

Are your MRI contrast agents cost-effective?

Learn more about generic Gadolinium-Based Contrast Agents.



**FRESENIUS
KABI**

caring for life

AJNR

Sensitivity and Prognostic Value of Early CT in Occlusion of the Middle Cerebral Artery Trunk

Thomas A. Tomsick

AJNR Am J Neuroradiol 1994, 15 (1) 16-18

<http://www.ajnr.org/content/15/1/16.citation>

This information is current as
of April 10, 2024.

Sensitivity and Prognostic Value of Early CT in Occlusion of the Middle Cerebral Artery Trunk

Thomas A. Tomsick, *Director of Neuroradiology, University of Cincinnati Hospital*

Despite recent documentation of the utility of magnetic resonance (MR) in patients with acute onset of neurologic deficit caused by acute ischemia (1), emergency computed tomography (CT) remains the diagnostic procedure of choice in that setting when thrombolysis is being considered (2). To date CT has been primarily a test of exclusion: to exclude hemorrhage that would eliminate anticoagulant therapy or require surgery, and to exclude a disease process mimicking ischemia (eg, subdural hematoma or tumor).

Early hypodensity on CT has previously been suggested to predict subsequent hemorrhage (3). The article by von Kummer et al (4) in this issue identifies early hypodensity greater than 50% of the middle cerebral artery distribution and swelling as predictive findings on early CT in the setting of acute ischemia. Local swelling and hypodensity, once identified, both have predicted poor outcome despite treatment with thrombolytic agents in all patients, or glycerol and/or decompressive craniectomy in selected patients. Presuming no statistical difference in outcome between intravenously and intraarterially treated patients, two conclusions can be drawn from the study: 1) early CT signs do exist that predict poor outcome; and 2) the experimental treatment regimen did not open occluded vessels rapidly enough or completely enough to overcome the deleterious effects of ischemia on neurologic function.

The reader may rightly object to conclusion 1, pointing out that considerable interobserver variability might exist in determining the presence or absence of local swelling and early hypodensity greater than 50% of middle cerebral artery distribution. Perhaps the findings should have been subjected to Kappa analysis before publication of

the manuscript; notwithstanding, the authors rejoiner that this testing is forthcoming.

A holo-middle cerebral artery distribution infarct measures approximately 300 cc and will not be difficult to estimate. But in a previous study in which 15 patients had documented middle cerebral artery occlusion, patients (26%) had infarct volumes measured at the cathode-ray terminal console of approximately 50% middle cerebral artery distribution volume (5). How would they be categorized on the basis of eyeball estimates? Therefore, the adequacy of guesstimation of 50% middle cerebral artery distribution involvement is suspect.

The hyperdense middle cerebral artery sign has correlated with a large neurologic deficit in 80% to 90% of individuals but has not attained statistical significance as a predictor of outcome in several studies (6, 7). Furthermore, an equal number of patients may have neurologic deficits similar to patients with hyperdense middle cerebral artery signs, but not exhibit the sign, despite arteriographically proved middle cerebral artery occlusion (4, 8). However, it is of interest that the hyperdense middle cerebral artery sign still may be the most predictive ultra-early CT sign of a bad or fatal outcome in von Kummer et al's study (CT performed in 120 minutes or less).

The prevalence of the sign within 2 hours, multiplied by the number of patients with poor outcome or death with the sign, divided by the total number with the sign, shows the hyperdense middle cerebral artery sign may be more predictive than large hypodensity or swelling in the first 2 hours (35% versus 28% and 24%, respectively). When enough time for swelling or hypodensity to develop is allowed, those signs become more predictive.

Address reprint requests to Thomas A. Tomsick, MD, Department of Neuroradiology, University of Cincinnati Hospital, 234 Goodman St, Cincinnati, OH 45267-0742.

Index terms: Arteries, cerebral, middle (MCA); Arteries, stenosis and occlusion; Brain, computed tomography; Brain, infarction; Brain, ischemia; Commentaries

The author's definition of the sign may falsely increase its prevalence in this study: that is, a "unilateral tubular structure denser than brain in the basal sylvian fissure." A loose definition decreases the utility of the sign in predicting middle cerebral artery occlusion (9, 10). The hyperdense middle cerebral artery sign should indicate a middle cerebral artery denser than its counterpart and denser than any visualized vessel of similar size, not attributable to calcification. The normal vascular pool of the middle cerebral artery can appear denser than brain and, therefore, can be visualized (11).

