BRAIN COMMUNICATIONS

Frequent neurocognitive deficits after recovery from mild COVID-19

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Neuropsychiatric complications associated with coronavirus disease 2019 caused by the Coronavirus SARS-CoV-2 (COVID-19) are increasingly appreciated. While most studies have focussed on severely affected individuals during acute infection, it remains unclear whether mild COVID-19 results in neurocognitive deficits in young patients. Here, we established a screening approach to detect cognitive deficiencies in post-COVID-19 patients. In this cross-sectional study, we recruited 18 mostly young patients 20–105 days (median, 85 days) after recovery from mild to moderate disease who visited our outpatient clinic for post-COVID-19 care. Notably, 14 (78%) patients reported sustained mild cognitive deficits and performed worse in the Modified Telephone Interview for Cognitive Status screening test for mild cognitive impairment compared to 10 age-matched healthy controls. While short-term memory, attention and concentration were particularly affected by COVID-19, screening results did not correlate with hospitalization, treatment, viremia or acute inflammation. Additionally, Modified Telephone Interview for Cognitive Status scores did not correlate with depressed mood or fatigue. In two severely affected patients, we excluded structural or other inflammatory causes by magnetic resonance imaging, serum and cerebrospinal fluid analyses. Together, our results demonstrate that sustained sub-clinical cognitive impairments might be a common complication after recovery from COVID-19 in young adults, regardless of clinical course that were unmasked by our diagnostic approach.

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Keywords: COVID-19; post-COVID-19; neurocognitive deficits; neurocognitive screenings

Abbreviations: CNS = central nervous system; COVID-19 = Coronavirus disease 2019 caused by the Coronavirus SARS-CoV-2; CSF = cerebrospinal fluid; FDR = false-discovery rate; MCI = mild cognitive impairment in elderly adults; MERS-CoV = Middle-east-respiratory-syndrome-coronavirus; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2; TICS-M = Modified Telephone Interview for Cognitive Status

Received August 3, 2020. Revised October 31, 2020. Accepted November 6, 2020. Advance Access publication November 23, 2020
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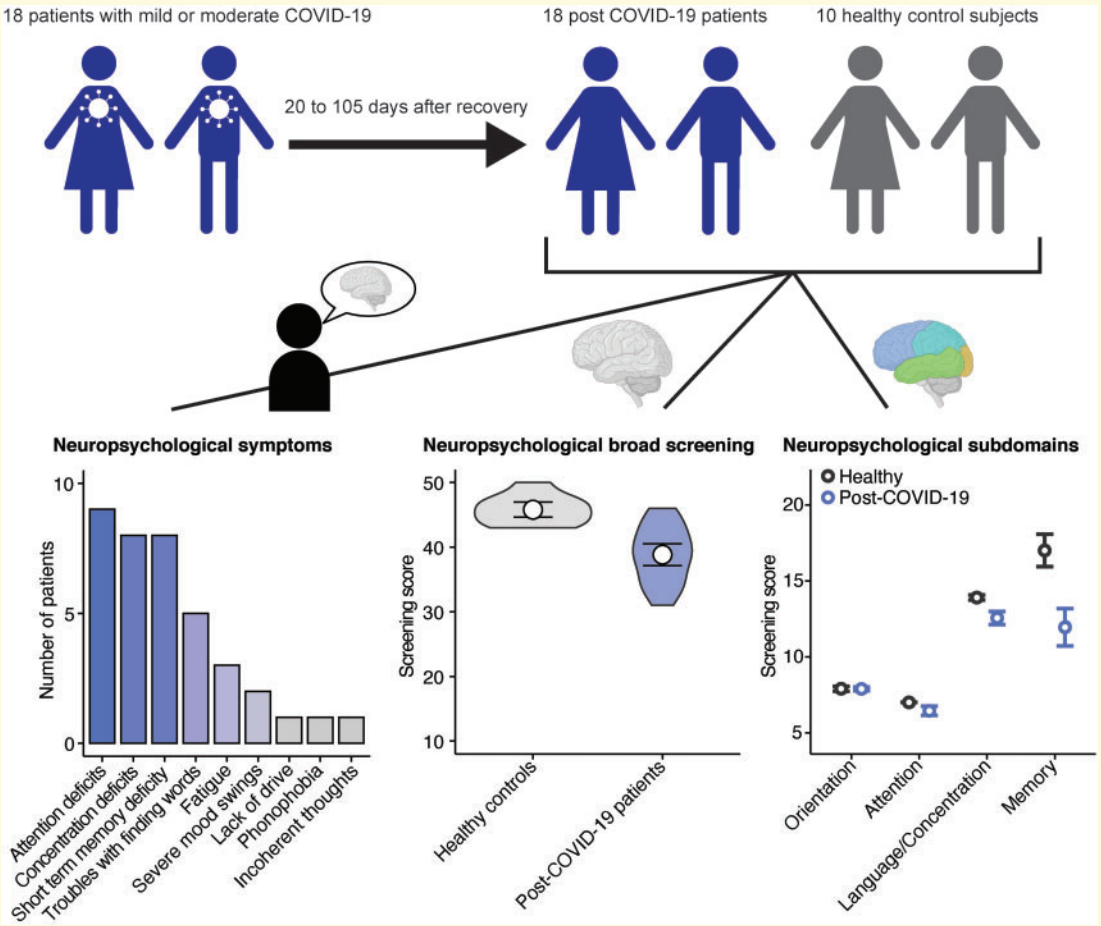
Graphical Abstract



Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was first identified in December 2019 as the cause of a respiratory illness designated coronavirus disease 2019 (COVID-19) (Huang *et al.*, 2020). While patients with COVID-19 frequently suffer from respiratory symptoms, neurologic and neuropsychiatric complications have been increasingly reported (Ellul *et al.*, 2020; Varatharaj *et al.*, 2020). Moreover, histopathologic examination of brains from deceased COVID-19 patients indicate the potential of SARS-CoV-2 to infiltrate the central nervous system (CNS) (Solomon et al., 2020).

Reported neuropsychiatric manifestations include milder symptoms like dizziness and anosmia (Hornuss et al., 2020) but also in rare cases severe manifestations such as acute demyelinating encephalopathy (Reichard *et al.*, 2020), meningitis (Moriguchi *et al.*, 2020) and strokes (Helms *et al.*, 2020; Oxley *et al.*, 2020). Recently, the symptoms of 153 patients with COVID-19 from the UK who reported neurologic and psychiatric complications during the acute phase of the disease were reported and intra-cerebral haemorrhages and altered mental status were the most common complications (Varatharaj *et al.*, 2020). Similarly, in a study from Wuhan with 214 patients, 78 patients reported unspecific neurological symptoms and 13 patients had a new cerebrovascular diagnosis during acute infection (Mao *et al.*, 2020).

These studies have focussed on severe neurologic and neuropsychiatric complications during the acute infection but did not include sustained neuropsychological deficits after full recovery from COVID-19. Moreover, severe neurologic complications have been mostly investigated in patients with multiple risk factors who developed severe COVID-19 with complications but not in young adults after recovery. For the outbreaks of the closely related SARS-CoV-2 and middle-east-respiratory-syndrome-coronavirus (MERS-CoV), acute delirium and encephalitis have been reported during acute disease but also sustained neuropsychologic syndromes (Rogers *et al.*, 2020). Thus, deeper analysis and epidemiologic (Ritchie *et al.*, 2020) studies as well as development of screening tools for mild cognitive deficits in young adults are an important unmet clinical need to detect sub-clinical neuropsychologic symptoms and help to differentiate unspecific post-illness-manifestations.

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Here, we established a facile screening approach for cognitive deficits in 18 young patients without diagnosed cognitive pre-conditions after recovery from COVID-19 and discovered widespread sub-clinical deficits.

Materials and methods

Patient cohorts

For this cross-sectional study, we randomly interviewed patients from the outpatient clinic of the University Medical Centre Hamburg-Eppendorf (UKE) and included only the patients who did not stay at intensive care unit. In total, 21 patients were approached and 18 agreed to participate in our study. The severity of COVID-19 into mild, moderate, severe, critical and lethal disease courses was classified using the WHO criteria (WHO reference number: 451 WHO/2019-nCoV/clinical/2020.5). We included only the patients who suffered from mild and moderate COVID-19 and were not admitted to our intensive care unit. Since our patient cohort was considerably younger (mean age, 42.2 years; SD 14.3 years), we additionally tested healthy individuals with similar age (mean age, 38.4 years; SD 14.4 years). Randomly selected healthy individuals were the employees of the University Medical Center Hamburg-Eppendorf, who did not have prior knowledge of the Modified Telephone Interview for Cognitive Status (TICS-M) or similar neuropsychological screenings and were matched for age within a range of 5 years but not for sex. Except for one patient who still visited high school, all patients and healthy participants received in total more than 12 years of education. The patient-reported symptoms were collected by individual reports and documented symptoms during the inpatient and subsequent outpatient stays at the University Medical Center Hamburg-Eppendorf. During the interviews, we questioned the patients in a structured manner that included all organ systems and specifically asked for neuropsychological deficits.

