Occipital Infarction With Hemianopsia
From Carotid Occlusive Disease

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Extracranial internal carotid artery occlusive disease usually produces stroke in the middle cerebral artery territory or the border zone between the middle and anterior cerebral arteries. It is unusual for occipital infarction in the posterior cerebral artery territory to be caused by internal carotid artery disease despite the fact that the posterior cerebral artery may arise directly from the internal carotid artery as an anatomic variation. We describe a patient with a fetal posterior cerebral artery originating from the internal carotid artery, and the initial manifestation of his extracranial internal carotid artery occlusive disease was hemianopsia from occipital infarction. (Stroke 1989;20:409–411)

The usual clinical manifestations of extracranial internal carotid artery (ICA) atherosclerotic disease are well known.1–4 Transient ischemic attacks (TIAs) of the monocular or hemispheral type are common. When it occurs, stroke usually involves the middle cerebral artery (MCA) territory or the watershed region between the MCA and the anterior cerebral artery and produces contralateral sensorimotor abnormalities and dysphasia or behavioral abnormalities, depending on the hemisphere affected. Rarely, ischemia due to ICA occlusive disease includes infarction in the posterior cerebral artery (PCA) territory when the PCA arises primarily from the ICA. This report is prompted by our recent experience with a patient whose initial sign of ICA occlusive disease was hemianopsia from an occipital infarct related to a fetal origin of the PCA from the ICA.

Case Report

A 70-year-old man was driving his automobile when he suddenly lost vision throughout the left half of space in both eyes. He described his visual loss to the left as a “blank or nothing,” and it persisted unchanged from the onset. An ophthalmologist documented a complete left homonymous hemianopsia using automated testing (Humphrey Instruments, San Leandro, California) and saw a cholesterol plaque in a right retinal arterial branch at the disk margin.

On examination 13 days after the onset, a right carotid bifurcation bruit was present, the right retinal cholesterol plaque remained, and the patient’s left homonymous hemianopsia was noted. Otherwise, the examination was normal.

The patient gave no history of transient monocular blindness, transient hemispheral attacks, or other neurologic events. The patient had a history of polymyalgia rheumatica and peripheral vascular disease. He had smoked two packs of cigarettes a day for many years.

The results of routine blood studies were normal. An echocardiogram showed normal left ventricular size and no areas of dyskinesia or thrombus. He had 2+ aortic and mitral regurgitation and slight left atrial enlargement. Twenty-four-hour cardiac monitoring showed no significant arrhythmias.

A computed tomogram (CT scan) showed a right occipital infarct (Figure 1) in the PCA territory and a right frontal infarct. Cerebral angiography revealed a subtotal occlusion of the right ICA, with minimal antegrade blood flow and patent cervical and intracranial ICA on later films (Figure 2). A direct communication was present between the right ICA and the vertebrobasilar circulation via a fetal right posterior communicating artery (Figure 3). The right PCA was patent, with no missing branches.

The patient had a right carotid endarterectomy to remove a potential embolic source. The endarterectomy was performed uneventfully, and the patient was discharged home after 3 days. On the afternoon of his discharge, however, he complained of occipital headache and nausea and developed left-sided weakness and falling to his left. En route to the hospital by ambulance, he became unresponsive. Admission examination showed a comatose patient...
with 3-mm nonreactive pupils and flexion of all extremities to pain stimulation. CT scan showed a large parenchymatous hemorrhage in the right basal ganglia, with extension into the ventricular system. The patient remained comatose and died 2 days after admission. Autopsy was not performed.

Discussion

Usually, PCA occlusion producing occipital infarction results from either cardiac source or local embolism from vertebrobasilar atheroma.5-7 A few autopsy studies8,9 have described PCA territory infarction resulting from ICA occlusion, but no clinical features were discussed. In their text on extracranial cerebrovascular disease, Wylie and Ehrenfeld9 mentioned the occurrence of hemianopsia resulting from occipital infarction when the PCA anomalously arises from the ICA; however, the authors provide no supporting data. Although it is well known that one or both PCAs may arise primarily from the ICA rather than from the basilar artery, surprisingly, we were unable to find any descriptions of the clinical consequences of this anatomic variation.

Our patient illustrates the anatomic relation between the ICA and the PCA and highlights the initial clinical presentation of hemianopsia from an occipital infarct in the PCA territory likely caused by local embolism from a subtotal ICA occlusion. Our patient’s right PCA also filled from the basilar artery, making embolism from the posterior circulation a possibility. However, the absence of vertebrobasilar atheromatous disease on angiography or a cardioembolic source plus the compelling clinical

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features of carotid bruits and retinal embolus make carotid occlusive disease the probable cause of the occipital infarct. Although right ICA angiography did not fill the right PCA, this resulted from poor transmission of the arterial pulse pressure beyond the high-grade ICA stenosis. The fetal right posterior communicating artery seen on the left vertebral artery angiogram provided the anatomic channel for passage of an embolus from the ICA to the PCA.

Our experience should alert clinicians to the important but unusual clinical association between the PCA and extracranial ICA occlusive disease. If a patient presenting with occipital infarction has appropriate clinical signs and symptoms of ICA disease, clinicians should recognize the possible association and pursue the diagnosis of extracranial ICA occlusive disease as well as the more common causes of PCA territory ischemia.

References


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