MMSE score remained unchanged between the pre-pandemic assessment and t2, indicating cognitive stability. Absence of infection was also associated with higher scores on the Lawton scale and lower scores on the Katz scale, both indicating less pronounced functional decline in the non-COVID-19 group. Non-COVID-19 status was also associated with a significantly lower risk of abnormal total MMSE and abnormal CDR orientation and personal-care domains; the between-group difference in total CDR abnormality at t2 did not reach statistical significance (relative risk 0.26, 95% CI 0.06–1.07), suggesting that domain-specific measures captured impairment earlier or more consistently than the global CDR classification at that time point. The longitudinal comparison (pre-pandemic to t2) showed cognitive deterioration in the COVID-19 group, reflected in a mean reduction in total MMSE score and a greater risk of abnormal classification, together with significantly increased Katz scores and a higher risk of dementia classification by CDR. Notably, although the COVID-19 group had a shorter interval between assessments — implying reduced aging

exposure relative to the non-COVID-19 group — the observed decline remained significant after adjustment for follow-up duration, suggesting that the deterioration was independently associated with prior infection rather than merely reflecting differences in observation time. Systematic assessment during follow-up did not identify any participant meeting criteria for long COVID, indicating that the deterioration observed cannot be attributed to unresolved acute-phase symptomatology captured by that diagnostic category.

Although this study did not measure inflammatory, vascular, or neurodegenerative biomarkers, the pathophysiological mechanisms proposed for post-COVID-19 cognitive dysfunction — including systemic inflammation, blood-brain barrier dysfunction, and tau-related pathways — offer a plausible, though untested, explanation for the deterioration observed in our cohort. Available evidence supports models in which COVID-19 accelerates pre-clinical alterations, unmasks subclinical dementia, or produces persistent cerebral injury rather than initiating primary neurodegenerative disease; the most prudent interpretation is that SARS-CoV-2 acts as a biological stressor capable of accelerating or revealing neurodegenerative and vascular processes in vulnerable individuals [21–24,27,41]. A summary of the molecular mechanisms and plasma/cerebrospinal fluid biomarker alterations described in the literature is provided in Additional files 1 and 2 (Tables S1 and S2).

Comparisons between studies of long-term cognitive outcomes in older adults are challenging because of differences in study design, populations, and screening tests. A previous study by Corbett et al. [42] is methodologically closest to ours, although they used

the FLAME neuropsychological battery instead of screening tests. In their longitudinal cohort, performance declined during the pandemic in both infected and non-infected participants, yet those with COVID-19 showed greater declines, including a 50% reduction in working memory and a 46% reduction in executive function. A large community-based study of 112,964 participants also reported long-term cognitive sequelae of COVID-19, but did not include pre-pandemic data [43]. Relative to a non-COVID-19 group, a subgroup similar to our COVID-19 group showed a deficit of −0.23 SD (95% CI [−0.33, −0.13]). Cognitive performance was worst in individuals infected during the waves dominated by original or alpha variants; in our cohort, 59% of the COVID-19 group was infected during these waves and 45% had not received any vaccine dose at the time of infection (Table 1) — a profile associated with worse cognitive outcomes in that study. Taquet et al. [44] examined 475 hospitalized patients using the Cognitron battery 2–3 years after COVID-19 hospitalization, a follow-up comparable to that in our study. Across all assessed domains, these participants performed worse than non-infected controls (median deficit −0.71 SD, IQR 0.16–1.04, $p < 0.0001$), and cognitive performance at 2–3 years was only moderately correlated with earlier Montreal Cognitive Assessment scores, suggesting that screening tools may underestimate persistent deficits. Another study of patients similar to those in our COVID-19 group, who did not require ongoing oxygen therapy, indicated worsening in both instrumental and basic activities of daily living compared with pre-pandemic levels [45], reinforcing the link between functional decline and cognitive loss.

