Collateral blood supply in acute stroke

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Authors: M. Radzina, J. Savlovskis, A. Balodis, K. Kupcs, E. Miglane; Riga/LV
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Learning objectives

To show cerebral collateral pathways anatomy and pathophysiology in acute ischemic stroke and describe various imaging patterns of collateral circulation in angiography.

Describe role of collateral blood supply in stroke management and prognosis.
Background

Stroke has very variable clinical presentations due to capacity of autoregulatory mechanisms. Collaterals are one of the factors that help to maintain brain perfusion in acute ischemic stroke area. There is still inconsistency in imaging methods and grading of cerebral collateral circulation that plays an important role in appropriate patient selection for acute therapeutic strategies.
Findings and procedure details

Angiography is best suited to determine the anatomy of collateral circulation [Liebeskind et al., 2003]. There are 2 levels: (1) Circle of Willis (with anatomical variations up to 50%) [Hoksbergen et al., 2003] (see Fig.1) and (2) Leptomeningeal or extracranial to intracranial collaterals (see Fig.2) that can be easily detected on computed tomography angiography (CTA), magnetic ressonance angiography (MRA) [Kim et al., 2014] or digital subtraction angiography (DSA). Perfusion study (CT Perfusion or MR Perfusion) can be added to determine the functional adequacy of collaterals to preserve brain perfusion in ischemic area [Romero et al., 2009] (see Fig. 3). First level starts to work within first seconds after ischemic onset, but second level collaterals evaluation has been recently widely debated.

CT angiography collateral grading systems have several grades from 2 (good, poor) to 5 (absent, diminished >50%, <50%, equal, more) and show huge variability in parameters [Tan et al., 2009; Souza et al., 2012; Nambiar et al., 2014] e.g. rapidity of filling, amount, volume, intensity of contrast, comparison to contralateral side, occlusion site [McVerry et al., 2012]. We suggest to use 2 grades system in CTA evaluation - poor and good collaterals with added special conditions as very poor collaterals (1) malignant profile) and very good collaterals (2) almost normal (see Fig.4). Also CTA images should be adjusted to optimal slice thickness (MIP=45) that reveal better M3 and M4 segmental branches of MCA and better evaluation of contrast enhancement can be achieved on inverted images (see Fig.5).

More simple grading is used in DSA (slow/rapid and complete/partial filling or absence of collaterals) by ASITN/SIR system (see Fig.6) together with antegrade flow evaluation by TICI system before and after recanalization therapy (grades 0 to 3) (see Fig.7) [Lau et al., 2012].

In our study of 38 patients (2 certified, 1 fellow) we revealed optimal interobserver reliability in evaluation of collateral grades (certified vs. certified (Kappa=0,80) and Certified vs. Fellow (Kappa=0,78-0,87) [Radzina et al., 2014].

Recent literature proposes a number of hypotheses regarding the development mechanisms of intracranial collaterals and their role in clinical outcome of acute cerebral ischemia. The first hypothesis states that a long-term haemodynamic insufficiency observed in intracranial atherosclerosis contributes to development of intracranial collaterals and the tissue adapt to subsequent ischemia. Collaterals have a protective effect on the brain tissue in severe intracranial stenoses [Liebeskind et al., 2011; Lima et al, 2010].

In cases of impaired antegrade blood supply collaterals provide cerebral blood flow compensation and stabilization within the ischemic area and help to wash-out
microemboli [Caplan et al., 1998]. The effectiveness of collateral circulation may vary in the setting of acute and chronic brain ischemia resulting from intracranial or extracranial artery stenosis/occlusion [Romero et al., 2009]. For example - hypertension impair collateral development in carotid occlusion and increase stroke risk. Presence of good collateral circulation is favorable prognostic indicator in acute and chronic carotid artery disease [Radzina et al., 2013]. With continuing of long-term ICA occlusion, a dilatation of the posterior circulation arteries for 40% proximally was observed after 1 week, and about 72% - after three weeks [Meyer et al., 1957]. However, the actual process of angiogenesis in the human brain still has not been fully explored [Romero et al., 2009].

