

Cerebral Complications Induced by Covid-19

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Received: May 27, 2020; **Published:** June 15, 2020

Fever, cough, diarrhoea, fatigue are the clinical manifestations of COVID 2 from Wuhan China. Four neurologic manifestations are described type 1 affect the CNS/1 headache, dizziness, CVA, ataxia, epilepsy, 24%, type 2 peripheral system anosmia, dysgeusia, vision impairment 19% type 3 musculoskeletal 10% increased CK, C reactive protein, D-dimer observed in 214 patients age 52.7, 40% males.

The neurologic manifestations were predominant in severe patients. Hypogeusia is present in 5% anosmia in 5.1%, headache 13.6%, myalgia 14.9%, ICU 5%.

The receptor ACE2 is present in the brain and muscle. Necropsy studies in china show neuronal damage, oedema, hyperemic brain. Quimiosensory disorder was found in Iran, Italy, USA, 33% in COVID 19. The clinic resolution of the anosmia and dysgeusia depends on the clinical resolutions. The clinical course was different between outpatients and hospitalized patients.

Are the neurologic complications of real concern in COVID? Is there evidence of brain involvement? Patients with MS, Parkinson, Alzheimer have a high risk of mortality with COVID.

How is the mechanism of potential invasion through ACE2 receptor tmpr2/transmembrane serine protease and the S spike protein?

What is the Potential mechanism? Do the patients with COVID had cerebrospinal fluid, and brain with COVID is the same mechanism of MERS? And SARS?

And is there a way that ACE2 expression in the glial cell, neurons, hematogens allows viral spread or retrograde neural transmission throw olfactory neurons to the cribriform plate?

Is there another way? Could be a direct infection be possible?

The target of CNS finding is the anosmia which could be due to the viral invasion of the olfactory bulb.

Is neurotrophic the virus? Speculation is entirely possible. The isolated finding of Guillain Barre syndrome and hemorrhagic brain encephalopathy is it part of the intracranial storm?

Are the neurology symptoms of concern?

Anecdotal report and observational data is a concern? We need to use conventional logic. Lumbar puncture and neuroimaging are very important. There is no evidence that COVID2 attack the CNS of PNS the speculation of the mechanisms are 1 direct like herpes simplex infection, there is no evidence of encephalitis. 2 cytokine storm like GBS. 4 indirect hypoxemia in very ill patients.

We need more information, but the evidence shown that the virus is not neurotrophic.

Volume 12 Issue 7 July 2020

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