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Global Neurological Involvement in Covid-19 Presenting as Guillain Barre Syndrome

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ABSTRACT

COVID 19 most commonly presents with flu like symptoms but neurological involvement is not unknown. Cases with neurological involvement in association with COVID 19 are being rapidly reported from around the globe. The spectrum of neurological involvement is wide and the exact mechanism is still not understood. We hereby report a case of a young male with extensive neurological who presenting as Guillain barre syndrome in association with COVID 19.

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Introduction

COVID 19 most commonly affects the respiratory system but extra pulmonary involvement has also been reported. Several neurological illnesses like meningo-encephalitis, encephalopathy, stroke and Guillain Barre syndrome have been reported in SARS CoV 2 infected patients [1,2]. We hereby report a young patient who has central, peripheral, cranial nerve and autonomic nervous system involvement in association with COVID, with COVID antibodies positive in CSF. Such extensive nervous system involvement in association with COVID has not been described earlier in the literature.

Case Report

A 19 years old male, resident of New Delhi, India presented with sudden onset history of inability to walk since one day. The weakness started in the form of difficulty in climbing stairs associated with mild pain in both lower limbs, the weakness gradually progressed to inability to stand or walk without support over a period of 24 hours. There was history of low grade fever 10 days prior to this weakness, which was self-limiting and had no systemic features. There was no history of visual disturbances, trauma, abnormal body movements and urinary or stool incontinence. At the time of admission, his vitals were stable, general examination did not reveal any significant abnormality. On Nervous system examination, both lower limbs had decreased tone, power was 2/5 bilaterally, lower limb DTRs were diminished (1+) and bilateral plantar reflex was flexor, Sensory system examination was unremarkable. Upper limb and cranial nerve examination were within normal limits. MRI screening of the whole spine showed a focal syrinx formation at D6 level. Nerve Conduction Study was done which showed evidence of a proximal and distal mildly asymmetrical large fibre motor demyelinating and axonal polyradiculopathy affecting bilateral upper and lower limbs. CSF examination showed proteins 54.0mg/dl, glucose 68mg/dl and 2 cells (100% lymphocytes). A provisional diagnosis of Guillain Barre Syndrome was made based on acute ascending paralysis and NCV findings and patient was started on intravenous immunoglobulins (IVIG), in a dose of 2mg/kg body weight. On day 2 of admission, patient's weakness worsened to involve both upper limbs along with urinary incontinence. On 4th day of admission, the patient developed features of neurological weakness in the form of bulbar involvement, in the form of difficulty in swallowing, gag reflex was bilaterally absent and the patient was shifted to the intensive care unit in view of pooling of secretions and difficulty in breathing for which he was intubated and started on mechanical ventilation. After 5 days of IVIG therapy patient did not show any improvement, also developed left sided lower motor neuron facial palsy and autonomic nervous system involvement in the form of episodic tachycardia and hypertension. A repeat infusion of IVIG was administered in a dose of 1mg/kg body weight as there was minimal response. Patient was evaluated for the probable etiology, EBV and CMV PCR were negative. Considering the history of self-limiting fever, COVID PCR was sent which was negative but serum COVID antibodies were found to be positive with a high titre of 237 (positive >1). A repeat CSF examination was done which showed cyto-albumin dissociation [glucose 72 mg/dl, protein 358.6 mg/dl, albumin 116.3 mg/dl, 8 cells (100% lymphocytes)], CSF COVID antibodies were found

to be positive with a titre of 9.11. Bedside EEG was done which revealed moderate degree of diffuse encephalopathy and MRI Brain was normal. At one month follow up the patient showed improvement in the form of flickering movements in his fingers and toes and his gag reflex is bilaterally sluggish but was dependent on ventilator.

Discussion

Our patient had COVID associated global neurological involvement and presented GBS with the CSF showing presence of SARS CoV 2 specific antibodies. To the best of our knowledge this is amongst the first such cases to be reported.

The exact incidence of neurological involvement in association with SARS CoV 2 is known. Also the exact neurotropic nature of the virus remains unclear, but may be related to the virus crossing the blood brain barrier reaching to the hypothalamus through the olfactory pathway, as indicated by post mortem studies in SARS.

Multiple cases of GBS associated with COVID have been reported, which include all types of GBS based on the electrophysiological testing, in our case we could demonstrate specific antibodies against COVID 19 in the CSF. CSF analysis from COVID-19 associated GBS patients have been negative for SARSCoV-2 by RT-PCR and post-mortem biopsy of the brain tissues from COVID-19 patients were negative for any evidence of viral infection by immunohistchemical analysis[3]. However, few cases of encephalitis associated with SARS CoV 2 RNA positive in CSF have been reported[4]. Andriuta et al have reported two cases of COVID 19 associated encephalopathy with presence of antibodies against SARS CoV 2 in CSF[5].

Presently due to paucity of litrature, the neuropathogenesis and tropism of the nervous system involvement in SARS CoV 2 is not fully understood. Ling Mao et al proposed that SARS-COV-2 may enter the CNS via hematogenous or retrograde neuronal route[6]. SARS-CoV-2 attachment to the cell surfaces occurs through the viral spike (S) Protein, S protein binds to ACE 2 and gangliosides containing sialic acid residues including the GalNAc the residue of GM1. Cross-reactivity between the viral protein associated gangliosides and peripheral nerve gangliosides has been suggested to result in molecular mimicry [7]. The mechanism of nerve damage has also been proposed to be facilitated through the T-cell activation and release of inflammatory mediators from the macrophages. The immune system deregulation in COVID-19 patients as described as systemic hyper inflammation with a macrophage activation syndrome [8].

The disruption of blood brain barrier possible occurs due to a direct effect of SARS CoV 2 but is likely that it occurs secondary to generalised endothelitis. This hypothesis is supported by a study done by Bellon et al who studied the CSF features in 31 patients who tested positive for SARS CoV 2 in respiratory specimen and developed neurological manifestations, they concluded that there was no evidence of direct viral infection in the central nervous system based absence of SARS CoV 2 RNA in CSF using RTPCR; no signs of CNS inflammation and absence of SARS CoV 2 specific intrathecal IgG antibodies [9].

Presently, the literature available on neurological involvement in association with COVID 19 is in the form of case reports. Garg suggested that neurological manifestations associated with COVID 19 has more commonly been observed in cases with severe disease. Studies suggest that COVID 19 patients admitted with neurological manifestations have significantly higher in hospital mortality [10].

Key message

With this case we intend to bring to notice that COVID 19 can present with extensive neurological involvement even without involvement of the respiratory system. Thus, it is important to screen patients presenting with different neurological manifestation during the pandemic.

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Conflict of interest: none

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