Case Report

Fusiform Basilar Aneurysm Simulating Carotid Transient Ischemic Attacks

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SUMMARY Fusiform aneurysms of the basilar artery are extremely unusual. Most show signs of a posterior fossa mass. One case is reported which presented with recurrent transient ischemic attacks suggesting carotid vascular disease. Failure of an appropriate endarterectomy to relieve the symptoms, and later brain stem infarction, led to the correct diagnosis. Whenever recurrent or new symptoms appear following carotid vascular surgery, other diagnoses must be considered.

FUSIFORM ANEURYSMS of the basilar artery are rare. They are known to present with subarachnoid hemorrhage, with brain stem signs indicating compression of the hindbrain by a mass, with transient ischemic attacks, or with dementia and gait disturbance secondary to hydrocephalus. One case of a fusiform aneurysm of the basilar artery is reported which presented with a focal brain stem infarction after recurring carotid territory transient ischemic attacks. This case emphasizes the need to consider other diagnoses which can simulate transient ischemic attacks, especially when the attacks are recurrent after appropriate treatment.

Case Report

A 51-year-old male was admitted to the hospital Feb. 13, 1977, after 12 hours of headache, lethargy, dysarthria, nausea and vomiting, and the onset of right-sided weakness. During the 3 days prior to admission the patient reported his right side was gradually becoming weaker.

Previously, (Aug. 6, 1974) the patient had been admitted with a complaint of transient right-sided weakness and slurred speech. No carotid bruits were heard and blood pressure was 120/80. Angiography revealed a very small left internal carotid ulcerated arteriosclerotic plaque. An endarterectomy was performed, and the patient had a smooth postoperative course. A brain scan, EEG, and computed tomography (CT) scan were normal. A 5-hour glucose tolerance test suggested early diabetes because of late hypoglycemia.

The patient was then well until Sept. 17, 1974, when he developed right-sided weakness and dysarthria. Examination revealed a right central 7th paresis and a mild right hemiparesis, both of which cleared within 24 h. A repeat left carotid arteriogram revealed a normal appearing left internal carotid. A CT scan and EEG were normal. Holter monitoring for 24 h was normal. Blood coagulation profile and phonoelectrocardiography and ECG were normal. The patient was then well until Jan. 30, 1976, when he fainted. On awakening he vomited and had a severe headache. The next morning he noted right-sided weakness and slurred speech. Blood pressure was 140/100. A spinal tap revealed 64 red cells, 3 white cells, and a protein of 58. The patient recovered from his neurologic deficit over several hours. He was begun on warfarin sodium (Coumadin), and continued it as an outpatient.

The patient was then well until Aug. 7, 1976 when he developed right-sided weakness and dysarthria. Blood pressure was 118/80 and serum glucose was 110. The patient quickly recovered from his neurologic deficit and was continued on anticoagulant therapy.

For 3 days prior to this admission the patient had not been taking warfarin.

The general physical exam was normal, except for a surgical scar along the left side of the neck. No carotid bruits were heard. There was no neck rigidity. Blood pressure was 150/100. The patient was lethargic but followed verbal commands. The neurologic examination revealed the pupils to be equal, round, and reactive to light. The fundi were normal. There was first degree nystagmus on right lateral gaze. The patient could not look to the left, and the eyes could not be made to cross the midline with cold caloric testing. On gaze to the right there was incomplete adduction of the left eye. A left peripheral facial nerve paresis was present. The corneal reflexes were equal and present. A severe right spastic hemiparesis was present as were bilateral Babinski signs. Occasional ocular bobbing was visible.

An injected CT scan revealed a mass with the density of blood lying behind the clivus and extending up into the third ventricle (figs. 1, 2). A right brachial arteriogram revealed the presence of a large ectatic fusiform basilar aneurysm (figs. 3, 4). The irregular
walls suggested partial thrombosis or intramural dissection of blood.

The patient was begun on dexamethasone and antihypertensives. On Feb. 23, 1977, the patient suddenly became obtunded. A lumbar puncture revealed an opening pressure of 300 mm of H₂O and the spinal fluid was grossly bloody.

Although the brain stem signs did not fluctuate, the patient remained obtunded. Persistent hydrocephalus was demonstrated by CT scanning, and on Mar. 3, 1977, the patient underwent a right ventriculoperitoneal shunt. He became more alert and on April 17, 1977, was transferred to a rehabilitation center. At discharge he was fully alert. There was incomplete adduction of the left eye with first degree nystagmus on right lateral gaze. The patient could not
look to the left. A severe spastic right hemiparesis was present.

Over the next 3 months in the rehabilitation center the patient made a full intellectual recovery. He demonstrated emotional lability. He had a persistent mild right hemiparesis but was able to walk with a 4-legged cane. He was discharged home but died suddenly on October 23, 1977. Autopsy was not permitted.

Discussion

Of a series of 7,500 consecutive autopsies, Haynes, Bernhardt, and Young found only 5 cases of fusiform basilar aneurysms. Such aneurysms may present with subarachnoid hemorrhage, with brain stem or cranial nerve compression which simulates a tumor, with transient ischemic attacks, or with dementia and gait disturbance typical of normal pressure hydrocephalus. This case illustrates that a fusiform basilar aneurysm can also present as a localized brain stem infarction. Only one similar case in a 10-year-old has been reported. The presence of the aneurysm was suggested by CT scanning as reported by Peterson and associates. Occlusion of perforating pontine blood vessels leading to the infarction was caused by partial thrombosis of the fusiform aneurysm or by dissection of blood within the wall of the basilar artery obstructing the origins of the small brain stem vessels. Dissecting aneurysms of the intracranial vessels have been reported in young individuals in the postpartum period, following a tonsillectomy, or associated with cystic medial necrosis of the basilar artery, homocystinuria, trauma, migraine, syphilitic arteritis, segmental arteriosclerotic changes, or congenital medial defects. Dissection could have occurred at the site of arteriosclerotic changes in this basilar artery producing this fusiform aneurysm. However, most dissections are not preceded by any warning ischemic signs and survival is very rare when the basilar artery is involved.

Ventricular shunting was successful in reversing this patient's obtundation which was secondary to hydrocephalus caused by posterior third ventricular obstruction by the ectatic basilar artery. No specific therapy was possible for the pontine infarction. Because the symptoms produced by this aneurysm were ischemic in origin, treatment by vertebral artery ligation would probably have been disastrous. This patient's later death may have been due to subarachnoid hemorrhage or complete basilar occlusion.

This case emphasizes that whenever transient ischemic attacks recur in spite of apparent adequate
therapy or are associated with neurologic signs in a different vascular territory, another cause for the symptoms should be sought. This fusiform basilar aneurysm may have produced the transient ischemic attacks which were attributed to a small left internal carotid arteriosclerotic plaque. Recurrence of these symptoms after carotid endarterectomy and the later development of a brain stem infarction suggests that the basilar artery aneurysm may have been the real cause of these symptoms all along.

Although some recovery occurred following ventricular shunting and rehabilitation, the patient's disability and later death indicate that ectatic fusiform basilar aneurysms are not simply benign involutional vessel distortions caused by arteriosclerosis. Direct treatment of these lesions is not presently possible.

References

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