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Cognitive Assessment in Patients with Acute COVID-19: A Cross-Sectional Study

Akut COVID-19 Hastalarında Bilişsel Değerlendirme: Kesitsel Bir Çalışma

Yusuf KOCAK[1] Asuman CELİKBİLEK[1] Burc Esra SAHİN[1] Duygu ZORLU[2]

Gökmen ZARARSIZ[3,4]

ÖZ

Amaç: COVID-19'a bağlı kognitif bozukluk daha önce yapılan çalışmalarda yüksek oranda bildirilmiştir. Enfeksiyonun ilk solunum semptomlarından sonraki 5 gün içinde, hafif COVID-19 hastalarında, COVID-19 olmayan hastalara kıyasla, Montreal Bilişsel Değerlendirmesini (MoCA) kullanarak bilişsel işlevleri araştırıldı.
Araçlar ve Yöntem: Bu prospektif kesitsel çalışmaya hafif COVID-19'lu 113 hasta ve yaş ve cinsiyet olarak eşleştirilmiş 109 kontrol alındı. Bilişsel işlevler, MoCA kullanılarak değerlendirildi.
Bulgular: MoCA skorlarının COVID-19 hastalarında kontrollere göre anlamlı derecede düşük olduğunu bulduk (P<0.001). Kontrollerle karşılaştırıldığında, görsel uzamsal (P<0.001), bellek (P=0.017) ve dikkat (P<0.048) alanları gibi bazı MoCA öğelerinde puan kayıpları bulduk. Çok değişkenli modelde kadın cinsiyet (2.06 [1.02–4.16], P=0.044), düşük eğitim düzeyi (15.05 [5.16–43.90], P<0.001), yüksek açlık kan şekeri düzeyi (0.98 [0.96–1.00]), P=0.043) ve COVID-19 (24.24 [9.52–61.72], P<0.001) varlığı bağımsız olarak kognitif bozuklukla ilişkiliyidi (OR, %95 CI).
Sonuç: Hafif COVID-19'lu hastalarda akut faz sırasında görsel uzamsal, bellek ve dikkat alanlarında bilişsel işlev bozukluğu tespit edildi.

Anahtar Kelimeler: biliş; bilişsel değerlendirme; covıd-19; montreal bilişsel değerlendirme; salgın

ABSTRACT

Purpose: Many reports have revealed a high percentage of patients suffering from cognitive impairment due to COVID-19. We investigated cognitive functions using the Montreal Cognitive Assessment (MoCA) in mild COVID-19 patients compared with non-COVID-19 patients within 5 days after the initial respiratory symptoms of infection.
Materials and Methods: 113 patients with mild COVID-19 and 109 age- and sex-matched controls were enrolled in this prospective cross-sectional study. Cognitive functions were evaluated using the MoCA.
Results: We found that the MoCA scores were significantly lower in the COVID-19 patients than in the controls (P<0.001). Compared to the controls, we found point deficits within some MoCA items such as visuospatial (P<0.001), memory (P=0.017), and attention (P<0.048) domains. In the multivariate model, female sex (2.06 [1.02–4.16], P=0.044), low education level (150.05 [5.16–43.90], P<0.001), high fasting blood glucose level (0.98 [0.96–1.00], P=0.043), and the presence of COVID-19 (24.24 [9.52–61.72], P<0.001) were independently associated with cognitive impairment (OR, 95% CI).
Conclusion: We detected cognitive dysfunction, involving the visuospatial domain, memory, and attention, during the acute phase in patients with mild COVID-19.

Keywords: cognition; cognitive assessment; covid-19; montreal cognitive assessment; pandemic

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[1]Kirsehir Ahi Evran University, Faculty of Medicine, Department of Neurology, Kirsehir, Türkiye.
[2]Kirsehir Ahi Evran University, Faculty of Medicine, Department of Chest Diseases, Kirsehir, Türkiye.
[3]Erciyes University, Faculty of Medicine, Department of Biostatistics, Kayseri, Türkiye.
[4]Erciyes University, Drug Application and Research Center (ERFARMA), Kayseri, Türkiye.

