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Original Article

Association between COVID-19 and mild neurocognitive disorder due to possible Alzheimer's disease in pregeriatric adults

Asociación entre COVID-19 y trastorno neurocognitivo leve subtipo posible Alzheimer en adultos de edad pregeriátrica

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ABSTRACT

Introduction: Signs of cognitive dysfunction have been reported in post-COVID syndrome. However, a significant association between COVID-19 and Alzheimer's disease, which shares many pathophysiological similarities, has not been established.

Objective: To evaluate the association between COVID-19 infection and the presence of mild neurocognitive disorder due to Alzheimer's disease in pre-geriatric adults.

Methods: An analytical observational study was conducted with 149 subjects with mild neurocognitive disorder due to possible Alzheimer's disease (cases) and 298 subjects with normal cognitive function (controls, 1:2 ratio) by simple random sampling. The crude odds ratio (OR) for COVID-19 were calculated using a contingency table, and the ORs by strata were determined with homogeneity test by the Breslow-Day method (p <

0.05), and the adjusted ORs were determined using the Mantel-Haenszel test with a 95 % confidence interval and significance level $p < 0.05$, to control for possible confounders.

Results: COVID-19 was associated with mild neurocognitive disorder due to possible Alzheimer's disease (OR = 8.57; 95 % CI: 5.34–13.72; $p < 0.001$). A consistent association was found among the different strata evaluated (OR: 4.821–12.173), and modification of the effect was ruled out with Breslow-Day tests, which showed significant heterogeneity between strata ($p > 0.05$). The association remained significant after adjustment for the different factors evaluated using the Mantel-Haenszel method (adjusted OR: 6.927–10.059).

Conclusions: SARS-CoV-2 infection may be a relevant risk factor for mild neurocognitive disorder due to Alzheimer's disease. Longitudinal studies with long-term follow-up are needed to elucidate the underlying mechanisms and confirm the association.

Keywords: Alzheimer's disease; cognitive dysfunction; COVID-19; risk factors; SARS-CoV-2.

RESUMEN

Introducción: Se han reportado signos de disfunción cognitiva pos-COVID y similitudes fisiopatológicas entre COVID-19 y enfermedad de Alzheimer (EA); no obstante, no se ha logrado establecer una asociación significativa.

Objetivo: Evaluar la asociación entre la infección por COVID-19 y la presencia de trastorno neurocognitivo leve (TNCL) debido a EA en adultos de edad pregeriátrica.

Métodos: Se realizó un estudio observacional analítico de 149 sujetos con TNCL debido a EA posible (casos) y 298 sujetos con funciones cognitivas normales (controles, relación 1:2), mediante muestreo aleatorio simple. Se calculó la *odd ratio* (OR) cruda para COVID-19 mediante tabla de contingencia, y se determinaron las OR por estratos con prueba de homogeneidad por método de Breslow-Day ($p < 0,05$), y las OR ajustadas mediante la prueba de Mantel-Haenszel con su intervalo de confianza de 95 % y nivel de significación $p < 0,05$ para controlar posibles confusores.

Resultados: COVID-19 fue asociado con TNCL subtipo Alzheimer posible (OR = 8,57; IC 95 %: 5,34–13,72; $p < 0,001$). Se determinó asociación consistente en los diferentes

estratos evaluados (OR: 4,821-12,173) y se descartó modificación del efecto con las pruebas de Breslow-Day que mostraron heterogeneidad significativa entre los estratos (p > 0,05). La asociación se mantuvo significativa tras el ajuste por los diferentes factores evaluados al aplicar el método de Mantel-Haenszel (OR ajusatdos: 6,927-10,059).

Conclusiones: La infección por SARS-CoV-2 puede ser un factor de riesgo relevante de la enfermedad de Alzheimer. Se requieren estudios longitudinales para esclarecer los mecanismos subyacentes y confirmar la asociación.

