CT Techniques for Detecting Acute Stroke and Collateral Circulation: In Search of the Holy Grail

The management of acute “brain attack” is in evolution. A proliferation of new treatment approaches directed at lysing thrombus, normalizing cerebral perfusion, and reversing or minimizing neuronal ischemic damage are recently approved or currently under investigation. Preservation of “tissue at risk” within the ischemic penumbra is the goal of many of these newer therapies. The ischemic penumbra is a volume of metabolically unstable brain at the margins of the ischemic core that undergoes a reduction of cerebral blood flow in the range of 20% to 40% of normal (1–3). Experimental and clinical studies have shown that a narrow window of opportunity, probably no longer than 3 to 4 hours after the onset of acute ischemic symptoms, exists before complete and irreversible ischemic infarction extends throughout the ischemic penumbra (1). The promotional slogans “brain attack” and “time is brain” are a response to the new interest in timely interventions directed at salvaging as much of the penumbra as possible.

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The volume of this potentially salvageable tissue at risk in a given patient is dependent on many factors, including the length and severity of cerebral ischemia, the degree of collateral flow, and the metabolic status of the patient. Identifying and characterizing the volume of potentially reversible ischemic tissue is the holy grail of stroke imaging. An imaging technique capable of verifying the presence and volume of the ischemic penumbra at the time of acute infarction may provide important information that could influence the choice of treatment options and offer a surrogate method of comparing the efficacy of various treatment modalities.

In search of this holy grail, Hunter et al in this issue of the AJNR propose a bolus contrast technique using helical CT to detect and measure a region of “perfused blood volume” in patients with cerebral ischemia. These data are generated during the routine evaluation of a patient with a suspected acute stroke. A precontrast CT study provides information regarding acute hemorrhage, and is followed by a helical CT scan during the bolus infusion of contrast media. The bolus of contrast opacifies vascular structures throughout the brain, permitting the detection of perfusion deficits. The contrast-enhanced images can also be postprocessed to provide CT angiograms of the circle of Willis that could be useful in determining the site of arterial occlusion and the choice of appropriate therapies. The authors also show that a quantitative map of perfused cerebral blood volume can be obtained using the same data sets. This represents a map of the “delivery of contrast to those vessels still receiving blood, despite ischemia or infarction,” and thus may offer the clinician insight into the total volume of infarcted tissue plus penumbra. Thus it is not a map of salvageable tissue, but rather a map of perfused brain. The images appears to offer additional important information over routine CT scanning, which may occasionally appear normal within the first several hours after the onset of cerebral infarction.

While this apparently easy technique appears to improve the sensitivity of CT to early cerebral ischemia in some cases, in reality this diagnostic impact is probably rather minimal. The majority (about 75%) of noncontrast CT findings are abnormal within the first 6 hours after the onset of cerebral infarction (personal observation from the Abbott trials of prourokinase in acute middle cerebral artery occlusion). Indeed, in each figure in Hunter et al’s article one can detect the cerebral infarction on the precontrast images alone. However, the presence and volume of salvageable brain and the site of arterial occlusion are important pieces of information not available from noncontrast CT studies. Of acute cerebral stroke patients who are candidates for middle cerebral artery thrombolysis, early determination of the site of arterial occlusion with CT angiography could obviate the need for cerebral angiography in those with internal carotid occlusion who might not be candidates for thrombolysis. It would also be invaluable to quantify the potential viability of ischemic tissue before embarking on acute angiography, deployment of middle cerebral artery microcatheters, and expensive and perhaps dangerous antithrombolytic therapy.

Unfortunately, the technique described by Hunter et al does not yet uncover the holy grail of tissue at risk. While the technique apparently provides a measure of perfused cerebral blood volume, the thresholds levels that distinguish salvageable tissue from tissue dead or destined to die have yet to be defined. Nevertheless, this technique holds the promise of uncovering this information.

As of now, the best and most quantifiable technique for determining tissue at risk is xenon CT, which actually measures cerebral blood flow in regions of interest throughout the brain. This technique is unfortunately difficult, but not impossible, to implement in the patient with acute stroke. Diffusion/perfusion echo-planar MR techniques hold promise for quantification of the volume of ischemic penumbra surrounding an infarcted region of interest, but require easy access to magnets on an immediate basis throughout the day and night. The appeal of Hunter et al’s technique is the ease and availability of CT and the potential to quantify threshold levels of tissue perfusion. However, if the definition of tissue at risk cannot be precisely defined using this technique, then
it will not represent as valuable a tool as suggested by the authors. We look forward to the investigation of these threshold levels, and wish the authors luck in their search of the holy grail of cerebral ischemia.

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References

Can MR Help Predict Enlargement of Posttraumatic Spinal Cord Cysts?

Progressive enlargement of posttraumatic syrinx cavities occurs in a small percentage of patients who have suffered a severe spinal cord injury in the past. The exact mechanisms that cause this enlargement and why such enlargement is found in some patients but not in others remains uncertain. However, there are observations that could help explain the insidious progression of this posttraumatic complication, including the presence of scarring and adhesions within the subarachnoid space, alterations in cerebrospinal fluid flow dynamics, and fluid turbulence within the syrinx cavity itself. The article by Jinkins et al in this issue of the AJNR presents us with an MR finding not previously emphasized in patients with clinically pro-

gressive posttraumatic syringomyelia. A significant extent of increased signal on T2-weighted images cephalic to the syringomyelia is suggested by the authors to be present in those patients who are worsening clinically and whose cysts are enlarging. That observation raises a number of questions worthy of further investigation.

The fact that this extensive abnormal signal disappeared after a cyst shunting procedure suggests that this is a situation analogous to the periventricular edema frequently observed in obstructive hydrocephalus. Clearly, if these signal changes were caused by neural tissue destruction alone, signal reversibility would not be seen after cyst peritoneal shunt placement. A possibility is that this pericystic edema might, in combination with other dynamic changes, hasten or potentiate cyst enlargement and therefore account for subsequent clinical deterioration. This interesting observation is limited, however, by the small number of patients who formed the basis of the report; only six patients were studied, three of whom were clinically unstable, and three of whom were clinically stable and had short-segment cysts with minimal or no abnormal signal adjacent to the syrinx.

A larger number of both clinically progressive and stable patients is required to determine the reproducibility of these findings and to determine whether the signal changes will serve as a useful sign for predicting cyst expansion. The extension over time of the hyperintense parenchymal signal in conjunction with enlargement of the syrinx would lend further weight to the postulate that the abnormal signal represents spinal cord edema. Were that the situation, one could argue for surgical treatment even in the absence of obvious progressive symptoms, or at a minimum one could recommend more frequent MR monitoring of the affected spinal cord. In any event, the wisdom of obtaining T2-weighted images when a well-documented posttraumatic syringomyelia is identified on the T1-weighted images is suggested by this article. Evaluation of all these MR features should deepen our understanding of the pathophysiology of progressive posttraumatic cyst enlargement.

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