Corresponding Author: Asuman Çelikbilek, Kirsehir Ahi Evran University, Faculty of Medicine, Department of Neurology, Kirsehir, Türkiye.
e-mail: asunebioglu@yahoo.com

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INTRODUCTION

COVID-19 pandemic had destructive effects worldwide.[1,2] However, the pathologic mechanisms underlying the central nervous system (CNS) involvement are not fully understood.[3] Pronounced neurological manifestations of COVID-19 infection, such as headache, ageusia or anosmia, and dizziness, have been well-described, as they are easily noticed by patients.[3,4] However, cognitive dysfunction is often overlooked particularly if mild or if occurring in asymptomatic people.[5,6]

Multiple studies have revealed a high percentage of patients suffering from cognitive impairment due to COVID-19.[5-8] Several mechanisms have been proposed. First, the neurotropic SARS-CoV-2 can enter epithelial cells, depending on their expression of the angiotensin-converting enzyme 2 receptor, that is also found on neurons.[9,10] Notably, the virus has been found in the cortex and hypothalamus presenting with edema and neuronal degeneration in some COVID-19 patients, confirming that coronaviruses can invade the CNS.[11] Second, a systemic proinflammatory state and subsequent neuroinflammation and hypoxia have also been suggested to promote cognitive decline in patients with COVID-19 infection.[12,13] Some studies were performed during the sub-acute phase at the time of hospital discharge[6,9-11,14] and some after discharge.[8,15-20] Thus, data are limited for the acute phase of COVID-19, within a few days after symptoms initiate.[5,21] Early identification of cognitive impairment and supportive cognitive training if needed, may be preventive from further neurocognitive deterioration.[11] Beaud et al. revealed cognitive deficits during the acute stage in patients with COVID-19.[21] Rogers et al. performed a meta-analysis and reported that nearly one-third of patients in the acute phase and one-fifth of patients in the later stage experience impaired memory and concentration, or attention difficulties.[22] However, cognitive dysfunction remains obscure in mild cases of COVID-19, as it is unknown which cognitive areas are affected and to what extent. Amalakanti et al. reported deficits in visuoperception, naming, and fluency on cognitive screening in asymptomatic COVID-19 patients who were recruited from relatives of COVID-19 patients on admission.[5] We hypothesized that cognitive problems may occur even in mild cases. The Montreal Cognitive Assessment (MoCA) tool is useful to assess cognitive functions, as no obvious cognitive deterioration is expected in mild COVID-19 cases, and this tool is superior in detecting mild cognitive impairment.[6,10,11] Moreover, the MoCA has been widely validated in many cultures,[23] and it covers multiple cognitive domains on one page.[24] Beaud et al. revealed cognitive decline in executive, memory, attentional and visuospatial functions on the MoCA during the acute stage in COVID-19 patients.[21] Daroische et al. concluded that attention and executive functioning are more prone to deterioration than are other domains.[7] Taken together, subtle cognitive impairment limited to some domains is likely to present in even mild cases of COVID-19. Thus, we investigated cognitive functions using the MoCA in mild COVID-19 patients compared with non-COVID-19 patients in the course of the acute stage within the infection.

MATERIALS and METHODS

Study Design

This prospective study included 222 patients <50 years old who were consecutively selected from Kirsehir Ahi Evran University Hospital, Kirsehir, Turkey between May 2021 and January 2022. Patients who were admitted to our pandemic clinic were examined by a chest disease specialist for COVID-19 infection. The diagnosis of COVID-19 infection was made based on the real-time reverse-transcription polymerase chain reaction in nasal and throat swab specimens.[25] Acute COVID-19 was defined as the phase within 5 days after the initial respiratory symptoms. COVID-19 severity was classified as mild (not requiring hospitalization), moderate (requiring oxygen supplementation and/or hospitalization), or severe (requiring intensive care support) disease.[26] 113 patients who were in the mild group and 109 age- and sex-matched control subjects were included in this study. COVID-19 patients were examined by the same neurologist and questioned for all infectious and neurological symptoms. None of the COVID-19 patients was vaccinated, and none were receiving any treatment. Demographic data such as age, sex, body mass index, and education level were noted in all participants. Patients <18 years, those who were pregnant, those who had a

documented neurological or psychiatric disorder, hearing and/or visual impairments, thyroid disease, or vitamin B12 and/or folate deficiency, and those who were confused due to an acute infection were excluded.