The 4.2-fold increased risk of dementia (CDR) observed in the COVID-19 group is consistent, in direction, with associations reported in three recent meta-analyses (Rahmati et al. [46]: HR 1.66; Shan et al. [47]: RR 1.58; Zhang et al. [48]: HR 1.49, rising to 1.68 in

adults over 65) (Additional file 3, Table S3). However, this estimate was derived from a small number of events and carries considerable statistical uncertainty (95% CI 1.31–14.0). Given this wide interval, the point estimate should not be interpreted as indicating a substantially larger effect than that reported in larger studies; rather, our data are compatible with a wide range of possible effect sizes, including those reported in the literature, and a more precise estimate would require a larger sample. Additional contributors to the observed point estimate may include differences in outcome ascertainment — clinician-administered CDR classification versus diagnostic codes extracted from electronic health records in the larger registry-based studies — and the relatively high burden of vascular comorbidity in our cohort (hypertension 83%, dyslipidemia 64%). Furthermore, the specificity of this association to SARS-CoV-2 remains uncertain, as excess risk is substantially attenuated when comparisons involve other respiratory infections [49,50], suggesting that part of the excess risk reported in the literature may reflect disease severity, diagnostic surveillance bias, or residual confounding rather than a pathogen-specific effect. Given that COVID-19 is a relatively recent disease and dementia typically develops over many years, longer follow-up is required to determine whether this risk estimate is confirmed with greater precision.

This study has some strengths. First, we monitored cognitive function over an extended period (mean duration of 32 months between infection and the second cognitive test) in a relatively old group of participants (mean age 78.8 years). Second, we included a control group without COVID-19. Third, we used standardized tests to assess both cognition and functional status. Fourth, we included pre-pandemic test results, which allowed us to compare each participant's performance over time.

However, our study also has several limitations. First, COVID-19 status and infection characteristics were based on self-reported data and medical records where available, as systematic serological screening was not performed — an approach similar to that used in other large observational studies [42,43]. This introduces some uncertainty related to potential recall bias, a concern that is particularly relevant in older populations, because asymptomatic or mildly symptomatic carriers may have gone undetected in the non-COVID-19 group. Such bias would likely weaken the observed association, potentially underestimating the true magnitude of the long-term cognitive and functional impact of COVID-19. Nevertheless, for our study population, the data provided sufficient evidence to support the observed association. Second, our recruitment strategy excluded the most frail older adults who could not attend geriatric services after the pandemic. Accordingly, our findings may not apply to the most vulnerable segment of the population. Third, the requirement for pre-pandemic data also reduced the sample size, limiting statistical power, the precision of the effect estimates, and the generalizability of our findings. Fourth, pre-pandemic cognitive and functional data were extracted retrospectively from medical records rather than administered under a standardized protocol controlled by the research team, which may have introduced variability in testing conditions and documentation quality. Fifth, evaluators performing the t1 and t2 assessments were not blinded, which could introduce observer bias, although the unavailability of t1 results at the time of the t2 assessment likely reduced the risk of anchoring or recall-based bias in test administration. Finally, although the COVID-19 and non-COVID-19 groups did not differ significantly in measured sociodemographic and clinical variables, residual confounding cannot be ruled out.

Future studies with larger, multicenter cohorts and longer follow-up — ideally exceeding five years — are needed to distinguish transient decline from stabilization, recovery, or conversion to mild cognitive impairment or dementia. Comparison groups with other respiratory infections would help clarify whether the pattern observed here is specific to SARS-CoV-2 or reflects a broader consequence of systemic infection in older adults.

5. CONCLUSIONS

In this cohort of adults aged 65 years and older, a history of COVID-19 was associated with long-term cognitive impairment and functional decline. Compared with their own pre-pandemic performance, older adults who had COVID-19 showed lower MMSE scores, a higher risk of dementia based on the total CDR, and increased dependence in basic and instrumental activities of daily living; cross-sectional comparisons with non-infected peers at t2 showed a consistent pattern across most measures, although the difference in overall CDR-based dementia risk between groups did not reach statistical significance. The mechanisms underlying this association remain unclear and warrant further investigation.

DECLARATIONS

Ethics approval and consent to participate

All procedures involving human participants were carried out in accordance with the 1964 Declaration of Helsinki and its later amendments, and with Brazilian National Health Council Resolution 466/2012. This study was approved by the Research Ethics Committee of the Fluminense Federal University Faculty of Medicine (CAAE 56346822.2.0000.5243). All participants provided written informed consent prior to inclusion.

Consent for publication

Not applicable.

Permission to use the MMSE

An unauthorized version of the Brazilian Portuguese MMSE was used by the study team without permission, however this has now been rectified with PAR. The MMSE is a copyrighted instrument and may not be used or reproduced in whole or in part, in any form or language, or by any means without written permission of PAR.