Assessment tools

The interviews were either conducted by phone or directly with the patient. Individuals were recruited until 14 July 2020. To sensitively screen for mild cognitive deficiencies, we utilized the TICS-M that was originally developed to broadly screen for mild cognitive impairment in elderly adults (MCI) by telephone. This questionnaire has been validated for amnestic mild cognitive impairment (reference for mean age, 74.9 years; amnestic mild cognitive impairment, <34) (Cook *et al.*, 2009) and Alzheimer's dementia (Manly *et al.*, 2011). We performed TICS-M according to the previously published protocols (Cook *et al.*, 2009). The total interview lasted 15–20 min. Four domains are tested by the TICS-M: (i) orientation, (ii) recent memory and delayed memory, (iii) attention and (iv) semantic memory, comprehension and repetition (language/concentration) (De Jager *et al.*, 2003). The TICS-M included the following items: (i) name, (ii) age, (iii) date, (iv) weekday, (v) season, (vi) phone number (each 1 point), (vii) counting backward (2 points), (viii) first, a 10-word list learning exercise and then a delayed (21) recall of that word list (10 points each), (ix) subtractions (5 points); (x–xiii) responsive naming (4 points), (xiv–xv) repetition (2 points), (xvi) current chancellor and (xvii) president of Germany (each 2 points), (xviii) finger tapping (2 points) and (xix, xx) word opposites (2 points). The total score was 50 points. The TICS-M score has been validated to test episodic memory for words, episodic memory for non-verbal information and attention (Crooks *et al.*, 2006). We controlled for possible biases by the Patient Health Questionnaire-9 Depression Scale (PHQ-9) and Fatigue Assessment Scale. Two patients with peculiarly low TICS-M scores (Vignette A, 39 points; Vignette B, 31 points) who reported severe restrictions in their everyday life due to their reported COVID-19-associated neuropsychological symptoms underwent further neurologic and neuropsychologic evaluation.

Neuropsychological and neurological assessment

Two patients were invited for further neuropsychological and neurological assessment at the outpatient clinic for neuroimmunological diseases at the Department of Neurology of the University Medical Center Hamburg-Eppendorf, Germany. Neurological examination was performed by board-certified neurologists. Further diagnostic measures included analyses of serological parameters, cerebrospinal fluid (CSF) and cranial imaging.

SARS-CoV-2 diagnostic procedures

We used Cobas6800 system (Roche, Mannheim, Germany) for the detection of SARS-CoV-2 RNA from nasopharyngeal smears by polymerase chain reaction as previously described in detail (Pfefferle *et al.*, 2020). For serology, we used Liaison XL system for quantitative SARS-CoV-2 IgG detection according to the manufacturer's recommendation.

Statistical analysis

Data were analysed within the R environment (Version 1.2.5.002) on a Mac OS X. Unless stated otherwise, comparisons between two experimental groups are presented as violin plot with median or 95% confidence intervals and differences were determined using two-tailed, unpaired Wilcoxon–Mann–Whitney test and were false-discovery rate (FDR)-corrected for multiple comparisons. To find the predictors of screening results, we used multiple linear regression models. The results were FDR-

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corrected for multiple comparisons. Exact *P*-values are reported in respective sections of the article and figure legends. We analysed the scores of Fatigue Assessment Scale and Patient Health Questionnaire-9 Depression Scale as well as age, length of hospitalization, sickness duration, time from recovery to neurocognitive assessment, maximal PCR cycle threshold values, maximal antibody titres, maximal CRP, IL-6, ferritin and D-dimers as predictors. The outcome variable was the TICS-M score. For effect size of non-parametric comparisons, we calculated *Rosenthals r*. Significant results are indicated by $*P < 0.05$, $**P < 0.01$ and $***P < 0.001$.

Ethics statement

The study was approved by the *Ärztekammer Hamburg*. All patients and healthy participants gave consent for participation, data analysis and publication.

Data availability

Data are available from the corresponding author, upon reasonable request. Data are not publicly available due to ethical restrictions because the information contained on those data could compromise the privacy of the reported patients.

Results

The aim of this study was to establish a screening that sensitively and specifically detects subtle neurocognitive deficits. Therefore, we screened our post-COVID-19 outpatient clinic for mostly young patients with mild to moderate disease courses according to the WHO criteria (WHO reference number: 451 WHO/2019-nCoV/clinical/2020.5) without known cognitive pre-conditions who recovered without complications. We included 10 females and 8 males at ages ranging from 17 to 71 years (mean, 42.2 years; SD, 14.3 years). Our cohort consisted of 11 inpatients (61%), 6 outpatients (33%) and 1 patient did not seek medical care (6%). During the acute infection, four patients received supplementary oxygen, two patients were treated with remdesivir and one patient with tocilizumab due to cytokine storm. None of the patients received intensive care and no vascular or structural neurological event was recorded. All patients recovered without severe complications 20–105 days (median, 85 days from COVID-19 recovery to assessment time) prior to the timepoint of our screening (a summary of patients' characteristics is provided in Table 1). In addition, we tested 10 healthy individuals with similar age ($n = 10$; mean age, 38.4 years; SD, 14.4 years; a summary of healthy controls' characteristics is provided in Table 1) as control group.