Laboratory analyses were carried out using the standard methods. Informed consent was obtained from each subject and Kırşehir Ahi Evran University Clinical Research Ethics Committee approved the study (date: 10.12.2020 and approval number: 2020-18/131).

Cognitive Assessment

All patients were screened for cognitive functions by a trained neuropsychologist blinded to the participants using the MoCA.[24] The MoCA consists of assessments of visuospatial abilities (4 points), memory (5 points), executive functioning (4 points), attention (6 points), language (5 points), and orientation (6 points), as reported previously.[24] The perfect score is 30 points, and a cut-off score less than 26 points identifies mild cognitive impairment with high sensitivity (80–100%) and specificity (50-87%).[24] In this study, we used the Turkish version of MoCA (MoCA-TR) with a cut-off score of less than 21 points indicating poor cognition.[27,28]

Statistical Analysis

Histograms, q-q plots, and the Shapiro–Wilk test were used to test the normality of the data, and Levene's test was applied to assess variance homogeneity. The two-sided independent-samples *t*-test, Welch's *t*-test, or Mann–Whitney *U*-test was used to compare differences between continuous variables, and Chi-square analyses were used to assess differences between categorical variables. Effect sizes were calculated by Cohen's *d* (difference in the means/pooled standard deviations) for normally distributed continuous data, Rosenthal's *r* ($z/\sqrt{n}$) for non-normally distributed continuous data, and the Phi coefficient ($\sqrt{X^2/n}$) for categorical data. Univariate and multiple logistic regression analyses were performed to identify predictors of cognitive dysfunction in control subjects and COVID-19 participants. Variables with a univariate *p*-value < 0.25[29] were taken to the multivariate model, and forward elimination (Forward: LR) was conducted to detect independent risk factors. Estimated coefficients and odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. The goodness of fit of the multiple logistic regression model was evaluated using the Omnibus test, Hosmer and Lemeshow test, and Nagelkerke R^2 statistics. The data were analyzed using TURCOSA (Turcosa Analytics Ltd Co., Turkey, www.turcosa.com.tr) statistical software. Statistical significance was set at $p<0.05$.

RESULTS

The demographic data of controls and COVID-19 patients are summarized in Table 1. With respect to age or gender, no significant difference was found between the groups ($p>0.05$). The groups were also similar in terms of education level, smoking, and alcohol use ($p>0.05$). However, body mass index was higher in the COVID-19 patients than in the controls ($p=0.023$, Table 1). Table 2 shows that the MoCA scores were significantly lower in the COVID-19 patients than in the controls ($p<0.001$). Compared with the controls, we found point deficits within some MoCA items such as visuospatial ($p<0.001$), memory ($P=0.017$), and attention ($p<0.048$) domains. Table 3 summarizes the infectious and neurological symptoms of COVID-19 patients according to the MoCA cut-off scores. Amnesia, a lack of concentration, and inattention were higher in patients with low MoCA scores than in those with high scores ($p<0.001$). Similarly, headache, ageusia, anosmia, arthralgia, dizziness, and diarrhea were also more frequent in patients with low MoCA scores than in those with high scores among COVID-19 patients ($p<0.05$). In the multivariate model, female sex (2.06 [1.02–4.16], $p=0.044$), low education level (150.05 [5.16–43.90], $p<0.001$), high fasting blood glucose level (0.98 [0.96–1.00], $p=0.043$), and the presence of COVID-19 (24.24 [9.52–61.72], $p<0.001$) were independently associated with cognitive impairment (OR, 95% CI, Table 4).

