ANTI-NMDAR AUTOIMMUNE ENCEPHALITIS IN COVID-19 PNEUMONIA PATIENT

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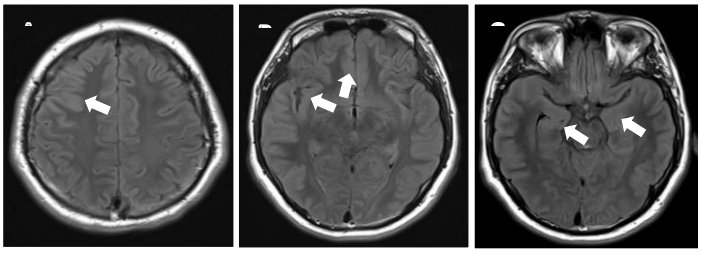
There are 7 identified strains of coronavirus capable of infecting humans (HCoV).[1] Five of these strains can cause nervous system disturbances (229E, OC43, SARS-CoV, MERS-CoV and SARS-CoV2)[2]. There is scientific evidence of their ability to penetrate to central nervous system (CNS) through different routes (neuroinvasion) and infect neurons and glia cells (neurotropism) to possibly induce neurological diseases (neurovirulence). According to murine models, there are two routes by which HCoVs can reach CNS: through hematogenic dissemination and through retrograde transport across the neurons of the olfactory bulb[1, 3]. SARS-CoV and SARS-CoV2 are known to use angiotensin-converting enzyme receptor 2 (ACE2) as the binding site for protein-S to subsequently penetrate the host cell; there is abundant presence of ACE2 in different CNS regions, and this may be binding site for neurons to[4]. The most common neurologic symptoms reported to be associated to SARS-CoV2 infection are headache, confusion, dizziness, hypogeusia an hyposmia/anosmia[5]. So far, there are few reports of patients with autoimmune inflammatory damage to CNS triggered by SARS-CoV2. Panariello et al, in a hospital in Milan, reported a 23-year-old patient with initial symptoms of COVID-19, who subsequently presented disorganized thinking, hallucinations, dyskinesia, autonomic instability, and positive autoantibodies against NMDA receptor (anti-NMDAr) in CSF, who responded clinically to immunomodulatory therapy with intravenous immunoglobulin[6].

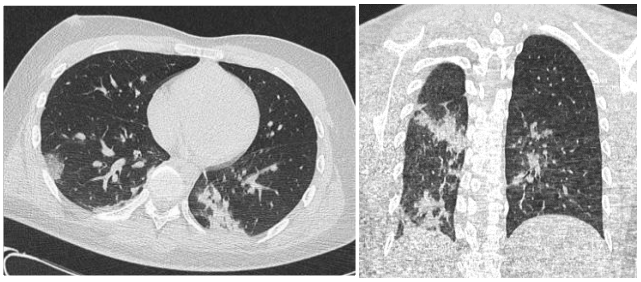
Case presentation

16-year-old male, high school student, no history of previous illnesses or significant risk factors, developed two acute generalized tonic-clonic seizures for which treatment with magnesium valproate was started. For the next two weeks, neuropsychiatric symptoms were added: soliloquies, restlessness, insomnia, suspicion, mystical-religious thoughts, visual and auditory hallucinations, sustained and prolonged postures periods, bizarre facial expressions, as well as memory problems, disorientation, judgment errors and language disturbances. One month later developed a cluster of five seizures in 24 hours and was took to our emergency department. In neurologic examination he presented catatonic syndrome (catatonia rating scale 4 points); the cerebrospinal fluid (CSF) study with 87 cells/mm^3 and protein levels at 25 mg/dL. Cerebral magnetic resonance imaging with hyperintensities in dorsolateral, medial and orbitofrontal prefrontal cortex, both insular cortex and hippocampus, predominantly on the right side in FLAIR.



Diagnosis of autoimmune encephalitis was suspected and treatment with high doses of intravenous methylprednisolone (1g per day) was started for 5 days, followed by 5 plasma exchanges (PLEX) at 48-hour intervals. Anti-NMDAR antibodies were tested positive in CSF (cell-based assay [CBA]); PCR multiplex, Gene Xpert MTB RIF, cultures, Gram, Zihel-Neelsen and China ink stain was negative. V.D.R.L, HIV serology and other tests reported negative.

Ten days post hospitalization, after his second PLEX, the patient began with fever, chills, myalgia, and malaise. A chest computed tomography scan (CT) revealed unilateral subpleural nodular ground glass opacities and pulmonary consolidation in lung bases compatible with SARS-CoV2 infection. The nasopharyngeal swap PCR test was positive.



PLEX was suspended due to decreased platelet count to 80,000/mm^3, spontaneously recovered to normal after which treatment was restarted one week later. Finally, the patient evolved satisfactorily with disappearance of neuropsychiatric symptoms and seizures, with only slight residual cognitive alterations. Rituximab 2g was administered for maintenance therapy.

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