We chose the TICS-M as primary tool (Cook *et al.*, 2009) as it has been extensively validated for screening of mild cognitive deficiencies by telephone. This we considered important when conducting larger studies that include affected individuals who might not seek professional health care and stay in home quarantine. Strikingly, post-COVID-19 patients scored significantly lower results in the TICS-M (mean, 38.83; range, 31–46) compared to healthy controls (mean, 45.8; range, 43–50) (Fig. 1A), especially regarding short-term memory, attention and concentration/language tasks (Fig. 1B). Notably, results from screening for depression (Personal Health Questionnaire 9) and for fatigue (Fatigue Assessment Scale) did not show significant correlation with TICS-M scores (Fig. 1C and D). In terms of patients' self-reported symptoms, out of 18 included individuals 9 (50%) reported attention deficits, 8 (44.4%) concentration deficits, 8 (44.4%) short-term memory deficits, 5 (27.8%) troubles in finding words, 3 (16.7%) fatigue, 2 (11.1%) severe mood swings and 1 (5.6%) sustained lack of energy, phonophobia and incoherent thoughts (Fig. 1E).

Next, we aimed to find the predictors of our observed cognitive deficits. First, we analysed patients' characteristics and found that neither sex (Fig. 2A) nor age (Fig. 2B) could explain the observed differences. Additionally, we investigated whether severity of the acute COVID-19 disease could be an explanation. Therefore, we next analysed whether hospitalization had an impact on post-COVID-19 manifestations. However, we found that TICS-M scores in post-COVID-19 patients did not correlate with the time interval from our interview to recovery (Fig. 2C), length of sickness (Fig. 2D) and length of inpatient stay (Fig. 2E). To further evaluate the impact of disease severity, we correlated the neurocognitive deficits with acute and sustained somatic symptoms (Fig. 2F) and treatments during the acute infection. Our analysis revealed that the number of somatic symptoms did not correlate with the number of sustained self-reported neurocognitive deficits (Fig. 2G) and the results in our screening (Fig. 2H). Moreover, treatments such as oxygen supplementation (Fig. 3A) and drugs such as remdesivir, tocilizumab or antibiotics (Fig. 3B) in acute infection could not predict the observed cognitive impairments. Thus, our data imply that post-COVID-19 neuropsychologic deficits are independent from hospitalization and disease severity.

Subsequently, we analysed the impact of acute inflammation and maximal viremia in acute COVID-19 on neurocognitive deficits. Therefore, we accounted the cycle threshold from SARS-CoV-2 PCR, antibodies against SARS-CoV-2 and inflammatory serum markers. Our analysis revealed that maximal SARS-CoV-2 IgG-titres (Fig. 3C), SARS-CoV-2-PCR cycle threshold values (Fig. 3D), CRP (Fig. 3E), ferritin (Fig. 3F), IL-6 (Fig. 3G) and D-dimer (Fig. 3H) serum concentration during acute COVID-19 were not significant predictors of our observed neurocognitive deficits.

For further diagnostic measures, we investigated the two most severely affected patients by cranial MRI and lumbar puncture that excluded structural pathologies and

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Table 1 Summary of characteristics and manifestations of post-COVID-19 patients and healthy control subjects

Characteristics	Post-COVID-19 patients	Healthy controls
Mean age (range)	42.11 (17–71)	38.4 (22–59)
Age distribution (%)		
<20	1 (5.6)	0 (0)
20–40	8 (42.2)	6 (60)
40–60	8 (47.4)	4 (40)
>60	1 (5.2)	0 (0)
Sex (%)		
Female	10 (57.9)	4 (40)
Male	8 (42.1)	6 (60)
Pre-conditions (%)		
Asthma bronchiale	3 (16.7)	Not assessed
Hypothyreosis	3 (16.7)	Not assessed
Hypertonus	2 (11.1)	Not assessed
Coagulation disorder	2 (11.1)	Not assessed
Diabetes mellitus type 2	1 (5.6)	Not assessed
Multiple sclerosis	1 (5.6)	Not assessed
Autoimmune hepatitis	1 (5.6)	Not assessed
Follicular lymphoma	1 (5.6)	Not assessed
Clinical stay (%)		
Outpatient clinic	6 (68.5)	Not assessed
Inpatient clinic	11 (31.5)	Not assessed
Treatment (%)		
Oxygen supplementation	6 (33.3)	Not assessed
Remdesivir	3 (16.7)	Not assessed
Antibiotics	2 (11.1)	Not assessed
Tocilizumab	1 (5.6)	
Neuropsychiatric symptoms (%)		
Attention deficits	9 (50.0)	0 (0)
Concentration deficits	8 (44.4)	0 (0)
Short-term memory deficits	8 (44.4)	0 (0)
Troubles in finding words	5 (27.8)	0 (0)
Fatigue	3 (16.7)	0 (0)
Severe mood swings	2 (11.1)	0 (0)
Lack of energy	1 (5.6)	0 (0)
Phonophobia	1 (5.6)	0 (0)
Incoherent thoughts	1 (5.6)	0 (0)
Test screening results (range)		
TICS-M	38.83 (31–46)	45.8 (43–50)
Fatigue Assessment Scale	24.17 (13–40)	18.1 (18–19)
Patient Health Questionnaire-9 Depression Scale	2.83 (0–9)	0.7 (0–2)