Table 1. Demographic data of the controls and COVID-19 patients.

Variables	Control (n=109)	COVID-19 (n=113)	p
Demographic variables			
Sex (female)	52(47.7)	56(49.6)	0.783
Age (years)	35.13([8.84])	35.59([7.74])	0.678
Body mass index (kg/m2)	25.51([2.99])	26.59([4.00])	**0.023**
Education level			
Primary school	9(8.3)	9(8.0)	0.994
Secondary school	12(11.0)	12(10.6)	
High school	58(53.2)	59(52.2)	
University	30(27.5)	33(29.2)	
Smoking (yes)	44(40.4)	37(32.7)	0.238
Alcohol use (yes)	24(22.0)	16(14.2)	0.128
Laboratory markers			
Fasting blood glucose (mg/dl)	98.17([18.72])	99.16([15.19])	0.667
TC (mg/dl)	192.83([47.62])	185.43([44.81])	0.234
LDL-C (mg/dl)	103.0(87.0-116.5)	101.0(90.0-130.0)	0.767
HDL-C (mg/dl)	51.0(44.0-59.5)	54.0(45.0-65.0)	0.065
TG (mg/dl)	157.0(105.5-211.0)	126.0(91.0-186.0)	**0.037**
Vitamin D (IU/L)	17.0(11.0-23.0)	16.0(12.0-21.0)	0.256

Values are expressed as n(%), mean([SD]) or median(1st-3rd quartiles). TC indicates total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. Effect size calculations were given as Cohen's d for normally distributed continuous data, Rosenthal's r for non-normally distributed continuous data, Phi coefficient for categorical data. Bold values indicate statistically significant (p<0.05).

Table 2. The MoCA scores and the number of the affected individuals displaying point deficits within each cognitive domain in controls and COVID-19 patients.

Variables	Control (n=109)	COVID-19 (n=113)	p
MoCA scores*	24.0(23.0-26.0)	20.0(18.0-22.0)	<0.001
Frequency of cognitive dysfunction**	14(12.8)	70(61.9)	<0.001
MoCA items***			
Visuospatial	66(60.6)	95(84.1)	<0.001
Memory	101(92.7)	112(99.1)	0.017
Executive	86(78.9)	95(84.1)	0.321
Attention	73(67.0)	89(78.8)	0.048
Language	78(71.6)	91(80.5)	0.117
Orientation	2(1.8)	0(0.0)	0.240

Values are expressed as n(%). MoCA indicates Montreal Cognitive Assessment. *Cognitive functions were evaluated using the Turkish version of the MoCA, with a cut-off score for mild cognitive impairment of less than 21 points. **The low MoCA score represents the poor cognitive performance (over 30 points). ***The numerical data indicates the number of the affected individuals displaying point deficits within each MoCA item. Bold values indicate statistically significant (p<0.05).

Table 3. The frequency of the infectious and neurological symptoms of COVID-19 pati-ents according to the MoCA cut-off scores.

Variables	MoCA scores		Total	p
	<21 (n=70)	≥21 (n=43)	(n=113)	
Fatigue	65(94.2)	37(86.0)	102(91.1)	0.179
Amnesia	66(94.3)	31(72.1)	97(85.8)	**0.001**
Unconcentration	66(94.3)	31(72.1)	97(85.8)	**0.001**
Inattention	66(94.3)	31(72.1)	97(85.8)	**0.001**
Fever	59(84.3)	36(83.7)	95(84.1)	0.937
Cough	59(84.3)	32(74.4)	91(80.5)	0.195
Headache	57(81.4)	27(62.8)	84(74.3)	**0.028**
Ageusia	56(80.0)	23(53.5)	79(69.9)	**0.003**
Anosmia	55(78.6)	24(55.8)	79(69.9)	**0.010**
Arthralgia	53(75.7)	21(48.8)	74(65.5)	**0.004**
Dyspnea	44(62.9)	19(44.2)	63(55.8)	0.053
Dizziness	40(57.1)	11(25.6)	51(45.1)	**0.001**
Diarrhea	24(34.3)	7(16.3)	31(27.4)	**0.037**
Syncope	2(2.9)	2(4.7)	4(3.5)	0.634
Autonomic dysfunction	1(1.4)	0(0.0)	1(0.9)	0.999
Impaired consciousness	1(1.4)	0(0.0)	1(0.9)	0.999

