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Editorial

Neurological Manifestations of COVID-19

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Although, the most common and important clinical presentation of COVID-19 is respiratory illness, reports of neurological symptoms are increasing. The neurological features can be as a result of a combination of non-specific complications of systemic disease, the effects of a direct viral infection and inflammation of the nervous system and its vasculature [1]. The neurotropic and neuro-invasive properties of the coronaviruses enable them to easily enter the CNS through the olfactory bulbs, most commonly; causing inflammation and demyelination in cases implicating neurological involvement. The virus can even reach up to the CSF upon further infectious spread [3]. At 36.4%, over a third of the cases of COVID-19, had neurological manifestations, ranging from milder symptoms such as headache, to more complicated ones such as seizures and strokes [2]. Anosmia and ageusia, headache and dizziness were amongst the most common neurologic manifestations.

Decreased to no sensation of smell: hyposmia/anosmia, and decreased to no sensation of taste: hypogeusia/ageusia; have a prevalence of 44.1% and 43.3% respectively; the former now heralded as conclusive of a diagnosis of COVID-19, even in the absence of an RTPCR indicating the same. The deficit is transient – lasting days to two weeks, onset sudden, and recovery rapid. Anosmia precedes ageusia, symptomatically. These neurological symptoms provide a fortunate opportunity to be utilised early on in the course of the disease, as a rapid screening tool, to identify otherwise asymptomatic cases and potential carriers of infection. These symptoms are independent of age and disease severity [4]. Anosmia is also regarded as an independent prognostic factor of a less severe COVID-19 infection [5]. Headache is reported in 17% of the cases of COVID-19 [1]. The headache is usually bilateral, diffuse, intense and of a migraine phenotype [6]. Some patients may also complain of headache as an otherwise rare adverse effect of the drugs employed for the treatment of COVID-19 infection: azithromycin, amoxicillin, ivermectin, doxycycline, favipiravir and remdesivir [6]. Dizziness, occurring at a frequency of 13%, is one of the main clinical manifestations. Albeit non-specific, if investigated, it can point towards underlying pathologies of the symptom [7].

Encephalopathy and Encephalitis are the major and serious CNS complications associated with SARS-CoV-2 infection, more often seen in the presence of comorbidities. Encephalopathy manifests as altered consciousness, delirium or even coma; additionally, as seizures, headaches and extrapyramidal signs. Encephalitis is a diffuse and inflammatory condition of the brain - and is clinically characterised by fever, headache, focal neurological deficits and altered levels of consciousness. The SARS-CoV-2 virus has been demonstrated in the CSF of some cases of associated Encephalitis. Such patients require ICU supervision and may also need mechanical ventilation. The prognosis associated with such patients remain poor [8].

Seizures, although a rare occurrence, have been reported in 0.5% patients suffering from COVID-19. Owing to hypoxia, multi-organ failures and severe metabolic and electrolyte derangements - it is reasonable to expect clinical/subclinical seizures [3]. No clinical evidence is available for worsening of seizures in patients of epilepsy. Three percent of the COVID-19 patients suffered from Cerebrovascular Accidents (CVA). CVA was identified in most cases upon further investigating patients with decreased levels of consciousness; more commonly seen in elderly, critically ill and those with prior cardiovascular and cerebrovascular comorbidities.

Embolic events are indicated by elevated levels of D-dimer, troponin and pro-BNP. A pro-thrombotic state as evidenced by increased levels of D-dimer, LDH and other markers of coagulation, are also implicated in the pathogenesis. The events precipitating cerebrovascular accidents need further understanding [9]. Critical Illness Polyneuropathy and Myopathy, a post intensive care syndrome (PICS), may be seen due to long durations of admissions, mechanical ventilations, hypoxia and sepsis that patients are subjected to when infected; the underlying mechanism being the resultant Systemic Inflammatory Response Syndrome [2].

With the onset of the pandemic and the ensuing lockdowns and quarantines, global prevalence of insomnia was recorded to be 20-45%, indicating an increased burden of COVID-19 related economic disruptions, psychosocial issues, substance abuse, depressive features, anxiety disorders, fatigue in the society. During the periods of lockdown and isolation, the resultant restricted mobility and social contact; worries about the unforeseeable future, finances, supplies; fear of infection; excessive usage of electronic devices – all resulted in features of anxiety, depression, stress; which culminate in poor sleep quality and quantity [10]. Studies have also indicated a potential cognitive dysfunction in patients who have been infected by COVID-19, significantly impacting attention, memory, executive functioning and psychomotor activities [11].

However even with a wide array of neurological manifestations, insufficient details reflect the challenges faced in studying such patients. A clear delineation of manifestations and burden of neurological manifestations of COVID-19 require further clinical, diagnostic and epidemiological studies. Even though the proportion of infections having neurologic diseases will be small, but the sequelae may be severe and debilitating. Furthermore, considering the huge number of cases of COVID-19, the resultant numbers of patients with neurological illness may potentially end up incurring large social and economic costs. Health-care planners and government policy makers should foresee this eventuality, whilst simultaneous studies and researches keep nourishing our knowledge [1].

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