Evidence for Embolization From a Posterior Cerebral Artery Thrombus by Transcranial Doppler Monitoring

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Background: Transcranial Doppler monitoring enables the detection of emboli passing through intracranial arteries. Comparison of the different intracranial vessels with respect to emboli detection can be of use in identifying the source of embolism.

Case Description: We report the case of a patient with an acute posterior cerebral artery (PCA) infarct on the right side, with evidence for high-degree ipsilateral PCA narrowing on admission. During transcranial Doppler monitoring 3 days later, we found frequent emboli-like signals in the power spectrum of the right PCA distal to the stenosis but not in any other intracranial vessels. Four days later, angiography and transcranial Doppler failed to show PCA narrowing, and transcranial Doppler monitoring of the right PCA showed no further emboli-like signals. A stenosis of the right vertebral artery was regarded as the possible source of thromboembolic narrowing of the PCA.

Conclusions: We assume that in our patient a thrombotic clot in the PCA was resolved autolytically through detachment of small emboli into the distal part of the PCA. We speculate that in the early course of autolysis a larger fragment of the clot had occluded one of the distal branches, thus leading to the small PCA territory infarct. (Stroke 1993;24:606-608)

KEY WORDS • cerebral infarction • embolus • ultrasonics

Small emboli in intracranial arteries can be detected by transcranial Doppler (TCD) ultrasonography. The characteristic sign of an embolus is a short-lived increase in the intensity of the power spectrum within a limited frequency range. Acoustically, such an event is accompanied by a loud whistle. Emboli from various sources can be detected by TCD. During cerebral angiography, changes in the TCD spectrum of the middle cerebral artery (MCA) can be demonstrated after the injection of the contrast material. The detection of air bubbles in the MCA was observed after the reclamping of the aorta during open-heart operations. Air bubble emboli in the MCA were also reported during carotid endarterectomy after the release of common carotid artery cross clamps. Moreover, spontaneously occurring emboli-like signals were recorded in 24 of 91 patients with severe carotid stenosis during TCD monitoring of the MCA ipsilateral to the carotid lesion. These events were interpreted as "formed-element emboli" originating from ulcerations of the carotid artery.

We report the case of a patient with an acute infarction in the territory of the right posterior cerebral artery (PCA). TCD revealed frequent emboli-like signals in the right PCA but in no other cerebral artery, which indicated embolization from a narrowing in the PCA.

Case Report

A 63-year-old, right-handed housewife one evening noted the sudden onset of severe occipital headache, nausea, and dizziness. Observing her face in the mirror, she realized that she could not see the left part of her face. The next morning, her visual disturbance was still present, and she was admitted to our neurological department. A left lower quadrantanopia was found, and complete neurological and neuropsychological examination showed no further deficits.

A computed tomographic scan performed 2 days after the onset of the symptoms demonstrated a small (diameter, 2 cm), fresh infarct in the calcarean area of the right occipital lobe. Cardiac assessments (transthoracic echocardiography and Holter monitoring) and extracranial arterial assessments (Doppler ultrasonography and color-coded duplex) were normal. TCD examination showed increased flow velocities in the PI segment of the right PCA (250 cm/sec), which suggested narrowing. Cerebral angiography performed 6 days later revealed a 50% stenosis located at the beginning of the right vertebral artery (VA) (Figure 1, left panel). No abnormality was found in the right PCA (right panel). A reexamination with TCD performed on the day of angiography showed normal flow velocities in the right PCA. A magnetic resonance imaging investigation 17 days after onset showed a small hypointense lesion in the T1-weighted pictures of the right calcarean area with superimposed hyperintense spots, suggesting a
hemorrhagic transformation of the infarct (Figure 2). The patient was discharged from our hospital after 3 weeks. At that time, no significant change in the lower left quadrantanopia had occurred.

Methods
To study visually evoked blood flow velocity changes in the affected right PCA, we performed TCD monitoring by fixing the probe with a headband over the temporal bone. During this investigation, we detected several emboli-like events and decided to study these signals more systematically. We used a trans-scan TCD apparatus (EME, Überlingen, FRG) that enabled the recording of the power spectra for off-line analysis. The patient rested on a couch with her eyes closed. Signals from the right PCA (P2 segment; depth, 69 mm) were measured over a recording time of 20 minutes. The following vessels were monitored for 5 minutes each: right PCA (P1 segment; depth, 58 mm); left PCA (P2 segment; depth, 67 mm); right MCA (depth, 57 mm); and left MCA (depth, 56 mm). The basilar artery (BA) was assessed with a hand-held Doppler probe at insonation depths of between 75 and 95 mm for a total time of about 4 minutes.