acute inflammation. Detailed neuropsychologic evaluation confirmed deficits of attention, executive functions and memory (detailed case vignettes are reported in the supplementary material).

Discussion

SARS-CoV-2 affects multiple organ systems by infiltrating endothelial cells of blood vessels throughout the whole body (Varga et al., 2020). Thus, a multitude of symptoms and clinical disease courses have been described (Gupta *et al.*, 2020). Here, we focussed on the evaluation of neurocognitive post-COVID-19 manifestations in mostly young adults who recovered from acute uncomplicated COVID-19. This study demonstrates substantial neurocognitive deficits that sustain after recovery and advocate screening routines for cognitive deficits during medical care of post-COVID-19 patients.

Cerebrovascular events and altered mental status have been described to be common neuropsychiatric manifestations in acute COVID-19 in a nationwide surveillance study in the UK (Varatharaj *et al.*, 2020). In total, 59% of patients with altered mental status could be assigned to a neuropsychiatric disorder, underlining the diversity of COVID-19-associated manifestations. We screened patients after recovery from COVID-19 and found substantial neurocognitive deficits that sustained after acute infection. Moreover, we detected subtle cognitive deficits that did not restrain most patients in daily life and were only unmasked by our specific screening, including deficits in short-term memory, attention and concentration. Retrospective meta-analysis of SARS and MERS outbreaks has revealed acute and long-term neuropsychological deficits. Similar to our findings, most-common post-illness manifestations included impaired concentration and attention in 19.9% and impaired memory in 18.9% of patients after recovery (Rogers *et al.*, 2020).

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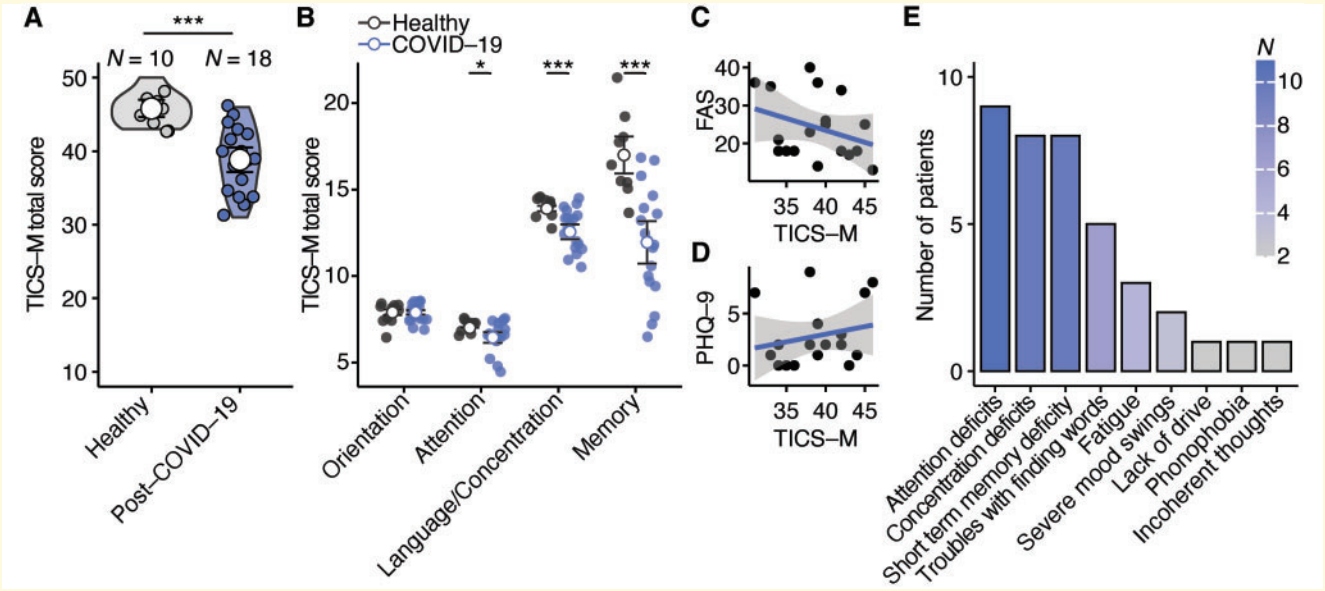

