Special Issue

Reperfusion in Ischemic Stroke

Message from the Guest Editors

Not all patients clinically improve after acute ischemic stroke early recanalization therapy: the restoration of blood flow (reperfusion) could lead to a paradoxical worsening of brain damage through a complex series of biochemical and cellular pathophysiological mechanisms, the so-called reperfusion injury. The disruption of the neurovascular unit, namely the cellular and extracellular components involved in the regulation of cerebral blood flow and blood–brain barrier (BBB) function (endothelial cells, basal lamina matrix, astrocyte, pericytes, neurons, and supporting cells), is responsible for the major clinical–radiological complications of acute ischemic stroke, cerebral edema, and hemorrhagic transformation. The current literature suggests that cerebral edema and hemorrhagic transformation share both pre-transcriptional and transcriptional factors and should be considered to belong to the same physio-pathological continuum, pointing out that blood components' extravasation could represent the result of a complex process triggered by ischemic cascade.

Guest Editors

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