Basilar Artery Migraine: Transcranial Doppler EEG and SPECT From the Aura Phase to the End

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Transcranial Doppler, electroencephalography, and single photon emission computed tomography were performed in a case of basilar migraine during the different phases of the attack. In the aura phase, the patient had bilateral blindness and ataxia. Doppler ultrasound studies showed a reduction in the mean flow velocity of the posterior cerebral arteries, electroencephalography showed slow activity confined to the posterior regions, and single photon emission computed tomography, an area of hypoperfusion in the right parietal and occipital regions. During the headache phase, when the neurological examination was normal, transcranial Doppler showed an increase in the mean flow velocity of both posterior cerebral arteries and the electroencephalogram revealed an increase in slow activity over the occipital regions. When the pain subsided, the electroencephalogram showed a progressive reduction of the slow abnormalities and transcranial Doppler was reported as normal. After a week, single photon emission computed tomography and cranial magnetic resonance imaging were normal. After a month, a follow-up electroencephalogram was also normal. All these findings indicated a transient focal reduction of cerebral blood flow during the aura phase.

Key words: basilar migraine, transcranial Doppler sonography, electroencephalogram, single photon emission computed tomography

Abbreviations: TCD transcranial Doppler, CBF cerebral blood flow, rCBF regional cerebral blood flow, SPECT single photon emission computed tomography, PCA posterior cerebral artery, MCA middle cerebral artery, MFV mean flow velocity, PI pulsatility index

Transcranial Doppler (TCD) is an effective technique for the study of cerebral blood flow (CBF) in patients with migraine. The available data refer to the pain phase and the intericta period.1,2 The possibility of documenting the CBF variation starting with the aura phase is a rare event limited to cases with prolonged aura.3 In this case, the data suggest a decrease of CBF in the cortical area responsible for the neurological manifestations during the aura phase.

Determinations of regional cerebral blood flow (rCBF) with Xenon-133 during migraine with aura have already demonstrated initial focal hypoperfusion followed by hyperemia.4 More recently, the use of technetium Tc 99m hexamethyl propyleneamine oxime (Tc 99m HMPAO) single photon emission computed tomography (SPECT) in patients with migraine without aura has failed to show evidence of CBF focal alterations.5 A relative rCBF reduction during migraine attacks might account for the different electroencephalographic abnormalities described in these patients, the meaning of which is uncertain.

We performed an EEG, TCD, and cerebral SPECT using Tc 99m HMPAO, during all phases of a basilar migraine attack in order to correlate the focal electroencephalographic abnormalities with the dynamic alterations of the rCBF.

In view of the neurological deficit in the aura phase, we evaluated the TCD parameters with respect to the posterior cerebral arteries (PCAs) and compared them with those of the middle cerebral arteries (MCAs). The normal values for mean flow velocity (MFV) suggested by the Italian Society of Neurosonology6 (PCA 51 to 26 cm/sec, MCA 76 to 44 cm/sec, pulsatility index [PI] 0.99 to 0.69 cm/sec) were taken into account.

CASE HISTORY

A 25-year-old woman was hospitalized with complaints of positive scotomata and bilateral blurred vision for about 3 hours.

For some years, she had been experiencing rare episodes of headache at the vertex. The headaches were sometimes pulsatile but without any other symptoms or prodromata. The pain resolved spontaneously after a few hours.
She had been taking an oral contraceptive for 3 years.

Three other members of her family had experienced episodes of migraine with aura.

On admission, the visual deficit prevented her from recognizing people and objects that were perceived like shadows. She needed to be steadied when walking and standing. Otherwise, the objective general and neurological examinations, including the optic fundi and pupil reactivity to light, were normal.

The initial TCD examination was performed very shortly after admission and showed a remarkable reduction in the MFV of both PCAs (or the left MFV 20 cm/sec, PI 1.39; on the right MFV 22 cm/sec, PI 1.04 [Figure 1]), but MFV values of the MCAs were within normal limits (on the left 70 cm/sec, PI 0.69; on the right 64 cm/sec, PI 0.78).

![Fig 1.](image1.png)

An EEG revealed continuous runs of theta waves over the posterior regions, mostly on the right side (Figure 2).

Shortly after the TCD examination, she underwent cerebral SPECT which showed an area of relative hypoperfusion in the right parietal and occipital regions (Figure 3A).

Three hours after admission, and as soon as the SPECT examination was completed, she, started complaining of a biparietal pulsatile headache without nausea. The visual deficit had completely subsided and the neurologic examination was normal.

The second TCD, performed 2 hours after the onset of the headache, showed an MFV increase of the PCAs (on the left MFV 42 cm/sec, PI 0.62 on the right MFV 48 cm/sec, PI 0.59 [Figure 4] and normal values for the MCAs (on the left MFV 76 cm/sec, PI 0.78; on the right MFV 72 cm/sec PI 0.67).