Palabras clave: enfermedad de Alzheimer; COVID-19; disfunción cognitiva; factores de riesgo; SARS-CoV-2.

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Introduction

Alzheimer disease (AD) is linked to many risk factors including genetic mutations, aging, and other environmental factors such as viral and bacterial infections. SARS-CoV-2, the viral pathogen responsible for COVID-19, has been linked to a variety of comorbidities and risk factors shared by AD. While COVID-19 usually results in a wide range of symptoms, including respiratory distress, infection has been associated with changes in brain structure and function which are also associated with neuroinflammation and potential neurodegeneration. Additionally, long-term cognitive impairments have been observed post-COVID-19.[1]

The review by Golzari et al.[2] suggested that the mechanisms involved in the pathogenesis of AD are also involved in COVID-19, with interchangeable inflammatory and AD biomarkers observed in recovering patients. Li et al.[3] expressed there is a strong correlation: both factors influence and potentiate each other, ultimately leading to a poor prognosis. Inflammation and the immune response likely play a key role in this process.

Doskas et al.[4] showed in their review that COVID-19 shares many epidemiological, molecular, and imaging similarities with AD; however, it remains unclear whether COVID-19 alters the natural history and onset of AD or accelerates its development. Therefore, they suggested that long-term, population-based studies are needed to establish its global contribution. The authors advised COVID-19 survivors with risk factors for AD should be closely monitored and evaluated periodically.

The predictive model of mild neurocognitive disorder (MNCD) due to AD in Cuban adults of Esquivel and Montoya[5] can distinguish subjects with risk factors for AD who are at higher risk of MNCD, using easily identifiable clinical and epidemiological variables, including COVID-19 history, which had a great impact. COVID-19 may interact with other risk factors, such as advanced age and other comorbidities, and may have complicated or accelerated cognitive decline in people who were already at risk. Therefore, any research in subjects with this factor is valid to understand its relationship with other factors.

The present research was carried out with the objective of evaluating the association between COVID-19 infection and the presence of MNCD due to AD in pre-geriatric adults.

Methods

An analytical observational case-control study was conducted in the health area of Manuel Fajardo Rivero Teaching Polyclinic, in the Cuban municipality of Las Tunas and province of the same name, during the period from January 1, 2024 to June 30, 2025, and for which the guidelines of the statement Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) were taken into account.[6]

The study population included 864 subjects aged 50 to 64 years and both sexes who agreed to participate in the research. The inclusion and exclusion criteria were based on the diagnostic guidelines for MNCD due to possible AD from Diagnostic and Statistical Manual of Mental Disorders (DSM-5).[7]

Prior to neurocognitive assessment, subjects with evidence of mixed etiology were excluded. This included cerebrovascular disease, other neurodegenerative disease, patients with the effects of a substance, or any other mental, neurological, or systemic

disorder, which constitute other etiologies of MNCD described in DSM-5,[7] or any other condition with a high probability of contributing to cognitive impairment, such as anemia, alcoholism, depression, and head trauma with brain injury. Subjects with visual and hearing impairments that clearly impacted performance on the neurocognitive assessment were also excluded, thus controlling for confounding variables. Subjects who did not cooperate with the neurological/neuropsychological assessment were also excluded.

Cases presented evidence of mild cognitive impairment documented by the Cuban version of the Addenbrooke's Cognitive Examination Revised (ACE-R),[8] and met the other diagnostic criteria for MNCD due to possible AD from DSM-5,[7] guaranteed by the exclusion criteria. The controls did not show cognitive impairment in the test results.

