Bilateral Posterior Cerebral Artery Strokes in a Young Migraine Sufferer

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We report a young migraine sufferer who developed bilateral posterior cerebral artery territory infarcts during the course of his classic migraines, the second of which was associated with intraluminal clot in the posterior cerebral artery. To our knowledge, bilateral posterior cerebral artery stroke from spontaneous migraine has not been reported. Head computed tomographic, magnetic resonance imaging, and angiographic correlation is presented. The mechanism of migrainous infarction may be in part explained by caliber changes in arterioles and capillaries leading to flow reduction in the more proximal conduit arteries combined with the associated coagulopathy that has been previously documented during migraine attacks. (Stroke 1988;19:525–528)

Ischemic cerebral infarction that occurs during the progress of a migraine attack is rare and the mechanisms are uncertain.1–4 Although the neurologic symptoms associated with migraine are most commonly visual,7 localized to the cerebral cortex supplied by the posterior cerebral artery (PCA),4 ischemic strokes attributed to migraine most commonly occur within the territory of the middle cerebral artery.1,8–11 Only rarely is arterial occlusion demonstrated on angiography and, paradoxically, most often in a single PCA.1,8,10,11 To our knowledge, bilateral PCA stroke from spontaneous migraine has not been reported. We describe a young migraine sufferer with bilateral PCA infarcts, the second of which was associated with an intraluminal clot in the PCA. The angiographic findings in this patient support recently postulated novel mechanisms of migrainous stroke,14 which we discuss.

Case Report

A 37-year-old man had suffered from confusional migraine since the age of 20 years. His attacks always began with the acute onset of visual images “shifting” in front of him, associated with scotoma and confusion for the memory of recent events. These symptoms lasted from minutes to hours and were then always followed by a mild to moderate bifrontal throbbing headache, photophobia, and malaise. They generally occurred once to twice a month but recently had increased in frequency, accompanied by a bifrontal headache. However, on one occasion his altered memory and vision did not disappear. Head computed tomography (CT scan) revealed a left temporoparietal infarct. Catheter vertebral angiography revealed an occluded left PCA (Figure 1). Bilateral carotid angiography was normal. He was placed on aspirin and dipyridamole. One week later he again noted shifting visual images, severe trouble recollecting recent events, and a more diffuse throbbing headache.

Medical history was unremarkable for head injury, loss of consciousness, cardiac disease, rheumatic fever, coagulopathy, chiropractic manipulation, dyslipidemia, diabetes mellitus, or hypertension. He had smoked 20 cigarettes/day for 10 years.

A sister had suffered from migraines associated with right orbital swelling and blurred vision. A maternal uncle went blind suddenly after a severe headache. The patient’s mother also had severe migraines.

General physical, neurovascular, and cardiac examinations were normal. Neurologic examination revealed absent anterograde memory and moderately impaired retrograde memory.

Cognitive function testing conducted 2–3 weeks after the initial onset of symptoms suggested a severe amnestic syndrome without global cognitive impairment. Testing also revealed substantial but incomplete disorientation to time, place, and person, and mild impairment of digit span and mental tracking. Immediately after presentation, the patient reproduced some of the themes and details from paragraph-long passages and nonverbal designs; 30 minutes later he could recall none of the content from either set of materials, even though strong cues were provided. He obtained a Wechsler memory quotient of 64, > 2 SD below his full-scale Wechsler intelligence quotient. Although testing revealed him to have mild to moderate impairment in verbal fluency, speech-sound perception, visual-motor coordination, and constructional praxis, he displayed normal to superior performance on concept formation, tactile problem solving, rhythm discrimination, and motor speed tests. Adequate scores on visual search and oral reading tests indicate that his visual field deficit did not interfere substantially with registration of stimuli presented in the memory tests.

There were bilateral superior quadrantanopsias and impaired vertical saccades in both directions.
FIGURE 1. Selective left vertebral angiogram, anterior-posterior view. There is occlusion of left posterior cerebral artery (PCA) (arrow) and paucity of arterial filling distal to occluded PCA. Right PCA appears normal.

