Pattern of cognitive deficits in severe COVID-19

The severe form of COVID-19 tends to be associated with neurological deficits. Among patients with acute respiratory distress syndrome (ARDS), who benefited from mechanical ventilation and were examined after discontinuation of sedation and neuromuscular blockade, 69% presented agitation, 65% confusion, 67% corticospinal tract signs and 33% dysexecutive syndrome.

We describe here the pattern of cognitive deficits in a series of 13 consecutive inpatients hospitalised in the Lausanne University Hospital, whom we examined during the post-acute critical stage of severe COVID-19 (table 1). Inclusion criteria were COVID-19 diagnosed by PCR and ARDS that required intubation and mechanical ventilation in intensive care unit (ICU). Exclusion criteria were prior psychiatric or neurological diseases, including neurocognitive impairment or dementia. At the time of testing, patients were no longer sedated and ICU delirium symptoms, which were present in seven patients, resolved in six of them (P5–P7, P10, P11, P13) or subsided to a great extent (P12).

The neuropsychological evaluation comprised two standardised test batteries. The Montreal Cognitive Assessment (MoCA; https://www.mocatest.org), which covers main cognitive functions, revealed normal cognitive performances in four patients (P1–P4), mild deficits in four (P5–P8) and moderate to severe deficits in five (P9–P13). MoCA subtests revealed selective cognitive pattern with lower performances in executive functions for patients with normal MoCA scores and more extensive cognitive impairment in executive, memory, attentional and visuospatial functions, with relatively preserved orientation and language, for patients with mild to severe MoCA deficits.

The Frontal Assessment Battery (FAB; www.psychdb.com/cognitive-testing/fab) revealed executive dysfunction in eight patients (table 1; P6–P13). Among the FAB subtests, the most affected was lexical fluency, impaired in all patients except in one (with normal MoCA: P3).

Pearson (r) and Spearman (rho) correlation analyses were conducted. MoCA and FAB scores were correlated (r=0.88; p<0.001). Mental fatigue and cognitive slowness, assessed with observational scales, correlated with MoCA (respectively: rho=−0.67; p=0.012; rho=−0.74; p=0.004) and FAB scores (respectively: rho=−0.85; p<0.001; rho=−0.72; p=0.006). Age correlated with FAB (r=−0.591; p=0.033) but not with MoCA scores. There was no significant correlation between MoCA or FAB scores and the following measures: gender, length of ICU stay, duration of mechanical ventilation, delay between ICU discharge and cognitive assessment, mood and anxiety disturbances. Mood disturbance, assessed in self-report, was considerable (table 1; ≥5/10) in 38% of patients and 23% reported anxiety about breathing difficulties, fear of dying or reminiscences of intensive care. Seven patients presented ICU delirium (table 1); its occurrence was correlated with MoCA score (rho=−0.619; p=0.024) and cognitive slowness (rho=0.585; p=0.036), but not with FAB, length of ICU stay, duration of mechanical ventilation, delay between ICU discharge and cognitive assessment, age, gender, mental fatigue, mood and anxiety disturbances.

Eleven patients had brain imaging. None of them sustained acute stroke or ischaemic damage; among the eight patients who had MRI-based morphometry, focal brain atrophy was, however, present in patients with normal (table 1; P1) or deficient performance at MoCA (P10–P13) as it was absent in patients with normal (P3, P4) or deficient performance at MoCA (P7). Four patients had lumbar puncture, revealing enhanced proteinorachia (P3, P11, P13) and barrier index (P11, P13) or normal values (P12).

Two cognitive profiles characterise the post-acute critical phase of severe COVID-19: (1) normal score at MoCA, but tendency for lower performances
Our sparse data on cerebrospinal fluid suggest that increased blood–brain barrier permeability may contribute to neurological symptoms, as previously described for mild central nervous system inflammation in ICU patients.1

In conclusion, pattern of cognitive deficits, present during the acute stage in our 13 patients without history of cognitive, psychiatric or neurological disorders, is probably linked to critical illness as part of ARDS due to COVID-19, since it is very similar to those reported in ARDS of other aetiologies. Further investigations are needed to determine predictive factors and underlying neural mechanisms, and clarify with a long-term follow-up whether patients will completely recover.

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PostScript

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