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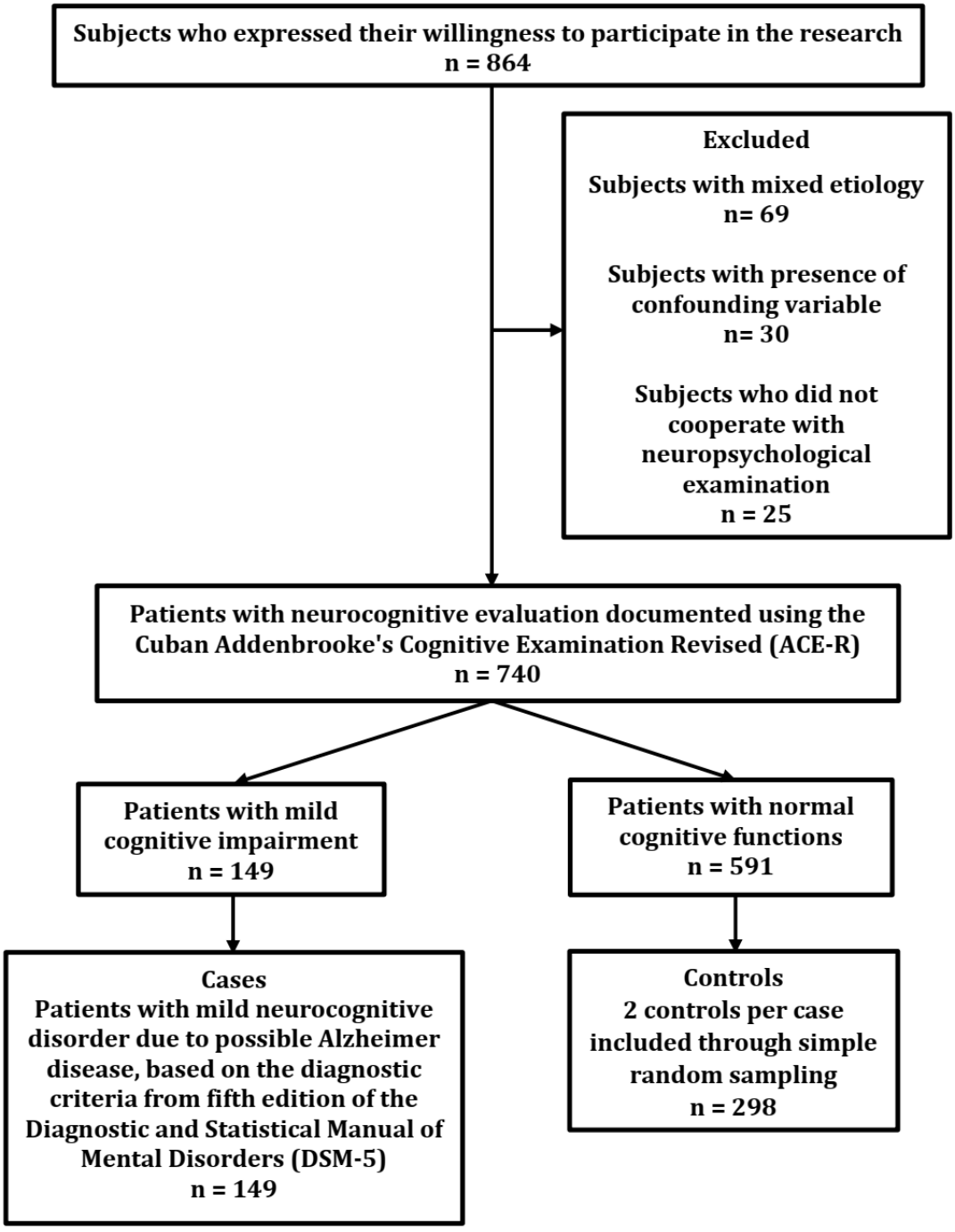

Fig. 1- Participant selection flow

Finally, the sample was formed by 149 subjects with MNCD due to possible AD (cases), and 298 subjects with normal cognitive function (controls, 1:2 ratio), who were included by simple random sampling, (figure 1).

