Transcranial Doppler Ultrasound Findings in Middle Cerebral Artery Occlusion

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We evaluated the efficacy of transcranial Doppler ultrasonography in 23 patients suffering from acute middle cerebral artery occlusion. The diagnosis of occlusion was most suggestive when all basal arteries except the affected middle cerebral artery were detectable. Enhanced blood flow velocity in the anterior cerebral artery due to leptomeningeal collateralization was used as a corroborating criterion. With frequent follow-up examinations, we monitored reperfusion of the M1 segment resulting from recanalization or embolus migration in 16 patients. Those patients undergoing recanalization within days after onset of the first symptoms revealed variable clinical courses and lesion patterns on computed tomography, indicating the crucial importance of early and efficient leptomeningeal collateral blood supply. Transcranial Doppler ultrasonography was able to exclude middle cerebral artery occlusion with accuracy, which provides important clinical information. However, distal branch occlusions could not be detected with sufficient exactness. (Stroke 1990;21:532–537)

Transcranial Doppler ultrasonography (TCD) can provide the diagnosis of middle cerebral artery (MCA) main stem occlusion under specific circumstances. This opens new vistas for research regarding the frequency, course, and prognosis of MCA occlusion. The noninvasive TCD method permits examination of patients unfit for angiography during the acute phase of stroke and at short intervals. The aim of our study is to describe TCD criteria allowing the diagnosis of MCA occlusion and to present TCD follow-up findings observed during MCA recanalization.

Subjects and Methods

Twenty-three patients suffering from acute MCA occlusion (Table 1) received a complete clinical and computed tomographic (CT) work-up. Criteria for MCA occlusion were derived from 11 patients who underwent angiography (patients 1–11). Twenty patients (4–23) received follow-up studies starting ≤24 hours after clinical onset; 18 (patients 6–23) were consecutive during 6 months. The diagnosis of MCA occlusion was confirmed in the consecutive series by angiography (patients 6–11), necropsy (patients 12 and 13), or retrospectively, follow-up TCD examinations showing reperfusion of the MCA (patients 14–21). In these eight patients, CT was repeated to exclude the possibility of hemorrhage into the infarcted brain tissue. Two patients (22 and 23) had certain MCA occlusion, with a proven cardiac source of emboli, simultaneous arterial occlusions in multiple other organs, and clinical and CT findings compatible with embolic MCA occlusion. Five patients (4–6, 18, and 19) suffered additional carotid artery occlusion.

In the consecutive series, follow-up TCD studies were carried out daily during the first week, every 3 days during the second week, and weekly until discharge in cases with persistent abnormal blood flow velocities. Death prohibited follow-up in six patients.

The basal cerebral arteries were investigated using a standard pulsed 2-MHz Doppler device (TC 2-64 B, EME, Überlingen, FRG) in accordance with accepted criteria. Extracranial continuous-wave (CW) Doppler sonography was carried out in all 23 patients.

Results

Figure 1 shows criteria for the diagnosis of MCA occlusion. Conventional CW Doppler sonography of the extracranial cerebral arteries must precede every TCD study because extracranial carotid stenoses and occlusions cause considerable changes in intracranial hemodynamics.

Having ruled out extracranial occlusive disease, demonstration of the basal arteries of the asymptomatic side is advisable initially. As physiologic blood flow conditions are to be expected there, information can be obtained regarding ultrasound penetration of the temporal bone and depth of the contralateral MCA (assuming symmetry).
orthograde
confirmation of periocclusive MCA artery-to-artery for technical reasons. Under these circumstances, the lack of an MCA signal may be explained by hemodynamic reasons as well. Very low blood flow velocities (<10 cm/sec) are not detectable.

The next step is to identify the distal segment of the internal carotid artery (ICA), the anterior cerebral artery (ACA), and the posterior cerebral artery (depth settings 50–75 mm). This procedure is the most important prerequisite since it guarantees the existence of an appropriate acoustic bone window. Compression tests may contribute to artery differentiation.