Values are expressed as n(%). MoCA indicates Montreal Cognitive Assessment. Cognitive functions were eval-uated using the Turkish version of the MoCA, with a cut-off score for mild cognitive impairment of less than 21 points. The low MoCA score represents the poor cognitive performance (over 30 points). Bold values indicate statistically significant (p<0.05).

Table 4. Univariate and multiple logistic regression analysis to identify the predictors of cognitive dysfunction in the controls and COVID-19 patients

Variables	Univariate		Multiple	
	OR(95%CI)	p	OR(95%CI)	p
Sex (female)	1.73(1.00-3.00)	**0.049**	2.06(1.02-4.16)	**0.044**
Age (years)	1.05(1.01-1.08)	**0.007**	-	-
Body mass index (kg/m2)	1.09(1.01-1.18)	**0.036**	-	-
Education level				
-High school or university	1.00	-	1.00	-
-Primary or secondary school	5.07(2.45-10.49)	**<0.001**	15.05(5.16-43.90)	**<0.001**
Fasting blood glucose (mg/dl)	0.99(0.97-1.00)	0.128	0.98(0.96-1.00)	**0.043**
TC (mg/dl)	1.00(0.99-1.01)	0.928	-	-
LDL-C (mg/dl)	1.00(0.99-1.01)	0.318	-	-
HDL-C (mg/dl)	1.01(0.99-1.03)	0.358	-	-
TG (mg/dl)	1.00(0.99-1.01)	0.120	-	-
D vitamin (IU/L)	0.99(0.96-1.02)	0.337	-	-
Smoking status (yes)	1.03(0.59-1.81)	0.920	-	-
Alcohol use (yes)	0.86(0.42-1.76)	0.683	-	-
COVID-19 status (present)	11.05(5.61-21.75)	**<0.001**	24.24(9.52-61.72)	**<0.001**

Cognitive functions were evaluated using the Turkish version of the MoCA, with a cut-off score for mild cognitive impairment of less than 21 points. TC indicates total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol. OR: Odds ratio, CI: confidence intervals. Bold values indicate statistically significant (p<0.05). Omnibus test chi-square test=4.121, p-value=0.042; Hosmer and Lemeshow test=10.076, p-value=0.260 and Nagelkerke R2=0.501.

DISCUSSION

An association between COVID-19 and cognitive impairment is being increasingly recognized.[6] Many studies were performed at the late stages of COVID-19 and mainly in recovered patients.[8,15-20] However, a few studies were conducted during the acute stage.[5,21,22] In this study, we examined the cognitive functions of mild COVID-19 patients on admission and found that nearly two-thirds of the patients presented with cognitive impairment. We assessed the acute period of COVID-19 because subtle cognitive impairment often goes unnoticed at this stage.[5] We used the MoCA for cognitive screening, as it is more sensitive to mild cognitive changes in wide ranges.[24] We found lower MoCA scores on the visuospatial, memory, and attention domains in the COVID-19 patients than healthy controls. Although the exact pathophysiological mechanism remains speculative, the low scores may result from multiple interacting factors. According to Matos et al.,[10] direct virus-induced injury to the frontal cortical pathways may disrupt the neuronal communication circuits towards the thalami (e.g., the pulvinar region where mostly visual connections are located), which may explain the visuospatial deficits.[30] Additionally, the hippocampus is vulnerable to coronavirus infection, and neuroinflammation caused by the virus may increase memory impairment via high cytokine levels during a "cytokine storm", resulting in hippocampal atrophy, especially in severe cases.[31,32] Moreover, acute psychological stress induced by a patient's clinical conditions may impair attentional processing and increase the risk of memory deficits.[31] Furthermore, Hugon et al. reported two cases of cognitive deficit due to COVID-19, as demonstrated by abnormal hypometabolic regions in the cingulate cortex detected by fluorodeoxyglucose positron emission tomography.[16] The cingulate cortex is involved in emotions, memory, depression, and decisions of action.[16,33] Inputs from the orbitofrontal cortex enter the anterior cingulate cortex and then the posterior cingulate cortex of which the projections are towards the hippocampus.[16] This may provide clues for the memory deficits and abnormal attentional functions in COVID-19-infected patients. In this study, the executive deficits were similar between COVID-19 patients and control subjects. This may be because executive dysfunction potentially occurs during the subacute phase of COVID-19. COVID-related stress during the initial phase of COVID-19 may impair attention in the foreground, whereas executive dysfunction occurs later during the subacute period, as supported by Chang et al.[34] We suggest that cognitive impairment in each domain occurs according to the phases of COVID-19.

