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The Adaptive Role of Entorhinal Cortical Thickness in Post-COVID 19 Cognitive Impairment

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ABSTRACT

Introduction: Only limited information is still available concerning cognitive dysfunctions and cortical thickness in individuals who recovered from mild COVID-19 infections and did not require hospitalization. Our aim was to evaluate if the highly adaptive potential of cortical thickness might play a critical role in COVID-19-related cognitive disorder in a compensatory manner.

Methods: Fifteen individuals with no history of medical, neurological, or psychiatric disease and with positive COVID-19 test results, and sixteen healthy age and education-matched healthy controls identified from the official hospital health system were evaluated in terms of cognitive scores using Alzheimer Disease's Assessment Scale-cognitive subscale (ADAS-Cog) and brain MRI cortical thickness measurements using FreeSurfer, Version 7.4.0.

Results: An increased cortical thickness in the right entorhinal cortex (EC) and impaired cognition (increased ADAS score) were observed in the post-COVID 19 group as compared to the controls confirmed by the student's t test (respectively, p=0.006, p<0.001).

The apparent correlation observed between cognitive impairment and increased entorhinal cortical thickness in our COVID-19 patients might suggest a continuum pathophysiology between healthy and COVID-19 affected brains that was not evident in previous COVID-19 cases with cognitive impairment.

Conclusion: Our findings of increased entorhinal cortical thickness, together with impaired cognitive scores, may indicate a flexible role of EC thickness in compensatory mechanisms of cognition.

Keywords: cognitive impairment, entorhinal cortical thickness, post-COVID-19

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INTRODUCTION

COVID-19 involves a wide range of (1) neurological changes that have been reported in 36.4% of cases (2), including notable cognitive symptoms manifested even in individuals who recover from COVID-19 (3). Despite several studies indicating the role of cognitive manifestations in severe COVID-19 infections (4–8), limited information is still available regarding cognitive dysfunctions in individuals who recovered from mild COVID-19 infections and did not require hospitalization.

Within that context, there have been few investigations of mild cases of COVID-19 revealing comparable impairments in critical cognitive functions, aligning well with previous observations in patients with severe COVID-19 (9–11). Nevertheless, it remains uncertain whether any associated structural and functional changes occur even during the early stages of mild COVID-19 patients with cognitive impairment. Herein, several functional magnetic resonance imaging (fMRI) studies have suggested the presence of critical functional connectivity patterns within major functional networks (default mode network and dorsal attention network) in individuals with impaired cognition due to COVID-19 infection (12–14). Despite providing valuable imaging data, these studies either lacked a control group or else assessed connectivity differences only among COVID-19 patients at different disease stages, thus yielding an insufficient perspective for illuminating the relationship between specific cognitive regions and cognitive functions. Although the novel static UK Biobank study found significant structural abnormalities in individuals with cognitive impairment post COVID-19, it was limited to the limbic system for findings explaining changes in cognition (7). These findings align well with other structural studies showing general alterations in cerebral white matter associated even with disseminated leukoencephalopathic changes (15,16), despite other research data obtained using brain computed tomography or MRI has not identified any specific alterations at structural neuroimaging examinations (17).

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Highlights

- **Mild COVID-19 patients have increased in the right entorhinal cortical thickness.**
- **Patients have more impaired cognitive performance than controls.**
- **Cortical thickness might play an adaptive role in cognitive deficits in COVID-19.**

These findings suggest a special role for structural imaging in patients with apparent cognitive manifestations, showing only the robust and permanent effects of COVID-19 on brain structures compared to dynamic neuroimaging approaches, indicating a pre-degenerative phase that might be associated with subtle dynamic alterations. This is consistent with the unique potential of structural brain imaging for identifying neurobiological markers and serving as a guide to diagnosis and treatment, as opposed to earlier dynamic alterations in these initial stages (16).

