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COVID-19, prevention & management of epilepsy

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SARS-CoV-2 virus (a single-straded RNA virus) can break the blood–brain barrier and enter into the brain. The virus, possibly, travels to the brain by a hematogenous route. The virus can also enter trans-neuronally to the brain via the olfactory system, across the cribriform plate. Angiotensin-converting enzyme 2 receptors that are present on endothelial cells of cerebral vasculature act as cell entry points for virus. At the microscopic level, the SARS-CoV-2 engages ACE2 as an entry receptor and employs the host cell enzyme, Transmembrane Serine Protease 2 (TMPRSS2) or furin for spike protein priming. Priming of the spike protein occurs by cleavage of the spike protein (by host cell enzymes), exposing fusion peptides (that fuse the viral membrane with the host cell). Once fusion occurs, the virus can enter the cell.

Competitive blockage of angiotensin-converting enzyme 2 by the SARS-CoV-2 virus downregulates angiotensin-converting enzyme 2 expression leading to uncontrolled blood pressure and the enhanced possibility of cerebrovascular accidents. Cytokines are crucial mediators of the inflammation in COVID-19. Severe COVID-19 is characterized by markedly elevated levels of proinflammatory cytokines, lymphopenia, an increased number of neutrophils. This kind of cytokine profile is termed as "cytokine storm or bradykinin storm". Interleukin 6 is a key element of the cytokine storm. Other proinflammatory cytokines that are elevated in cytokine storm areinterleukin-1β, interleukin-2, interleukin-8, interleukin-17, chemokine ligand 3, granulocyte-colony stimulating factor, granulocyte-macrophage colony-stimulating factor, the human interferon-inducible protein, and tumor necrosis factor-alpha. Cytokine storm is associated with enhanced vascular hyperpermeability, coagulopathies, and multisystem dysfunction. Postinfectious autoimmune reactions can affect neuronal cells.

The SARS-CoV-2 virus epitopes bear a structural resemblance to several human proteins. Molecular mimicry between virus epitope and myelin basic protein results in autoimmune postinfectious demyelinating syndromes. Dysregulation of the angiotensin-converting enzyme 2 receptor also contributes to the pathogenesis of experimental autoimmune encephalomyelitis. Spike surface glycoprotein plays a crucial role in immunopathology.

Common laboratory findings include lymphopenia (mostly), increased neutrophil count, eosinopenia along with prolonged prothrombin time. raised lactate dehydrogenase, raised alanine aminotransferase, raised aspartate aminotransferase, a high troponin and markedly elevated D-dimer, and C-reactive protein levels. Increased ferritin level is an indicator of the imminent cytokine storm.[Recently, IgM and IgG antibody tests to detect antibodies, against SARS-COV-2 infection in human blood, serum/plasma, have been made available. These tests are valuable as they can be used for screening purposes in a large population Recently, IgM and IgG antibody tests to detect antibodies, against SARS-COV-2 infection in human blood, serum/plasma, have been made available. These tests are valuable as they can be used for screening purposes in a large population.