On the other hand, amnesia, lack of concentration, and inattention were very common in the group with poor cognitive performance, which was expected, as these three symptoms are closely linked to cognitive functioning.[8] Headache, ageusia, anosmia, and dizziness were frequently detected in COVID-19 patients with poor cognition. Additionally, patients with infectious symptoms, such as arthralgia and diarrhea, during an acute

infection had a worse cognitive performance on the MoCA as reported by Almeria et al.[8] All of these concomitant symptoms suggest a common underlying mechanism of ongoing neuroinflammation, which further affects cognitive status.[34] In the present study, the presence of COVID-19, female sex, and low education level were independently and strongly related to cognitive decline. As older age seems to influence cognitive status,[5,14] we excluded older people to avoid age-related cognitive impairment. The relationship between cognition and sociodemographic factors, such as sex and age, is controversial in COVID-19 patients. Woo et al.[35] and Beaud et al.[21] found a relation of cognitive functioning neither with age nor gender, while Alemanno et al.[14] showed that cognitive impairment is correlated with age. Herein, we found a correlation between cognitive decline and female sex, which was linked to the lower education status of the women compared with the men in our cohort. Education has a potential effect on cognition by promoting healthy behaviors regardless of age.[36] Confirming this observation, a primary or secondary school education level was predictive of cognitive impairment in COVID-19 subjects compared with high school education.

This study had certain limitations. First, the sample size was relatively small representing a single-center data. Second, our study was cross-sectional; therefore, a causal link is unclear. Third, some potential cofactors of cognition as nutritional, sleeping, and physical exercise habits, could not be excluded. Fourth, structural brain imaging was lacking due to infection control measures. Finally, we lacked follow-up assessment for cognitive status during recovery from COVID-19. Three- or six-months' follow-up are needed in further research.

In conclusion, some recent studies indicate that cognitive impairment may occur regardless of COVID-19 disease stages. In line with the literature, we detected cognitive dysfunction, mainly involving the visuospatial domain, memory, and attention, during the acute phase in patients with mild COVID-19. Despite the aforementioned limitations, our findings may provide some clues that may help guide future meta-analyses.

Conflict of Interest

The authors declare that there is not any conflict of interest regarding the publication of this manuscript.

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Ethics Committee Permission

The study was approved by Kırşehir Ahi Evran University Clinical Research Ethics Committee (Date: 10.12.2020 and number: 2020-18/131).

Authors' Contributions

Concept/Design: YK, AÇ, BEŞ, DZ, GZ. Data Collection and/or Pro-cessing: YK, AÇ, BEŞ, DZ, GZ. Data analysis and interpretation: YK, AÇ, BEŞ, DZ, GZ. Literature Search: YK, AÇ. Draft-ing manuscript: YK, AÇ. Critical revision of manuscript: AÇ. Supervisor: AÇ.

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