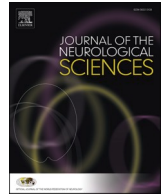Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Demographic and social determinants of cognitive dysfunction following hospitalization for COVID-19

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A B S T R A C T

Background: Persistent cognitive symptoms have been reported following COVID-19 hospitalization. We investigated the relationship between demographics, social determinants of health (SDOH) and cognitive outcomes 6-months after hospitalization for COVID-19.
Methods: We analyzed 6-month follow-up data collected from a multi-center, prospective study of hospitalized COVID-19 patients. Demographic and SDOH variables (age, race/ethnicity, education, employment, health insurance status, median income, primary language, living arrangements, and pre-COVID disability) were compared between patients with normal versus abnormal telephone Montreal Cognitive Assessments (t-MOCA; scores<18/22). Multivariable logistic regression models were constructed to evaluate predictors of t-MoCA.
Results: Of 382 patients available for 6-month follow-up, 215 (56%) completed the t-MoCA ($n = 109/215$ [51%] had normal and $n = 106/215$ [49%] abnormal results). 14/215 (7%) patients had a prior history of dementia/cognitive impairment. Significant univariate predictors of abnormal t-MoCA included older age, ≤ 12 years of education, unemployment pre-COVID, Black race, and a pre-COVID history of cognitive impairment (all $p < 0.05$). In multivariable analyses, education ≤ 12 years (adjusted OR 5.21, 95%CI 2.25–12.09), Black race (aOR 5.54, 95%CI 2.25–13.66), and the interaction of baseline functional status and unemployment prior to hospitalization (aOR 3.98, 95%CI 1.23–12.92) were significantly associated with abnormal t-MoCA scores after adjusting for age, history of dementia, language, neurological complications, income and discharge disposition.
Conclusions: Fewer years of education, Black race and unemployment with baseline disability were associated with abnormal t-MoCA scores 6-months post-hospitalization for COVID-19. These associations may be due to undiagnosed baseline cognitive dysfunction, implicit biases of the t-MoCA, other unmeasured SDOH or biological effects of SARS-CoV-2.

1. Introducion

Cognitive dysfunction following COVID-19 infection has been described in both the acute and post-acute phases of illness [1–16], and constitutes an important component of the Post-Acute Sequelae of SARS-CoV-2 Infection (PASC) syndrome. Several potential mechanisms related to secondary effects of SARS-CoV-2 infection may contribute to cognitive impairment, such as hypoxia, hyperinflammation, hypercoagulability, blood brain barrier disruption, uremia, septic encephalopathy or autoimmune mechanisms [17]. Beyond these biological effects, extensive literature also suggests a role for sociodemographic factors—including race, ethnicity, education, healthcare access and economic environment—in modulating cognitive outcomes and

recovery following hospitalization [18–21]. Additionally, pandemic-related lockdowns, school closures and economic stressors appear to contribute to both mental health and cognitive sequelae [22,23]. In the context of COVID-19, racial/ethnic disparities and other social determinants of health (SDOH) have been linked to higher rates of SARS-CoV-2 infection, severity of illness and mortality rates [24–26]. However, the role that SDOH may play in the development of PASC-related cognitive dysfunction has not been addressed.

In this study, we evaluated the relationship between demographics, SDOH and cognitive outcomes 6-months after index COVID-19 hospitalization. We further characterized the subdomains of cognitive dysfunction within this patient cohort. We hypothesized that racial/ethnic minority groups, patients with less access to the health care

system, and those with lower education levels would have higher rates of cognitive deficits following severe COVID-19.

2. Methods

2.1. Study design

We conducted a retrospective analysis of COVID-19 patients who were prospectively enrolled in the Study of Neurologic and Psychiatric Events in Acute COVID-19 (SNaP Acute COVID) [27] and who underwent follow-up interviews 6-months post-hospitalization [27].

2.2. Participants

Consecutive COVID-19 patients hospitalized in the New York City metropolitan area between March 10, 2020 and May 20, 2020 were prospectively screened for new neurological events by a team of neurologists [16,27]. We then conducted 6-month telephone follow-up interviews in patient populations with and without new neurological events during hospitalization. Patients underwent batteries which included cognitive, functional and neuropsychiatric assessments.

