Journal of Neuroradiology (2014) 41, 234—242

Available online at ScienceDirect www.sciencedirect.com

Elsevier Masson France EM|consulte www.em-consulte.com/en

ORIGINAL ARTICLE

Assessment of collateral flow in patients with cerebrovascular disorders

Joseph Donahue, Suna Sumer, Max Wintermark*

Division of Neuroradiology, University of Virginia, Charlottesville, VA, United States

Available online 30 December 2013

KEYWORDS
Collateral circulation;
Cerebrovascular circulation;
Perfusion Imaging;
Cerebrovascular accident

Summary The ability to maintain cerebral parenchymal perfusion during states of acute or chronic ischemic insult depends largely on the capacity of the cerebral collateral circulation. Perfusion techniques, including perfusion-CT and arterial spin labeling, may not only describe the overall status of the collateral network, but can also quantify the pathophysiologic collateral reserve, which is occult to conventional imaging techniques. The following review details advanced imaging modalities capable of resolving pathophysiologic collateral circulation in a functional and dynamic manner, with regards to the evaluation of both acute ischemic penumbra and chronic cerebral vascular reserve. Specifically, the applications of perfusion-CT, arterial spin labeling MRI techniques, and transcranial Doppler are reviewed in the context of collateral circulation with emphasis on perfusion techniques and proposed clinical utility.

Introduction

In general terms, cerebral collateral circulation is the network of vascular anastomoses providing supplemental blood flow in states of acute and/or chronic principal perfusion pathway insufficiency. Collateral circulation pathways encompass a number of entities which may be broadly categorized into large structural collateral vessels (primary and secondary) and pathophysiological collateral vessels [1].

The Circle of Willis constitutes the primary structural collateral circulation pathway between the hemispheres, as well as between the anterior and posterior circulations. The Circle of Willis, and its well-known variations or deficiencies, are readily detailed by conventional imaging modalities such as CTA/MRA and direct catheter angiography [1,2].

Secondary structural collateral pathways include both extracranial and intracranial anastomoses, which are expected normal anatomic vascular structures that may become hypertrophied with ischemic stimulus. The largest and most clinically relevant example of these secondary structural collateral pathways is the intracranial leptomeningeal anastomoses between the distal cortical branches of the intracranial arteries. These vessels are resolvable with CTA and MRA techniques [1,3,4].

Pathophysiologic collateral vessels are potential anastomotic pathways, which are recruited over time by ischemic parenchyma when principle arterial pathways are insufficient to provide stabilized tissue perfusion [1]. Although not

Abbreviations: CTA, computed tomography angiography; MRA, magnetic resonance angiography; MTT, mean transit time; rCBF, regional cerebral bold flow; rCBV, regional cerebral blood volume; ASL, arterial spin labeling.

* Corresponding author. Radiology and Medical Imaging P.O. Box 800170 [FedEx: 1215 Lee Street] Charlottesville, VA 22908, United States. Tel.: +1 434 243 9312.
E-mail address: max.wintermark@gmail.com (M. Wintermark).

0150-9861/$ - see front matter © 2013 Published by Elsevier Masson SAS.
http://dx.doi.org/10.1016/j.neurad.2013.11.002

necessarily derived from a specific and anatomically constant arterial plexus, the pathophysiologic collaterals may be of significant importance to the overall cerebrovascular reserve capacity.

Cerebral collateral circulation is a dynamic entity on a number of levels. Within seconds of a large artery occlusion, changes in the direction and velocity of flow within the primary structural collaterals are mediated by changes in blood pressure gradients and reflexive vasodilation. Such changes are noted to occur within several heart beats during carotid endarterectomy. An incompletely understood complex interplay of metabolic and hemodynamic factors are proposed to initiate and sustain recruitment of secondary structural and pathophysiological collateral vessels in states of chronic hypoperfusion. Additionally, various comorbidities such as hypertension may variably decelerate or endanger incipient collateral network growth and the overall tissue perfusion capacity [2].

