AJNR

Transcranial Doppler Sonography and CT Angiography in Patients with Atherothrombotic Middle Cerebral Artery Stroke

Nijasri C. Suwanwela, Kammant Phanthumchinda and Nitaya Suwanwela

AJNR Am J Neuroradiol 2002, 23 (8) 1352-1355
http://www.ajnr.org/content/23/8/1352

AJNR Am J Neuroradiol 23:1352–1355, September 2002

Transcranial Doppler Sonography and CT Angiography in Patients with Atherothrombotic Middle Cerebral Artery Stroke

Nijasri C. Suwanwela, Kammant Phanthumchinda, and Nitaya Suwanwela

BACKGROUND AND PURPOSE: **Atherothrombotic disease of the middle cerebral artery (MCA) frequently occurs in Asian populations. This abnormality can be noninvasively assessed with transcranial Doppler sonography (TCD) and computed tomographic angiography (CTA). To our knowledge, the usefulness of TCD sonography compared with CTA in the diagnosis of nonembolic MCA disease has not been studied.**

METHODS: **We prospectively examined 70 patients with clinically suspected atherothrombotic MCA stroke by using TCD sonography and CTA. We excluded patients with a known source of cardiac emboli, significant carotid stenosis, or classic lacunar syndrome. TCD sonography was performed within 2 days of admission, followed by CTA within 7 days after stroke onset.**

RESULTS: **CTA demonstrated MCA stenosis of more than 50% in 57 patients (81%), whereas only 29 patients (41%) had abnormal TCD findings. CTA showed proximal M1 stenosis, distal M1 stenosis, and M2 disease in 29%, 29%, and 24% of the patients, respectively. Stenotic sites differed between patients with normal TCD results and those with abnormal results. TCD findings correlated well with CTA findings in all patients with proximal M1 stenosis. In contrast, TCD sonography correctly depicted distal M1 or M2 disease in only 24% of the patients.**

CONCLUSION: **In this population, CTA is superior to TCD sonography in the diagnosis of MCA disease. Abnormal TCD results are highly suggestive of MCA stenosis. However, normal TCD findings do not exclude such lesions, especially in patients with distal M1 or M2 disease. Because distal M1 and M2 disease was found in half of our patients, TCD sonography should not be used as a method to screen for MCA stenosis.**

Ischemic stroke in the middle cerebral artery (MCA) is commonly caused by embolism. A small percentage of patients have in situ atherosclerotic disease of the MCA. According to Fisher (1), atherosclerotic thrombosis is found in only 7% of patients with MCA occlusion, as assessed with clinical, angiographic, or pathologic criteria. Intracranial stenotic lesions are believed to be much more common in Asians and African Americans than in other populations. In contrast, cerebral embolism usually has a sudden onset that causes a maximal deficit. The clinical findings in patients with MCA atherosclerotic disease include progressive and fluctuating stroke.

Transcranial Doppler (TCD) sonography is a useful technique for measuring blood flow velocities in the intracranial arteries. It is widely used for the early detection of vasospasm after subarachnoid hemorrhage. It also aids in the evaluation of intracranial hemodynamics and the collateral circulation in patients with extracranial carotid stenosis (2). Furthermore, TCD sonography may depict occlusion and stenosis of the basal cerebral arteries around the circle of Willis (3–5). Although TCD sonography is widely available and highly affordable, its use is limited to patients with sufficient acoustic windows. Computed tomographic angiography (CTA) is another alternative noninvasive method that permits visualization the anatomy of the intracranial arteries (6–8). To evaluate the diagnostic accuracy of TCD sonography in the assessment of intracranial vascular stenotic lesions, we compared TCD findings with CTA findings in patients with stroke and a presumptive diagnosis of MCA atherosclerosis.

Methods

We examined consecutive patients who presented to the neurology service between August 1999 and June 2000 with the

Received December 18, 2001; accepted after revision April 15, 2002.

From the Neurological Unit, Department of Medicine (N.C.S., K.P.), and the Department of Radiology, Faculty of Medicine (N.S.), Chulalongkorn University, Bangkok, Thailand.