Second hypothesis says that collateral blood vessels develop at embryonic stage together with the central nervous system and their number in a small volume changes after birth. In animal studies genetic variability is a major determinant factor for development of collaterals [Zhang et al., 2010]. Based on this hypothesis the collateral status in case of progressive ischemia should not deteriorate.

Collateral blood supply role in acute stroke.

Patients with good collateral flow have less hypoperfused tissue and also less infarct growth within penumbra zone, because reperfusion can occur in absence of recanalization due to retrograde flow [Pop et al., 2014].

Good collateral flow is predictor of high recanalization rates. It is more related with intravenous thrombolysis treatment. A possible explanation is the better penetration of medication into thrombus via retrograde flow [Pop et al., 2014].

Poor collaterals lead to unfavorable outcome, particularly in the setting of partial/poor recanalization and extensive baseline lesion (see Fig.8,10). [Bang et al., 2008; Radzina et al., 2014].

With ASPECTS score higher than 1 unit, the possibility for favorable outcome increases 1.3 times and in combination with good collateral flow 3.2 times (see Fig. 9,11) [Radzina et al., 2013].

Collaterals also may promote complications as hemorrhagic transformation that should be considered in pretreatment planning [Liebeskind et al., 2003; Christophoridis et al., 2005].

Multivessel angiography is impractical for all subjects, the development of noninvasive approaches that combine angiographic information with perfusion data promote our understanding of collateral circulation considerably (see Fig.10,11).

Therefore patients should not be selected for the recanalization treatment based solely on the proportion of collaterals, each case should be evaluated individually considering timing, hemodynamic status and vascular alterations as well as careful evaluation.
of imaging technical details as contrast media dynamic, planes, duration of stroke, procedure and site of occlusion.
Fig. 1: Variations of Circle of willis. Hypoplastic posterior communicating artery or hypoplastic posterior cerebral artery proximal segment

**Fig. 2:** Intracranial leptomeningeal collaterals (ACA - anterior cerebral artery, MCA - middle cerebral artery, PCA - posterior cerebral artery, ACoA - anterior communicating artery)

© Liebeskind DS. Stroke 2003;34:2279-2284

**Fig. 3:** CT perfusion imaging. Patient 65 years, male, 2 hours from onset, left hemiparesis IRF to map shows slow transit time in the right hemisphere. CBV and CBF maps show corresponding lesion in right side basal ganglia suggesting necrosis (core lesion) that is surrounded by hypoperfused area (penumbra). (CBV - cerebral blood volume, CBF - cerebral blood flow, CT - baseline computed tomography of the brain)

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Fig. 4: Suggested CTA Collateral grading system. 2 major grades: 1) poor (0 and 1) 2) good (2 and 3) 0 - malignant pattern (absent collateral supply to the occluded MCA territory); 1 - poor patterns - collateral supply filling >50% but <100% of the occluded MCA territory; 2 - moderate pattern - collateral supply filling >50% but <100% of the occluded MCA territory; 3 - good pattern- 100% collateral supply of the occluded MCA territory.

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Fig. 5: Optimization of CTA images for collateral evaluation 1) correct selection of slice thickness (MIP) 2) correct axial plane for comparison of hemispheres 3) inverted image for evaluation of contrast intensity.
**Table**  
The ASITN/SIR Collateral Flow Grading System for determining angiographic collateral grade on pretreatment angiography

<table>
<thead>
<tr>
<th>ASITN/SIR collateral grade</th>
<th>Definition</th>
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<tr>
<td>0</td>
<td>No collaterals visible to ischemic site</td>
</tr>
<tr>
<td>1</td>
<td>Slow collaterals to the periphery of ischemic site, with persistence of some of the defect</td>
</tr>
<tr>
<td>2</td>
<td>Rapid collaterals to the periphery of ischemic site, with persistence of some of the defect, and to only a portion of the ischemic territory</td>
</tr>
<tr>
<td>3</td>
<td>Collaterals with slow but complete angiographic blood flow of the ischemic bed by the late venous phase</td>
</tr>
<tr>
<td>4</td>
<td>Complete and rapid collateral blood flow to the vascular bed in the entire ischemic territory by retrograde perfusion</td>
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**Fig. 6:** The ASITN/SIR Collateral Flow Grading System for determining angiographic collateral grade on pretreatment angiography

