Local Embolism From Vertebral Artery Occlusion

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Basilar artery territory stroke may result from embolism arising from the site of vertebral artery occlusion. This stroke mechanism (local embolism) has been well documented in the middle cerebral artery territory from extracranial internal carotid artery disease but not fully appreciated in the vertebral basilar circulation. We report two patients whose clinical presentation indicated major basilar artery territory infarction documented by angiography to be the result of vertebral artery occlusion and artery-to-artery embolism. Vertebral artery occlusion has often been associated with a benign course, but under certain circumstances embolism to the basilar artery may complicate the outcome. (Stroke 1988;19:112–115)

Artery-to-embolism embolism, so-called local embolism,1 refers to dislodgement and distal migration of material from an athero-occlusive plaque or thrombus. In the carotid circulation, local embolism is the major mechanism for middle cerebral artery (MCA) territory stroke as a result of extracranial internal carotid artery disease.2–4 This mechanism is not as well recognized in the vertebral basilar system although Fisher and Karnes1 called attention to it more than 20 years ago. Recognition that specific large artery occlusion may cause distal embolization may help in planning initial therapy, perhaps in the form of short-term anticoagulation, in an effort to avoid this unwanted complication of local embolism. We report two patients with vertebral artery (VA) occlusion and distal embolism to the basilar artery.

Case Reports

Patient 1. A 34-year-old man noted the onset of a persistent nonthrobbing headache localized to the left posterior neck area 4 days prior to admission. On the evening before admission, while watching television, he developed a transient, prickly sensation in his right arm and leg. A few hours later, while in bed, his left arm and leg became weak and his vision was fuzzy. There was no history of trauma, transient ischemic attacks (TIAs), or medical conditions. He was a smoker and admitted to regular beer drinking.

On admission evaluation, the general physical and neurovascular examinations were normal. On neurologic examination, he was alert with normal speech and mental status. Cranial nerves were normal except for definite upbeating nystagmus present in the neutral position, accentuated with upgaze. Horizontal nystagmus was also present, most prominent with left lateral gaze. Left corneal sensation was diminished compared with the right, but sensations on his face were intact. There was a left hemiparesis involving his arm more than his leg with sparing of his face. Deep tendon reflexes were brisker on the left side. Primary and cortical sensations were normal.

Routine laboratory data included normal hemoglobin, hematocrit, white blood cells, platelet count, prothrombin time, partial thromboplastin times, sedimentation rate, and glucose. An electrocardiogram (ECG) and echocardiogram were both normal.

Computed tomography (CT scan) on admission was normal, but a repeat CT scan several days later showed a right pontine infarction. Angiography within 24 hours of admission revealed an occlusion of the left VA at the C2 level and an intraluminal filling defect (embolus) in the distal basilar artery and proximal superior cerebellar artery (Figure 1).

The patient was anticoagulated with heparin and then warfarin. His neurologic status gradually improved, and the left posterior head and neck pain disappeared in 3 weeks.

He was reevaluated 6 months later, at which time he had only mild impairment of fine and rapid alternating movements in his left limbs and increased deep tendon reflexes on his left side. His gait showed spasticity and slight foot slapping on the left. Repeat angiography showed an unchanged left VA occlusion, but the distal basilar artery was now normal (Figure 2).

Patient 2. A 62-year-old man awakened one night to go to the bathroom; his wife heard an unusual sound and found him leaning on the sink, poorly responsive. The emergency medical technicians found him lethargic and unresponsive to verbal commands. Purposeful movements of his left hand were noted. His medical history was unremarkable; there was no history of TIAs.

