COGNITIVE IMPAIRMENT IN PATIENTS WITH COVID -19

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Annotation: COVID-19 has a wide range of clinical manifestations. During the pandemic, neurological manifestations were demonstrated in patients with COVID-19, including cognitive impairment. In a study conducted in Wuhan, Hubei Province, 36.4% of patients had some neurological manifestations, while central organs were more often affected (dizziness, headache, changes in consciousness, stroke, ataxia and epilepsy). In addition, patients with severe course of the disease more often developed neurological disorders, especially disorders of consciousness, acute cerebrovascular diseases and diseases of the musculoskeletal system. In addition to neurological manifestations, cognitive impairment was assessed in patients with COVID-19. In a Chinese study, the cognitive functions of 29 patients with COVID-19 were assessed using digital questionnaires linking cognitive complaints with high levels of C-reactive protein in the acute phase of the disease. Another study assessed cognitive impairment in outpatient patients using the Mini Mental Health Assessment (MMSE), the Montreal Cognitive Assessment (MoCA), the Hamilton Rating Scale for Depression and the Functional Independence Measurement (FIM), detecting 80% of cognitive impairment. In addition, various cognitive manifestations, such as encephalopathy, have been described.

Conclusion: Cognitive impairment was found after other infections, including coronavirus infections. In addition, since COVID-19 can cause critical illness in some patients, ARDS and delirium, which have previously been associated with cognitive impairment, may occur. Thus, Rass et al. cognitive impairment was detected in 23% of patients with COVID-19 (in patients with severe COVID-19 — 29%, moderate — 30% and mild — 3%). In addition, a disease leading to hypoxemia with consequences for memory is expected, since the hippocampus is sensitive to low oxygen concentrations. Another possible cause, which has not yet found support in the medical literature, was the direct effect of the virus. Such a direct role of the virus does not provide convincing evidence in studies of general neurological manifestations with assessment of cerebrospinal fluid or autopsy.

Used literature

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