Address reprint requests to Nijasri C. Suwanwela, MD, Neurological Unit, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok 1033 0, Thailand.

clinical syndrome of stroke or transient ischemic attack (TIA) in the MCA territory (9). Clinically, these included patients who had hemiparesis in which the arm had more severe weakness than the leg and patients with evidence of weakness plus cortical dysfunction in the MCA territory. Patients with infarction in the MCA territory involving the cerebral cortex and giant lacunar infarction (1.5 cm.) in the deep MCA territory on CT scans were also included. Patients who had a known source of emboli (eg, atrial fibrillation, valvular heart disease, recent myocardial infarction, marked carotid stenosis) and those with clinical and CT findings compatible with classic lacunar syndrome were excluded. In addition, patients with renal insufficiency (serum creatinine level 1.5 mg/dL) in whom contrast medium injection for CTA was therefore contraindicated were not enrolled. In every case, TCD sonography was performed within 2 days of admission, followed by CTA within 7 days after the onset of stroke. During the study period, 11 patients in whom TCD examination was limited because of insufficient temporal acoustic windows were excluded.

TCD Examination

TCD was performed transtemporally by using the standard procedure (10) with an EME TranScan (Nicolet Vascular Inc., Palm Springs, CA) device and a 2-MHz transducer to record the MCA flow directions and velocities. Patients with insufficient temporal windows for TCD were excluded. Stenosis was diagnosed if the mean velocity was more than 80 cm/s or if the peak systolic velocity exceeded 140 cm/s. To ascertain focal stenosis, the high flow velocity in the MCA must be circumscribed within a 4–10 mm insonation depth.

CTA Examination

Spiral CTA was performed with a Somatom Plus 4 (Siemens Medical Systems, Erlangen, Germany) helical scanner. A preliminary test bolus of 10 mL nonionic contrast medium (Ultravist 300 [iopromide]; Schering, Berlin, Germany) was administered by means of a power injector at a rate of 3 mL/s via the antecubital vein. Scanning was initiated after an 8-second delay to generate a time-attenuation curve. The spiral CTA protocol was set according to the peak enhancement of the test bolus. A total volume of 90 mL of contrast medium injected at a rate of 3–3.5 mL/s by using the power injector. Scanning was performed caudocranially, starting at the floor of the sella turcica for a length of 40–50 mm. A 1-mm collimation and a pitch of 1.5 were used. The total scanning time was 29–35 seconds, and the increment of reconstruction was 0.5 mm. After scanning, 100 serial images were transferred to a Prominence workstation (Siemens) to produce maximum intensity projections and surface-shaded display images. Significant MCA stenosis was diagnosed when the diameter of the residual lumen was less than 50%.

Results

During the study period, 70 patients with a history of stroke or TIA in the MCA territory fulfilled the selection criteria. The patients included 37 men and 33 women aged 45–87 years with a mean age of 61 years. Their clinical histories included 69 strokes and one TIA. In 47 patients (67%), the onset of stroke was either acute with progression of the deficit over 2–4 days or fluctuating in the same MCA territory, which suggested an atherothrombotic nature. Twelve patients (17%) woke up after the episode with a deficit, and the remaining 11 had a sudden onset of the illness. Ischemic symptoms in the left cerebral hemisphere were found in 28 patients. Forty-two patients had ischemic symptoms attributed to the right hemisphere.

TCD sonography was able to demonstrate the MCAs at depths of 44–64 mm in all patients examined. Focal, high flow velocities that suggested MCA stenosis were found in 29 patients (41%).

Abnormal CTA findings indicated an MCA stenosis of more than 50% in 57 patients (81%). All had a corresponding stroke, which was identified on the basis of the clinical data or an infarction on a CT scan. In four patients, CTA demonstrated bilateral MCA stenosis; in these patients, only the stenotic side, which matched the clinical findings, was analyzed. Twenty patients (29%) had stenosis in the proximal part of the M1 segment. In this group, two patients had stenosis involving the entire M1 segment (proximal and distal). Another 20 patients (29%) had distal M1 stenosis just proximal to the MCA bifurcation (trifurcation). Fifteen patients had an MCA branch (M2) stenosis, and two patients had M2 occlusion. Thirteen patients had normal CTA findings.

Twenty-nine patients with abnormal TCD findings were found to have a corresponding lesion at CTA. The stenoses were detected at the proximal M1 segment in 20 patients and in the distal M1 segment in nine. Among those patients with normal TCD findings, 17 had stenosis or occlusion of the M2 segment and 11 had distal M1 stenosis. None of these patients had proximal M1 stenosis.

Discussion

Most MCA strokes are believed to be embolic in nature. Atherothrombotic disease of the MCA is rather uncommon, especially among Caucasians (11). However, many findings have suggested that intracranial stenosis, especially that of the MCA, is more prevalent in African American, Hispanic, and Asian persons (11–12). Our study excluded patients with a known source of cerebral embolism and lacunar infarction to focus on in situ atherothrombotic disease of the MCA. Moreover, 77% of our patients had progressive or fluctuating symptoms and another 17% were found to have deficit upon awakening; this observation suggests the presence of thrombus (13).