<table>
<thead>
<tr>
<th>TICI Score</th>
<th>Description</th>
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<tbody>
<tr>
<td>0</td>
<td>No perfusion</td>
</tr>
<tr>
<td>1</td>
<td>Perfusion past the initial obstruction but limited distal branch filling with little or slow distal perfusion</td>
</tr>
<tr>
<td>2a</td>
<td>Perfusion of less than half of the vascular distribution of the occluded artery</td>
</tr>
<tr>
<td>2b</td>
<td>Perfusion of half or greater of the vascular distribution of the occluded artery</td>
</tr>
<tr>
<td>3</td>
<td>Full perfusion with filling of all distal branches</td>
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**Fig. 7:** Thrombolysis in Cerebral Infarction angiographic response scale.

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**Fig. 8:** Stroke size and collateral flow correlation. Alberta Stroke Program Early CT score (ASPECTS) is a 10-point quantitative topographic CT scan score. Lower score correspond to larger stroke area. Higher score correspond to smaller lesion size. Collaterals are graded as poor, moderate and good. Higher ASPECTS score (smaller lesion size) correlate with good collaterals.

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**Fig. 9:** Neurological status outcome and collateral flow correlation in acute stroke. (NIHSS - National Institute of Health Stroke Scale) Collaterals are graded as poor, moderate, good. Good collaterals correlate positively with mild impairment and poor collaterals - with more severe impairment.

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Fig. 10: Case. Male, 72 years, wake up stroke, NIHSS 20, T occlusion right side, ASPECTS 3 on CT perfusion with malignant collateral pattern on CT angiography. 24h follow-up CT shows malignant stroke with extensive hemisphere lesion and hemorrhagic transformation and unfavorable clinical outcome.

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Fig. 11: Case. Female, 70 years, MCA left M1 occlusion, NIHSS score at admission 14, ASPECTS 9 on CT perfusion with moderate collateral pattern on CT angiography. 24h follow-up CT shows left side lacunar type defect in basal ganglia and patient had favorable clinical outcome with NIHSS score 1.

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Conclusion

The protective action of collaterals in acute stroke depends on a number of factors: anatomic variations, duration and site of occlusion.

CT-based techniques demonstrate good interobserver reliability and correlation with clinical outcome, and may provide an accessible and reliable assessment method for grading collateral flow in acute stroke. Evaluation of brain collaterals along with cerebral perfusion and clinical indicators in acute stroke patients provides a wider insight into the assessment of cerebrovascular conditions, particularly with the development of dynamic CTA combined with perfusion imaging.

Acute stroke patients should not be selected for the recanalization treatment based solely on the proportion of collaterals, each case should be evaluated individually considering timing, hemodynamic status and vascular alterations.
Personal information

Maija Radzina
MD, PhD, Radiologist
Paula Stradina clinical university hospital
Diagnostic Radiology Institute
Riga, Latvia

Janis Savlovskis
Interventional radiologist
Paula Stradina clinical university hospital
Diagnostic Radiology Institute
Riga, Latvia

Arturs Balodis
Radiologist (in training)
Paula Stradina clinical university hospital
Diagnostic Radiology Institute
Riga, Latvia

Karlis Kupcs
MD, PhD, Interventional radiologist
Paula Stradina clinical university hospital
Head of Diagnostic Radiology Institute
Riga, Latvia

Evija Miglane
MD, PhD, Neurologist
Paula Stradina clinical university hospital
Neurology clinic, Head of Stroke unit
Riga, Latvia
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