What insights regarding therapy can be gleaned from this study? von Kummer reports generally poor outcomes in many patients despite therapy. No intravenously administered lytic drug has been proved to save lives or improve outcome. A previous study of intravenous tissue plasminogen activator has shown it to be ineffective at lysing proximal thrombi yet capable of opening smaller more distal middle cerebral artery branches (8). An ongoing double-blind NIH-sponsored study of intravenous tissue plasminogen activator has randomized over 400 patients to date, with 200 still to come (T. Brott, personal communication). A registry of intraarterial thrombolytic treatment has been created for the use of urokinase delivered into middle cerebral artery thrombus in hopes of rapid enough clot lysis to allow useful survival and reduce morbidity. Although not randomized, this registry may allow insights into alteration of the natural history of the disease process (13–15).

Patients with middle cerebral artery occlusion, in general, have relatively high stroke scale scores. In a previous study of patients treated as rapidly as possible with intravenous tissue plasminogen activator without pretreatment arteriography, 17 patients had the hyperdense middle cerebral artery sign and presumed middle cerebral artery occlusion (5). All 17 had NIH stroke scale scores (a narrower stroke scale score range than von Kummer et al's, in which 0 is normal, 42 maximal deficit) of 10 or greater (15). (A patient with a stroke scale score of 10 might exhibit drowsiness, minor facial paresis, no resistance to gravity of the arm and leg, partial neglect and sensory loss, and mild to moderate dysarthria and dysphasia). Of 20 patients with similar stroke scale scores greater than 10, but not exhibiting the hyperdense middle cerebral artery sign, 14 posttreatment arteriograms demonstrated nine internal carotid artery occlusions

and/or occlusions of the M-1 or M-2 segments of the middle cerebral artery. These 37 patients had six deaths and mean stroke scale score deficits at 3 months of approximately 50% of their initial stroke scale scores, despite treatment with intravenous tissue plasminogen activator. This reflects the clinical shortcomings of intravenous treatment for major vascular occlusion. This may reflect the arteriographic results after intravenous treatment reported by Wolpert et al, in which one-third of M-1 or M-2 segment occlusions partially recanalized with intravenous tissue plasminogen activator (8).

By the same token, in patients with stroke scale scores less than 10 ($n = 17$; range 3–9; mean = 6.0), none died and the residual neurologic deficit at 3 months was 25% of the initial, with 50% normal neurologically. Arteriography in that group showed that none had internal carotid artery or M-1 or M-2 occlusions within days after treatment ($P < .001$). It seems, then, that patients with relatively low stroke scale scores may have more distal occlusive changes and recover fairly well with intravenous tissue plasminogen activator. The ongoing randomized study should give us some insight as to whether they do better than with no treatment. However, the observation stands that a mean higher stroke scale scores seems to be associated with internal carotid artery or M-1 and M-2 occlusive changes and a greater percent residual neurologic deficit at three months. Patients with lower stroke scale scores likely may have less major occlusive disease and will do better at 3 months.

Arteries recanalize normally, and patients do improve spontaneously (16–18). A neurologic deficit at presentation may determine how aggressively we diagnose and treat occlusive disease. Perhaps we will define a group in which benefits from intravenous therapy may be sufficient to allow a reasonable recovery without undue risk. Similarly, patients with larger deficits may have a higher likelihood of major occlusive changes and be less likely to respond to intravenous lytic therapy and may need more than intravenous thrombolysis. Unfortunately von Kummer et al do not separate intravenously ($n = 46$) and intraarterially ($n = 7$) treated groups, so we have no insight as to whether they fared equally poorly when the hypodensity or mass effect were already present. Perhaps intravenous therapy will be begun after CT to initiate treatment as rapidly as possible, followed by arteriography. If middle cerebral artery occlusion is

shown, additional intraarterial thrombolysis can be instituted. More aggressive thrombus manipulation may ultimately be indicated, as our colleagues in cardiology are discovering (19). Under these circumstances, the neuroradiology team will have to be involved at a very early clinical stage to begin the evaluation and treatment as rapidly as possible. Neuroradiologists on call will have to be familiar with microcatheter techniques to achieve intraarterial thrombolytic recanalization or clot removal. This will create a major test for the neuroradiology team and will challenge us to respond adequately to "brain attacks" in the Decade of the Brain and beyond. Will it improve outcomes (20)? Time will tell!