The following variables were analyzed: neurocognitive assessment, COVID-19 history, sociodemographic characteristics (low educational attainment, low skill level, age and sex), and comorbidity (diabetes mellitus, hypertension and obesity):

- Neurocognitive assessment was performed by the Cuban version of the ACE-R, with reliable internal consistency (Cronbach's alpha coefficient α=0.879) and an optimal cutoff score of 84/85 (≤ 84 for mild cognitive impairment), in a study that validated this instrument in a Cuban population for a sensitivity of 0.896 and a specificity of 0.831.[8] However, given the sociodemographic characteristics of the study sample, lower cutoff scores than the normative values for other versions of the instrument applied in populations with low socio-educational levels were used as a reference,[5,9,10] and a score ≤ 70 was defined as mild cognitive impairment. Then, a possible Alzheimer's subtype of MNCD was established with compliance of diagnostic guidelines described in the DSM-5.[7]
- For a history of COVID-19, a biomolecular diagnosis by polymerase chain reaction was required for all participants at least two years prior to the present study. A history of COVID-19 was considered in subjects with positive SARS-CoV-2 test results.
- In the case of educational attainment, it was considered low for completed primary or secondary education and high for upper secondary and higher education, according to the educational levels in Cuba.[11]
- In the case of the skill level, it was defined based on the complexity and variety of tasks and responsibilities to be performed in an occupation, according to the criteria of the International Standard Classification of Occupations (ISCO-08).[12] Two categories were considered: a high skill level for ISCO-08 levels 3 and 4, with greater intellectual demands, and a low skill level for ISCO-08 levels 1 and 2, with predominantly manual work.
- The presence or absence of the remaining variables (age, sex, diabetes mellitus, hypertension, and obesity) was determined based on medical records. In the case of obesity, a body mass index greater than 30 kg/m^2 was considered.

Individual medical records were reviewed to obtain sociodemographic and clinical variables. Neurocognitive assessment was performed by ACE-R. The data obtained were stored in a Microsoft Excel 2016 spreadsheet and processed using SPSS version 25 for Windows. A description of the sample was prepared, and the crude odds ratio (OR) for COVID-19 was calculated using a contingency table. Subsequently, ORs were calculated for each stratum, with homogeneity assessed using the Breslow-Day method ($p < 0.05$). Adjusted ORs were then determined using the Mantel-Haenszel test with a 95 % confidence interval and a significance level of $p < 0.05$ to control for potential confounders (all stratum frequencies had to be higher than 5). A forest plot was constructed to visualize the adjusted associations and their confidence intervals.

The ethical standards of the Declaration of Helsinki were followed.[13] It was approved by the Scientific Council and the Research Ethics Committee of Manuel Fajardo Rivero Teaching Polyclinic. All participants expressed their willingness to participate in the research through informed consent.

Results

Regarding the sociodemographic and clinical characteristics of the sample (table 1), age over 57 years and female sex were observed in similar proportions between the case and control groups. In contrast, higher proportions of low educational and skill levels, hypertension, diabetes and obesity were observed in subjects with MNCD.

Table 1. Sociodemographic and clinical characteristics

Characteristics	Mild neurocognitive disorder (%)	No neurocognitive disorder (%)	Total (%)
Age >57 years	74 (49.66)	150 (50.33)	224 (50.11)
Female sex	64 (42.95)	128 (42.95)	192 (42.95)
Low educational attainment	113 (75.84)	87 (29.19)	200 (44.74)
Low skill level	127 (85.23)	80 (26.85)	207 (46.31)
Hypertension	116 (77.85)	94 (31.54)	210 (46.98)

Diabetes	116 (77.85)	89 (29.87)	205 (45.86)
Obesity	75 (50.34)	41 (13.76)	116 (25.95)

COVID-19 was considerably more frequent among participants with MNCD than among those without (table 2). COVID-19 was associated with MNCD possible Alzheimer's subtype (OR = 8.57; 95% CI 5.34–13.72; $\chi^2 = 91.6$; $p < 0.0001$).