2.3. Inclusion and exclusion criteria

Inclusion criteria were: age ≥ 18 years, hospital admission for COVID-19 illness, reverse-transcriptase-polymerase-chain-reaction (RT-PCR) positive SARS-CoV-2 infection from nasopharyngeal sampling, and consent to participate in a follow-up interview. Exclusion criteria were: negative or missing SARS-CoV-2 RT-PCR test, in-hospital death outpatient or emergency department encounter only or inability to complete the t-MOCA assessment. Detailed screening and data acquisition protocols have been reported previously [16,27]. Patients were characterized as having a new neurological complication during hospitalization following acute SARS-CoV-2 infection (excluding recrudescence of prior neurological symptoms) based on evaluation of a board-certified neurologist. Neurological diagnoses – including toxic-metabolic encephalopathy, hypoxic-ischemic encephalopathy, stroke (ischemic or hemorrhagic), seizure, neuropathy, myopathy, movement disorder, encephalitis/meningitis, myelopathy, myelitis—followed established criteria [28–37].

Telephone interviews were conducted to obtain longitudinal 6-month follow-up assessments, as previously described [16]. Contact was attempted at 6-months (±1 month) from COVID-19 diagnosis. Three attempts at contact were required before patients/surrogates were coded as "unreachable".

2.4. Data collection

Demographic variables included: age, sex, race, ethnicity and zip code. SDOH variables included: health insurance status, median income, education level, non-fluency in English, employment status, living arrangements, disability status (as measured by the mRS) and marital status. Demographics, past medical history, hospital course, and discharge disposition were collected through review of medical records. Race, ethnicity, education, employment, primary language and non-fluency in the dominant language of the region, marital status and living arrangements (alone, with family, nursing home, rehabilitation center or other institution) were reported by the patient or their surrogate. A past history of dementia was coded for patients with pre-existing diagnoses of mild cognitive impairment, Alzheimer's type dementia, vascular dementia, Lewy body/Parkinson's related dementia, progressive supranuclear palsy, multiple system atrophy, corticobasal degeneration, frontal-temporal dementia, normal pressure hydrocephalus or Creutzfeld-Jakob disease. Pre-morbid baseline modified Rankin scale scores (mRS scores [38]) were collected based on patient/surrogate report at the 6-month interview [16]. Pre-COVID-19 employment status was categorized as: retired, part-time employment, full-time employment, not employed, student or other. Unemployment pre-COVID was coded only for subjects who described themselves as "not employed", excluding retired subjects and students. Median income was determined by zip code and obtained from online databases for New York City [39,40] and Nassau counties [41].

2.5. Study outcomes

We administered the Telephone/Blind Montreal Cognitive Assessment [42] (t-MoCA: 22 = perfect score; <18 = cutoff for cognitive impairment) in the primary language for each patient using standardized forms (English, Spanish, Chinese, and Russian). Interviews were conducted over the phone by trained physicians and medical students who were native speakers or trained translators of the patient's primary language. Patients were required to complete all components of the t-MoCA for scoring, and incomplete assessments or surrogate responses were excluded from analyses. Patients were required to complete the t-MoCA by themselves without surrogate assistance.

The t-MoCA consists of 22 items divided into the domains of attention (6 points, 3 items: digits forwards and backwards, identifying a target in a series, and serial subtraction), language (2 points, 1 item: sentence repetition), memory (5 points, 3-trial recall of 5 items with short-term uncued delayed recall), executive function (3 points, 2 items: verbal abstraction, categorical verbal fluency) and orientation (6 points, assessment of orientation to time and place) [43]. The score is total points earned plus an additional point given for those with ≤12 years of education [44]. Anxiety and depression symptoms were measured to assess for confounding of cognitive outcomes due to psychiatric comorbidities with the Quality of Life in Neurological Disorders [45] (Neuro-QoL) short form self-reported health measures of depression and anxiety. Neuro-QoL raw scores were converted into T-scores with a mean of 50 and standard deviation of 10 in a reference population (U.S. general population or clinical sample); T-scores ≥60 were considered abnormal (mean + 1SD). Higher T-scores indicate worse self-reported depression or anxiety.

2.6. Standard protocol approvals and patient consents

This study was approved by the NYU Grossman School of Medicine Institutional Review Board. All patients or their surrogates provided consent for participation.

2.7. Statistical analyses

Impaired t-MoCA scores were defined as <18 points based on published criteria [42]. Patients were grouped as with or without t-MoCA impairment and compared according to demographic variables, SDoH, premorbid medical comorbidities, clinical data, peri-COVID neurological complications, medical complications, medications administered during hospitalization, and in-hospital outcomes using the Mann-Whitney U test for non-normally distributed continuous variables and Chi-squared or Fisher's exact test, as appropriate, for categorical values. Multivariable, backward, step-wise logistic regression models were constructed to identify independent predictors of t-MoCA impairment 6-months post hospitalization utilizing univariate variables associated with p values<0.100. Patients with a baseline history of dementia or cognitive abnormality were excluded from multivariable analysis. All interactions were tested.

