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

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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-19[5]. 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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