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-critical acute phase of severe COVID-19: (1) normal score at MoCa, but tendency for lower performances...
in executive than in other cognitive functions; (2) mild to severe deficits at MoCA with extensive cognitive impairment in executive, memory, attentional and visuospatial functions, but relatively preserved orientation and language, executive dysfunction being confirmed by the FAB score. These cognitive profiles together with mood and anxiety disturbances, which we observed in the acute stage, in the absence of stroke, are reminiscent of those reported in the aftermath of ARDS of other aetiologies, where up to 70% of ARDS survivors had presented at hospital discharge cognitive deficits, affecting predominantly attention, mental processing speed, memory and executive functions,\(^3,4\) with a high prevalence of depression and anxiety.\(^5\)

Furthermore, cognitive impairment in severe COVID-19, as in ARDS of other aetiologies,\(^1\) does not correlate with length of mechanical ventilation or length of ICU stay and thus severity of the acute illness. However, the occurrence of ICU delirium tends to be associated with poorer cognitive performance.\(^1\)

Structural damage, such as ischaemic or hypoxemic lesions of the hippocampus, basal ganglia or cerebellum lesions as well as brain atrophy (in particular hippocampal) or disruption of functional connectivity, which occur frequently in ARDS survivors,\(^3,4\) may contribute to cognitive dysfunction. In the context of COVID-19, stroke and perfusion abnormalities have been reported,\(^1,2\) but were excluded here in all 11 patients, who had brain imaging during the acute stage.

Prior brain atrophy may confer worse outcome as shown for the risk to develop delirium and cognitive disorders in ARDS of other aetiologies.\(^1,4\) Patchy grey and/or white matter atrophy was present in five patients (of the eight who had MRI), most likely reflecting a prior condition; it was associated with cognitive impairment in four patients. However, brain atrophy did not always induce cognitive impairment and conversely, cognitive impairment was also present without imaging abnormalities, as in previous non-COVID-19 studies.\(^4\)

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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Funding This work was supported by the Biaggi de Blasys Foundation for SCH.

Competing interests None declared.

Patient consent for publication Not required.

Ethics approval The procedure was approved by the Cantonal Ethics Committee of Vaud and informed consent was obtained in agreement with the requirements of the Ethics Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

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