Frequent neurocognitive deficits after recovery from mild COVID-19

Short title: Post-COVID-19 neurocognitive deficits

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Abstract

Neuropsychiatric complications associated with coronavirus disease 2019 caused by the Coronavirus SARS-CoV-2 (COVID-19) are increasingly appreciated. While most studies have focused 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 to 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 (TICS-M) 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 hospitalisation, treatment, viremia or acute inflammation. Additionally, TICS-M 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 subclinical 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.

Key words
COVID-19; post-COVID-19; neurocognitive deficits; neurocognitive screenings

Abbreviations
CANTAB = Cambridge Automated Test Battery; CNS = central nervous system; COVID-19 = Coronavirus disease 2019 caused by the Coronavirus SARS-CoV-2; CSF = Cerebrospinal fluid; Ct = Cycle threshold; FAS = Fatigue Assessment Scale; MCI = mild cognitive impairment in elderly adults; MERS-CoV = Middle-east-respiratory-syndrome-coronavirus; PHQ-9 = Patient Health Questionnaire-9 depression scale; SARS-CoV-2 = Severe acute respiratory syndrome coronavirus 2; TICS-M = Modified Telephone Interview for Cognitive Status
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 COVID-19 patients 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 COVID-19 patients from the United Kingdom who reported neurologic and psychiatric complications during the acute phase of the disease were reported and intracerebral 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 focused 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 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 subclinical neuropsychologic symptoms and help to differentiate unspecific post-illness-manifestations.

Here, we established a facile screening approach for cognitive deficits in 18 young patients without diagnosed cognitive preconditions after recovery from COVID-19 and discovered widespread subclinical 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 only included 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 only included 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; SD 14.3 years) we additionally tested healthy individuals with similar age (mean age 38.4; SD 14.4 years). Healthy individuals were randomly selected employees of the University Medical Center Hamburg-Eppendorf who did not have prior knowledge of the TICS-M or similar neuropsychological screenings and were matched for age within a range of five 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 July 14th, 2020. To sensitively screen for mild cognitive deficiencies, we utilised the Modified Telephone Interview for Cognitive Status (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, < 34 amnestic mild cognitive impairment) (Cook et al., 2009) and Alzheimer’s dementia (Manly et al., 2011). We performed TICS-M according to published protocols (Cook et al., 2009). The total interview lasted 15 to 20 minutes. Four domains are tested by the TICS-M: (1) orientation, (2) recent memory and delayed memory, (3) attention and (4) semantic memory, comprehension and repetition (language/concentration) (De Jager et al., 2003). The TICS-M included the following items: (1) name, (2) age, (3) date, (4) weekday, (5) season, (6) phone number (each 1 point); (7) counting backward (2 points); (8) first, a 10-word list learning exercise and then a delayed (21) recall of that word list (10 points each); (9) subtractions (5 points); (10–13) responsive naming (4 points); (14–15) repetition (2 points); (16) current chancellor and (17) president of Germany (each 2 points); (18) finger tapping (2 points), and (19–20) 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 (PHQ-9) depression scale and Fatigue Assessment Scale (FAS). 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 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 recommendation.

Statistical analysis

Data was 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 FDR-corrected for multiple comparisons. To find predictors of screening results we used multiple linear regression models. The results were FDR-corrected for multiple comparisons. Exact $P$-values are reported in respective sections of the article and figure legends. We analysed the scores of FAS and PHQ-9 as well as age, length of hospitalisation, sickness duration, time from recovery to neurocognitive assessment, maximal PCR ct 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 Rosenthal's r. Significant results are indicated by *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

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 their containing information 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 preconditions 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 to 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 ten 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 Modified Telephone Interview for Cognitive Status (TICS-M) as primary tool (Cook et al., 2009) as it has been extensively validated for screening of mild cognitive deficiencies by telephone. 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) (Figure 1A), especially regarding short term memory, attention and concentration/language tasks (Figure 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 (Figure 1C and D). In terms of patient self-reported symptoms, out of 18 included individuals nine (50%) reported attention deficits, eight (44.4%) concentration deficits, eight (44.4%) short term memory deficits, five (27.8%) troubles in finding words, three (16.7%) fatigue, two (11.1%) severe mood swings, and one (5.6%) sustained lack of energy, phonophobia, incoherent thoughts (Figure 1E).

Next, we aimed to find predictors of our observed cognitive deficits. First, we analysed patient characteristics and found that neither sex (Figure 2A), nor age (Figure 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 hospitalisation 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 (Figure 2C), length of sickness (Figure 2D) and length of inpatient stay (Figure 2E). To further evaluate the impact of disease severity, we correlated the...
neurocognitive deficits with acute and sustained somatic symptoms (Figure 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 (Figure 2G) and the results in our screening (Figure 2H). Moreover, treatments such as oxygen supplementation (Figure 3A) and drugs like remdesivir, tocilizumab or antibiotics (Figure 3B) in acute infection could not predict the observed cognitive impairments. Thus, our data implies that post-COVID-19 neuropsychologic deficits are independent from hospitalisation 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 (Ct) from SARS-CoV-2 PCR, antibodies against SARS-CoV-2 and inflammatory serum markers. Our analysis revealed that maximal SARS-CoV-2 IgG-titres (Figure 3C), SARS-CoV-2-PCR Ct-values (Figure 3D), CRP (Figure 3E), ferritin (Figure 3F), IL-6 (Figure 3G) and D-dimer (Figure 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 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 focused 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). 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 have 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). 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 characterised by severe fatigue (Thomas, 1987). Smartphone-App based patient-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 are 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 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. Nonetheless, our test results should be validated by neuropsychological tests that were established in young adults such as the Cambridge Automated Test Battery (CANTAB) (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.

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Competing interests
The authors declare no competing interests.
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| 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)           |
| Preconditions (%)                |                        |                  |
| 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.    |
| Diabetis 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)   |
| FAS                              | 24.17 (13 – 40)        | 18.1 (18 – 19)   |
| PHQ-9                            | 2.83 (0 – 9)           | 0.7 (0 – 2)      |

Table 1: Summary of characteristics and manifestations of post-COVID-19 patients and healthy control subjects.
Figure Legends

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 FAS (C; $t = –1.3653$, FDR-adjusted $P = 0.3820$, Estimate = –0.165) and PHQ-9 (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 and 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).

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), Ct values in SARS-CoV-2 PCR (D; $t = 0.8422$, FDR-adjusted $P = 0.4358$, 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.
Figure 1
Figure 3
Abbreviated summary

Long-lasting sequelae after COVID-19 recovery are increasingly reported. In this study, Woo et al. performed a telephone-based neuropsychological screening in 18 patients who recovered from mild to moderate COVID-19 and 10 healthy control subjects. Post-COVID-19 patients frequently reported neuropsychological symptoms and showed deficits in attention, concentration and especially short-term memory.
Graphical Abstract