Delayed TIAs Distal to Bilateral Occlusion of Carotid Arteries — Evidence for Embolic and Hemodynamic Mechanisms

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SUMMARY We studied 4 patients with bilateral carotid artery occlusion who suffered delayed TIAs in one of the occluded internal carotid or common carotid areas. Hemodynamic mechanisms were prominent in two patients, in head turning and orthostatic hypotension. In the other two, embolic phenomena through the homolateral external carotid collateral pathways were probable, because this artery (or the common carotid artery) showed atheromatous stenosis and major collateral supply to the brain and retina. Different mechanisms may be responsible for further ischemia after bilateral occlusion of carotid arteries.

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Case Reports

Case I

This 59-year-old man suddenly lost consciousness without warning symptoms. After one day the patient regained consciousness, but was aphasic and hemiparetic on the right side of the body. When hospitalized 5 weeks later he showed a right-sided facio-brachial weakness and a moderate expressive speech disturbance. Archography and bilateral carotid arteriography showed bilateral ICA occlusion, with stenosis of left external carotid arteries (ECA) and left common carotid artery (CCA) (fig. 1). ECA collateral pathways to the brain and retina were well developed on both sides. Intracerebral arteries did not show significant changes. Doppler ultrasonography showed reversal of ophthalamic flow bilaterally. There was no heart disturbance. During the next 6 months the patient experienced three times amaurosis fugax in the left eye and once numbness of the right face and arm, of 5' duration. Acetylsalicylic acid was begun, but the patient was not seen again.

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Comment

BCO was discovered in this patient after he suffered an acute left hemispheric infarct. Follow-up disclosed the occurrence of 4 homolateral retinal and hemispheric TIAs without hypotensive phenomenon. As angiography had demonstrated left ECA and left CCA atheromatous stenosis and excellent ECA collateral pathways to the retina and brain, it appears probable that embolic phenomena through the left ECA channel were responsible for the TIAs.

Case 2

A 65-year-old man was admitted after a 2-day history of left facio-brachial weakness. Two years before he had suffered a similar but transient (4 hours) episode. Neurological examination showed a slight weakness of the right face and arm and constructive apraxia. Archography showed bilateral ICA occlusion with a suspicion of stump on the right side. Right ECA and left subclavian arteries (SCA) showed marked atheromatous stenosis (fig. 1). The left ECA was only slightly narrowed. Doppler ultrasonography showed reversal of the ophthalmic flow on the right side. No flow was detected on the left side. No cardiac dysrhythmia was present. Acetylsalicylic acid was begun. During the month following admission the patient experienced 4 transient (10–15') episodes of increased weakness and tingling of the left face and hand. Antiaggregant was changed for anticoagulant therapy. During the next 8 months there was no recurrence of TIAs.

Comment

This patient with chronic VBI experienced left sylvian TIAs shortly before and during one year after right ICA and left CCA occlusions were discovered on angiography. ECA collateral pathways to the brain were inconspicuous. On the other hand, TIAs were closely related to head turning, suggesting a hemodynamic mechanism.

Case 4

For 10 years, this 50-year-old woman had suffered amaurosis fugax in the right eye (duration: 30') after heavy meals or when quickly passing from a lying or sitting to a standing position. Fifteen days before admission she began to experience bilateral numbness of hands and lips, of a few seconds duration. Twelve days later she twice suffered motor aphasia during one minute. Neurological examination was normal. Archography showed bilateral ICA occlusion without stump, and normal ECAs (fig. 1). Doppler ultrasonography showed bilateral reversal of ophthalmic flow. During the hospitalization the patient continued to suffer orthostatic amaurosis fugax in the right eye and experienced dizziness, diplopia and perioral numbness after an episode of cardiac dysrhythmia.
Figure 2. Case 3, archography and right carotid angiogram. A) Occlusion of the left CCA (oblique view). B) Occlusion of the right ICA, with normal ECA. C) The right ECA fills the ophthalmic artery but brain supply is poor.

Comment

This patient with bilateral ICA occlusion clearly suffered hypotensive amaurosis fugax in the right eye. She also showed VBI symptoms of similar origin. No embolic phenomenon could explain these TIAs, which appeared to be of hemodynamic origin.

Discussion

When studying the outcome of BCO, most authors did not mention the eventuality of further TIAs after the angiographic demonstration of BCO. Some studies reported TIAs as the presenting complaint of BCO, but no mention was made of their continuation after the angiographic demonstration of BCO. When TIAs were noted before admission, many of them were of the VBI type, suggesting that hemodynamic disturbances and steal phenomena may be prominent in BCO. On the other hand recent reports showed that most delayed TIAs and strokes distal to occlusion of one internal carotid artery are of embolic origin. In these cases, emboli may arise from an atheromatous stenosis of homolateral ECA, when this artery is the main collateral to the occluded ICA. When both ICAs are occluded, further TIAs may be due more often to hemodynamic disturbances than in unilateral occlusion, because intracranial perfusion is more impaired.

In our cases 1 and 2, embolic phenomena through ECA pathways explained the occurrence of the delayed TIAs, whereas hemodynamic disturbances were most probable in our cases 3 and 4. Our cases 1 and 2 showed an atheromatous stenosis of ECA or CCA homolaterally to the side of the brain that suffered further TIAs. In case 2 a homolateral stump was suspected. In both cases ECAs were a major collateral system to the occluded ICA areas. Embolization from the ECA or CCA atheromatous plaques appears probable because TIAs were not related to hypotensive events and they disappeared in case 2 after anticoagulant therapy was introduced. Embolization from the stump through collateral pathways could also be considered in case 2. Such a mechanism has already been reported and ECA collateral channels appear to have the same pathogenic role than in ECA atheromatous stenosis. In contrast to these cases, in case 3 head turning suggesting recurrent compression of VAs was responsible for transient aphasia. VAs were the main collaterals to the occluded ICAs areas, because the left CCA was occluded and the right ECA poorly supplied the brain. Intracerebral arteries did not show any significant modifications. In case 4 hemispheric TIAs were closely related to hypotensive episodes. It is not possible to establish if the brain hypoperfusion occurred through the ECA or VA systems, but its hemodynamic origin appears obvious. Most probably both collateral channels were involved together.
Our study confirms that BCO may not be a major disabling condition. As in unilateral ICA occlusion, delayed TIAs may occur, but they seem to be more often related to hemodynamic factors. However, embolic phenomena through homolateral ECA pathways may also be responsible. Patients with BCO and VBI symptoms (cases 3 and 4) seem more exposed to show delayed hemodynamic carotid TIAs, because "steal VBI" discloses a major instability of intracerebral perfusion. Medical therapy may be tried in order to suppress embolic phenomena. In hemodynamic ischemia ECA-ICA bypass procedures alone are probably not satisfying when ECA channels are not the main collaterals to the involved part of the brain, or when ECA shows atheromatous stenosis. ECA endarterectomy should first be performed. Good results may be obtained by correcting extracranial stenosis of the collateral pathways.

References
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