Figure 1 Cognitive deficiencies in post-COVID-19 patients. (**A**) Comparison of TICS-M total scores ($P = 0.0002$) between healthy individuals ($n = 10$) and post-COVID-19 patients ($n = 18$). Two-tailed Wilcoxon-test was used and mean with 95% confidence interval is shown. (**B**) Comparison of the different cognitive domains orientation ($P = 0.9643$), attention ($P = 0.029$), language and concentration ($P = 0.009$) and memory ($P = 0.004$) that were tested with the TICS-M. Two-tailed Wilcoxon-test was used and mean with 95% confidence interval is shown. (**C, D**) Linear regression analysis of TICS-M scores and Fatigue Assessment Scale (**C**; $t = -1.3653$, FDR-adjusted $P = 0.3820$, Estimate = –0.165) and Patient Health Questionnaire-9 Depression Scale (**D**; $t = 0.8957$, FDR-adjusted $P = 0.3836$, Estimate = 0.324) scores of post-COVID-patients. (**E**) Reported neuropsychiatric symptoms that sustained after recovery.

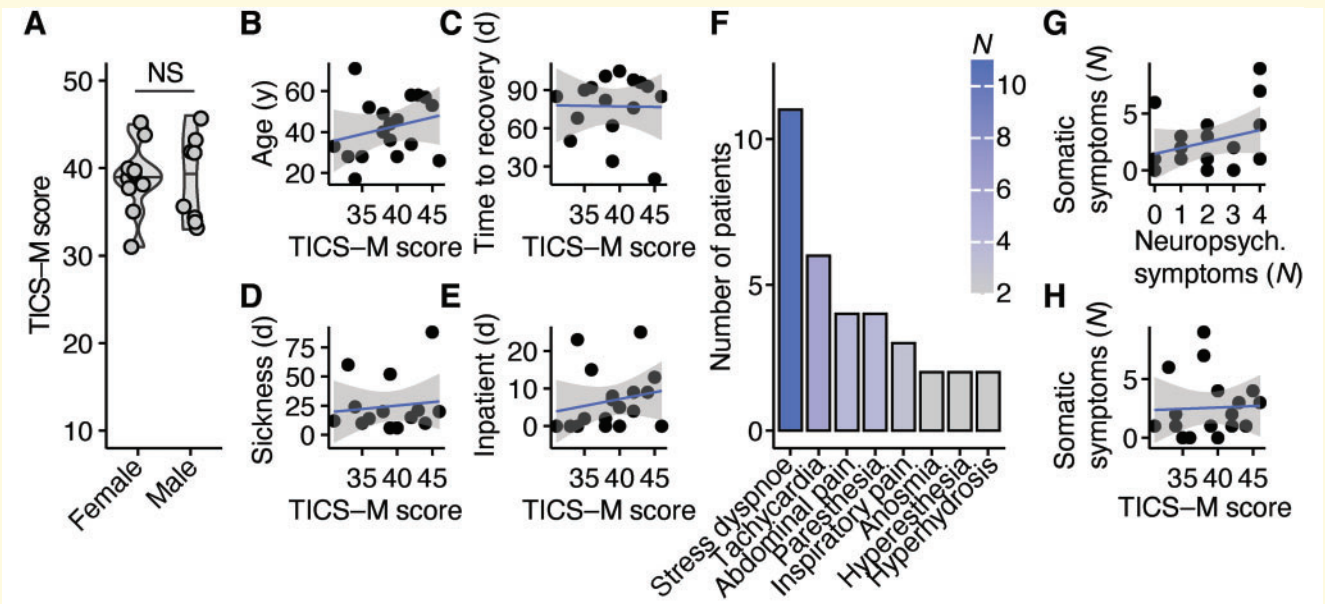

Figure 2 Cognitive deficits are independent from hospitalization and sickness duration. (**A**) Comparison of TICS-M total scores ($P = 0.9644$) between female ($n = 10$) and male ($n = 8$) post-COVID-19 patients. Two-tailed Wilcoxon-test was used. (**B–E**) Linear regression analysis of TICS-M scores and age in years (**B**; $t = 1.0241$, FDR-adjusted $P = 0.6420$, Estimate = 0.057), time to recovery from acute COVID-19 in days (**C**; $t = -0.0576$, FDR-adjusted $P = 0.9548$, Estimate = −0.003), duration of sickness in days (**D**; $t = -0.0576$, FDR-adjusted $P = 0.9548$, Estimate = 0.023) and duration of inpatient treatment in days (**E**; $t = 0.8254$, FDR-adjusted $P = 0.7021$, Estimate = 0.112) of post-COVID-patients. (**F**) Self-reported somatic symptoms that appeared at least once after recovery from COVID-19 and were reported from at least two patients. (**G,H**) Linear regression analysis of number of somatic and neurocognitive symptoms (**G**; $t = 1.282$, FDR-adjusted $P = 0.2181$, Estimate = 0.177) and number of somatic symptoms and TICS-M scores (**H**; $t = 0.161$, FDR-adjusted $P = 0.874$, Estimate = 0.068).