References

1. Shimosegawa E, Inugami A, Okudera T, et al. Embolic cerebral infarction: MR findings in the first three hours after onset. *AJR Am J Roentgenol* 1993;160:1077-1082
2. Brott TG, Haley EC, Levy DE, et al. Urgent therapy for stroke. Part I. Pilot study of tissue plasminogen activator administered within 90 minutes. *Stroke* 1992;23:632-640
3. Bozzao L, Angeloni V, Bastianello S, et al. Early angiographic and CT findings in patients with hemorrhagic infarction in the distribution of the middle cerebral artery. *AJNR Am J Neuroradiol* 1992;12:1115-1121
4. von Kummer R, Meyding-Lamadé U, Forsting M, et al. Sensitivity and prognostic value of early CT in occlusion of the middle cerebral artery trunk. *AJNR Am J Neuroradiol* 1994;15:9-15
5. Tomsick T, Brott T, Barsan W, Broderick J, Haley EC, Spilker J. Thrombus localization with emergency cerebral CT. *AJNR Am J Neuroradiol* 1992;13:257-263
6. Leys D, Pruvok JP, Godefroy O, et al. Prevalence and significance of hyperdense middle cerebral artery in acute stroke. *Stroke* 1992;23:317-324
7. Ricci S, Caputon, Aija G, Celan MG, et al. Prognostic value of the dense middle cerebral artery sign in patients with acute ischemic stroke. *Ital J Neurol Sci* 1991;12:45-47
8. Wolpert SM, Bruckman H, Greenlee R, et al. Neuroradiologic evaluation of patient with acute stroke treated with recombinant tissue plasminogen activator. *AJNR Am J Neuroradiol* 1993;14:3-13
9. Rauch RA, Buzan G, Larrson EM, Jinkins JR. Hyperdense middle cerebral arteries identified on CT as a false sign of vascular occlusion. *AJNR Am J Neuroradiol* 1993;14:669-673
10. Tomsick T, Brott T, Chambers AA, et al. Hyperdense middle cerebral artery sign on CT: efficacy in detecting middle cerebral artery thrombosis. *AJNR Am J Neuroradiol* 1990;11:473-477
11. New P, Scott W, eds. *CT of the brain and orbit*. Baltimore: Williams & Wilkins 1975:263-267
12. Jones-Bey H. Logistical hurdles loom for stroke intervention. *Diagn Imaging* 1993;45-51
13. Barnwell S. Direct intra-arterial thrombolytic therapy for acute stroke. Proceedings of the 31st annual meeting of the American Society of Neuroradiology, Vancouver, 1993:38
14. Zeumer H, Freitag HJ, Zanella F, Thie A, Arning C. Local intra-arterial fibrinolytic therapy in patients with stroke: urokinase vs recombinant tissue plasminogen activator (rTPA) *Neuroradiology* 1993;35:159-162
15. Brott T, Adams HP, Olinger CP, et al. Measurements of acute cerebral infarction: a clinical examination scale. *Stroke* 1989;20:864-870
16. Taneda M, Shimeda N, Tsuchiya T. Transient neurological deficits due to the embolic occlusion and reopening of the cerebral arteries. *Stroke* 1985;16:522-524
17. Irino A, Mamoru T, Minami T. Angiographic manifestations in postrecanalized cerebral infarction. *Neurology* 1977;27:471-475
18. Biller J, Love BB, Marsh EE, et al. Spontaneous improvement after acute ischemic stroke. *Stroke* 1990;21:1008-1012
19. Lange RA, Hillis LD. Immediate angioplasty for acute myocardial infarction. *N Engl J Med* 1993;328:726-728
20. Wardlaw JM. Thrombolysis in acute ischemic stroke: does it work? *Stroke* 1992;23:1826-1839