Availability of data and materials

The dataset(s) supporting the conclusions of this article are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Leandro Galdiano: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. Ronaldo Gismondi: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Supervision, Writing – original draft, Writing

– review & editing. Yolanda Boechat: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Supervision, Writing – original draft, Writing – review & editing.

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Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author(s) used Claude (Anthropic) to assist with translation of the text into English and with structuring and formatting the manuscript in accordance with the submission requirements of BMC Geriatrics. This technology was not used to generate references, analyze data, or generate conclusions. After using this tool, the author(s) reviewed and edited the content as needed and take full responsibility for the content of the publication.

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Figure Legends

Figure 1. Flowchart of the selection, monitoring, and analysis of study participants

Figure 2. Cross-sectional comparison between the non-COVID-19 and COVID-19 groups at t2. (A) Relative risk of abnormal classification for the total MMSE; the total CDR and its six subdomains (memory, orientation, judgement and problem solving, community affairs, home and hobbies, and personal care); Katz≥3; and Lawton<21. (B) Relative effect on the total MMSE score and its subitems (orientation to time, orientation to place, registration, attention and calculation, recall, verbal language, and written language), Katz, and Lawton. Estimates are expressed as non-COVID-19 vs. COVID-19. Poisson regression models with robust variance (panel A) and negative binomial regression models (panel B), adjusted for age, sex, and income. Error bars represent 95% confidence intervals.

Figure 3. Longitudinal change within the COVID-19 group, comparing the pre-pandemic assessment with t2. (A) Relative risk of abnormal classification for the total MMSE, total CDR, Katz≥3, and Lawton<21. (B) Relative effect on the total MMSE, Katz, and Lawton scores. Regression models adjusted for age, sex, income, and the time interval between assessments. Error bars represent 95% confidence intervals.

Figure 4. Box plot of total MMSE score by COVID-19 status and time point (pre-pandemic, t1, t2).

Figure 5. Percentage of participants with abnormal classification on the MMSE, CDR, Katz, and Lawton tests, by COVID-19 status across time points.

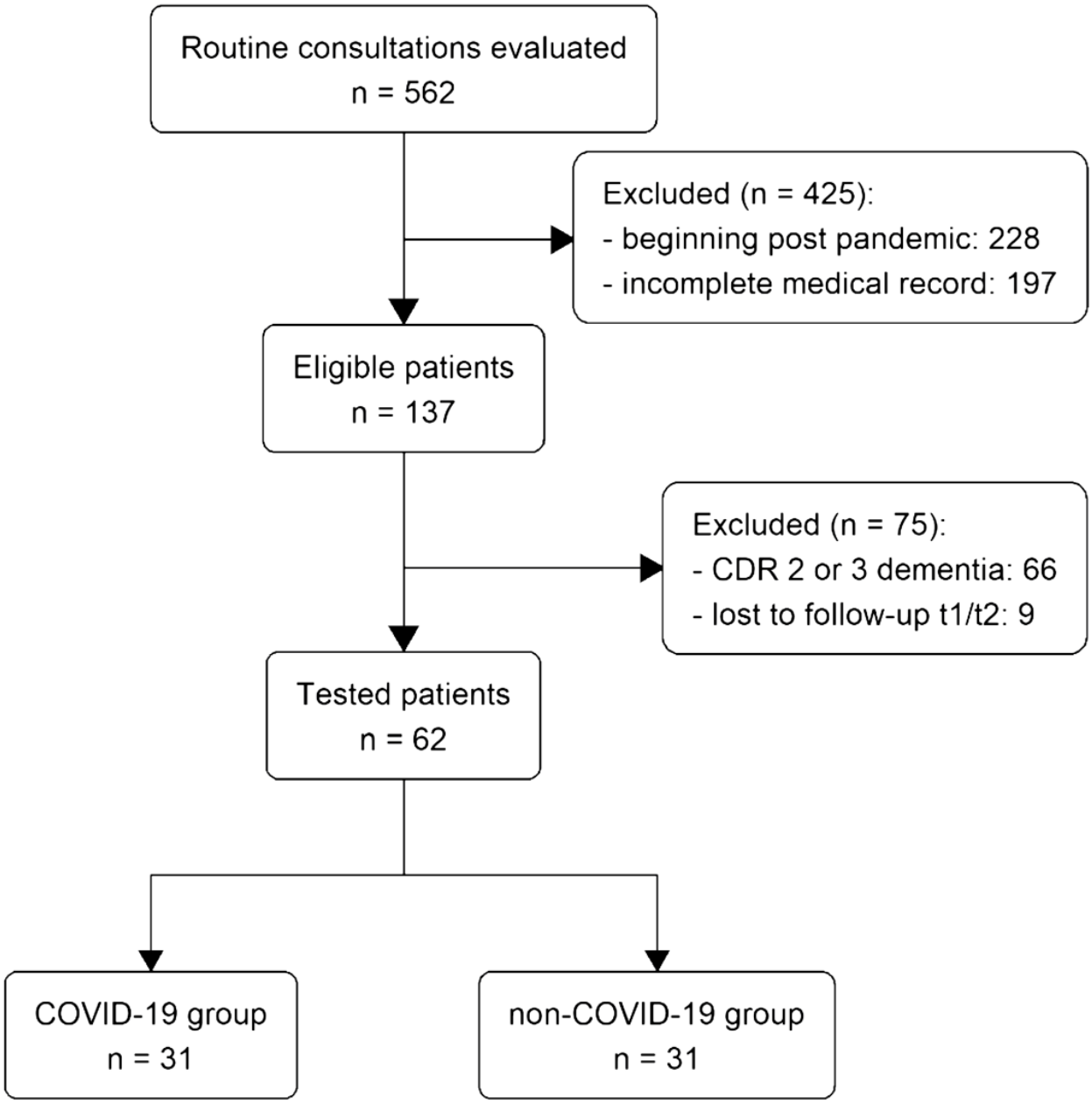
Routine consultations evaluated
n = 562
Excluded (n = 425):
- beginning post pandemic: 228
- incomplete medical record: 197
Eligible patients
n = 137
Excluded (n = 75):
- CDR 2 or 3 dementia: 66
- lost to follow-up t1/t2: 9
Tested patients
n = 62
COVID-19 group
n = 31
non-COVID-19 group
n = 31

