Editorial

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COVID-19's Neurology: A Retrospective

Bello Figuim*

Department of Neurosurgical unit, Central Hospital, Messa Yaounde , Cameroon

Corresponding author: Bello Figuim, Department of Neurosurgical unit, Central Hospital, Messa Yaounde, Cameroon, Email:bellofiguim@gmail.com

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Infection with SARS-CoV-2 affects the central nervous system (CNS), the peripheral nervous system (PNS), and the muscle, according to a comprehensive evaluation of neurological problems documented during the current COVID-19 epidemic. Anosmia, hyposmia, hypogeusia, and dysgeusia are common early symptoms of coronavirus infection; headache and decreased responsiveness are regarded early markers of probable neurological involvement. COVID-19 can cause respiratory failure, which is fatal. It is likely neurogenic in nature and may be caused by viral invasion of cranial nerve I, which then spreads to the rhinencephalon and brainstem respiratory centres, resulting in 264,679 deaths globally. Cerebrovascular disease, particularly large-vessel ischemic strokes, and cerebral venous thrombosis, which are less common, are two of the most common causes of death in the United States. Intracerebral haemorrhage and subarachnoid haemorrhage are usually caused by viral attachment to ACE2 receptors in endothelium, resulting in extensive endotheliitis, coagulopathy, and arterial and venous thromboses. The review's key conclusion is that it is critical to clarify COVID-19's neurology, including its frequency, symptoms, neuropathology, and causation. We cordially invite national representatives on behalf of the World Federation of Neurology. During the ongoing pandemic, regional neurological societies will build local databases to report cases with neurological signs. International neuroepidemiological collaboration could aid in determining the natural history of this global issue. During the ongoing pandemic, regional neurological societies will build local databases to report cases with neurological signs. International neuroepidemiological collaboration could aid in determining the natural history of this global issue. We wanted to see if giving students an extra week of neurology exposure would improve their clerkship experience and spark interest in the field. These can happen in individuals with malignancies that aren't generally associated with paraneoplastic neurological autoimmunity, such as melanoma and renal-cell carcinoma, and should be questioned if new neurological symptoms appear while on ICI and aren't explained by disease progression or metabolic dysfunction. Renal disease patients are more likely to be admitted to the neurological critical care unit because of overlapping risk factors for renal and cerebrovascular disease, as well as distinct hazards associated with renal failure alone. In these individuals, managing acute neurological damage necessitates personalised attention to diagnostic and therapeutic aspects such as coagulopathy, immune function problems, encephalopathy, and renal replacement methods. To optimise therapy for this unique patient population and improve neurological and renal outcomes, careful evaluation of these brain-kidney interactions is required. Intensive attention Acute brain damage, as well as the neurological repercussions of chronic and acute medical illness, are the focus of neurology. Chronic kidney disease (CKD) is estimated to affect 15% of individuals in the United States, and the WHO estimates that 864,226 world deaths were caused by CKD in 2012, putting it 14th on the list of main causes of death. Physiological changes during admission and long after release that raise the risk of neurological disease and complicate the management and outcome of neurological patients. 3 Given these brain-kidney interactions, it is critical for clinicians caring for patients with brain damage to understand how CKD interacts with acute neurological impairment in order to provide the best possible treatment for these complicated patients.