Domains of cognitive dysfunction were compared between patients with and without neurological complications during index hospitalization and among subgroups with significantly higher rates of t-MoCA impairment using Mann-Whitney U tests.

Correlation of t-MoCA scores and NeuroQoL depression and anxiety scores was assessed using Spearman's correlation coefficients. Statistical significance was set at $P < 0.05$. All analyses were conducted using IBM

SPSS Statistics for Mac version 25 (IBM Corp., Armonk, NY).

3. Results

Of 382 patients/surrogates who consented to 6-month follow-up, 215 (56%) completed the t-MOCA (Fig. 1). Nearly half of the sample had t-MoCA scores <18 (106/215 or 49% impaired, vs 109/215 or 51% not impaired). Impairment in t-MoCA scores was significantly associated with older age, Black race, ≤12 years of education, worse baseline mRS scores, unemployment prior to hospitalization, and a history of cognitive impairment (all *P* < 0.05, Table 1). There was a trend toward higher rates of t-MoCA impairment among lower income and non-English speaking patients. There were no differences in NeuroQoL anxiety or depression scores at 6-months between those with or without abnormal t-MoCA scores.

Comparing Black to non-Black patients, there were no significant differences in age, sex, health insurance status, rates of employment pre-COVID, baseline mRS score, education level, past medical history of diabetes, hypertension, COPD/asthma, chronic kidney disease, dementia, smoking, or coronary artery disease, severity of COVID-19 illness (SOFA scores or ventilator requirement) or discharge disposition. However, median household income was significantly lower in Black compared to non-Black patients ($65,112 [IQR $53,660–$75, 008] vs. $77,056 [IQR $54,646-93,488], *P* = 0.011). Rates of past psychiatric history were lower in Black than non-Black patients (0% vs. 12.5%, *P* = 0.035), however, NeuroQoL T-scores for self-reported depression and anxiety did not differ between Black patients versus patients of other races at 6-month follow-up.

Notably, we did not detect a relationship between cognitive outcomes and any hospital metric, including severity of illness as measured by ventilation status or Sequential Organ Failure Assessment (SOFA) scores [46–48], inpatient medications, medical or neurological complications or discharge disposition (Table 1). Though t-MoCA scores were significantly worse among patients with neurological complications during hospitalization (median scores of 17 [IQR 13–19] versus 18 [IQR 15–19], *P* = 0.036), the percentage of patients who scored in the impaired range (t-MoCA<18/22) did not differ between those with and without neurological complications (55% of patients with neurological events had abnormal 6-month t-MoCA versus 45% without neurological events, *p* = 0.155). There was no significant correlation between t-MoCA scores and NeuroQoL depression (Spearman's rho = −0.82, *p* = 0.240) or anxiety (Spearman's rho = −0.001, *p* = 0.988) scores measured 6-months post discharge.
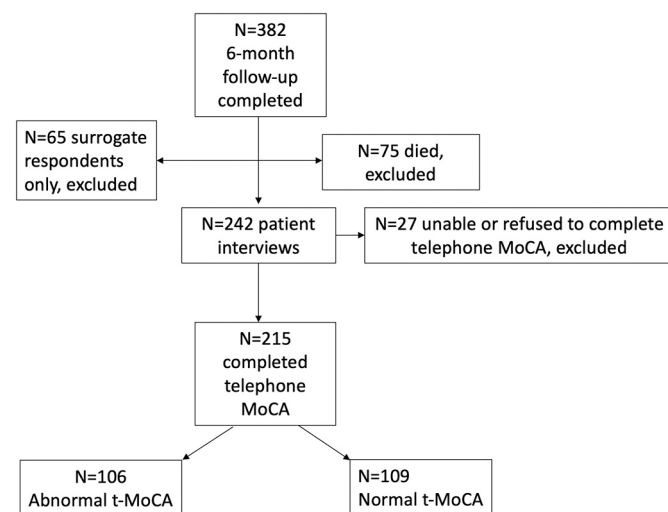

Fig. 1. Flow diagram of inclusion and exclusion for 6-month telephone Montreal Cognitive Assessment (MoCA) testing.

Table 1

Demographic and clinical factors associated with telephone MoCA scores 6-months after COVID-19 hospitalization.

