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
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 quadrant anopia had occurred.

Methods

To study visually evoked blood flow velocity changes in the affected right PCA,4 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), appearing at the time of the main spot and extending to maximal positive and negative velocities.

A majority of the detected emboli (10 of 19; 53%) were seen at the time of the maximal systolic blood flow velocity (Figures 3A, 3B, and 3E). An additional five events (26%) occurred shortly before or after systole (Figure 3D). Only four emboli (21%) were detected shortly before or during the minimal diastolic blood flow velocity (Figure 3C).

Although the maximal blood flow velocity in the P1 segment of the PCA (100 cm/sec) was higher than that in the P2 segment (80 cm/sec), the averaged velocity of the emboli (measured as mean velocity of the high-intensity spots) was much higher in the P2 segment (58.9±12.4 cm/sec [mean±SD]) than in the P1 segment (12.8±2.1 cm/sec; p<0.001 by Wilcoxon's test).

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, aVA 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 calcarean branch and not directly by a proximal PCA occlusion.
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