Cerebral hypoperfusion

Etiology

Multiple trauma (MT) associated with hemorrhagic shock (HS) might lead to cerebral hypoperfusion and brain damage.

see Chronic cerebral hypoperfusion.

see Orthostatic Cerebral Hypoperfusion Syndrome.

see Aneurysmal Subarachnoid Hemorrhage

Diagnosis

Cerebral Ultrasound Perfusion Imaging is feasible to enable detection of cerebral tissue hypoperfusion after aSAH, and the left-right difference of TTP values is the most indicative result of this finding [1].

The development of noninvasive approaches for identifying hypoperfused brain tissue at risk is of major interest. The temporal-shift (TS) maps estimated from resting-state blood oxygenation level-dependent (BOLD) signals have been proposed for determining hemodynamic state.

BOLD TS analysis has the potential as a non-invasive alternative to current methods based on CVR for the identification of tissue at risk of ischemic stroke [2].

In a retrospective study, Warnert et al. focused on quantifying the anatomical differences in the posterior circulation of 133 hypertensive subjects diagnosed with essential hypertension and compared these data to that of 306 healthy controls. Blood pressure measurements were taken in both arms in seated position using standard automated sphygmomanometry. In more than half of patients, 24-h ambulatory blood pressure monitoring was completed. Then, they performed 3-dimensional (3D) time-of-flight MR angiography at 1.5 T with dedicated coil to study posterior circulation anatomy. They defined vertebral hypoplasia as vessel diameter less than 2 mm throughout the vessel. They noted a higher percentage of congenital cerebrovascular variants such as vertebral artery hypoplasia and incomplete Circle of Willis in hypertensive subjects (53% and 64% vs 27% and 36% in normotensive subjects, respectively, with $P < .0001$). In fact, the odds ratios indicated that individuals with vascular artery hypoplasia and incomplete circle of Willis were about 3 times more likely to have hypertension.

Following the results of the above study, the authors then performed a case-control study with 77 hypertensive and 49 normotensive subjects to determine whether anatomical differences in posterior circulation translate into differences in cerebral perfusion and vascular resistance. High-resolution 3 T MRI gradient echo 3D-fast spoiled gradient echo was used for structural scan, 3D time of flight angiography for arterial anatomy. Phase-contrast pulse-sequences were used to measure blood flow

in internal carotids and basilar artery at baseline and pseudocontinuous arterial spin-labeling was used to measure regional cerebral blood flow. Posterior circulation variants were associated with increased vascular resistance and decreased cerebral blood flow. Using binary logistic regression, the authors found that individuals with vascular artery hypoplasia, an incomplete circle of Willis, or both, were 3.0, 2.6, and 3.2 times more likely to have hypertension (odds ratios, 95% confidence intervals 1.4-6.3, 1.2-5.6, and 1.4-7.6, respectively). Incomplete anterior circle of Willis had no bearing on predicting whether an individual would have hypertension (P = .46).

Total arterial cerebral blood flow was lower and vascular resistance was higher in hypertensive than normotensive group using MR contrast-phase imaging (analysis of variance [ANOVA], P < .0001, for both). Next, the authors found that those with vertebral artery hypoplasia had a lower total arterial cerebral blood flow and higher cerebral vascular resistance (ANOVA, P < .0001). Interestingly, in subjects with vertebral artery hypoplasia, the larger contralateral artery did not fully compensate for the hypoplastic vessel. Also, in what may become a controversial point in the future, they found that subjects treated with antihypertensive therapy had decreased cerebral blood perfusion.

Data indicate that congenital cerebrovascular variants in the posterior circulation and the associated cerebral hypoperfusion may be a factor in triggering arterial hypertension. Therefore, lowering blood pressure may worsen cerebral perfusion in susceptible individuals [3].

1)
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2)
Nishida S, Aso T, Takaya S, Takahashi Y, Kikuchi T, Funaki T, Yoshida K, Okada T, Kunieda T, Togashi K, Fukuyama H, Miyamoto S. Resting-state Functional Magnetic Resonance Imaging Identifies Cerebrovascular Reactivity Impairment in Patients With Arterial Occlusive Diseases: A Pilot Study. Neurosurgery. 2018 Sep 20. doi: 10.1093/neuros/nyy434. [Epub ahead of print] PubMed PMID: 30247676.
3)
Warnert EA, Rodrigues JC, Burchell AE, Neumann S, Ratcliffe LE, Manghat NE, Harris AD, Adams Z, Nightingale AK, Wise RG, Paton JF, Hart EC. Is High Blood Pressure Self-Protection for the Brain? Circ Res. 2016 Dec 9;119(12):e140-e151. Epub 2016 Sep 26. PubMed PMID: 27672161.