Entorhinal cortex (EC) is located in the anterior part of the parahippocampal gyrus between the hippocampus and the trans-entorhinal region. The EC plays a significant role within the limbic circuity by serving as an interface connecting the neocortex and hippocampus (18). Several studies have suggested that EC plays a significant role in encoding episodic memory, which is particularly vulnerable to early pathological alterations associated with Alzheimer's disease (AD) (19,20), hence making it reasonable that neuronal loss in EC might lead to significant attentional and memory deficits. Nevertheless, despite the prevailing notion that the EC primarily serves as a conduit connecting cortical regions and the hippocampus, recent imaging data revealed that the EC and the hippocampus possess separate, albeit partially overlapping, networks of connections, implying that impairments concerning these brain regions may result in distinct functional and cognitive states (21).

Considering all these, we evaluated specific cortical thickness parameters in connection with neuropsychological performance in a homogenized population of patients with mild COVID-19 infection within a specific timeframe (<12 months). To our knowledge, this timeframe represents a potential intermediate phase between functional and structural changes in cognitive impairment. Our main motivation was to evaluate if the highly adaptive potential of cortical thickness might play a critical role in COVID-19 related cognitive disorder in a compensatory manner, which is a hot topic in cognitive neuroscience as was previously shown by our dynamic neuroimaging study in COVID-19 patients with cognitive impairment (22).

METHODS

Participants

Fifteen individuals with a positive SARS-Cov-2 test without a history of medical or related neurological/psychiatric diseases (major depressive disorder, Parkinson's disease, stroke, normal pressure hydrocephalus, subdural hematoma, brain tumors, etc.) and an age-education matched healthy control group (n=16) were included in the study. The COVID-19 group participants were scanned within 12 months of positive COVID-19 test results emerged. All participants completed medical histories and physical examinations and underwent structural imaging. The Alzheimer's Disease Assessment Scale - Cognitive (ADAS-Cog) was used as the global cognitive assessment. The total ADAS-Cog score ranges from 0–70, with higher scores suggesting greater impairment. This study was approved by Alanya Alaaddin Keykubat University (2023/09) Ethics Committee.

The Alzheimer's Disease Assessment Scale - Cognitive (ADAS-Cog)

Alzheimer's Disease Assessment Scale (ADAS) is a scale specifically designed for the assessment of the cognitive and noncognitive dysfunctions seen in Alzheimer's disease. ADAS-Cog is the most widely used test in clinical trials dealing with Alzheimer's disease. Its adaptation to Turkish society and its validity and reliability studies were carried out by Mavioğlu et al. (23).

The original ADAS-Cog (24) included 11 items assessing cognitive function. The domains include memory, language, praxis, and orientation. There are 70 possible points: 48 for the first 9 items, and 22 for the last two items; word recall and recognition. Test performance was assessed for errors in following ordered commands, the naming of real objects and of fingers, constructional praxis, ideational praxis, orientation, a 10-item word recall task and a 12-item and 12 foils word recognition tasks. Higher scores reflect greater cognitive impairment.

Data Processing

Cortical morphometry analyses encompassing cortical thickness, surface area, and gray matter volume were conducted on FreeSurfer version 7.4.0 software (25) for a comprehensive (http://surfer.nmr.mgh.harvard.edu) evaluation of the cortex using intensity and continuity information. Segmentation of participants' entire brains was carried out through the automated recon-all pipeline integrated within FreeSurfer. This pipeline encompasses a sequence of steps, including motion correction, averaging of multiple volumetric T1-weighted images, skull stripping utilizing a deformable template model, automated registration to the Talairach space, segmentation of subcortical white matter and deep gray matter volumetric structures, intensity normalization, tessellation of gray matter and white matter boundaries, automated topology correction, and surface deformation guided by intensity gradients for the precise placement of gray/white and gray/cerebrospinal fluid borders (26). Rigorous visual inspections were carried out, and manual adjustments were executed when necessary, guided by control points outlined in Fisch et al. (27). Following registration, the brain surface was subdivided into 148 regions (74 per hemisphere) using the Destrieux Atlas parcellation scheme. Within the context of this study, we derived mean cortical volume (in mm^3), surface area (in mm^2), and cortical thickness (in mm) values for each individual region by employing the cortical parcellation defined by the Destrieux Atlas. Intracranial volume was also quantified for each participant in order to account for individual differences in overall brain size and structure.