Table 2. Distribution of cases and controls according to history of COVID-19

Groups	Mild neurocognitive disorder (%)	No neurocognitive disorder (%)	Total
No COVID-19	29 (19.46)	201 (67.45)	230
COVID-19	120 (80.54)	97 (32.55)	217
Total	149 (33.33)	298 (66.67)	447

Stratified analysis showed the association between COVID-19 history and MNCD remained consistent across the different strata evaluated (table 3). Stratified OR ranged from 4.821 to 12.173, with strong associations observed in all subgroups analyzed. Breslow-Day homogeneity tests showed no evidence of significant heterogeneity between strata, suggesting no significant effect modification. COVID-19 was significantly associated with the presence of MNCD after adjustment for the different factors evaluated, applying the Mantel-Haenszel method. Adjusted OR ranged from 6.927 to 10.059, with statistical significance in all cases (table 3; figure 2).

Table 3.- Stratified analysis

Strata		Controls		Cases		OR	*p* (Breslow-Day)	Adjusted OR Mantel-Haenszel (95 % CI)	*p* (Mantel-Haenszel)
		No COVID-19	COVID-19	No COVID-19	COVID-19				
Age	> 57	96	54	11	63	10.181	0.492	9.253 (5.396-15.863)	<0.001
	≤ 57	105	43	18	57	7.732			

Sex	Female	82	46	8	56	12.173	0.179	8.902 (5.206-15.219)	<0.001
	Male	119	51	21	64	7.105			
Educational attainment	Low	63	24	23	90	10.271	0.849	10.059 (6.001-16.861)	<0.001
	High	138	73	6	30	9.452			
Skills level	Low	57	33	22	105	8.238	0.246	7.77 (4.606-13.110)	<0.001
	High	144	64	7	15	4.821			
Hypertension	Yes	66	28	22	94	10.04	0.575	9.236 (5.907-14.437)	<0.001
	No	135	69	7	26	7.236			
Diabetes	Yes	50	39	23	93	5.217	0.081	6.927 (4.399-10.911)	<0.001
	No	151	58	6	27	11.689			
Obesity	Yes	25	16	13	62	7.483	0.963	7.841 (5.007-12.280)	<0.001
	No	176	81	16	58	7.889			