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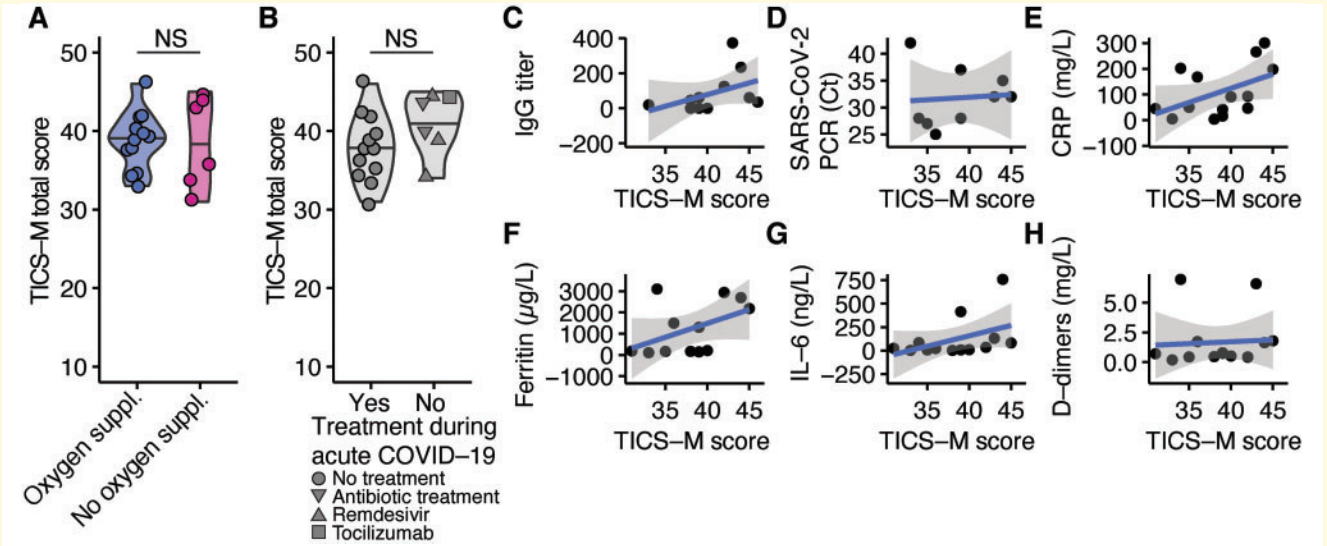

Figure 3 Cognitive deficits are independent from acute disease severity and viremia. (**A**) Comparison of TICS-M total scores ($P = 0.9251$) between post-COVID-19 patients who received supplementary oxygen during acute disease ($n = 6$) and patients who recovered without supplementary oxygen ($n = 12$). Two-tailed Wilcoxon-test was used. (**B**) Comparison of TICS-M total scores ($P = 0.1589$) between post-COVID-19 patients who received no treatment ($n = 12$), antibiotics ($n = 2$), remdesivir ($n = 2$) or tocilizumab ($n = 1$) during acute COVID-19. Two-tailed Wilcoxon-test was used. (**C–H**) Linear regression analysis of TICS-M scores and maximal anti-SARS-CoV2-IgG titre (**C**; $t = 1.4352$, FDR-adjusted $P = 0.4626$, Estimate = 0.014), cycle threshold values in SARS-CoV-2 PCR (**D**; $t = 0.8422$, FDR-adjusted $P = 0.9358$, Estimate = 0.064), CRP (**E**; $t = -0.0811$, FDR-adjusted $P = 0.4363$, Estimate = 0.021), ferritin (**F**; $t = 0.1266$, FDR-adjusted $P = 0.4363$, Estimate = 0.002), IL-6 (**G**; $t = -0.1309$, FDR-adjusted $P = 0.4363$, Estimate = 0.009), D-dimers (**H**; $t = 0.8330$, FDR-adjusted $P = 0.4363$, Estimate = 0.121) during acute COVID-19 infection.

Therefore, our study represents first indications that warrant broad screenings in post-COVID-19 patients to clarify the diversity of neuropsychological deficits and prevent potential further harm.