Characteristic	Normal t-MoCA (N = 109)	Abnormal t-MoCA (N = 106)	P
Demographics and Social Determinants			
Median Age (IQR)-yr	62 (51–69)	68 (57–77)	**0.004**
Male sex-no./total no. (%)	72/109 (66%)	67/106 (63%)	0.662
Body Mass Index-median (IQR)	27 (23–33)	27 (25–33)	0.457
Years of education (≤12 years)	14/109 (13%)	30/106 (28%)	**0.005**
Unemployed prior to COVID hospitalization	7/109 (6%)	17/106 (16%)	**0.025**
Median household income by zip code (IQR)	$75,009 ($58,603–$99,193)	$70,089 ($53,669–$87,892)	0.078
Health Insurance Status			
Uninsured vs other	4/109 (4%)	2/106 (2%)	0.427
Uninsured or Medicaid vs other	8/109 (7%)	9/106 (9%)	0.754
Commercial Insurance vs other	67/109 (62%)	63/106 (59%)	0.760
Government insurance (Medicare or Medicaid) vs. other	38/109 (35%)	40/106 (38%)	0.661
Not fluent in English	22/109 (20%)	32/106 (30%)	0.091
Married (vs unmarried)	74/108 (69%)	65/106 (61%)	0.270
Lives alone	19/108 (18%)	19/100 (19%)	0.793
Race- no./total no. (%)			**0.014**
White	64/109 (59%)	45/106 (43%)	
Black	8/109 (7%)	26/106 (25%)	
Asian	12/109 (11%)	8/106 (8%)	
Native Hawaiian/Pacific Islander	1/109 (1%)	0	
American Indian	1/109 (1%)	0	
Other	23/109 (20%)	27/106 (25%)	
Ethnicity- no./total no. (%)			0.372
Latinx	27/108 (25%)	32/105 (31%)	
Non-Latinx	81/108 (75%)	73/105 (70%)	
Past Medical History Prior to COVID- no./total no. (%)			
Dementia	3/109 (3%)	11/106 (10%)	**0.023**
Psychiatric Illness	10/108 (9%)	9/106 (9%)	0.843
Stroke (ischemic or hemorrhagic)	9/109 (8%)	11/106 (10%)	0.593
Seizure	7/109 (6%)	8/106 (8%)	0.746
Movement Disorder	1/109 (1%)	0	0.323
Multiple sclerosis/demyelinating disease	2/109 (2%)	0	0.161
Chronic kidney disease	10/109 (9%)	14/106 (13%)	0.348
Hypertension	47/109 (43%)	38/106 (36%)	0.276
Diabetes	26/109 (24%)	29/106 (27%)	0.556
Coronary artery disease	15/109 (14%)	12/106 (11%)	0.589
COPD/Asthma	11/109 (10%)	11/106 (10%)	0.945
Atrial Fibrillation	7/108 (7%)	11/106 (10%)	0.305
Baseline modified Rankin scale score-median (range)	0 (0)	0 (0–1)	**0.024**
6-month Depression Neuro-QoL T-scores scores-median (IQR)	43 (37–48)	45 (37–51)	0.252
Abnormal depression scores (T-score ≥ 60)- no./total no. (%)	3/108 (3%)	1/102 (1%)	
6-month Anxiety Neuro-QoL T-scores scores-median (IQR)	48 (42–54)	48 (36–54)	0.995
Abnormal anxiety scores (T-score ≥ 60)no./total no. (%)	46/108 (43%)	48/103 (47%)	
Hospital Course			
Date of Index COVID-19 Admission -median (IQR)	April 3, 2020 (Mar 26-Apr 9)	April 4, 2020 (Mar 27-Apr 12)	0.759
Intubation- no./total no. (%)	41/109 (38%)	33/106 (31%)	0.317
Worst SOFA score- median (IQR)	4 (3–8)	4 (3–7)	0.663
Lowest Oxygen saturation (%), median (IQR)	86% (77–90%)	88% (77–92%)	0.277
Lowest Mean Arterial Pressure (mmHg), median (IQR)	64 (55–72)	67 (53–74)	0.335
Acute renal failure- no./total no. (%)	15/109 (24%)	16/106 (15%)	0.781
Hypotension requiring vasopressors- no./total no. (%)	33/109 (30%)	27/106 (26%)	0.432
Neurological Disorders during Hospitalization			

Table 1 (*continued*)