Starting from the intracranial ICA or from the ACA, one must now search for the MCA by shortening the ultrasound focusing depth in a stepwise fashion (depth settings 35–40 mm) with roving changes of direction of the ultrasound beam. If this painstaking procedure fails to uncover an ultrasound signal related to the MCA, occlusion of the artery must be suspected. A characteristic finding corroborating the diagnosis of MCA occlusion is accelerated blood flow in the A1 segment of the ACA due to activation of leptomeningeal anastomoses.

The diagnosis of MCA occlusion secondary to a lodging embolus is complicated when there is additional obliteration of the extracranial ICA or of the siphon. Under these circumstances, increased orthograde blood flow in the ipsilateral ACA is not to be expected and the lack of an MCA signal may be explained by hemodynamic reasons as well. Very low blood flow velocities (<10 cm/sec) are not detectable for technical reasons. Under these circumstances, confirmation of periocclusive MCA artery-to-artery embolism is possible, retrospectively, if MCA recanalization and sufficient collateralization via basal communicating arteries can be demonstrated (Figure 2, patient 6).

Follow-up studies were conducted repeatedly during the acute period after occlusion; recanalization of the MCA could be monitored by TCD in 16 cases (Table 1). Recanalization occurred gradually; a high-frequency stenotic signal indicating a partially patent lumen was subtle at first, but later the TCD signs of MCA stenosis became more obvious. Further increases in the vascular lumen led to decreases in blood flow velocity in the M1 segment. Finally, turbulent signal irregularities disappeared (Figure 3). Recanalization was seen in this series of 20 patients until 17 days after stroke onset. Augmented blood flow velocity in the M1 segment disappeared between 11 days and 4 weeks after onset. Three patients (14, 15, and 17) died after recanalization of the MCA secondary to temporal herniation after developing extensive postschismic edema. In three patients (5, 8, and 10; Figure 4), blood flow velocity enhancement persisted for weeks. Unfortunately, it was not possible to repeat angiography to demonstrate a residual MCA stenosis in these patients.

In another patient with carotid occlusion (15), blood flow velocity enhancement was seen at the initial TCD examination 2 days after onset of the first
hemispheric symptoms. Periocclusive embolization of the proximal MCA and subsequent thrombus migration was diagnosed, taking into account the finding of extensive multiple MCA branch infarction on CT. 

Differentiating posts ischemic hyperemia from residual MCA stenosis during recanalization caused considerable problems. Hemorrhagic transformation causing clinical deterioration in relation to arterial recanalization was not observed (under conditions of hemodilution therapy and 300 mg/day aspirin).

Discussion

One prerequisite for the TCD diagnosis of MCA occlusion is a viable acoustic bone window, allowing examination of uninvolved basal cerebral arteries. Time, patience, and some experience are needed to be sure that an MCA signal is really lacking.

According to the literature, difficulties due to insufficient ultrasound penetration through the temporal bone arise in up to 30% of cases depending on age, sex, and race. In personal prospective TCD studies currently including 80 consecutive patients admitted ≤24 hours after acute stroke, we have 35% drop-outs due to insufficient ultrasound penetration. Furthermore, false-positive diagnosis of MCA occlusion may result from a tortuous course of the artery and is possible when the MCA branches vertically from the siphon.

The recorded Doppler shift is at first unable to differentiate residual MCA stenosis from increased blood flow velocity due to decreased peripheral arteriolar resistance (i.e., posts ischemic hyperperfusion syndrome). A combination of both phenomena during recanalization seems likely. On the basis of only one TCD examination shortly after stroke, one must take care not to misdiagnose reactive hyperemia as MCA stenosis because erroneous conclusions regarding pathogenesis would result. However, significant obstruction of the main stem of the MCA causes a "resistance profile" in the feeding ICA, whereas reactive hyperemia leads to increased blood flow velocity even proximal to the M1 segment. The latter is sometimes so pronounced that velocity enhancement after intracranial arterial recanalization can be detected by extracranial CW Doppler sonography.2 Thus, calculating the relation between blood flow velocity in the MCA and that in the ipsilateral ICA3 may help differentiate MCA stenosis from hyperemia after recanalization.