Various noninvasive diagnostic tools have been used to identify intracranial vascular lesions. TCD sonography is one of the most commonly used examinations because of its simplicity and affordability. It also provides additional information about the hemodynamic status of the intracranial circulation. Nonetheless, TCD sonography has some limitations. For example, during our study we found that 11 patients had insufficient temporal acoustic windows that resulted in a limited insonation signature. Moreover, stenosis of the distal part of the MCA (M1) or the M2 segment may not be detected at TCD sonography because of technical difficulties.

CTA is a relatively noninvasive method that enables visualization of the intracranial vascular system. With its superior digital and three-dimensional images, it has become widely used to evaluate the intra-

cranial circulation (14). A recent study has shown that high-resolution CTA is almost as good as digital subtraction angiography for the detection of intracranial steno-occlusive disease, with a 100% sensitivity and 93.4% predictive value (15). Unlike MR angiography (MRA), which measures the physiology of blood flow in the arteries, CTA depicts the anatomy of blood vessels. Hence, CTA is superior to MRA in the evaluation of vascular stenosis, especially stenosis at the bifurcation where turbulent flow is usually present. Moreover, CTA can be performed simultaneously with routine CT of the brain in patients with acute stroke. Thus, CTA can immediately provide information about the vascular disease underlying the stroke (14). In this study, we used a 50% reduction in vascular diameter as the criterion for significant stenosis. Because CTA depicts the inner surface anatomy of the blood vessels, the measurement of the diameter reduction with CTA should be an accurate method for determining the percentage of stenosis. According to the findings of previous flow studies, a diameter reduction of more than 50% can cause a hemodynamic change. Therefore, we believe that a reduction of more than 50% in the major intracranial arteries might be responsible for low-flow stroke in the same vascular territory. In addition, significant stenosis may also lead to thromboembolic events.

In this prospective study, TCD sonography was performed before CTA to exclude the possibility of a thrombus dislodging before the examination and resulting in normal velocities at TCD sonography. All TCD and CTA examinations were performed during the acute phase of stroke (within 7 days). At transtemporal TCD imaging, abnormal findings in the MCA at a depth of 44–64 mm that suggested MCA stenosis were present in only 29 patients (41%). In contrast, CTA demonstrated a stenotic lesion in the MCA in 81% of the patients. The CTA findings can be categorized into three groups: proximal M1 stenosis, distal M1 stenosis, and M2 stenosis.

We previously reported the results of a pilot study in which we compared TCD sonography and CTA in 10 patients (16). At that time, we did not use a 50% criterion for significant stenosis. However, we found that CTA was notably superior to TCD imaging with respect to the detection of focal MCA stenosis, especially in the distal M1 and M2 segments. A report of CTA compared with TCD sonography in 19 patients with acute basilar artery ischemia was recently published (17). CTA was found to be superior to TCD in the assessment of basilar patency. Findings from another study in which MCA TCD and CTA results were correlated in 10 patients have been reported (8). In that study, TCD was used as a screening test for MCA stenosis, and CTA was performed only in patients with abnormal TCD results. The authors established a good correlation between the results with both methods. Our findings confirm that patients with abnormal TCD results always have corresponding lesions at CTA. Focusing on the stenotic site, we found a 100% correlation between abnormal TCD findings and CTA findings in patients with proximal M1 disease. On the other hand, 69% of those with abnormal TCD results had proximal M1 disease. Therefore, we conclude that abnormal TCD findings are highly suggestive of stenosis, particularly stenosis in the proximal M1 segment of the MCA.

Studies in Caucasians have shown that most MCA stenoses are located in the proximal M1 segment (12). In contrast, reports from Asia have demonstrated a higher prevalence of distal MCA stenosis. Distal M1 or M2 disease is found in 40–60% of Asian patients with MCA atherosclerosis (18–19). On the basis of our observations, CTA is definitely superior to TCD sonography in the diagnosis of distal MCA disease. TCD failed to depict distal M1 and M2 stenosis (as shown at CTA) in 28 (76%) of 37 patients. Because those with distal M1 or M2 stenosis accounted for approximately half of our patients, TCD sonography is not recommended as a screening test for intracranial atherothrombotic disease.

