Surgical Correction and SPECT Imaging of Vertebrobasilar Insufficiency Due to Unilateral Vertebral Artery Stenosis

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**Background and Purpose:** Incapacitating vertebrobasilar insufficiency is generally associated with bilateral vertebral artery disease, whereas unilateral vertebral artery stenosis usually is clinically silent. Regional brain perfusion has not been part of the routine evaluation of patients with vertebrobasilar insufficiency. This report describes two patients who had isolated unilateral vertebral artery stenosis operatively corrected to eliminate their incapacitating vertebrobasilar insufficiency. Hindbrain hypoperfusion was identified preoperatively and evaluated postoperatively, then correlated with patient presentation and response to revascularization.

**Case Description:** Two patients with incapacitating vertebrobasilar insufficiency presented with isolated unilateral vertebral artery stenosis with patent, nonstenotic internal carotid arteries. Hindbrain hypoperfusion was demonstrated by iodine-123-iodoamphetamine single-photon emission computed tomography preoperatively and demonstrated significant improvement following vertebral-carotid reimplantation. The patients' symptoms resolved following revascularization.

**Conclusions:** Although unusual, unilateral vertebral artery stenosis can cause incapacitating vertebrobasilar insufficiency. These cases demonstrate the value of imaging with single-photon emission computed tomography to evaluate regional brain hypoperfusion and to evaluate objectively the results of therapy. (Stroke 1992;23:602–606)

**KEY WORDS** • vertebrobasilar insufficiency • tomography, emission computed

In the mid-1950s Hutchinson and Yates suggested that atheromas at the origin of the vertebral arteries caused ischemia of the brain stem and cerebellar and occipital lobes. Many patients are incompletely evaluated because the symptoms of vertebrobasilar insufficiency frequently are nondescript, and complaints of dizziness, vertigo, syncope, and ataxia are commonly attributed to the aging process.

Vertebral artery reconstruction is successful and functionally important for patients with vertebrobasilar insufficiency resulting from bilateral vertebral artery disease. It is accepted that one nondiseased vertebral artery is sufficient to maintain adequate circulation to the brain stem. Current teaching suggesting that unilateral vertebral artery stenosis is clinically unimportant results from the observations that 1) not all flow-reducing lesions of the vertebral arteries cause symptoms, 2) previous correction of unilateral vertebral artery stenosis failed to relieve symptoms, and 3) correction of stenotic carotid lesions relieved symptoms of vertebrobasilar insufficiency.

We describe two patients with incapacitating vertebrobasilar insufficiency who had unilateral vertebral artery stenosis as their only significant lesion. Both patients had patent internal carotid arteries and showed posterior circulation hypoperfusion with single-photon emission computed tomography (SPECT) using the cerebral blood flow agent iodine-123-iodoamphetamine (IMP). After reimplantation of the involved vertebral artery into the ipsilateral common carotid artery, significant symptomatic relief occurred, and objective evidence of reperfusion was demonstrated by normalization of the SPECT scan in each case. This report calls attention to the fact that isolated unilateral vertebral artery stenosis can cause severe cerebellar ischemia that can be corrected with vertebral artery reconstruction. The IMP SPECT imaging identified significant regional hypoperfusion, which assisted in establishing the diagnosis and offered an objective, quantitative method of evaluating the revascularization procedure.

**Case Reports**

We obtained preoperative and postoperative SPECT images after intravenous injection of 3 mCi iodine-123 monoamine-N-isopropyl-p-iodoamphetamine (IMP) (Spectamine Medi+Physics, Paramus, N.J.). The patients were kept supine with eyes open in a dimly lit room for 20 minutes after injection before imaging. To ensure similar positioning between studies, a laser positioning system was used to orient the patient's head at 15° parallel to the orbitomeatal line. Tomographic images were acquired using 360° rotation with 64 angular samples of 40 seconds each. This resulted in images of...
approximately 50K counts for reconstruction. The SPECT images were reconstructed in the transaxial, coronal, and sagittal planes. Images were acquired with a medium-energy 30° slanthole collimator to minimize the imaging distance. The rotational images were acquired using 128 projection angles over 360° rotation for 30 seconds using a 64 x 64 matrix. Reconstruction used commercially available SPECT software (Star-Cam, General Electric Corp., Milwaukee, Wis.) with a 9-point smooth and a ramp-Hanning filter with a 0.5 cycle per second cutoff frequency. Reconstructions were all 0.6 cm thick and oriented parallel to the orbitomeatal plane. Figure 1 shows the arteriogram demonstrating each subclavian artery and the proximal vertebral arteries of patient 1, and Figure 2 (top and bottom panels) demonstrates the SPECT images in the sagittal plane. The sagittal plane illustrates more clearly the relative hypoperfusion of the cerebellar hemispheres.