Statistical Analysis

Statistical analysis of the data, as well as verification of normality, was performed using the Shapiro-Wilks test due to the small number of patients. Normally distributed data were analyzed using the Student's t Test (presented as mean, standard deviation), and non-normally distributed data were analyzed using the Mann–Whitney U test (given as median, interquartile range). Pearson's correlation analysis was used to determine the relationship between ADAS-Cog score and right entorhinal thickness. Data was analyzed using IBM Statistical Package for Social Sciences (SPSS) program for Windows, version 23.0 (Armonk, NY: IBM Corp). A value of $p < 0.05$ was considered significant.

RESULTS

Demographic Features and Clinical Characteristics

All the participants' (n=31) demographic features and clinical characteristics are summarized in Table 1. The Mann-Whitney U test revealed no differences in terms of age and education between the two groups (p=0.549 and p=0.857, respectively). However, the post-COVID-19 group (n=15) registered significantly worse ADAS-Cog scores than the control group (n=16) (Student's t-test, p <0.001). Also, right EC was significantly increased in the patient group (Mann-Whitney U test, p=0.006) (Table 1).

In addition, correlation analysis in the whole sample revealed a significant correlation between entorhinal cortical thickness and ADAS-Cog scores (Pearson's r=0.363, p=0.045), while no correlation was found between ADAS-Cog and entorhinal volume (Table 2).

In comparing the post-COVID 19 patients with (n=7) and without anosmia (n=8), we detected that seven of fifteen patients had anosmia that revealed no significant difference in the mean of right EC thickness (Student's t-test, p=0.789), although patients with anosmia had significantly higher ADAS-Cog scores than those without anosmia (Student's t-test, p=0.046) (Table 3), indicating a significant cognitive impairment in patients with anosmia.

Group Differences in the Cortical Measurements

In order to test the hypothesis that changes occur in cortical measurements after post-COVID 19 infections, we performed region of interest (ROI)-based analyses following cortical parcellation on Freesurfer. A group comparison of the post-COVID 19 and control participants revealed significantly (p=0.006) higher cortical thicknesses in the post-COVID 19 patients in the right EC than controls (Table 1, Fig. 1). Correlation analysis also revealed a significant correlation between entorhinal cortical thickness and ADAS-Cog scores (Pearson's r=0.363, p=0.045, Table 2) in the whole group. According to the latest literature adjustment for eTIV was not required since entorhinal thickness is not correlated with intracranial volume (28).

Table 1. Clinical characteristics between the patients with post-COVID 19 infection and control group at baseline

Variables	Post-COVID 19 (n=15)	Controls (n=16)	p
Age (years, median, IQR)	32 (10.5)	27 (21.3)	0.549
Education (years, median, IQR)	12 (4)	13 (4)	0.857
ADAS-Cog (mean ± SD)	8.06±2.26	5.16±2.09	**0.001***
Right entorhinal thickness (mean ± SD)	3.365±0.317	3.042±0.296	**0.006***
Right entorhinal volume (median, IQR)	1750 (306)	1840 (361)	0.346

ADAS-Cog: Alzheimer's disease assessment scale - cognitive; IQR: interquartile range; n: number of patients; SD: standard deviation (±); *p: Student's t test, <0.05; normally distributed data were analyzed with Student's t test; non-normally distributed data were analyzed with a Mann-Whitney U test.