The following day, the patient continued to complain of a pulsatile headache in the parietal regions. No analgesics were given.

![Fig 2.](image2.png)

![Fig 3.](image3.png)

A third TCD showed an MFV increase of the PCAs (on the left 62 cm/sec, PI 0.70; on the right 56 cm/sec, PI 0.70 [Figure 5]); MFV of the MCAs had slightly increased on the left side (MFV on the left 80 cm/sec, PI 0.85; on the right 70 cm/ sec, PI 0.83).

A follow-up EEG revealed a fairly considerable increase in the slow waves over the posterior regions, and a widespread increase in the
A fourth TCD revealed decreased MFV of the PCAs compared with those of the previous day (on the left 38 cm/sec, PI 0.86; on the right 42 cm/sec, PI 0.68 [Figure 7]); MFV of the MCAs had only slightly increased (on the left 78 cm/sec, PI 0.76; on the right 82 cm/sec, PI 0.71).

A further EEG showed a reduction in the slow, abnormalities over the posterior regions and in the low-frequency activity over the extracerebral regions (Figure 8). The neurologic examination was again normal.

A week after admission, cranial MRI and a follow-up cerebral SPECT (Figure 3B) were normal. The EEG was unchanged. A final TCD showed that PCA parameters (MFV 40 cm/sec, PI 0.71 on both sides) as well as MCA parameters (on the left MFV 70 cm/sec, PI 0.69; on the right MFV 66 cm/sec, PI 0.65) were normal. A month later the EEG was normal.

COMMENTS

Thié et al used terms such as "vasoconstriction" and "vasodilation" to define an increase or decrease in MFV of at least 10 cm/sec. Therefore, while describing the alterations revealed by TCD rather than the simple normal reference limits for changes in rCBF, we considered as significant, MFV variations equal to or superior to this value.

The most important data refer to the aura phase, when the patient still complained of a bilateral visual deficit. In that phase, TCD showed a sharp reduction of MFV of the PCAs together with PI values above normal limits. We ascribed this alteration to a constriction of resistance vessels leading to a real reduction in the blood flow, rather than to a dilation of conductance vessels, which would not have a significant effect on the blood flow. This suggestion was confirmed by cerebral SPECT, which revealed an area of relative decreased uptake of the tracer in the right parieto-occipital region compatible with ischemia in the same region. It is possible that if this examination had been performed earlier, it would have revealed a bilateral perfusion deficit.
To our knowledge, this is the first case of Tc 99m HMPAO SPECT examination during the aura phase of a basilar migraine attack.

The area of reduced flow, documented in the aura phase of the migraine attack, resolved with resolution of the pain. Therefore, we believe that it was not a preexisting defect, but was directly correlated with the migraine attack. Possibly, future SPECT examinations in the early phases of migraine aura attack may reveal a rCBF deficit in similar cases, in which ischemia cannot be revealed by sensitive examinations such as MRI.

During the headache phase, there was a progressive increase of MFV of the PCAs, which doubled in the initial phases of the pain, as compared with the MFV in the aura phase. Many hours after the onset of the pain, the MFV of the PCAs increased even further and reached absolute values above normal limits.

We explained this MFV increase of the PCAs together with a reduction in PI values as caused by a dilation of resistance vessels with consequent hyperemia. When the pain was over, the MFV of the PCAs decreased as compared with the headache phase. In this phase, the MFV absolute values had returned to, and remained at normal values.

During the different phases of the migraine attack, serial EEGs were performed in addition to TCD.

Different electroencephalographic alterations have been described in patients with basilar migraine. In some reports, however, the temporal relationship with the migraine onset is not always clear. In addition, the EEGs have rarely been recorded during the aura phase as was the case in our patient who showed continuous slow wave activity over the posterior regions, mostly on the right side. The correlation between the EEG findings and the clinical defect, the TCD data showing hypoperfusion in the region of the PCAs, the SPECT data, and above all, the subsequent disappearance of the electroencephalographic abnormalities, suggests that in our patient, the electroencephalographic alterations were also due to the reduced rCBF during the migraine attack. In the headache phase, the slow focal posterior discharges initially grew, but later improved with resolution of the headache. A week later, theta runs were still present in the posterior derivations at a phase when TCD and SPECT showed normal findings. A month later, a follow-up EEG was normal.

The monitoring of the different phases of the migraine attack with different modalities (TCD, EEG, SPECT, MRI) allows a better interpretation of the data obtained from each modality and, at the same time, a better understanding of the pathogenesis of this disease.

In our patient, the neurological defect during the aura phase corresponded with the evidence of a transient focal reduction in the cerebral blood flow, as revealed by TCD and cerebral SPECT.

Therefore, the corresponding slow abnormalities on EEG in a cerebral region responsible for the neurologic disorder were the electrophysiological equivalent of a focal reduction in the cerebral blood flow caused by the dynamic alterations of the cerebral vessels, as revealed by the TCD.

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