The TCD monitoring was conducted 3 days after the stroke and 24 hours after treatment with heparin was started, and it was repeated 4 days later.

Results
Fifteen events, which were acoustically and visually clearly recognizable as emboli, were recorded during the 20-minute monitoring of the right P1 segment (frequency, 0.75 per minute). An additional four emboli were detected during the 5-minute monitoring of the right P1 segment (frequency, 0.80 per minute). Emboli were not observed during monitoring of the left PCA, the BA, or either of the MCAs.

Figure 3 gives five examples of typical emboli signs in the Doppler spectrum. A small spot of high intensity...
occurs for the duration of one or two fast-Fourier transformations (16 or 32 msec, respectively). In all cases, the intensity was at least 15 dB above the intensity values obtained in the absence of emboli. These high-intensity spots were always striated, covering a velocity range of 10 to 40 cm/sec (difference between the highest and lowest velocity). Although the velocity range of these spots was always located within the power spectrum of the physiological blood velocity signal (in only three of 19 emboli did it extend slightly in a negative velocity range), in most embolic events there were additional spectral changes outside the normal Doppler curve. These changes were discrete points, sometimes at equidistant intervals (Figures 3B, 3C, and 3E). Only four emboli (21%) were detected shortly before or during the minimal diastolic blood flow velocity (Figure 3C).

A repetition of the TCD monitoring 4 days later yielded no additional emboli. Moreover, the flow velocity in the right P1 segment was then decreased from 250 cm/sec (day 2) and 100 cm/sec (day 3) to 57 cm/sec (day 7).

Discussion

In Western countries, the most important stroke etiologies are arteriosclerosis of large extracranial vessels and cardiac embolism. Primary arteriosclerosis of the main intracranial vessels is a rare cause of brain ischemia. One day after admission, our patient had, as evidenced by TCD, a high-degree stenosis in the proximal part of the right PCA. Six days later, normal TCD signals of the right PCA and a normal diameter of this vessel in the angiogram indicated complete resolution of the PCA stenosis. Because spontaneous resolution of a primary arteriosclerotic lesion within a few days is improbable, the PCA stenosis of our patient was presumably a thrombotic embolus originating from an extracranial arteriosclerotic stenosis. Consistent with this idea, a VA stenosis with 50% lumen narrowing was demonstrated by angiography. As was shown in a large study by Lammers et al., the VA stenosis is less than 75% in 37% of cases with symptomatic VA diseases.

Although thromboembolic occlusion of the proximal PCA is a frequent mechanism for infarctions in the PCA territory, the interesting feature in our patient was that progress of the thrombus resolution could be followed by TCD monitoring. The following observations indicate that the emboli-like signals found in our patient imaged, indeed, the process of thrombolysis of the PCA narrowing: 1) TCD follow-up clearly showed that within 1 week a high-degree stenosis in the P1 segment gradually disappeared. 2) Emboli-like signals were observed only in the right PCA, not in any other intracranial vessel. In particular, the normal TCD monitoring of the left PCA and the BA reduced the possibility that the observed emboli originated from the VA stenosis. 3) The flow velocity of the emboli was much higher in the P2 than in the P1 segment, suggesting that the emboli started in the P1 segment and were accelerated to a higher velocity in the P2 segment. 4) No further emboli were detected when the flow velocity in the P1 segment was normalized.

Spontaneous lysis of an embolic thrombus in an intracranial vessel is thought to be a frequent event in acute stroke cases: angiography within 6 hours after the onset of stroke symptoms shows intracranial occlusions in 66% of the cases, whereas occlusions are only rarely found a few days later, which suggests that recanalization occurs within a few days of onset. Our TCD findings give an impression of the progress of spontaneous thrombolysis. The thrombus shrinks successively by detaching small emboli into the distal part of the affected vessel. Although in our case these emboli gave a strong TCD signal, they were obviously too small to occlude distal branches of the PCA because the patient remained clinically unchanged during the embolization period. However, it is possible that one large embolus in the early course of autolysis was responsible for the occipital infarction. Both the very small extent of the infarct and the secondary hemorrhage indicate that the stroke was caused by embolic occlusion of a calcarine branch and not directly by a proximal PCA occlusion.

References

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