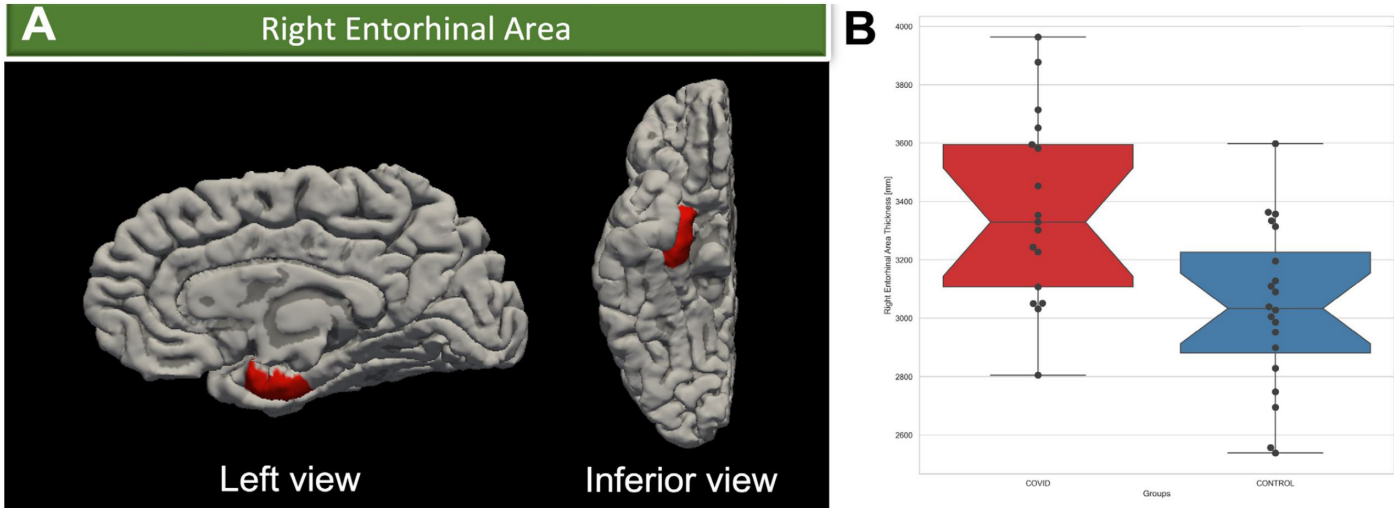

Figure 1. A, B. Group comparison of gray matter thickness analysis. Increased cortical thickness in the right entorhinal area was observed in the post COVID-19 group compared to the controls (A). Box plots of right entorhinal area thicknesses for the post COVID-19 and control individuals (B) (post-COVID 19 > control, p=0.006).

Table 2. The correlation of ADAS-Cog and right entorhinal thickness in the sample

		Right entorhinal thickness	Right entorhinal volume
ADAS-Cog	**Pearson's r**	0.363	-0.316
	df	29	35
	p	**0.045***	0.056

ADAS-Cog: the Alzheimer's disease assessment scale - cognitive; Pearson's r: Pearson's correlation coefficient; df: degree of freedom.

Table 3. The comparison of ADAS-Cog and right entorhinal thickness between post-COVID 15 patients with and without anosmia

Variables	Patients with anosmia (n=7)	Patients without anosmia (n=8)	p
Age (mean ± SD)	33 (11.6)	35 (8.6)	0.714
Education (years, median, IQR)	14 (0.27)	12 (0.42)	0.865
ADAS-Cog (mean ± SD)	9.28 (2.36)	7 (1.63)	**0.046***
Right entorhinal thickness (mean ± SD)	3.39 (0.3)	3.34 (0.35)	0.789
Right entorhinal volume	1728±212	1834±466	0.837

ADAS-Cog: Alzheimer's disease assessment scale - cognitive; IQR: Interquartile range; n: number of patients; SD: standard deviation (±); *p: Student's t test; <0.05; normally distributed data were analyzed with Student's t test, non-normally distributed data were analyzed with a Mann-Whitney U test.