Figure 2. Cross-sectional comparison between the non-COVID-19 and COVID-19 groups at t2

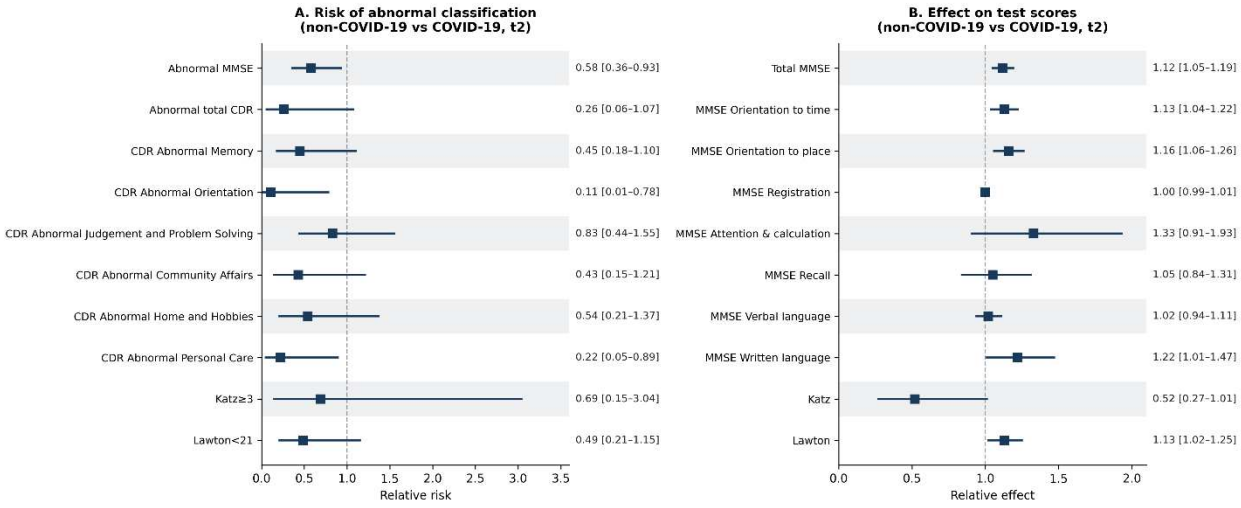

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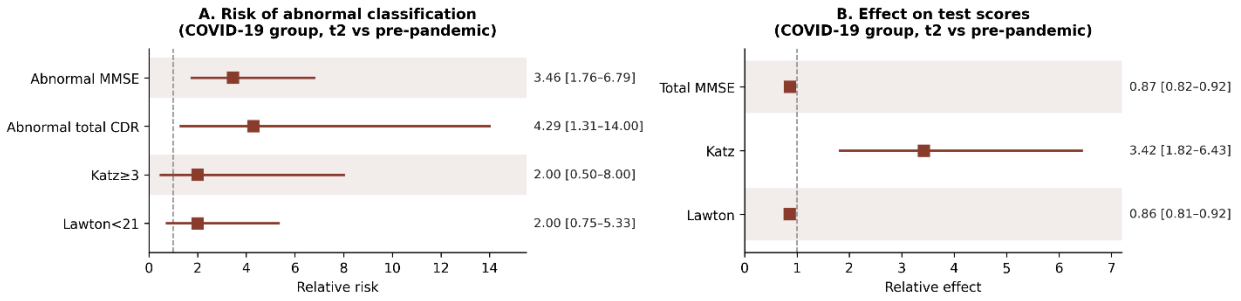
