Cerebral collateral circulation

The cerebral collateral circulation refers to the subsidiary network of vascular channels that stabilize cerebral blood flow when principal conduits fail. Arterial insufficiency due to thromboembolism, hemodynamic compromise, or a combination of these factors may lead to the recruitment of collaterals.

Leptomeningeal collateral status

Leptomeningeal collateral status

Primary collateral pathways

Primary collaterals include the arterial segments of the circle of Willis.

Secondary collateral pathways

Ophthalmic artery and Leptomeningeal collateral circulation

Importance

Acute ischemic stroke outcome depend in part on the extent of their collateral circulation. Good collateral circulation has also been associated with the greater benefits of intravenous thrombolysis and endovascular treatment. Acute ischemic stroke treatment decisions for these reperfusion therapies are increasingly guided by a combination of clinical and imaging parameters, particularly in later time windows. Computed tomography and magnetic resonance imaging enable a rapid assessment of both the collateral extent and cerebral perfusion. Yet, the role of the collateral circulation in clinical decision-making is currently limited and may be underappreciated due to the use of rather coarse and rater-dependent grading methods. Uniken et al. discussed determinants of collateral circulation in patients with acute ischemic stroke, reported on commonly used and emerging neuroimaging techniques for assessing collateral circulation, and discuss the therapeutic and prognostic implications of collateral circulation in relation to reperfusion therapies for acute ischemic stroke [1)].

The pial (leptomenigeal) collateral circulation is a key determinant of functional outcome following mechanical thrombectomy after large vessel ischemic stroke. Patients with good collateral blood flow benefit up to 24 hours after stroke onset, while those with poor collateral flow evidence less or no benefit. However, clues to why collateral flow varies so widely among patients have remained elusive. Recent findings in animal studies, which are currently being tested for confirmation in humans, have found that naturally occurring variants of a novel “collateral gene”, Rabep2, result in large differences

in the extent of anatomic collaterals and thus blood flow and infarct size in mice after stroke. The comprehension of collagerogenesis in humans and the evaluation of collateral status could aid in identifying patients who will benefit not only from mechanical thrombectomy in the extended time window, but also from any reperfusion strategy. We performed a literature review focused on radiographic, clinical and genetic aspects of the collateral circulation [2].

1)
Uniken Venema SM, Dankbaar JW, van der Lugt A, Dippel DWJ, van der Worp HB. Cerebral Collateral Circulation in the Era of Reperfusion Therapies for Acute Ischemic Stroke. Stroke. 2022 Aug 8:101161STROKEAHA121037869. doi: 10.1161/STROKEAHA.121.037869. Epub ahead of print. PMID: 35938420.

2)
Piedade GS, Schirmer CM, Goren O, Zhang H, Aghajanian A, Faber JE, Griessenauer CJ. Cerebral collateral circulation: A review in the context of ischemic stroke and mechanical thrombectomy. World Neurosurg. 2018 Oct 17. pii: S1878-8750(18)32363-5. doi: 10.1016/j.wneu.2018.10.066. [Epub ahead of print] Review. PubMed PMID: 30342266.