Imaging of Cerebral Ischemia
From Acute Stroke to Chronic Disorders

May Nour, MD, PhD[a,b], David S. Liebeskind, MD[a,*]

KEYWORDS

• Stroke • Ischemia • MRI • CT • Collaterals

KEY POINTS

- Cerebral ischemia spans a temporal continuum from hyperacute presentation and extends into acute, subacute, and chronic phases.
- Serial imaging of patients throughout the dynamic course of ischemia is highly informative.
- Selection of an appropriate imaging modality to answer the clinical question and to complement the clinical examination is crucial.
- Several neuroimaging modalities yield information regarding the integrity of brain parenchyma, changes in tissue demands and metabolism, severity of vascular disease, and neuronal repair over time.
- Taken in the appropriate context, with the correct selection of imaging tests, clinicians can effectively gain insight into disease mechanism, tailor therapeutic decision-making, and monitor patients for progression of disease over time.

INTRODUCTION

Cerebral ischemia spans a temporal continuum from hyperacute presentation into acute, subacute, and chronic phases. Imaging provides detailed information to the clinician that must be evaluated in light of the patient's symptomatic presentation and clinical examination. Imaging results in the context of the patient's examination are valuable in confirming diagnosis, ruling out pathologic conditions, evaluating the degree of disease progression, helping in selection of optimal treatment, and adjusting treatment based on patient response. It also provides invaluable information on patients who may carry the burden of cerebrovascular disease but are clinically asymptomatic. For these patients, findings from imaging will drive a return to the clinical examination and, certainly, affect treatment decision-making. The wide availability of imaging in the current era allows for the possibility of serial evaluations of patients

Disclosures: DSL is a consultant to Stryker, Inc and Covidien, Inc.
[a] Department of Neurology, David Geffen School of Medicine, UCLA Stroke Center, University of California, RNRC, RM 4-126, Los Angeles, CA 90095, USA; [b] Department of Radiology, Division of Interventional Neuroradiology, University of California, Los Angeles, 757 Westwood plaza Suite 2129, Los Angeles, CA 90095, USA
* Corresponding author. UCLA Stroke Center, 710 Westwood Plaza, Los Angeles, CA 90095.
E-mail address: davidliebeskind@yahoo.com

Neurol Clin 32 (2014) 193–209
http://dx.doi.org/10.1016/j.ncl.2013.07.005 **neurologic.theclinics.com**
0733-8619/14/$ – see front matter

throughout their disease course. This is of particular value in the monitoring of cerebral ischemic disorders, which inherently follow a dynamic course. Irrespective of clinical practice settings, the evolution and refinement of imaging techniques now permit treatment decisions to be made in real time.

REASON FOR CONSULTATION

One may propose that the reason for consultation is perhaps one of the most important guiding aspects in obtaining supportive imaging. Knowing how the specific clinical question can be asked and appropriately answered by a specific imaging modality is fundamental to selecting the appropriate imaging test. Whether the question is related to evaluation of the ischemic core, penumbral tissue, or areas at-risk, a specific vascular lesion or pathologic condition that may culminate in cerebral ischemia, understanding the advantages and limitations of neuroimaging techniques increases the yield of data gathered. In addition, much like a subspecialist consultation, obtaining ancillary imaging in an outpatient clinical setting requires providing a framework for the subspecialty-imaging expert who is analyzing and coordinating the studies. Providing a reason for consultation—whether it is assessing intracranial arterial stenosis in a specific vascular distribution, evaluating a pattern of cerebral ischemia to better understand disease mechanism, or shedding light on ischemic disease progression to modify disease management is essential in providing the evaluating imaging expert with a focus for the study interpretation. This promotes focus on a specific region of interest in the interpretation and it will lead to potential adjustment of the imaging protocol, if needed, to address best the question of interest. Improved patient care, more cost-effective measures, and reduced need for unnecessary repeat imaging will ultimately result. These will augment the clinical examination findings and aid in patient management.

PATHOPHYSIOLOGY OF CEREBRAL ISCHEMIA

The pathophysiology of cerebral ischemia extends beyond the direct effects of anatomic changes in the arterial system leading to brain tissue. Unlike focal ischemia of ischemic stroke, cerebral hypoperfusion or cardiac arrest may lead to global ischemic injury unrestricted to a specific vascular territory. The pathophysiology of ischemia is similar at the tissue and cellular level, involving metabolic dysfunction and cell death due to hypoxia. The regulation of tissue perfusion in the brain is modulated differently from any other organ in the body given that nearly half of cerebrovascular resistance relies on the large arteries at the circle of Willis, in addition to intracranial and extracranial vasculature.[1,2] These arteries and their end arterioles play a primary role in oxygen delivery to the brain parenchyma through their regulation of cerebral blood flow (CBF). Many studies in animals and humans have investigated the threshold below which a reduction in CBF manifests neurologic symptoms and those that correlate to pathologically irreversible neuronal damage.[3–5] Across studies, depending on study design, neurologic symptoms and ischemia have been reported to range in values from below nearly 20 mL/100 mL/min to between 8 and 12 mL/100 mL/min in which tissue oxygenation was no longer sufficient to support the cellular machinery.[5–8] Although conventionally cerebral ischemia was thought to result as a direct consequence of a reduction in CBF, Ostergaard and colleagues[9] recently discussed the concept of capillary transit time heterogeneity and its contribution to the brain's efficacy in extracting oxygen at a given CBF. Regional CBF changes can be demonstrated using CT or MRI (**Fig. 1**). Several fatal outcomes result from the final aftermath of tissue oxygen deprivation that, on a cellular level, includes cell body