

COVID-19: Expression of ACE2-receptors in the Brain Suggest Neurotropic Damage

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Editorial

COVID-19 is a severe coronavirus disease spreading all over the world. To date of this publication, 2,501,156 people were infected with COVID-19, and 171,810 deaths were found in 185 countries (John Hopkins Coronavirus Resource Center, date 21 april 2020). This overwhelming death rate makes intensive research activities necessary all over the world. To date, the US will be slayed by the number of deaths per day, suggesting an uncontrollable state of spreading infection. SARS-CoV-2 binds to the angiotensin II receptor on various tissues in the human body, especially in the oral cavity and tongue. SARS-CoV-2 requires the cheerful TMPRSS2 to pro-activate for this sluggishness. SARS-CoV-2 uses the ACE2 receptor as an access portal to the lungs [1]. The SARS-CoV-2 virus binds with the top protein to the ACE2 receptor [1]. The S-protein has a functional polyphasic furin cleavage side at S1/S2 by the insertion of 12 nucleotides and shows changes in the receptor binding region (RPD) with the sluggishness of 6 RBD amino acids. Because of the binding site at the angiotensin II receptor angiotensin receptor blockers are possible alternatives for the treatment of children with severe COVID-19 infection. To date, there is no clinical experience with COVID-19 positive children and no study on this topic. In adults, a randomized controlled trial with losartan in 200 participants with severe COVID-19 infection was initiated at the University of Minnesota on March 16, 2020 [2]. COVID-19 is more found in Afro-American people in the US. The expression and distribution of ACE2 in the

human body may indicate the possible routes of infection of 2019-nCoV. Using the developed single cell RNA sequencing technique (scRNA-Seq) and single cell transcriptomes based on the public database, the researchers have developed an ACE2 RNA expression profile with single cell resolution [3]. The high expression of ACE2 was identified in type II alveolar cells (AT2) of the lung [4], in upper and stratified epithelial cells of the esophagus, in absorptive enterocytes of ileum and colon in cholangiocytes in myocardial cells in proximal tubular cells of the kidney and in urothelial cells of the bladder. These results have shown that those organs with the strong ACE2 expression of cells should potentially be considered at high risk for nCoV infection in 2019 [1]. The study by Xu and others has shown a superiority of COVID-19 virus over ACE2 receptors in the oral cavity and tongue, indicating that COVID-19 reaches a part of the body in this way [1]. ACE2 and its receptors are also found in the brain, especially in CNS neurons of glial cells. The brain accelerators ACE-2 receptors in a broad number [5]. They have been discovered via glial cells and neurons, indicating that COVID-19 is a potential target for the human brain [6]. Former studies have shown the ability of SARS-COV to induce a neural death with mice, while it reaches by the nose near the smelling epithelium in the brain [7]. The contribution of the neurological potential of a circle of SARS CoV 2 must be determined with patients who were announced to the new outbreak of COVID-19. With the infections with SARS-COV about which was reported in the past the results of the necropsy of patients with electron microscopy, Immun- histochemie and reverse transcriptase-PCR have shown strong tips to the availability of SARS-COV [11]. Patients with an acute SARS COV illness have also proved the availability of the virus in the liquor. The role the blood brain barrier by the containment of the virus and the prevention of his access to nervous system must be examined with the patients with whom COVID-19 was diagnosed further. In addition, demands a discovery which was published with a patient who wears the involuntary breath control lost [2,7] during the new outbreak together with several other patients with acute breath failure, experts and clinicians to separate COVID-19 patients in patient with neurological manifestations those without neurological deficits. A study published in adults in medRxiv, 214 COVID-19 patients were included and 36.4% had neurological symptoms indicating a neurological potential of COVID-19 [8,9]. New case reports in adults shed light on brain damage from COVID-19 with decay, loss of speech, loss of taste and presence of COVID-19 have triggered necrotic encephalitis [9,10]. Typical CT and MRI characteristics were found in a female airline worker with a 3-day history of coughing, fever, and poor mental health and COVID-19 positive viral wipe [9]. Non-contrast head CT has shown symmetrical hypo attenuation in the area of bilateral middle thalami [9]. Images from brain MRI have shown hemorrhagic rim height lesions within the thalami on sides, medial temporal lobe area and supinely area [9]. The patient was treated with intravenous immunoglobulin. Acute necrotizing encephalopathy (ANE) is a rare complication and mostly caused by viral reagent. COVID-19 appears to induce an intracranial cytokine storm leading to blood-brain barrier depression with induction of direct viral invasion and Para

infectious demyelination [9]. COVID-19 appears to have a neurological potential that needs to be excluded by further research and retrospective case studies.

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