Ischemic stroke complications

- Efficacy and safety of trans-carotid approach for mechanical thrombectomy in acute ischemic stroke: a multicenter two-arm case series with systematic review and meta-analysis
- Prognostic Value of Pan-Immune Inflammation Value for Major Adverse Cardiac Events and Mortality in Patients with Aortic Stenosis After TAVI
- Emergent Endovascular Intervention for Acute Neurological Deficits Post-Carotid Endarterectomy: A Single-Institutional Analysis and Systematic Review of the Literature
- Psychiatric symptoms in stroke patients: Clinical features of depression and anxiety
- Unmet Needs in Acute Ischemic Stroke: Overcoming Recalcitrant Clots
- Efficacy of danhong injection adjuvant therapy in patients with acute ischemic stroke: a real-world, multicenter, retrospective study
- Prediction of mortality in severe ischemic stroke during COVID-19 in patients with multiorgan dysfunction
- Knockdown of RUNX2 Attenuated A1 Astrocyte Overactivation, Brain Injury, and Cerebral Edema During Ischemic Stroke

The systemic inflammation response to the ischemic stroke, followed by immunosuppression in focal neurologic deficits and other inflammatory damage, reduces the circulating immune cell counts and multiorgan infectious complications such as intestinal and gut dysfunction dysbiosis. Evidence showed that microbiota dysbiosis plays a role in neuroinflammation and peripheral immune response after stroke, changing the lymphocyte populations. Multiple immune cells, including lymphocytes, engage in complex and dynamic immune responses in all stages of stroke and may be a pivotal moderator in the bidirectional immunomodulation between ischemic stroke and gut microbiota [1].

Ischemic stroke (IS) is a risk factor for new coronavirus pneumonia, and patients with COVID-19 infection have a dramatically elevated risk of stroke. At the same time, patients with long-term IS are vulnerable to COVID-19 infection and have more severe disease, and carotid atherosclerosis is an early lesion in IS. A study used human-induced pluripotent stem cell (hiPSC)-derived monolayer brain cell dataset and human carotid atherosclerosis genome-wide dataset to analyze COVID-19 infection and carotid atherosclerosis patients to determine the synergistic effect of new coronavirus infection on carotid atherosclerosis patients, to clarify the common genes of both, and to identify common pathways and potential drugs for carotid atherosclerosis in patients with COVID-19 infection. Using several advanced bioinformatics tools, we present the causes of COVID-19 infection leading to increased mortality in carotid atherosclerosis patients and the susceptibility of carotid atherosclerosis patients to COVID-19. Potential therapeutic agents for COVID-19 -infected patients with carotid atherosclerosis are also proposed.

With COVID-19 being a relatively new disease, associations have been proposed for its connections with several ailments and conditions, including IS and carotid atherosclerosis. More patient-based data-sets and studies are needed to fully explore and understand the relationship [2].

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see Recurrent ischemic stroke.

Patients presenting with large-territory ischemic strokes may develop intractable cerebral edema that puts them at risk of death unless intervention is performed.

Stress ulcers

In ischemic stroke or patients with TIA less than five cerebral microbleeds (CMBs) should not affect antithrombotic decisions, although with more than five CMBs the risks of future ICH and ischaemic stroke are finely balanced, and antithrombotics might cause net harm. In lobar ICH populations, a high burden of strictly lobar CMBs is associated with cerebral amyloid angiopathy (CAA) and high ICH risk; antithrombotics should be avoided unless there is a compelling indication [3].

Hemorrhagic transformation

see Hemorrhagic Transformation After Ischemic Stroke

1)
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2)
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3)
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