

**Short Report**

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**An update on neurological outcome in Covid-19 crisis****Manik Mathur<sup>1\*</sup>; Rupjyoti Das<sup>2</sup>**<sup>1</sup>Consultant Neurologist, Department of Clinical Neurology, GNRC Hospitals, Guwahati, India.<sup>2</sup>Head of Department & Senior Consultant, Department of Clinical Neurology, GNRC Hospitals, Guwahati, India.**\*Corresponding Authors: Manik Mathur**

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**Abstract**

Recent pandemic surge of Covid-19 has made it necessary to further understand a detailed neurological association with the virus in-relation to sub-clinical picture. Additionally, in the presence of a sweeping corona virus wave throughout the globe, pre-liminary studies have indicated a strong association of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in addition to neuropsychiatric and neurological illness. Following a trivial research (clinical and paraclinical data), five major scenarios have been observed, namely, encephalopathies with no distinct MRI or CSF abnormalities, inflammatory CNS syndrome (including para-or-post-infections) with/out isolated myelitis, ischaemic strokes associated with pro-thrombotic state, peripheral neurological disorders such as Guillain-Barre syndrome and other miscellaneous neurological disorders. Therefore, an early recognition and understanding in the backdrop of neuropathology associated with COVID-19 is essential. Where hypercoagulability is a newfangled conduit in further complicating the pathogenesis to unreasonable levels. Here, a brief discussion on present neurology associated outcomes in COVID-19 scenario.

**Short report**

SARS-Cov-2 pandemic surge is one of the largest disaster ever happened to the life on the Earth. Headache, loss of consciousness, delirium and lately stroke stand out to be the major neurological manifestations in Covid positive patients or even in post-covid scenarios. Whereas, pulmonological complications have been the most frequent and life threatening, with/out Central (CNS) and Peripheral Nervous System (PNS) involvement. However, it still remains unclear that virus directly targets the CNS or promoted by generalised systemic response. Recent study has highlighted a possibility of acute ischemic stroke in patients with COVID-19, but risk factors, in-hospital events, and outcomes are not well studied in large cohorts [1].

The first study, published in *The New England Journal of Medicine* stated various complications include Meningo-Encephalitis [1,2], Encephalopathy [3], Ischaemic Stroke [4], and Guillain-Barré Syndrome (GBS) [5]. In addition, data published focusing the radiological findings suggests involvement number of infarcts, microhaemorrhages with/out features of posterior reversible encephalopathy syndrome [6,7] where a detailed neurological assessment is a challenge in-particular patients it limits the opportunity to understand the pathophysiology behind. Direct or in-relation to various inflammatory mediators the underlying mechanism targets the neuronal injuries [8], which further augments a hyperinflammation syndrome [9].

Furthermore, this evaluates amalgamation of both inflammatory and immune mediated reactions at the insult core, which then introduces consequences of sepsis, hypoxia and hypercoagulability.

Earlier, the virus was isolated in CSF samples of by RT-PCR in cases of encephalopathy with seizures [10] and was cultured from the brain tissue autopsy [11]. In context, virus association with neuropathology has been increasing with middle aged patients confirming encephalitis [12]. Moreover, similar age patients were also reported ischaemic stroke in the context of hypercoagulability [13]. Additionally, other pathologies such as GBS, two-thirds of patients with GBS were predecessor of respiratory or gastroenterological illness. In early phase of the pandemic, GBS associated virus infections were reported in Italy [5] although more data is required to enlighten this relationship. Patients with acute illness and poor outcome are considered to be at high risk of developing hypercoagulability post discharge [14]. Therefore, it is crucial to consider an anticoagulant treatment regimen post-COVID-19 infection. To prevent this, Low Molecular Weight Heparin (LMWH) has been introduced over direct anti-coagulants due to possibility of interaction covid treatment options [15]. Presumably, non-hospitalised patients or COVID-19 patients with no symptoms will also have a pressing need for anti-coagulation. In the time of uncertainty, we must follow guidelines introduced by CDC. Other parameters such as C-Reactive Protein (CRP), lactate dehydrogenase (LDH), procalcitonin with D-dimer, fibrinogen, and Prothrombin Time (PT) are being used in the inpatient setting. In present times, majority of consultations occur through telehealth to minimize transmission, the index of suspicion of hypercoagulability should remain high [16]. Moreover, upsurge in cytokines and activation of type II pneumocytes post insult seem to play dominant role [17]. The cytokine-dominated insult found to be similar to other pathologies such as secondary haemophagocytic lymphohistiocytosis syndrome [18]. Usually steroids have a good potency towards inflammation but here they have been identified as a comorbid risk factor for poor outcome.

### Conclusion

In conclusion, an effective clinical and non-clinical trials would help in future shaping of an approach to the covid patients and could reduce the burden of neurological associated outcomes. Further eye-catching findings would also highlight other post-treatment complications. The gap in various guidelines around the world would warrant associated severe neurological sequelae. With a recent 2<sup>nd</sup> and 3<sup>rd</sup> waves of infection will further impact social and economic costs. Although, this is a selective discussion, with the numerous limitations associated with research updates. Lastly, the upcoming tsunami of various vaccination options will also play a dominant role in shaping the COVID-19 conduit.

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