Isolated superior cerebellar artery infarction is rare, and the mechanism is often not readily apparent. We describe a patient with an isolated superior cerebellar artery infarction resulting from an ipsilateral vertebral artery dissection. Angiography demonstrated intraluminal clot in the superior cerebellar artery, suggesting artery-to-artery embolus as a mechanism of this uncommon stroke syndrome. 

**Case Report**

A 40-year-old man awoke one morning, turned, and abruptly experienced dizziness and double vision. He felt off-balance, noted left-sided weakness and clumsiness, and vomited. These symptoms were severe for 5-6 hours and then gradually resolved except for the left-sided weakness and clumsiness. Several days previous he had noted transient pain "between his ears" and periodic nausea. There was no history of neck manipulation, trauma, or pain. Medical history was unremarkable.

Findings of the general physical examination including cardiac auscultation were normal. Neurologic examination revealed anisocoria, with the left pupil 1 mm larger, and vertical diplopia that was worse on gaze to the right; pursuits were saccadic but there was no ocular dysmetria. Palatal myoclonus was present. A mild hypotonic left hemiparesis was present as was left-sided dysmetria and hyperreflexia; he fell to the left. Findings of the sensory examination were normal.

Results of hematologic and coagulation testing were normal, and there was no laboratory evidence of vasculitis. Electrocardiogram and chest x-ray were normal. On the day of admission, head CT revealed an early infarction in the medial left superior cerebellar hemisphere (Figure 1). Cerebral angiography demonstrated an irregular 70% narrowing of the left VA 1.5 cm long at the level of the atlas (Figure 2). Intraluminal clot was present in the left proximal ipsilateral SCA at its bifurcation along the anterolateral margin of the left mesencephalon (Figures 2 and 3). Echocardiography demonstrated minimal intermittent mitral valve prolapse. The patient was placed on intravenous heparin, discharged on warfarin sodium (Coumadin), and has gradually improved during 3 years of follow-up. Repeat cerebral angiography 1 year later demonstrated resolution of both the VA and the SCA lesions.

**Discussion**

SCA territory infarction is much less commonly diagnosed clinically, radiographically, and postmortem than the posterior inferior cerebellar artery (PICA) syndrome of Wallenberg. Although the SCA is the largest and most consistent branch of the vertebrobasilar tree, relatively few reports have documented isolated SCA infarcts and their mechanisms. Our patient had an angiographically documented distal extracranial VA dissection with ipsilateral SCA intraluminal clot. Clinical examination and CT supported an isolated SCA syndrome. Our patient had a mild hypotonic hemiparesis ipsilateral to the infarct. Limb weakness has been described with cerebellar lesions, although it is generally due to organizational and coordination deficits of muscle activity. Given the variability of supply of the SCA, it is conceivable that ischemia in the right brainstem may explain our patient’s weakness as hemiparesis is generally not a feature of the SCA syndrome.

An extensive search failed to reveal evidence of hypercoagulability, and minimal mitral valve pro-
lapse detected by echocardiography was probably not responsible for the irregular appearance of the VA on angiography. There was no clinical or angiographic evidence of a congenital or acquired diffuse systemic or cerebral arteriopathy.

Angiographic evidence of occlusion of the SCA with a corresponding clinical syndrome has only rarely been documented. Embolism isolated to the SCA is also distinctly unusual. Other specific etiologies for isolated SCA infarction include basilar artery atherothrombosis, cardiac embolism, dissection associated with fibromuscular dysplasia, and migraine associated with oral contraceptive and cigarette use.

Artery-to-artery embolism causing infarction in the vertebrobasilar circulation is becoming more commonly recognized as a specific mechanism of stroke. Embolus accompanying a basilar artery clot resulting in a right pontine infarction. The source of embolus was an occluded VA at the level of the second cervical vertebral body; cause of the VA occlusion was not readily apparent. Zimmerman et al reported a 7-year-old boy who developed a right cerebellar infarct after gymnastics and chiropractic treatments. Angiography revealed intraluminal clot in the distal basilar artery. Artery-to-artery embolus from a traumatic thrombosis of the left VA to the right SCA was postulated to explain his ataxia and right-sided cerebellar signs. The boy also had a right superior quadrantanopsia. In most angiographic studies of artery-to-artery embolism in the vertebrobasilar circulation, the local embolus was not documented by an intraluminal filling defect that subsequently cleared. This angiographic appearance in our case strongly supports embolism.

VA dissection is a well-known cause of vertebrobasilar stroke, generally within the PICA territory. However, Kase and colleagues reported the case of a 49-year-old man with mild headache and dysarthria, left hemiataxia, and normal power and sensation. Four days later, CT demonstrated an anteromedial left cerebellar infarct, and the next day angiography demonstrated a significant lesion of the left VA, suggestive of a dissection. A double SCA pattern was noted on the left. Proximal ste-
FIGURE 3. Selective vertebral angiogram in 40-year-old man, anteroposterior view. Intraluminal filling defect (arrow) documented at first major bifurcation of left superior cerebellar artery.

nosis of the upper SCA branch on the left (presumed to be partially recanalized embolism) was demonstrated. Slow improvement with mild residua occurred while the patient was receiving intravenous heparin. Katirji et al reported the case of a 57-year-old man with recurrent left occipitoparietal ischemia following heavy lifting. CT of his head was normal; angiography demonstrated a long area of arterial narrowing suggestive of a subintimal hematoma. Intraluminal thrombus attached both to a pedicle at the superior aspect of the subintimal dissection and in the left SCA was noted. Osteophytes were noted at C4-5 and at C5-6. Despite transient symptoms on anticoagulation, no further strokes occurred. Perez-Higuera et al recently reported the case of a 9-year-old boy who had transient recurrent episodes of loss of consciousness, myoclonus, and ocular deviation. He was noted to be somnolent with a right abducens palsy, general hypotonia, hyporeflexia, and gait ataxia. CT showed a large right cerebellar hemispheric infarction that led to obstructive hydrocephalus. Angiography demonstrated type I focal fibromuscular dysplasia of the left VA. An adjacent dissecting aneurysm was also present; either lesion may have caused distal embolism. Savoiardo et al published the CT scan of a patient with a SCA territory infarct in a single paravermian branch and contralateral hemispheral branch caused by embolism from an aneurysm of the ipsilateral VA at the C-2 level. Mas and colleagues could not demonstrate distal embolization from extracranial VA dissections in any of their 13 patients, even in those with early angiograms, and they found only two such reported cases in the literature. These authors surmised that distal embolization from extracranial VA dissection may be rare, but we believe that this stroke mechanism is probably underrecognized and not easily documented. VA dissection (due to minor trauma or chiropractic manipulation or spontaneous) has been reported to cause unilateral or bilateral cerebellar hemisphere infarction and should be considered in the differential diagnoses of all posterior circulation ischemia.

Our patient improved with anticoagulation, and follow-up angiography revealed resolution of the arterial lesions. The limited number of similar cases in the literature suggests that anticoagulation in this setting may reduce ischemic complications distal to the dissection.

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References


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