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Abdul Mannan Baig Aga Khan University, Karachi

Mohammad Wasay Aga Khan University, Karachi

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COVID-19 INFECTION; LOSS OF TASTE, SMELL, AND NEUROGENIC RESPIRATORY FAILURE

Correspondence to: Abdul Mannan Baig Email: abdul.mannan@aku.edu

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Neurological Signs and Symptoms in COVID-19: Like the rest of the world, Pakistan is been gripped by unprecedented and complex burden of COVID-19. While the diseases got upgraded to a pandemic, the preparedness of even the first world countries to combat COVID-19 is distressing, as there seems to be a lack of clear strategy to tackle the disease spread and treatment. Though the response of the clinicians and scientists worldwide was swift against, the causative agent of COVID-19, it is yet to see any measurable success against this coronavirus pandemic. Of the many ways in which the SARS-CoV-2 is duping its detection and evading the human immune response, it has been observed that it has also exhibited neuroinvasive potential that has gone undetected until recently 1, 2, 3. Of the neurological deficits it is inducing in COVID-19 patients, the most alarming is the respiratory failures that could be neurogenic in origin. According to multiple senior front-line Chinese doctors' feedback, COVID-19 associated respiratory failure is very difficult to treat which results in high mortality, whereas the autopsy report indicates less severe lung injury compared to SARS-CoV-1 comparatively. The loss of smell and taste hints that SARS-CoV-2 can target the nervous system and it is neurotropic ^{2, 3, 4}. After its first reports ^{2, 3,} loss of smell and taste were for the first time were hinted towards being one of the initial heraldings signs of COVID-19 on 13th March. Later the British 4, and then on 26th March 2020 the American academy of otolaryngology-head and neck surgery has also released a statement noting that anosmia and dysgeusia are 'significant symptoms' associated with COVID-195. Viewed with skepticisms initially 4, after observing anosmia and dysgeusia in a huge number of COVID-19 patients there was a consensus on them being a consistent feature in many serologically confirmed cases of COVID-19 patients 5. The last piece of evidence that was required, and was thought to unequivocally clarify the neuroinvasive potential of SARS-CoV-2, was the isolation SARS-CoV-2 from the cerebrospinal fluid (CSF) of COVID-19 patients. To end the debate, a very recently published work has reported the isolation of SARS-CoV-2 from the CSF by RTq-PCR 6. Furthermore, the radiological evidence of bleeding lacunae in thalami and adjacent zones as seen on MRI has provided the evidence of neurotropic cell damages caused by necrotizing encephalopathy 7. In light of the later and similar studies in mice that have shown neuronal death (within the respiratory center in the brainstem) as the main death cause of infected mice 8,9, it would be important to here eye for signs of neurological deficits early in the course of COVID-19 to define treatment protocols that could prevent the occurrence of a neurogenic respiratory failure for which the use of ventilators could not prove to be of any significant benefit.

Based on the similarity between the pathology of SARS-CoV-1 and SARS-CoV-2 neurovirulence and in the light of recent 6,7 measures to prevent the neuronal access uncommon routes 1,4 can not be overemphasized.

A neuroprotective therapy and strategies that could prevent the invasion of SARS-CoV-2 to the brain are the next needed approaches in our fight against COVID-19. Through this letter, it is intended to herald the healthcare workers and clinicians managing the COVID-19 patients that early signs and symptoms of neurological involvement should be carefully monitored to save lives in COVID-19 affected patients.

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Abdul Mannan Baig; concept, data collection, data analysis, manuscript writing, manuscript review **Mohammad Wasay;** data collection, data analysis, manuscript writing, manuscript review