Characteristic	Normal t-MoCA (N = 109)	Abnormal t-MoCA (N = 106)	P
Any neurological event during index hospitalization	63/109 (58%)	51/106 (45%)	0.155
Toxic/Metabolic Encephalopathy- no./total no. (%)	19/109 (17%)	28/106 (26%)	0.111
Stroke (any type) - no./total no. (%)	5/109 (5%)	4/106 (4%)	0.766
Ischemic/TIA	4/109 (4%)	4/106 (4%)	0.968
Intracerebral/Intraventricular hemorrhage	1/109 (1%)	1/106 (1%)	0.984
Spontaneous Subarachnoid hemorrhage	0	0	–
Seizure (clinical or electrographic) - no./total no. (%)	5/109 (5%)	4/106 (4%)	0.766
Hypoxic/ischemic brain injury- no./total no. (%)	9/109 (8%)	10/106 (9%)	0.761
Movement Disorder- no./total no. (%)	8/109 (7%)	2/106 (2%)	0.058
Neuropathy- no./total no. (%)	5/109 (5%)	6/106 (6%)	0.721
Myopathy- no./total no. (%)	5/109 (5%)	2/106 (2%)	0.265
Guillain Barre Syndrome- no./total no. (%)	1/109 (1%)	1/106 (1%)	0.984
Encephalitis/meningitis- no./total no. (%)	0	0	–
Myelopathy/Myelitis- no./total no. (%)	0	0	–
Medications during Hospitalization			
Corticosteroids- no./total no. (%)	31/109 (28%)	30/106 (28%)	0.982
Hydroxychloroquine- no./total no. (%)	86/109 (79%)	74/106 (70%)	0.127
Azithromycin- no./total no. (%)	80/109 (73%)	69/106 (65%)	0.187
Remdesivir- no./total no. (%)	0	0	–
Therapeutic anticoagulation- no./total no. (%)	38/109 (35%)	37/106 (35%)	0.995
Tocilizumab- no./total no. (%)	0	0	–
Inflammatory Markers at Hospital Admission			
Admission IL-6 (pg/mL) - median (IQR)	40 (15–73)	25 (13–51)	0.220
Admission D-Dimer (ng/mL)- median (IQR)	454 (254–685)	431 (264–926)	0.902
Admission ferritin (ng/mL)- median (IQR)	905 (329–1530)	625 (252–1221)	0.111
Admission CRP (mg/L)- median (IQR)	109 (35–183)	128 (47–187)	0.411
Hospital Outcomes			
Home- no./total no. (%)	76/106 (72%)	70/102 (69%)	0.628
Acute rehabilitation facility- no./total no. (%)	16/106 (15%)	8/104 (8%)	0.092
Nursing home - no./total no. (%)	11/106 (10%)	20/104 (19%)	0.071
LTAC- no./total no. (%)	2/106 (2%)	4/104 (4%)	0.394
Length of Stay- median (IQR)	11 (5–31)	8 (3–22)	0.133
Ventilator Days- median (IQR)	11 (2–20)	12 (7–27)	0.180

Abnormal MoCA<18/22.

In multivariable logistic regression analysis, excluding patients with a pre-COVID history of dementia and after adjusting for univariate predictors of 6-month t-MoCA impairment (Table 1) including age, Black race, years of education, pre-hospitalization employment status, primary language other than English, median household income, baseline mRS, movement disorder diagnosed during hospitalization, any neurological event during index COVID-19 hospitalization, discharge to nursing home, discharge to acute rehabilitation center and relevant interactions, significant, independent predictors of 6-month t-MoCA impairment were Black race (adjusted OR 5.54, 95% CI 2.25–13.66), ≤12 years of education (adjusted OR 5.21, 95% CI 2.25–12.09), and the interaction of baseline mRS score and unemployment prior to COVID-19 hospitalization (adjusted OR 3.98, 95% CI 1.23–12.92); Table 2, AUC 0.744 [95% CI 0.674–0.814]). The interactions of Black race with age, baseline mRS, education≥12 years, median household income and

Table 2

Multivariable predictors of abnormal telephone MoCA at 6-months post-hospitalization.

Variable	Adjusted Odds Ratio (95% CI)	P
Education≤12 years	5.21 (2.25–12.09)	<0.001
Black race	5.54 (2.25–13.66)	<0.001
Interaction of baseline mRS and unemployment pre-COVID hospitalization	3.98 (1.23–12.92)	0.021

mRS = modified Rankin Score.

