Case Reports

Transient Amnesia Resulting From Vertebral Artery Dissection

John Laterra, MD, PhD, Stephen Gebarski, MD, and J. Chris Sackellares, MD

Acute transient amnesia has not been previously associated with vertebral artery dissection. We describe two men with acute onset of dense anterograde amnesia and partial retrograde amnesia resulting from spontaneous vertebral artery dissection. Both amnesic syndromes completely resolved with conservative management. (Stroke 1988;19:98–101)

Spontaneous vertebral artery dissection is a not infrequent cause of posterior circulation ischemia.1,2 Although the amnesic state may result from posterior cerebral artery occlusion,3 memory impairment has not been associated with vertebral artery dissection. Head or neck pain associated with findings referable to the brainstem, cerebellum, and visual cortex are most characteristic.1,2,4

We report two patients with profound acute transient amnesia resulting from spontaneous vertebral artery dissection.

Subjects and Methods

Case 1

This 54-year-old right-handed man, well until approximately 20 days before admission to the hospital, experienced persistent severe pulsatile left occipital headaches. Soon after the onset of these headaches he was awakened from sleep by pulsatile tinnitus. He developed intermittent diplopia and was observed by others to have episodes of staring blankly for periods of up to 30 minutes. Ten days before admission, while looking upward with his neck extended, he became ashen gray and lost consciousness. Unconsciousness was followed by a 30-minute period of confusion. Neurologic examination 2 days later was normal, but his headaches persisted. He complained of transient visual difficulties and intermittent vertigo. Three days before admission, his wife noted memory deficits. He atypically misplaced objects and could not recall recent events. While asleep on the evening before admission he was awakened by urinary incontinence. He walked to his bathroom as if “drunk”; lost consciousness; and had transient eye fluttering, teeth clenching, and fist shaking. His medical history was significant only for peptic ulcer disease, well-controlled mild hyper tension, and mild hypercholesterolemia.

Examination showed a well-developed, well-nourished man who was very cooperative and pleasant but unable to recall his problems other than the presence of a persistent headache. He required constant reminders that he had been admitted to the hospital. Blood pressure was 155/100 mm Hg; the general examination was otherwise unremarkable. No murmurs or bruits were present. He was oriented to person and place but not to year or month. He was inappropriately jovial, but actions were otherwise appropriate and judgment was normal. Details concerning World War II and dates of his birth, marriage, and children’s births were stated correctly; however, he could not remember facts concerning his illness and failed to recognize them when being described by his wife. Only one of three items presented verbally could be recalled immediately; no items presented verbally or visually could be recalled at 5 minutes. He could not recall his physicians’ names or their instructions. Digit span was six forward and four reversed. He could calculate, identify fingers, and draw well. Speech was fluent, but reading comprehension of complex material was impaired. His pupils were 5 mm on the right, 4 mm on the left, and equally reactive. There was a mild weakness of the right inferior rectus. Gait was narrow-based and steady. There was diminished right arm swing and right arm posturing with toe walking. Extinction on the right to double simultaneous tactile stimuli was intermittently present. Neurologic examination was otherwise normal.

Serum chemistries, complete blood count, urine analysis, chest radiograph, and electrocardiogram were normal. Day of admission computed tomogram of the head both before and after administration of intravenous infusion contrast material was normal. Electroencephalogram on admission demonstrated occasional left temporal slow wave transients and sharp waves in the bilateral central/parietal/temporal derivations, especially on the left. On the second hospital day, arch and cerebral angiography was performed employing both conventional film screen and digital subtraction techniques (Figure 1). This demonstrated a long irregular narrowing of the left vertebral artery ending in occlusion at the level of the second cervical vertebra. The right vertebral artery was of small caliber.
but patent through the basilar artery. Both posterior communicating arteries failed to fill, suggesting possible absence or occlusion. Minimal different sites of atherosclerotic change were seen. Formal neuropsychological testing indicated a primary memory deficit involving both verbal and visual modalities. Nuclear magnetic resonance brain imaging obtained 3 weeks after admission demonstrated increased signal in the cerebellar hemispheres consistent with ischemic injury.

