COGNITIVE DECLAIN AS A CONSEQUENCES OF SARS-CoV-2 INFECTION

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COVID-19 is primarily a respiratory disease but up to two thirds of hospitalised patients show evidence of central nervous system (CNS) damage. Hypothetically, SARS-CoV-2 may affect the central nervous system (CNS) either by direct mechanisms like neuronal retrograde dissemination and hematogenous dissemination, or via indirect pathways. CNS complications associated with COVID-19 include encephalitis, acute necrotizing encephalopathy, diffuse leukoencephalopathy, stroke (both ischemic and hemorrhagic), venous sinus thrombosis, meningitis, and neuroleptic malignant syndrome. The elderly population and those who are suffering from Alzheimer’s disease and dementia related illnesses seem to be at the higher risk. Cerebral white matter is particularly vulnerable to ischaemic damage in COVID-19 and is also critically important for cognitive function. We present a patient with clinical picture of cognitive declain as consequences of SARS-CoV-2 infection.

61 old female was admitted in our Department of Intensive Care Unit, Clinic of Neurology under the clinical picture of stroke consequent occlusion of the M2 branch of the left artedia cerebri media (ACM) from the Clinic for Infectious Diseases Fran Mihaljević where she was treated and admitted because COVID 19 pneumonia. On the thirteenth day of hospitalization, she developed a picture of acute ischemic stroke with right hemiparesis and aphasia, and the urgent invasive treatment of stroke with mechanical thrombectomy was indicated. The procedure proceeded without complications and with the achievement of complete recanalization of the occluded artery. In the control neurological status, there were a regression of the neurological deficit. At the first follow-up examination in the outpatient clinic, 3 months after hospitalization, the patient was diagnosed with marked cognitive decline, with impaired verbal expression, executive functions, and depression.

There is many evidence that cerebral hypoperfusion accelerates amyloid-β (Aβ) accumulation and is linked to tau and TDP-43 pathology, and by inducing phosphorylation of α-synuclein at serine-129, ischaemia may increase the risk of development of Alzheimer’s disease. Also there are new evidence for frequent cognitive sequelae of COVID -19 that indicate an association with the severity of the lung affection and potentially restricted cerebral oxygen delivery. Cognitive follow-up of COVID-19 patients will be important, especially in patients who develop cerebrovascular and neurological complications during the acute illness. The mental health and illness aspect of COVID-19 are among the most important side effects of this pandemic which requires a national plan for prevention, diagnosis and treatment.