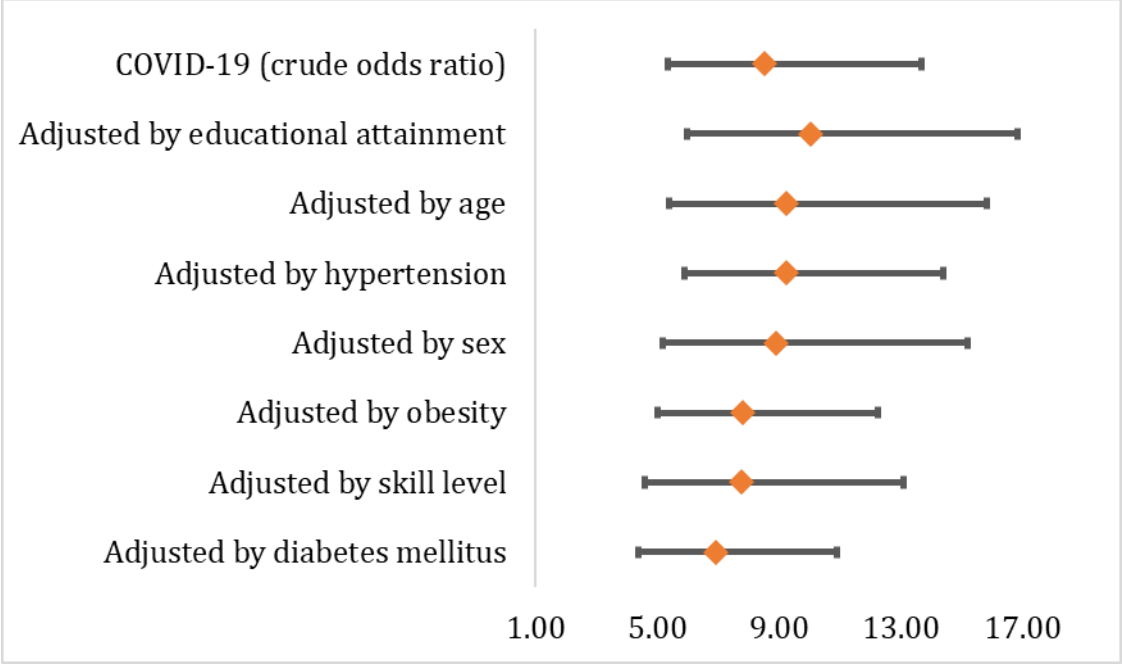

Fig.2-Forest plot of association: crude odds ratio, adjusted oods ratio by Mantel-Haenszel method, and their confidence intervals

Discussion

The results showed a significant association between COVID-19 history and MNCD due to possible AD, for that SARS-CoV-2 infection can constitute a relevant risk factor. These

findings are consistent with studies[1-4,14-19] that have described an increased frequency of cognitive impairment after SARS-CoV-2 infection, with higher rates of MNCD in subjects with a history of infection than in those who were not infected.

The association was significant at all levels, although it was greater in strata considered risk factors for AD:[20] advanced age, female sex, low educational attainment, low skill level and hypertension, except in the case of diabetes and obesity, which may be explained because individuals with these two diseases present a higher baseline risk of cognitive dysfunction (chronic inflammation, vascular dysfunction, metabolic alterations), which may attenuate the relative effect of COVID-19, and in healthy people the effect is observed cleanly with a higher OR.

The effect remained consistent even after its adjustment by age, sex, educational and skill level, hypertension, diabetes mellitus and obesity, applying the Mantel-Haenszel method; it means the observed effect is not explained by differences in the assessed sociodemographic and clinical factors. Variations in the magnitude of the adjusted OR correspond to slight modifications of the effect and not to a confounding phenomenon, indicating the association may be relatively independent of the presence of comorbidity.

The magnitude of the observed association reinforces the hypothesis that COVID-19 may alter the natural history and onset of AD through pathophysiological mechanisms. COVID-19 has been associated with structural and functional changes that are related to potential neurodegeneration;[1] markers of AD have been observed in patients recovering from COVID-19;[2] and common biological mechanisms have been identified: neuroinflammation, oxidative stress and neuronal damage, vascular dysfunction and microthrombosis, alterations in the immune response, hypoxia and metabolic stress, which probably play a fundamental role in the correlation.[3,4]

The previous studies[1-4, 14-19] presented notable differences that did not allow an effective comparison. They differ in methodological design, sociodemographic characteristics of the sample, diagnostic criteria, definition and collection of variables, neuropsychological instruments, and data analysis, among other elements. Unlike those studies, this research had an analytical case-control design with OR estimation and

adjustment for potential confounding factors using the Mantel-Haenszel method, at the primary health care in pre-geriatric subjects.

These findings highlight the importance of neuropsychological screening in patients with a history of SARS-CoV-2 infection, particularly in older populations or those with risk factors for AD. Early detection of cognitive impairment could allow for timely interventions aimed at preserving cognitive function.

Some limitations should be considered in the interpretation of the results. First, the case-control design does not allow for the establishment of definitive causal relationships. Furthermore, there is the possibility of biases inherent to the type of study, particularly selection and information biases. A stratified study was performed and adjustments were made to control for potential confounding variables, although the influence of other unmeasured factors cannot be ruled out.

The results of this research provide further evidence of the association between COVID-19 and mild neurocognitive disorder due to Alzheimer's disease, and indicate SARS-CoV-2 infection can be a relevant risk factor. Longitudinal studies with long-term follow-up are needed to clarify the underlying mechanisms, confirm the association, and identify the potential progression to major neurocognitive disorder.

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Conflicts of interest

The authors declare no conflicts of interest.

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