To compare preoperative and postoperative studies more objectively, we calculated a semiquantitative index of relative perfusion by obtaining the ratio of the average counts per pixel of the visually apparent areas of decreased perfusion to adjacent normal tissue. In both cases, manually defined regions of interest were applied to the same preoperative and postoperative areas.

In case 1, symmetrical regions of interest were drawn to obtain the average counts per pixel in both the right and left parieto-occipital cortices. The average counts per pixel in the distribution of the right and left middle cerebral arteries were then identified. A ratio of the parieto-occipital average counts per pixel, divided by the middle cerebral average counts per pixel, was calculated. (Regions of interest are demonstrated by boxes in Figure 2, top panel.)

In case 2, the average counts per pixel for each cerebellar hemisphere were obtained and divided by the average counts per pixel in the right and left middle cerebral artery territories.

Case 1

A 64-year-old woman with a 3-month history of incapacitating vertigo was admitted. The patient previously had undergone a coronary artery bypass for unstable angina and bilateral carotid endarterectomies for hemispheric symptoms of carotid artery disease. She had a history of depression.

Four-vessel angiography revealed bilaterally patent internal carotid arteries, a large left vertebral artery, and a stenosis at the origin of her small right vertebral artery (Figure 1). No abnormality of her intracranial circulation was identified. A computed tomographic scan of the head was normal. A dilemma existed similar to that in the second patient: the only lesion that could explain the patient's symptoms was the isolated stenosis at the origin of her relatively small right vertebral artery. An IMP SPECT scan was performed that showed symmetrically decreased perfusion of both frontal lobes with more marked hypoperfusion of both cerebellar hemispheres (Figure 2, top panel). Again, using an index of perfusion based on average counts per pixel in the cerebellar hemispheres compared with the middle cerebral artery distribution, this patient was found to have an 18% reduction in activity in the cerebellar hemispheres compared with the middle cerebral cortex territories.

A right vertebral-to-common carotid implantation was performed. Postoperatively, the patient was asymptomatic and able to walk normally. A postoperative arteriogram demonstrated a patent anastomosis, and a postoperative IMP SPECT scan showed increased perfusion to both cerebellar hemispheres (right greater than left) (Figure 2, bottom panel).

Postoperatively, the cerebellar perfusion index improved to only a 5% decrease. The patient recovered without complication and was discharged on the fourth postoperative day. The patient was asymptomatic at 1½ years after the operation, at which time she was lost to follow-up.

Case 2

A 71-year-old obese white woman presented with a 4-month history of progressively severe positional vertigo, diplopia, and blurred vision. Her symptoms became so severe that she was unable to function in the
upright position. Her past medical history included a previous aortic valve replacement and bilateral carotid endarterectomies. The patient had type II diabetes, was a controlled hypertensive, had type IV hyperlipidemia, and was a 52-pack-per-year cigarette smoker.

Physical examination and routine laboratory studies failed to demonstrate any unexpected abnormality. Four-vessel cerebral angiography demonstrated bilaterally patent internal carotid arteries with minimal plaque of the right internal carotid artery, tortuous vertebral arteries with a significant stenosis of the origin of the left vertebral artery, and no intracranial disease. A computed axial tomographic scan revealed a small infarct in the patient’s nondominant hemisphere, which was present before her first carotid endarterectomy. Because we believed that isolated unilateral vertebral artery stenosis should not cause incapacitating vertebrobasilar insufficiency, objective documentation of cerebellar hypoperfusion was necessary before any suggestion of operative correction.

An IMP SPECT study was performed to assess regional cerebral and cerebellar perfusion. This showed symmetrically decreased perfusion of both parieto-occipital hemispheres. In light of the patient’s incapacititating symptoms, the single vertebral artery stenosis, and objective documentation of posterior hypoperfusion, a
left vertebral–left common carotid artery reimplantation was performed. Postoperatively, the patient was completely relieved of her symptoms.