All univariate variables with $P < 0.100$ pushed into backward stepwise logistic regression analysis. Variables not in final model: age, history of dementia/ cognitive impairment, baseline mRS, movement disorder diagnosed during hospitalization, any neurological event during index COVID-19 hospitalization, discharge to nursing home, primary language non-English, median household income, discharge to acute rehabilitation center.

unemployment pre-COVID did not significantly predict abnormal t-MoCA scores.

We next evaluated individual t-MoCA cognitive domains (Tables 3 and 4). Overall, memory was the area of weakest performance, with only 14% of respondents achieving the maximum possible score, but without significant difference between those patients with and without neurological complications during hospitalization. However, those with neurological complications scored significantly worse than those without in the domains of attention and language (Table 3). Comparing Black respondents to other races, scores in the domains of attention, memory and executive functioning were significantly lower, while those with lower levels of education scored lower in these domains as well as the language domain (Table 4). Patients who were unemployed prior to COVID hospitalization demonstrated abnormalities across all domains of attention, language, memory, executive function and orientation (Table 4).

Table 3

Domains of t-MoCA compared between patients with and without neurological complications during hospitalization for COVID-19.

t-MoCA Domain	Overall	Neuro Event	No Neuro Event	P
Attention (max 6 points),				
Median (range)	5 (1–6)	5 (1–6)	5 (1–6)	**0.003**
N (%) with maximum score	87/223 (39%)	32/105 (31%)	55/118 (47%)	
Language (max 2 points)				
Median (range)	2 (0–2)	2 (0–2)	2 (0–2)	**0.039**
N (%) with maximum score	134/223 (60%)	56/105 (53%)	78/118 (66%)	
Memory (max 5 points)				
Median (range)	3 (0–5)	3 (0–5)	3 (0–5)	0.790
N (%) with maximum score	30/223 (14%)	17/105 (16%)	13/118 (11%)	
Executive Function (max 3 points)				
Median (range)	2 (0–3)	2 (0–3)	2 (0–3)	0.108
N (%) with maximum score	86/221 (39%)	37/105 (35%)	49/116 (42%)	
Orientation (max 6 points)				
Median (range)	6 (3–6)	6 (3–6)	6 (3–6)	0.967
N (%) with maximum score	190/222 (86%)	89/104 (86%)	101/118 (86%)	
Total Score (max 22 points)				
Median (range)	18 (7–22)	17 (7–22)	18 (8–22)	**0.036**
N (%) with normal score (≥18)	109/215 (51%)	46/101 (46%)	63/114 (55%)	

Patients were included in domain-specific analyses if all components of the domain were completed; Total score only calculated if all domains completed; P represents Mann-Whitney U for continuous variable.

Table 4

Domains of cognitive dysfunction in groups that were independently associated with abnormal t-MoCA scores in multivariable analyses.

t-MoCA Domain	Overall	Black	Other Races	P	Education >12 years	Education ≤12 years	P	Employed Pre-COVID	Unemployed Pre-COVID	P
Attention (max 6 points)				**0.012**			**<0.001**			**<0.001**
Median (range)	5 (1–6)	4 (1–6)	5 (1–6)		5 (1–6)	4 (1–6)		5.5 (1–6)	4 (1–6)	
N (%) with max score	87/223 (39%)	7/36 (19%)	79/185 (43%)		79/174 (45%)	6/46 (13%)		62/124 (50%)	25/99 (25%)	
Language (max 2 points)				0.155			**0.030**			**0.014**
Median (range)	2 (0–2)	1.5 (0–2)	2 (0–2)		2 (0–2)	1 (0–2)		2 (0–2)	2 (0–2)	
N (%) with max score	134/223 (60%)	18/36 (38%)	115/185 (62%)		109/174 (63%)	22/46 (48%)		82/124 (66%)	52/99 (53%)	
Memory (max 5 points)				**0.016**			**<0.001**			**<0.001**
Median (range)	3 (0–5)	2 (0–5)	3 (0–5)		3 (0–5)	2 (0–5)		3 (0–5)	2 (0–5)	
N (%) with max score	30/223 (14%)	1/36 (3%)	28/185 (15%)		26/174 (15%)	3/46 (7%)		22/124 (18%)	8/99 (8%)	
Executive Function (max 3 points)				**0.025**			**<0.001**			**<0.001**
Median (range)	2 (0–3)	2 (0–3)	2 (0–3)		2 (0–3)	1 (0–3)		2 (0–3)	2 (0–3)	
N (%) with max score	86/221 (39%)	9/36 (25%)	77/183 (42%)		75/173 (43%)	8/45 (18%)		61/124 (49%)	25/97 (26%)	
Orientation (max 6 points)				0.343			0.566			**0.005**
Median (range)	6 (3–6)	6 (4–6)	6 (3–6)		6 (4–6)	6 (3–6)		6 (5–6)	6 (3–6)	
N (%) with max score	190/222 (86%)	28/35 (80%)	160/185 (87%)		148/174 (85%)	40/45 (89%)		114/125 (91%)	76/97 (78%)	
Total Score (max 22 points)				**0.001**			**0.002**			**<0.001**
Median (range)	18 (7–22)	15 (9-22)	18 (7–22)		18 (8–22)	16 (7–22)		18 (8–22)	16 (7–21)	
N (%) with normal score (≥18)	109/215 (51%)	8/34 (24%)	100/179 (56%)		95/171 (56%)	14/44 (32%)		76/121 (63%)	33/94 (35%)	