Enhancement of blood flow velocity was not detectable by TCD in all patients undergoing recanalization. Partial blockade of the peripheral vascular bed persisting after distal migration of clot fragments may be an explanation.

Patients with MCA occlusion persisting at least 1 day revealed highly variable clinical courses and lesion patterns on CT. This was independent of later spontaneous recanalization, indicating the crucial importance of early collateral blood supply by leptomeningeal anastomoses.4 Five patients featured typical “deep” subcortical CT infarcts, which are considered to result from proximal MCA trunk occlusion and blockade of lenticulostriate end arteries.

Shortly after ischemic stroke, increased or diminished blood flow velocity compared with the opposite side can frequently be observed in the affected MCA. As the normal ranges of blood flow velocity in the cerebral arteries are wide (mean±SD in the MCA 62±12 cm/sec)1 and as side differences of up to 20% are not necessarily pathologic, clear-cut limits for physiologic and pathologic blood flow velocities are difficult to establish. Follow-up studies are especially helpful in this respect since reactive hyperemia decreases over time, enabling the correct diagnosis by TCD retrospectively. We classified a >50% increase in blood flow velocity as abnormal.

Exclusion of an MCA occlusion can also be of major importance, influencing further diagnostic and therapeutic strategies.5 This information is generally obtained by TCD with minimal delay and considerable accuracy.

The small number of cases in our series does not allow us to precisely estimate the sensitivity of TCD in the diagnosis of MCA occlusion. Moreover, statements concerning the diagnostic reliability of TCD in patients with MCA occlusion are biased if the examinations are performed in awareness of the clinical and CT findings, which is to be supposed from a practical point of view. Distal MCA branch occlusions cannot be diagnosed by means of TCD since there is no reliable way to investigate these segments of the artery.

Angiography remains the gold standard for verifying MCA occlusions diagnosed by TCD. However, angiography is seldom indicated during the acute phase of stroke. TCD is even more expedient concerning follow-up studies of recanalization. Individual angiograms can deliver only one-time information, initially showing MCA occlusion, later possibly showing stenosis; after recanalization, angiography is of no diagnostic value. This may explain occasional

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**Figure 1.** Criteria for diagnosis of left middle cerebral artery (MCA) occlusion. All basal arteries except MCA are detectable by transcranial Doppler ultrasonography. Blood flow velocity is enhanced in ipsilateral A1 segment of anterior cerebral artery due to activated leptomeningeal anastomoses. Brief compression of common carotid artery (bar in lower left tracing) elicits increasing blood flow in P1 segment of ipsilateral posterior cerebral artery.

**Figure 2.** Recanalizing right (R) middle cerebral artery (MCA) occlusion with concomitant internal carotid artery (ICA) occlusion (patient 6). Diagnosis can be established retrospectively by follow-up transcranial Doppler ultrasonography. Top: Stenotic signal related to M1 segment on day 4, indicating reperfusion, disappearing thereafter. Due to ICA occlusion, dampened pulse curve with decreased pulsatility index (PI, 0.39) persists in spite of good collateralization. Bottom: Left (L) MCA as reference.
FIGURE 3. Patient 9, 34-year-old woman suffering from cardiac embolism. Top: Recanalization of internal carotid artery beginning 24 hours after clinical onset. Initially stenotic signal is prominent; 28 days later transcranial Doppler ultrasound signal is symmetric and normal on both sides. Differentiating stenosis from postischemic hyperemia is difficult. Bottom: Right (R) middle cerebral artery (MCA) as reference. PI, pulsatility index.

Discrepancies between angiography and TCD studies carried out at different times. It is also possible that occlusion on an angiogram appears as severe stenosis on TCD. A single angiogram shortly after stroke cannot differentiate primary atherosclerotic MCA stenosis from recanalizing embolic occlusion, which is relevant for further therapeutic strategies.

FIGURE 4. Top: Late recanalization of right (R) middle cerebral artery (MCA), starting 12 days after clinical onset in patient 10. Five weeks later unequivocal blood velocity enhancement is demonstrable (residual stenosis?). Bottom: Normal left (L) MCA as reference. Note different scales! PI, pulsatility index.

References


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