The dynamic nature of these cerebrovascular reserve components emphasizes the need for a multifaceted interrogation of collateral vessel status. CTA, MRA, and direct catheter angiography are all well recognized and accurate modalities for the evaluation of structural primary and secondary collateral vessels [5]. However, the parenchymal perfusion capacity of the pathophysiologic collateral pathways may not necessarily be inferred from the status of primary and secondary structural collateral vessels [6]. As such, during periods of ischemia, tissue fate may depend on the status of these hemodynamically significant pathophysiologic collateral pathways that are occult to conventional vascular imaging modalities [7].

With this in mind, the following discussion is intended to explore the role of perfusion-CT in the assessment of cerebral collateral circulation with regards to two specific applications: acute ischemic stroke and chronic cerebrovascular disease.

Assessment of cerebral collateral circulation in acute ischemic stroke

The adequacy of collateral circulation in acute ischemic stroke is an important determinant of the fate of the ischemic tissue and the prognosis for vascular recanalization [8,9]. The blood flow supplied by the collateral arterial network helps maintain misery perfusion in areas of ischemic penumbra. Robust collaterals also likely facilitate the ''wash-out'' of thrombi fragments and mitigates against infarct progression. Similarly, collateral arterioles may also help deliver thrombolytic agent to more distal thrombotic regions, thereby assisting in more rapid recanalization.

Functional imaging of cerebral collateral circulation

Perfusion-CT

In states of hemodynamic perturbation, blood may arrive to parenchyma via collateral pathways in antegrade or retrograde direction; however, this is difficult to distinguish in practice. Perfusion-CT complements angiographic techniques by offering a dynamic assessment of parenchymal perfusion, regardless of occlusion site or directionality of blood flow [10]. This technique allows for the assessment of the pathophysiologic collateral vessels as inferred from the ischemic penumbra estimate [6,10].

	MTT	rCBF	rCBV
TIA	↑	normal	↑
Penumbra	↑↑	↓	↑
Infarct	↑↑↑	↓↓	↓

Figure 1 Interpretation of perfusion-CT maps. The green and red boxes highlight key differences in perfusion characteristics of endangered yet salvageable ischemic penumbra and irreversible ischemic core.

Perfusion-CT monitors the parenchymal wash-in and wash-out of an iodinated contrast material bolus with serial acquisitions, usually at several supratentorial levels. In the post-processing stage, the investigator or the processing software select a reference artery input and vein output functions. A series of time-concentration curves are then generated for each voxel, and perfusion-CT parameters are generated that describe these curves in each voxel: mean transit time (MTT), regional cerebral blood volume (rCBV), and regional cerebral blood flow (rCBF). MTT designates the average time required for blood cell passage through the parenchymal capillary network. rCBV reflects the volume of blood per unit of parenchyma, mL/100 g. rCBF is a rate unit derived from rCBV/MTT. MTT is the most sensitive perfusion indicator of ischemia, with changes in cerebral blood flow and cerebral blood volume are more specific for distinguishing penumbra from ischemic core [9].

As detailed in Fig. 1, MTT is elevated in all ischemic cerebrovascular events. Parenchymal autoregulatory mechanisms initially compensate with reactive vasodilation and collateral flow recruitment, thus increasing rCBV. Again, as rCBF is derived from rCBV/MTT, a proportional increase in both the numerator and denominator will result in an unchanged, normal rCBF value. This is a pattern typically observed during a transient ischemic attack. With worsening and more prolonged ischemia, MTT becomes more prolonged and rCBF eventually decreases. Where collateral circulation is robust enough to maintain rCBV, ischemic penumbra exists; that is, tissue at risk for infarction but that is still currently salvageable. When the autoregulatory mechanisms are overwhelmed such that the collateral circulation is inadequate to maintain threshold tissue blood flow, the rCBV falls and an irreversible infarct develops [10—12].

These principles are illustrated in Fig. 2 with the perfusion-CT map of an acute right MCA territory infarct. In the subcortical areas of ischemic penumbra, the leptomeningeal collateral circulation is adequate to maintain rCBV. However, the caudate head and putamen, perfused by an inadequately collateralized lenticulostriate system, demonstrate decreased rCBV and thus constitute the ischemic core.