Screening methods for mild cognitive deficits are mostly used in the diagnostics of dementia that were validated in elderly adults (Castanho *et al.*, 2014). Here, we chose TICS-M as it has been validated as a telephone screening method which is important for prospective studies that include patients who did not seek professional medical care during or after COVID-19. However, TICS-M has been validated for the diagnosis of mild amnestic dementia in a patient cohort with an average age of 74.9 years (Cook *et al.*, 2009). Since the average age of our patient cohort (42.1 years) was considerably lower, we additionally tested a healthy control group with similar age as control. Thus, further optimization of a standardized screening tool is needed. Our findings suggest to especially focus the screening on short-term memory, attention and concentration. In contrast to currently available screening tools, validation should include all age groups since we and others (Dinakaran *et al.*, 2020; Nalleballe et al., 2020) observed neurocognitive deficits in young adults.

Post-viral-syndromes have been described for multiple viral infections, such as Epstein–Barr–Virus or influenza (Hotchin *et al.*, 1989) and are characterized by severe fatigue (Thomas, 1987). Smartphone-App-based patients' reports assessing fatigue in COVID-19 (Menni *et al.*, 2020) and histopathological findings of viral infiltrates and diffuse immune cell activation in brains from deceased COVID-19 patients (Polak *et al.*, 2020) may indicate similar clinical presentations. However, systematic analysis with large patient cohorts in different cultural settings and different countries is needed for clarification. To exclude potential biases and classical post-viral-syndromes, we additionally screened for fatigue and depression. Our data indicate that neurocognitive deficits after recovery from COVID-19 are independent from fatigue and mood alterations and therefore might be different from the classical post-viral syndrome (Perrin *et al.*, 2020) but a specific post-COVID-19 manifestation. SARS-CoV-2 might infiltrate the CNS through the nose (Riel *et al.*, 2015) and trigger a reactive immune response in the brain that could alter neuronal signalling. In addition, exposure of human brain organoids to SARS-CoV-2 revealed direct infection of neurons with subsequent alterations of intracellular signalling and cell death (Ackermann *et al.*, 2020) that could disturb neuronal connectivity. Notably, TICS-M scores of post-COVID-19 patients did not significantly correlate with the maximal inflammatory response during the acute infection. Studies that investigated chronic fatigue syndrome in rheumatic disorders (Korte and Straub, 2019) and multiple sclerosis (Giovannoni, 2006) found that chronic but not acute dysregulation of the immune metabolism and especially

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cytokine composition correlated with fatigue and individual suffering. Although serum profiling during acute COVID-19 revealed distinct cytokine profiles that correlated with disease outcome (Lucas *et al.*, 2020), detailed immunological profiling of post-COVID-19 patients is sparse. Our study is limited by the sample size. Therefore, we cannot report representative frequencies of post-COVID-19 manifestations and longitudinal monitoring and analysis of large cohorts of post-COVID-19 patients is warranted to find clear correlates with long-lasting symptoms after recovery.

Furthermore, we used the TICS-M that was originally developed to screen for MCI in elderly adults because we aimed to establish a tool that was validated for telephone interviews. However, our test results should be validated by neuropsychological tests that were established in young adults such as the Cambridge Automated Test Battery (Crooks *et al.*, 2006). Furthermore, confounders for cognitive testing such as years of education and substance abuse were not assessed. However, we documented the patients' profession and except for one participant who still visited high school all patients received at least 12 years of education. Moreover, only two patients received detailed neuropsychological and neurological assessment that confirmed the results of our screening as well as cranial MRI and lumbar puncture to exclude other potential pathologies. However, we provide a clear description of the observed deficits and provide distinct symptoms that will instruct prospective screenings in larger cohorts to sensitively identify post-COVID-19 patients with cognitive deficits.

Together, the results of our study demonstrate that young patients who recovered from uncomplicated COVID-19 can have sustained neuropsychologic deficits that can be unmasked by targeted screening.

Supplementary material

Supplementary material is available at *Brain Communications* online.

Acknowledgements

The authors thank the UKE ID COVID-19 study team for their support. They thank members of the Friese and Schulze zur Wiesch laboratories for discussions. They also thank Marc Lütgehetmann for providing PCR and antibody titres. The authors thank Stephanie Lau for helping with neuropsychological assessments. The graphical abstract was created using biorender.com. M.A.F. received honoraria for consultation and travel expenses from Biogen, Merck KGaA, Novartis and Roche unrelated to this Correspondence.

Funding

A.H., M.M.A., J.S.Z.W. are funded by the Deutsche Zentrum für Infektionsforschung (DZIF).

Competing interests

The authors declare no competing interests and no conflict of interest.

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