A postoperative angiogram showed the anastomosis to be widely patent with good distal vertebral flow. On an intracranial intra-arterial digital subtraction angiogram, both vertebral arteries joined to perfuse the basilar without evidence of asymmetry. A postoperative SPECT study revealed significantly increased posterior perfusion compared with the preoperative study. Using a comparison of the counts, the posterior circulation, which had an average 26% reduction in the relative perfusion index preoperatively, improved to only a 5% reduction postoperatively. The patient recovered quickly and was discharged with no symptoms on the fourth postoperative day. She had no further symptoms of vertebrobasilar insufficiency for the next 28 months, at which time she died of congestive heart failure.

**Discussion**

Patients suffering from bilateral vertebral artery disease who are symptomatic and without significant carotid disease should be offered direct vertebral artery reconstruction. If, however, the lesion is unilateral with a patent contralateral vertebral artery, conflicting opinions exist regarding the appropriateness of surgical intervention. Berguer and Bauer and Imparato have maintained that unilateral disease is not an indication for operative correction. Reul et al recognized the possibility of surgical correction of unilateral vertebral artery disease in a small subpopulation of patients. In that report, six patients with apparent isolated unilateral vertebral artery stenosis underwent vertebral resection. All patients with unilateral disease presumably improved; the only patients with no reported improvement were those with multiple-vessel disease.

These previous reports failed to evaluate regional brain perfusion preoperatively, most likely due to the lack of available techniques. If such studies had been available, perhaps revascularization procedures would have been recommended for other patients with unilateral disease who had objective evidence of end-organ ischemia.

As in selected cases previously reported, our patients received prompt relief from their incapacitating symptoms. After excluding cardiac arrhythmia and cervical carotid artery disease, the disabling symptoms experienced by these patients were attributed to vertebrobasilar insufficiency. Unilateral vertebral artery stenosis was the only angiographic abnormality in each of these cases. In light of the existing controversy, a diagnostic test such as SPECT perfusion imaging, which can demonstrate posterior hypoperfusion, becomes crucial to making appropriate recommendations for care.

The recent introduction of new radiopharmaceuticals and the development of tomographic (SPECT) nuclear medicine cameras have now made it possible to study regional cerebral blood flow on a routine basis. Amines are important clinical mediators of brain function. Labeled amines such as 123I-iodoamphetamine are lipophilic and will cross the blood–brain barrier with a high extraction efficiency during a single passage through the cerebral circulation. The uptake within the brain has been previously correlated with cerebral blood flow, and regional cerebral perfusion can be displayed using SPECT computerized reconstruction images (Figure 2). Such studies are particularly suited to evaluation of regional hypoperfusion and assessment of the results of cerebral revascularization procedures. The frontal lobe hypoperfusion observed in patient 1 is an interesting finding, especially in view of the lack of any angiographic demonstration of a vascular lesion to account for the decreased perfusion. The patient had a history of depression, which has been associated with such perfusion abnormalities using SPECT and positron emission tomographic imaging.

The decreased perfusion associated with the diseased vertebral arteries in these patients was clearly demonstrated preoperatively. This suggests that the cerebellar function in these patients is dependent on both vertebral arteries. The question arises of whether the absence of demonstrable angiographic abnormalities of the intracranial vasculature is equivalent to a physiologically intact or nondiseased circle of Willis. Our experience with these two patients would suggest that this is not the case. We expected that in the absence of angiographically demonstrable intracranial disease and a normal contralateral vertebral artery, adequate cerebellar perfusion would be achieved. It is evident that the nondiseased vertebral artery did not adequately perfuse the hindbrain in either of these cases; this was particularly surprising in case 1, in which a large “dominant” vertebral artery was not adequate for normal hindbrain perfusion. Transcranial Doppler evaluation of the collateral pathways of the intracranial circulation was not available during evaluation of these patients, but should be helpful in clarifying hemodynamic abnormalities of such patients currently and in the future.

This report draws attention to the fact that, although unusual, unilateral vertebral artery disease can cause incapacitating vertebrobasilar insufficiency. It also highlights the value of SPECT perfusion imaging by demonstrating cerebellar hypoperfusion preoperatively and correlating the symptomatic improvement postoperatively with objective improvement of cerebellar perfusion.

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**References**

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