Echocardiography, electrocardiogram, Holter cardiac monitor, complete blood count, platelet count, prothrombin time, activated partial thromboplastin time, factor VIII, factor V, antithrombin III, protein C, protein S, plasminogen activity and antigen, α-antiplasmin activity, Westergren erythrocyte sedimentation rate, VDRL, serum protein electrophoresis, antinuclear antibody, rheumatoid factor, quantitative immunoglobulins, cryoglobulins, complement assays, and IFA anti-DNA were normal. CT scan revealed bilateral mesial temporo-occipital infarctions (Figure 2). Magnetic resonance imaging (1.5 T) documented involvement of both hippocampi, parahippocampal gyri, and amygdala, more extensive on the right (Figure 3). Cerebral angiography revealed an intraluminal clot in the right PCA and an occluded left PCA with visible collaterals now present compared with the previous angiogram (Figure 4). A magnified view of this intraluminal lesion is shown in Figure 5. Warfarin therapy was initiated after the finding of the intraluminal clot, and he has remained stable for 1 year.

Discussion

Our patient had regularly occurring stereotypic spells of confusional migraine in the context of a strong family history of migraine associated with focal neurologic symptoms. Extensive evaluation failed to reveal another adequate explanation for his strokes, although cigarette smoking may be an independent stroke risk factor in young adults. There was no supporting evidence of cardiac, hematologic, immunologic, or neoplastic disease. Both of his strokes occurred during the progression of a typical migraine attack and the neurologic deficits were typical of the transient migrainous symptoms of previous attacks, thus satisfying accepted criteria for stroke due to migraine.14,16
Previously, migrainous stroke or ischemia has been attributed to arterial vasospasm, cerebral edema, arterial wall dissection, and increased platelet aggregation. No specific pathologic changes that characterize migraine have been consistently identified on any autopsied cases of migrainous stroke. Thus, the mechanism of migrainous stroke remains open to discourse.

What, then, are the common mechanisms that produce these blood flow changes and precipitate intravascular clotting such as in this case report? Complete obstruction of the PCA or its branches has been documented angiographically in only a few reports of migrainous stroke. As in the case reported here, revascularization was noted when one patient was restudied.

Intraluminal clot within one PCA in the absence of a detectable embolic source, also found in our patient, has been documented previously. Migrainous occipital infarction was associated with intraluminal clot in the PCA of two patients. One, a 28-year-old man, developed retro-orbital headache and a left homonymous hemianopia while jogging. He was asymptomatic 5 months later while on warfarin. The other patient was a 23-year-old woman with a 3-day history of daytime headache who developed a left superior quadrantanopsia. She was asymptomatic 32 months later on no treatment. Both patients had a strong history of
common migraine. Vasospasm was not seen. Coagulopathy and cardiac or other embolic sources were extensively searched for and not found. Only one other case of migrainous infarction has been reported to have intraluminal clot.4

O’Brien26 first demonstrated global oligemia that outlasted the brief time associated with focal symptoms of the migraine aura. Olesen et al27 has shown this spreading oligemia during attacks to be analogous to the spreading depression of Léao. More recent evaluations have reported regional cerebral blood flows to be in the ischemic range during an attack.28 Caliber change in arterioles and capillaries can in part be mediated by local factors in the vascular wall, independent of blood flow,29 and by neurogenic factors.30 If the smaller resistance vessels are involved in the putative spreading depolarization of the migrainous process, flow reduction in the more proximal, larger conduit arteries such as the proximal PCA could be explained by altered downstream resistance. This, combined with the previously reported serum platelet factor abnormalities in young adults with cerebral infarction secondary to migraine,18 may produce blood flow stasis leading to intraluminal clot.

Note added in proof. Since submission of this manuscript, three additional reports of migrainous stroke have appeared, none with documented bilateral PCA strokes.31,32 Bogousslavsky et al31 reported 22 patients, 19 with normal angiograms; 9 had PCA infaracts. Rothrock et al32 reported 22 patients, 5 of 12 with abnormal angiograms, primarily “spasm.” One patient had PCA “beading.” Broderick and Swanson33 reported 20 patients. Five of 12 had abnormal angiograms including one each with intraluminal clot and filling defect.

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References

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