On admission, he was comatose. Blood pressure ranged from 158/87 to 230/120 mm Hg, and pulse was regular at 85 beats/min. Respirations were spontaneous and regular. His general physical and neurovascular examinations were normal. On neurologic examination, spontaneous decerebrate posturing of his left extremities was present. His right eye deviated to the right, his left eye rested down and in. Pupils were...
FIGURE 1. **Top:** Oblique view of selective left vertebral artery injection shows occlusion at C2 level (arrow). **Bottom:** Lateral projection in late arterial phase of right vertebral angiogram demonstrates smooth intraluminal filling defect (arrow) in distal basilar artery and proximal superior cerebellar artery (arrowhead).
small but reactive. Oculocephalic reflexes were full. There was a right central facial weakness and a dense, flaccid right hemiplegia, but after treatment with mannitol and intravenous steroids his left extremities showed normal movements and appropriate withdrawal responses to pain. Plantar responses were extensor.

The ECG showed no change from an earlier record with normal sinus rhythm, occasional premature ventricular contractions, and an inferior wall infarction of undetermined age. Chest x-ray was normal. Echocardiogram showed normal left ventricular cavity size, wall thickness, and systolic function. The distal septum was noted to be hypokinetic. Left atrial size was normal, and no thrombus was seen. Continuous bedside cardiac monitoring revealed no significant arrhythmias. Pertinent laboratory studies on admission included a normal hemoglobin, hematocrit, white blood cells, platelet count, prothrombin time, partial thromboplastin times, and cardiac enzymes. CT scan was limited by patient movement but showed no definite abnormality. Heparin was initiated.

Angiography on the day of admission showed occlusion of the right intracranial VA and an intraluminal filling defect (embolus) in the distal basilar artery (Figure 3). Later the same day, his condition deteriorated and he died despite supportive measures. No postmortem examination was obtained.

Discussion

Local embolism within the vertebral basilar circulation has not been well recognized as a stroke mechanism compared with MCA embolism from extracranial internal carotid artery occlusive disease. Pathologic studies have documented a few instances of artery-to-artery embolism from an occluded VA to the basilar artery proper, or more frequently to basilar artery branches, such as the posterior cerebral artery. Angiographic studies have also demonstrated the mechanism of local embolism to the basilar artery and/or its branches. Several patients in Caplan's report had evidence of VA occlusion and presumed distal embolization into the basilar artery or branches. George and Laurian describe two patients with VA occlusive disease and suspected basilar artery embolism. Most recently Koroshetz and Ropper, studying local embolism in the posterior circulation especially to PCA branches, identified at least one patient (No. 12) with additional evidence of a basilar artery embolus. In contrast to these angiographic studies in which local embolism was usually inferred, our two patients clearly had intra-arterial filling defects, with subsequent clearing in one patient, confirming the embolic nature of the angiographic findings. Early angiography (within 36 hours of stroke onset in our patients) enhances visualization of embolic occlusions before natural fragmentation and disappearance occur.
Occlusion of the distal VA has a variable but often favorable outcome. Some patients remain asymptomatic, especially if the other VA is functionally intact. In others, a lateral medullary or cerebellar infarction occurs because of compromise of the posterior inferior cerebellar artery branch of the occluded VA, but an excellent prognosis for recovery is expected. Other patients with VA occlusion may have devastating brainstem infarcts, either because clot propagates distally into the basilar artery or because a low-perfusion condition results when the other VA is occluded, atretic, or nonfunctional. In our patients, the clinical presentation was a pontine infarct, not lateral medullary as might have been expected. Local embolism to the basilar artery accounted for the distribution of brainstem infarction and probably occurred at, or subsequent to, the VA occlusion. No definite cardioembolic source was identified to account for the embolism, but Patient 2 did have an old myocardial infarction.

The clinician should be alert to the fact that local embolism and even antegrade propagation of clot distally may at times complicate the outcome of VA occlusion. It has been our practice, based on these observations, to use anticoagulation in patients with a lateral medullary syndrome during the acute period while clot organization occurs and for 6–8 weeks in an effort to prevent these unwanted effects, but a scientific study would be necessary to document efficacy of this treatment.

References


KEY WORDS • cerebral infarction • vertebral artery • embolism
Local embolism from vertebral artery occlusion.
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