Patients were included in domain-specific analyses if all components of the domain were completed; Total score only calculated if all domains completed; P represents Mann-Whitney U for continuous variable.

4. Discussion

In this study, we found that Black race, fewer years of formal education, and pre-hospitalization unemployment in the context of disability may be significant predictors of cognitive dysfunction as measured by the t-MoCA 6-months after hospitalization for COVID-19, even after adjusting for other factors. Contrary to our expectation, we found that cognitive impairment was independent of any hospital metric or recorded measure of illness severity including ventilator status, Sequential Organ Failure Assessment (SOFA) scores [46–48], inpatient medications, inflammatory markers, medical or neurological complications, and discharge disposition. This may be because the most severely ill patients either died in-hospital or were too impaired to complete the 6-month the t-MoCA assessment. Strengths of our study include its prospective ascertainment of data, self-reported race/ethnicity and employment status, a large sample size, initial neurological diagnoses by board-certified neurologists, adjustment for multiple possible confounders of cognitive dysfunction, and assessment of alterations in cognition that could be due to depression or anxiety.

Health disparities, including increased rates of SARS-CoV-2 infection, increased severity of illness and increased mortality have been reported among racial/ethnic minority groups, particularly among Black and Latinx populations [25,49]. Traditionally, racial health disparities for general medical conditions have been ascribed to differences in rates of chronic medical conditions, such as diabetes, hypertension, chronic kidney disease or smoking. However, we found that worse cognitive scores were independent of premorbid conditions that are found at higher rates in Black and Latinx populations, including hypertension, diabetes, obesity and chronic kidney disease. Furthermore, we did not detect any differences in baseline demographics, education, health insurance status, or employment status between Black and non-Black patients, apart from income level as estimated by zip code. This finding is important because it not only implies the significant role that financial status may play in health outcomes, but also suggests regional community variability in health outcomes. However, in multivariable analysis, even after controlling for household income/zip code, Black

patients still had worse cognitive outcomes than other race groups, suggesting that additional unmeasured factors are at play. Previous studies have shown an increased risk of testing positive for COVID-19 among Black and Latinx patients compared with White patients [25]. This suggests that differences in exposure to SARS-CoV-2 and access to outpatient care post hospitalization between race groups may also impact cognitive outcomes. For example, we did not collect data on certain SDOH such as access to outpatient care post-hospitalization, which may impact cognitive outcomes.

Studies have found that lower MOCA cut-points among Black and Latinx patients compared to non-Latinx White patients have better positive and negative predictive value for the clinical diagnoses of mild cognitive impairment and Alzheimer's Disease [50]. Therefore, it is also possible that there are biases intrinsic to the t-MOCA test that could unfairly impact different race/ethnic groups. Compared to the full MOCA, the shorter t-MOCA has been found to have reasonable, albeit less sensitive and specific, discriminative ability for detecting mild cognitive impairment or dementia across demographically diverse racial/ethnic populations independent of age, education level, depressive symptoms, and/or issues with telephone administration [51]. However, since the standard MOCA has been found to have varying levels of discrimination between mild cognitive impairment (MCI), normal aging, and dementia in Black and Latinx populations [52], it is possible that our results are affected by unmeasured testing biases that are specific to Black populations.