DISCUSSION

In our study, we found that SARS-COVID-19 infected patients with mild symptoms exhibited increased entorhinal thicknesses compared to healthy controls, significantly correlated with cognitive scores. Our findings aligned well with previous results showing that EC plays a specific cognitive role in cognitive impairment (20). For instance, Coutureau et al. (29) indicated the critical role of the EC in specific cognitive functions, such as working and spatial memory. Entorhinal cortex has also been implicated in some disease states, especially AD, where decreased volumes of entorhinal cortex might be a useful biomarker for early degenerative dementia (19,20,29).

Furthermore, the right EC is a well-known region bridging olfactory and cognitive functions. It is affected in COVID-19 patients, especially those who originally presented with cognitive impairment and smell dysfunction (30). The importance of EC also aligns well with recent data on the effect showing that SARS-CoV-2-related neuro-inflammation may result in decreased EC volumes, leading to critical memory changes observed in post-COVID 19 patients (30), which has already been confirmed in patients with mild cognitive impairment and AD (22). In contrast to previous literature, an increased thickness rather than a decreased EC volume emerged in the patients in our present study. This should be interpreted with caution and is distinct from the conventional view of the role of decreased EC volumes in cognitive impairment in neurological and non-neurological diseases, necessitating a dynamic view on the role of short-term EC thickness alterations in cognition.

For instance, several studies showed that the dynamic nature of the EC thickness-as opposed to solid volumetric changes-can better explain memory problems in patients with long COVID-19 (31). According to this view, our volume analysis revealed no significant differences in EC volumes between patient and control group. Hence, it seemed plausible to evaluate the EC thickness in addition to volume alterations given its dynamic nature and rapid adaptive capacity in diseased conditions compared to the volume alterations.

Given the robust evidence of the dynamic neuroimaging data our results may suggest a plausible increased compensatory morphological response in the EC thickness, one confirmed by a significant correlation between increased entorhinal thicknesses and poorer cognitive scores. Therefore, our findings of increased thickness values despite non-significant volumetric alterations align with recent dynamic neuroimaging data in post-COVID-19 patients with cognitive impairment, demonstrating increased hippocampal connectivity (32).

The proximity of the entorhinal cortex (EC) to the hippocampus and olfactory cortex suggests that neuropathological changes in the EC may contribute to early olfactory deficits and cognitive impairment in COVID-19 patients (33). Despite patients with anosmia demonstrating poorer cognitive function, our study found no significant difference in EC volume or thickness between patients with and without anosmia. This may be attributed to the small sample size (n=15) in the patient group, with only seven patients experiencing anosmia, which can be considered as a limitation of this study.

Here it is worth mentioning that the flexible role of EC thickness is consistent with recent evidence suggesting a biphasic pattern of changes in cortical thickness, in which a higher, rather than lower, thickness is associated with very early AD pathology, supporting the idea that an increased cortical thickness may precede future decline (34).

In conclusion, our findings not only confirm the neurodegenerative nature of COVID-19 (35–38), but also reveal that specific/rapid adaptive structural changes might be noted in addition to previously reported dynamic imaging data on impaired cognition and a specific network activity reported in COVID-19 patients and open a new therapeutic avenues for neurodegenerative and neuropsychiatric diseases (7,14,39-41). Our findings might be considered as unique in indicating the presence of both dynamic and static structural phases, in response to COVID-19.

It is important to note that the small sample size can be considered as a limitation in interpreting our findings of significantly decreased cognitive scores in the COVID-anosmia group, despite prior literature indicating a significant association between specific brain connectivity decreased cognitive scores, and anosmia (42, 43). Nevertheless, given the role of entorhinal cortex as a primary interface between the neocortex and hippocampus, future studies focusing on adaptive changes related to the entorhinal cortex will shed light on the neurodegenerative mechanism that develops following COVID-19 infection.

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Ethics Committee Approval: This study was approved by Alanya Alaaddin Keykubat University (2023/09) Ethics Committee.

Informed Consent: Written informed consent was obtained from all participants before their involvement in the study. The study was conducted in accordance with ethical standards and the Declaration of Helsinki.

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