His hospital course was one of slow gradual improvement. Although he remains amnesic of events surrounding the 2 weeks before admission, his bedside memory testing otherwise returned to normal. After 8 months of clinical follow-up he has had no further episodes of altered consciousness.

Case 2

This 44-year-old right-handed man was well until he awoke one morning with vertigo, nausea, diplopia, ataxia, and headache. Over the next hour he developed paresthesias of his left face and extremities. When admitted to a local hospital he had nystagmus, mild weakness of his left arm and face, and poor memory. His symptoms resolved in 3 hours. An hour later his physicians found him totally amnesic for events of the past few days and unable to incorporate new data into his memory. A new, dense, right homonymous hemianopsia was noted. His amnesia improved over the following 3 weeks, at which time we first examined him. He was an attentive man who was lucid and an excellent historian. Vital signs and general examination were normal. His mental status demonstrated normal immediate, intermediate, and remote memory. There was a dense right homonymous hemianopsia and very mild flattening of the left nasolabial fold. The remainder of the neurologic examination was normal.

Studies obtained at his initial presentation demonstrated normal echocardiogram, electroencephalogram, visual and brainstem auditory evoked responses, carotid Doppler studies, and brain computed tomography determined the day of onset. Nuclear magnetic resonance brain imaging 4 days later demonstrated a large area of increased signal in the left temporal and occipital lobes. The signal aberration was consistent with ischemia in the left posterior cerebral
artery distribution (Figure 2). Arch and cerebral arteriography obtained on the third hospital day demonstrated dissection of the transdural segment of the left vertebral artery. Left posterior cerebral artery distribution vascular hyperperfusion was consistent with ischemic injury. There was minimal disseminated intimal irregularity of the atherosclerotic type.

This patient has functioned well at 10 months after onset of symptoms with only minimal complaints of limitation of short-term memory, not detectable clinically or on neuropsychometric testing.

**Discussion**

Vertebral artery dissection has become an increasingly recognized cause of stroke. Each of our two patients developed symptoms and signs characteristic

**Figure 2.** Cranial nuclear magnetic resonance imaging, 10-mm axial sections, spin-echo technique, 0.5-T superconducting unit, repetition time 3,000 msec, echo time 90 msec. Top: Section just rostral to tentorium, nearly at tentorial incisura. Flocular high signal about medial aspect of both middle and posterior portions of left temporal lobe as well as basal occipital lobe. Mass effect demonstrated as distortion of deep white matter. Left: Section 20 mm rostral to Top. Well-defined high signal nearly restricted to left occipital lobe. Right: Section 10 mm rostral to Left. Signal aberration is relatively restricted to left occipital lobe. Configuration and imaging characteristics on these images are most consistent with left posterior cerebral arterial distribution ischemic brain injury, believed to be secondary to dissection of transdural portion of left vertebral artery (not illustrated).
of vertebral artery dissection. These included head and neck pain, vertebro-basilar ischemia, and appropriate angiographic findings. In addition, each case acutely developed severe transient anterograde amnesia associated with a retrograde amnesia of lesser extent. To our knowledge amnesia has not been clearly associated with vertebral artery dissection.

That the amnesia in both cases gradually improved may suggest that only unilateral infarction occurred. In fact, nuclear magnetic resonance imaging in Case 2 delineated unilateral temporal and occipital ischemic injury consistent with his most prolonged symptoms of amnesia and hemianopsia. The nuclear magnetic resonance image in Case 1 showed cerebellar infarction but no abnormalities in thalamic or hippocampal structures related to memory function, despite severe amnesia. The electroencephalogram in Case 1, however, indicated bilateral temporal dysfunction similar to reports of patients with transient global amnesia.7

Despite a usually favorable prognosis following arterial dissection, the use of heparin anticoagulation, antiplatelet agents, or no specific treatment remains controversial.8 The potential benefits of anticoagulation in treating apparent embolic events that may arise from the site of dissection must be weighed against the risk of hemorrhagic complication or extension of the acute subintimal hematoma. Both of our patients improved with conservative management.

Acute amnesia should now be included as a potential symptom of vertebral artery dissection, and vertebral artery dissection should be considered in the differential diagnosis of acute amnesia especially if associated with headache, neck pain, or other signs of vertebrobasilar ischemia.

References


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J Laterra, S Gebarski and J C Sackellares

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