Figure 3. Longitudinal change within the COVID-19 group (pre-pandemic vs t2)



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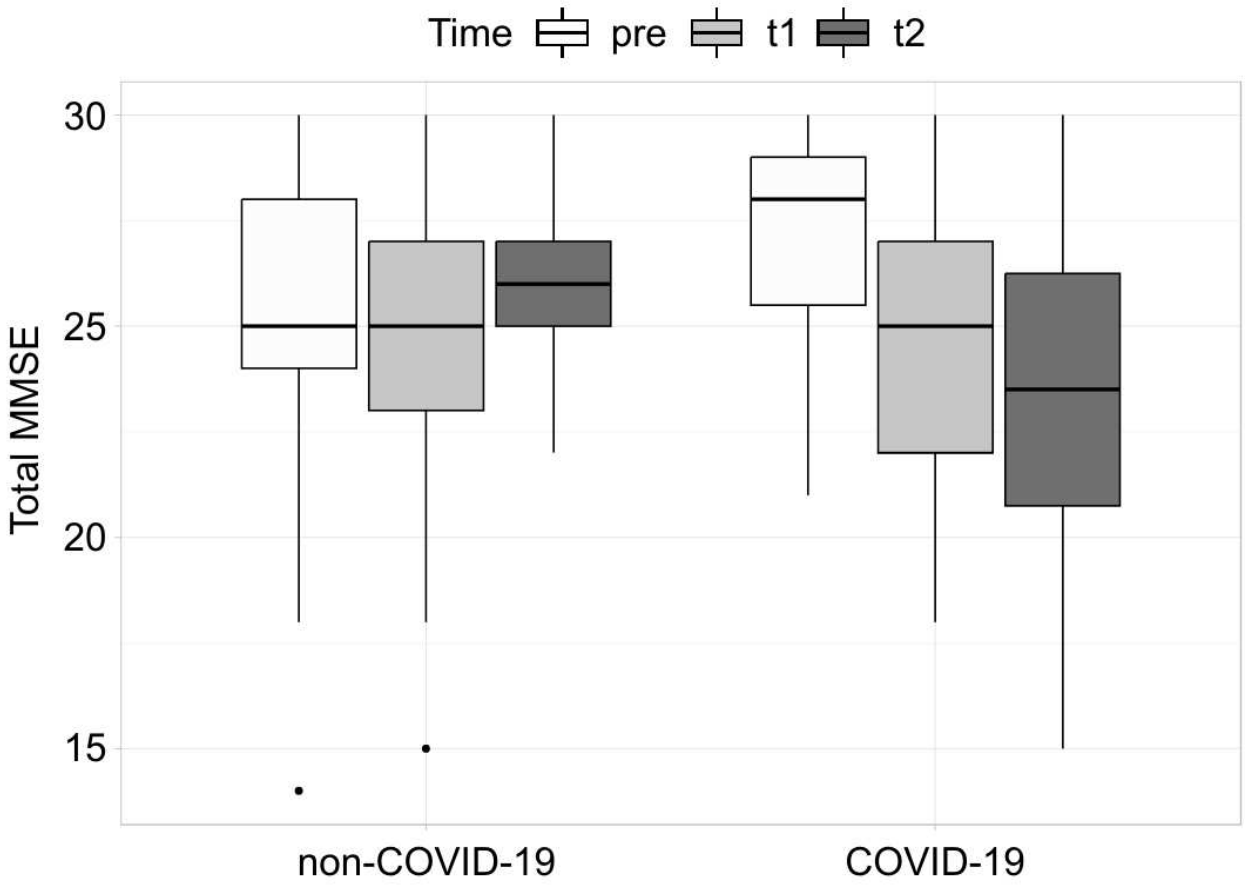