Another possible explanation may be that the biological effects of COVID-19 differ based upon genetic differences encountered in different racial/ethnic groups [53,54]. For example, genes regulating SARS-CoV-2 uptake, endosomal trafficking and cytokine signaling are differentially expressed in Black Americans compared to European White Americans with COVID-19 [55], which may contribute to more severe COVID-19 symptoms and worse cognitive outcomes among Black patients. However, we did not detect any differences in severity of COVID-19 illness (e.g. intubation status, oxygen or blood pressure nadirs, or SOFA scores) between patients with or without abnormal 6-month t-MoCA scores or between Black and non-Black patients. This suggests that the genetic

differences that have been identified, thus far [55], may not be clinically significant and that biological effects may not be the primary drivers of the observed differences in cognitive performance by race. Rather, social and economic disparities, nutritional variability [56], and systematic discriminatory environments may be contributing to observed cognitive differences. Apart from COVID-19, it is notable that the incidence of dementia, in general, in the U.S. is highest in Black Americans and Native Americans/Native Alaskans, intermediate in White, Latinx and Pacific IslanderAmericans and lowest among Asian Americans, after adjusting for age, sex, medical utilization and comorbidities [57]. Current literature suggests that this increased risk of dementia among Black individuals is more likely attributed to pervasive social inequities and racially related life-course factors, such as differences in education attainment, access to material and social resources, exposure to discrimination, and exposure to neurotoxins, rather than genetic predisposition [58].

The higher likelihood of cognitive impairment among patients with fewer years of formal education in our study was expected, given the well-established relationship between low education attainment and cognitive impairment [59]. This may be because individuals with more education possess a greater cognitive reserve and capacity to recruit eloquent brain regions, which allows them to sustain a larger degree of brain pathology before clinical impairment becomes apparent [59]. Though we adjusted for education level, there is no validated way to adjust for quality of education, which varies widely across metropolitan NYC [60]. Additionally, the role of education in moderating decline or promoting recovery after infection with SARS-CoV-2 remains unclear. In our cohort, patients with less education achieved lower scores in the domains of attention, memory, and executive function, and performed similarly in the domains of language and orientation compared with patients with more education. Previous studies have found similar patterns in patients with COVID-19 [61–64] and post sepsis [65]. Long-term monitoring of trajectories of cognition will be needed to determine the likelihood that this profile could progress to other neurocognitive disorders such as Alzheimer's disease (AD).

This study has limitations. First, we excluded patients with mild COVID-19 disease that was not severe enough to warrant hospitalization, which limits the generalizability of our findings to patients with mild COVID-19. Conversely, the most severely affected patients with profound deficits in cognition or level of alertness were unable to complete the t-MOCA, so we likely have underestimated the percentage of hospital survivors with abnormal cognition. Deficits in attention may also affect patients' ability to complete the t-MOCA, resulting in lower scores in all other cognitive domains. Though we accounted for baseline history of dementia and baseline mRS, it is possible that some patients had undiagnosed cognitive disorders. Indeed, patients who had worse baseline mRS and were unemployed before hospitalization for COVID-19 were also more likely to score poorly on the t-MOCA. It is possible that the underlying disability (as measured by the mRS), which was severe enough to preclude employment, also contributed to undiagnosed pre-COVID cognitive dysfunction. Despite this, the estimated prevalence of mild cognitive impairment in the general community among patients aged 65–69 years (the median age of our population) is only 8.4% [66] and the prevalence of dementia is <1% [67], suggesting that undiagnosed cognitive impairment is likely uncommon. Although our cohort derives from a large, diverse catchment area, it may not be representative of observations made in other parts of the country. Our screening for depression and anxiety with the Neuro-QoL batteries did not indicate that emotional distress contributed to the current findings. However, our study was limited by the use of this brief self-reported screening measure. We note also that rates of reported psychiatric history among Black patients in our sample were lower than would be expected. Underreporting of psychiatric illness, as well as concerns about prejudice and discrimination related to psychiatric diagnoses, have been shown to vary by race/ethnicity and gender [68–70]. Given the emotional distress that can occur with COVID-19 illness, future

studies should include a more comprehensive measurement of emotional health. Finally, lack of a SARS-CoV-2 negative control group in our study limits our ability to determine if cognitive deficits are a direct consequence of SARS-CoV-2 infection. Despite this limitation, we did adjust for confounders and serious abnormalities were identified in patients with no history of cognitive impairment, suggesting that either SARS-CoV-2 or its secondary complications may be implicated in the development of abnormal cognition.

5. Conclusions

Our study suggests that Black race, fewer years of education, and pre-hospitalization unemployment in the context of disability may predict worse cognitive performance, as measured by the t-MOCA, 6 months after hospitalization with COVID-19. To better characterize the nature and implications of these findings, future studies utilizing more extensive cognitive batteries in diverse populations with and without COVID-19 are needed. Such studies may guide future interventions and treatments to improve cognitive outcomes following hospitalization with COVID-19.

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