Conclusion

In our patients with atherothrombotic stroke in the MCA territory, CTA was superior to TCD sonography in the diagnosis of MCA stenosis. In 14% of the patients, no useful information was obtained from TCD sonography because of limited acoustic windows. Among the patients examined, CTA demonstrated an MCA stenosis of more than 50% in 57 patients (81%), whereas TCD sonography revealed stenosis in only 29 patients (41%). According to the site of stenosis, as depicted at CTA, TCD findings were abnormal in all patients with M1 segment stenosis, whereas TCD sonography depicted stenosis in only 24% of the patients with distal M1 or M2 disease. We conclude that abnormal TCD findings are highly suggestive of MCA stenosis. Normal TCD results, however, do not exclude such a lesion, especially in patients with distal M1 or M2 disease. Because distal M1 and M2 disease was found in approximately half of our patients, we do not recommend the use of TCD sonography as a screening test for MCA stenosis.

References

1. Fisher CM. **Cerebral ischemia**: **less familiar types.** *Clin Neurosurg* 1971;18:267
2. Can U, Furie KF, Suwanwela N, et al. **Transcranial Doppler ultrasound criteria for hemodynamically significant internal carotid stenosis based on residual lumen diameter calculated from en bloc endarterectomy specimens.** *Stroke* 1997;28:19661971
3. Ley-Pozo J, Ringelstein. **Noninvasive detection of occlusive disease of the carotid siphon and middle cerebral artery.** *Ann Neurol* 1990;28:640–647
4. Lindegaard KF, Bakke SJ, Aaslid R. **Doppler diagnosis of intracranial artery disorders.** *J Neurol Neurosurg Psychiatry* 1986;49: 510–518
5. Mattle H, Grolimund P, Huber P, Sturzenegger M, Zurbrugg HR. **Transcranial Doppler sonographic findings in middle cerebral artery disease.** *Arch Neurol* 1988;45:289–295
6. Katz DA, Marks MP, Napel SA, Bracci PM, Roberts SL. **Circle of Willis: evaluation with spiral CT angiography, MR angiography and conventional angiography.** *Radiology* 1995;195:445–449
7. Wong KS, Lam WW, Liang E, Huang NY, Chan YL, Kay R. **Variability of magnetic resonance angiography and computed tomography in grading middle cerebral artery stenosis.** *Stroke* 1996; 27:1084–1087

8. Wong KS, Liang EY, Lam WW, Huang YN, Kay R. **Spiral computed tomography angiography in the assessment of middle cerebral artery occlusive disease.** *J Neurol Neurosurg Psychiatry* 1995; 59:537–539
9. Caplan LR, Babikian V, Helgason C, Hier DB, Dewitt D, Patel D, Stein R. **Occlusive disease of the middle cerebral artery.** *Neurology* 1985;35:975–982
10. Aaslid R, Markwalder TM, Nornes H. **Noninvasive transcranial Doppler ultrasound recording of flow velocity in the basal cerebral arteries.** *J Neurosurg* 1982;57:769–774
11. Bogousslavsky J, Barnett HJM, Fox AJ, Hachinski VC, Taylor W. **Atherosclerotic disease of the middle cerebral artery.** *Stroke* 1986; 17:1112–1120
12. Feldman E, Daneault N, Kwan E, et al. **Chinese-white differences in the distribution of occlusive cerebrovascular disease.** *Neurology* 1990;40:1541–1545
13. Caplan LR. **Intracranial branch atheromatous disease. A neglected, understudied, and underused concept.** *Neurology* 1989;39: 1246–1250
14. Shrier DA, Tanaka H, Namaguchi Y, Konno S, Patel U, Shibata D. **CT angiography in the evaluation of acute ischemic stroke.** *AJNR Am J Neuroradiol* 1997;18:1011–1020
15. Skutta B, Furst G, Eilers J, Ferbert A, Kuhn FP. **Intracranial stenoocclusive disease: double detector helical CTA versus digital subtraction angiography.** *AJNR Am J Neuroradiol* 1999;20(5):791–799
16. Suwanwela NC, Suwanwela N, Phanthumchinda K. **Comparison of transcranial Doppler ultrasound and CT angiography in symptomatic middle cerebral artery stenosis.** *Australasian Radiol* 2000;44: 174–177
17. Brandt T, Knauth M, Wildermuth S, et al. **CT angiography and Doppler sonography for emergency assessment in acute basilar artery ischemia.** *Stroke* 1999;30:606–612
18. Ueda S, Fujitsu K, Inomori S, Kuwabara T. **Thrombotic occlusion of the middle cerebral artery.** *Stroke* 1992;23:1761–1766
19. Saito I, Segawa H, Shiokawa Y, Taniguchi M, Tsutsumi K. **Middle cerebral artery occlusion; correlation of computed tomography and angiography with clinical outcome.** *Stroke* 1987;18:863–868