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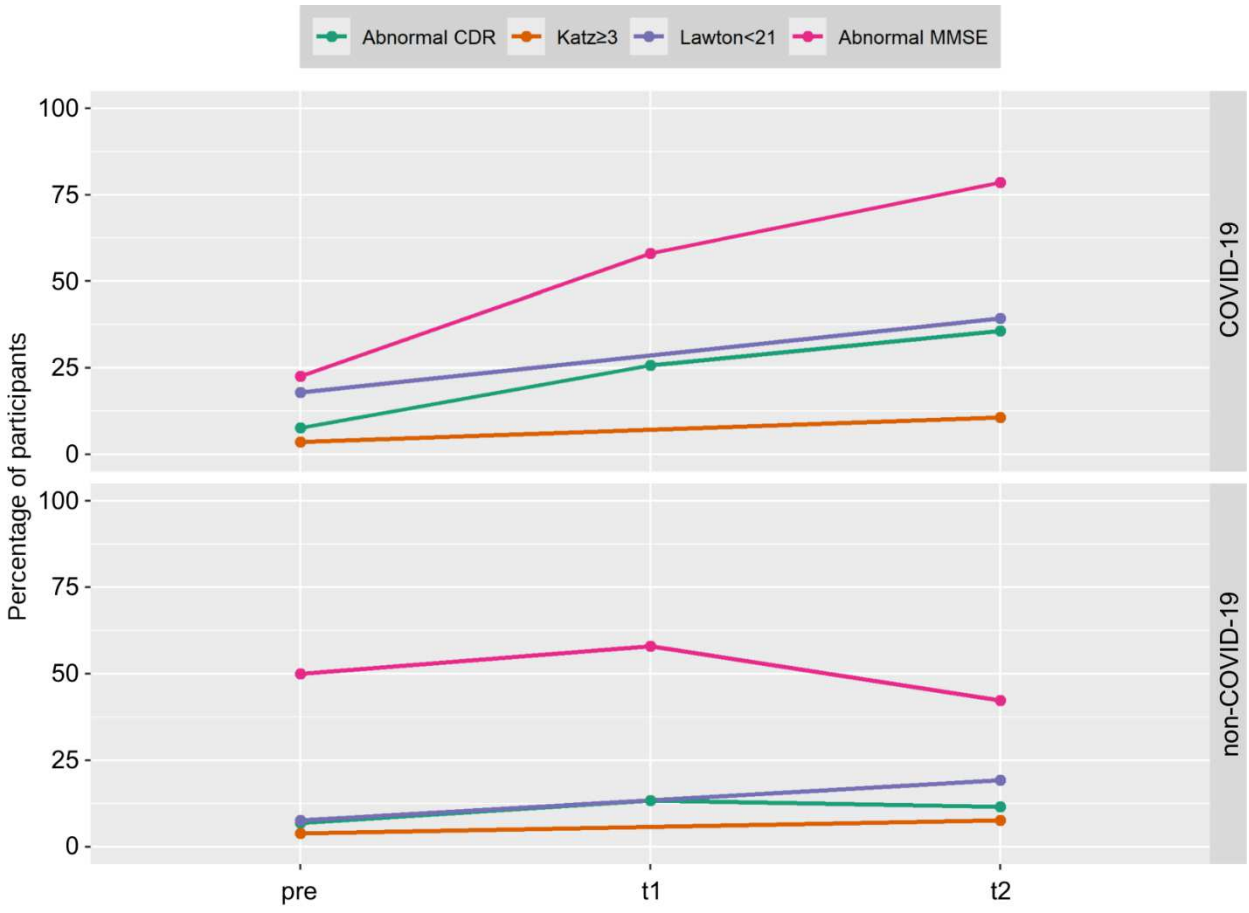
Abnormal CDR
Katz≥3
Lawton<21
Abnormal MMSE
Percentage of participants
100
75
50
25
0
COVID-19
non-COVID-19
pre
t1
t2

599

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

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- Table2.docx
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