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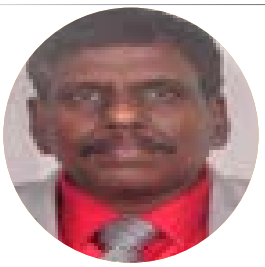

Ramachandran Muthiah
Morning star hospital, India

Neurovascular dysfunction in covid-19

Viral infection is initiated from the surface spike glycoprotein, which binds to ACE2 receptor in brain vascular endothelium and smooth muscle cells and enter the cells by endocytosis. After priming by TMPRSS2 (transmembrane protease serine 2), it is activated and then internalized, results in an imbalance in homeostatic procoagulant and anticoagulant pathways, Cytokine storm, endothelial dysfunction and dysregulated activation of macrophages contributes to hyperinflammation and thrombosis. Overactivation of NADPH oxidase-2 (Nox2), resulting in increased reactive oxidant species, is implicated in arterial vasoconstriction, clotting, and platelet activation. Virus directly infecting stromal cells via interaction with CD13 or CD66a adhesion molecules andinduce platelet aggregation via PAC-1 binding. Persisting changes of blood cell physical phenotypes contribute to long-term microcirculatory dysfunction. Cytokines induce cytoskeletal changes in myeloid cells and erythrocytes and impaired oxygen delivery. In severe COVID-19, low-density phenotype that is prone to neutrophil extracellular trap formation (NET), with elevated size and deformability as a source of vascular occlusion. Sars-CoV-2 propagates in hematopoietic progenitor, erythroid, and megakaryocytic cells as the main cause of thrombotic events. Viral cytopathic effects in peripheral smear and the peculiar morphological findings would suspect a diagnosis in the absence of a negative RT-PCR or antibody results. The coagulopathy characterized by an increase in procoagulant factors such as fibrinogen with a strong increase of D-dimers have been associated with higher mortality. The incidence of cerebral infarction in COVID-19 is 4.5%. Thromboprophylaxis with LMWHs is recommended in hospitalized patients with high levels of D-dimer indicating hypercoagulative state. Intravenous recombinant tissue plasminogen activator (rt-PA) for selected patients. MSCs therapy could help to cure the inflammation and coagulopathy by a vascular effect. Very small embryonic like stem cells (VSELs) have endothelial angiogenic potential. Exogenous ACE2 with human recombinant soluble ACE2 is a novel treatment for stroke and reverse endothelial dysfunction. JAK inhibitors reduce hyperinflammation.

Biography

Ramachandran Muthiah, Consultant at Zion hospital, Azhagiamandapam and Morning Star hospital, Marthandam, Kanyakumari District, India.. Completed primary education at Anaan vilai in keezhkulam and secondary education at Concordia Higher secondary school, Pootteti. MBBS in 1988 (Madurai Kamaraj), M.D. in 1996, D.M. in 2003 Dr.MGR Medical University, Chennai, 6 months course in Interventional cardiology at Batra Hospital, New Delhi in 2006 (Ministry of health, Govt of India). Worked as medical officer in Rural health services for 5 years (keezhachekkarakudi, Aryappapuram Primary health centres, ESI hospital, Singanallur, coimbatore), teaching category as Assistant Professor at Madras, Coimbatore and Thoothukudi medical colleges. Published papers in Cardiosource, American College of Cardiology Foundation, Case Reports in Clinical Medicine (SCIRP) and Journal of Saudi Heart Association.

cardioramachandran@yahoo.co.uk

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