

# AFTERIMAGES, JONATHAN WIRTSCHAFTER, EDITOR

## Value of Parasagittal Magnetic Resonance Images in Visualizing the Vascular Territories of the Cerebellum

CHIN-SANG CHUNG, MD, CLAUDIA J. CHAVES, MD, AND LOUIS R. CAPLAN, MD

*Department of Neurology, Tufts-New England Medical Center, Boston, Massachusetts*

**Abstract.** The outcomes of infarctions of the cerebellar arteries may range from self-limited neuro-ophthalmic symptoms to sudden death. Identification of the affected territory may assist in clinical management because the vascular syndromes, as well as vascular mechanisms of cerebellar infarcts, are different according to the involved cerebellar arteries. However, it is usually difficult to tell the exact vascular territories using only transaxial imagings. Magnetic resonance imaging is a good tool for obtaining parasagittal and coronal images of the cerebellum, which are better for showing the vascular territories. To illustrate this point we report a 61-year-old woman with bilateral cerebellar infarcts (right posterior inferior cerebellar artery and left superior cerebellar artery) who had vertigo, vomiting, headache, intermittent vertical diplopia, and difficulty walking. The clinical value of parasagittal imaging of the cerebellum in this case is discussed. (*Surv Ophthalmol* 39:399-402, 1995)

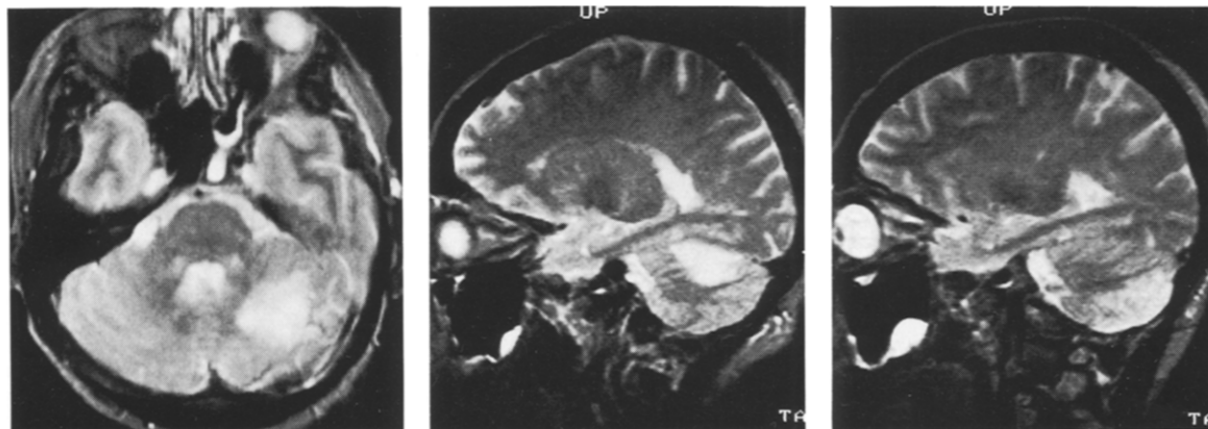
**Key words.** cerebellum • magnetic resonance imaging • parasagittal images • vascular territories

During the last decade advances in neuroimaging, particularly magnetic resonance imaging (MRI), have revolutionized *in vivo* study of the posterior fossa structures. Different from CT scan, which allowed only axial views, MRI has made it possible to obtain three-dimensional images of the brainstem and cerebellum using transaxial, parasagittal and coronal imaging.<sup>7</sup> We report a patient with bilateral cerebellar infarcts to illustrate the value of parasagittal MRI images in evaluating the vascular territories of the cerebellum.

### Case Report

A 61-year-old white woman, a past cigarette smoker, awakened at 3 a.m. 10 days before admission to the New England Medical Center Hospitals (NEMCH), Boston, MA, when she had

sudden severe vertigo and vomiting. She had difficulty walking, veering to the right. She had an occipital headache and intermittent vertical diplopia. The symptoms were maximal at onset and gradually improved over the next week. There were no hearing disturbances, no tinnitus; no history of vertigo, headache, syncope, tinnitus, amaurosis fugax, or other neurologic symptoms. She was referred to NEMCH because of bilateral cerebellar infarcts on CT scan and for further evaluation and management. She had no history of hypertension, diabetes mellitus, or heart diseases. On examination her blood pressure was 130/70. There was no ocular or carotid bruit. She was alert, oriented, and aware of current events. Her speech was normal. Mental status was normal. Cranial nerve examination revealed normal optic nerveheads and visual



*Fig. 1. A:* A transaxial T<sub>2</sub>-weighted MRI section through the midpons shows the high signal intensity in the central portion of the left cerebellar hemisphere. The plane of the section does not permit certain differentiation of the territory of the left superior cerebellar artery which is involved from the territory of the left cerebellar artery which is uninvolved. *B:* A parasagittal T<sub>2</sub>-weighted MRI section through the left cerebellar hemisphere (2 cm from the midline) shows the high signal intensity lesion in the territory of the superior cerebellar artery. *C:* A parasagittal T<sub>2</sub>-weighted MRI section through the right cerebellar hemisphere (2 cm from the midline) shows the high signal intensity lesion in the territory of the posterior inferior cerebellar artery.

fields bilaterally and there was no visual neglect. Extraocular movements were normal, but horizontal nystagmus was noted on horizontal gaze bilaterally. There was no vertical nystagmus. Facial sensation was normal and smile was symmetric. The soft palate elevated symmetrically. Sternocleidomastoid and trapezius muscles had good strength bilaterally. Tongue protruded in the midline. Motor power, tone, and bulk were normal and symmetric. Sensory system was intact to light touch, pinprick, temperature, vibration, and position throughout. Deep tendon reflexes were normal and symmetric with downgoing toes bilaterally. Cerebellar examination showed slight right limb incoordination with rapid alternating movements. Finger-to-nose and heel-to-shin tests were intact. Gait was wide-based and ataxic with veering to the right (lateropulsion). She was unable to walk unassisted or without learning on something.

All admission blood tests were within normal ranges. Electrocardiogram showed normal sinus rhythm and slight ST depression in leads II and III but no significant defects. An echocardiogram showed normal left ventricular size and ejection fraction of 50%. The valves were normal. Mild aortic insufficiency was noted. Brain computerized tomographic (CT) scanning showed bilateral cerebellar infarcts. MRI scanning revealed an infarct in the left superior cerebellar

artery (SCA) territory (Figs. 1A and B) and another in the right posterior inferior cerebellar artery (PICA) territory (Fig. 1C). The patient had ultrasound studies of the neck. In the right internal carotid artery (ICA) there was irregular calcified plaque on Duplex scan and mild to moderate stenosis on Doppler study. Left ICA showed 55–65% stenosis. The right vertebral artery had markedly diminished flow and the left vertebral artery was normal. Transcranial Doppler study was done on the vertebrobasilar system. Mean velocities were slightly diminished in the basilar artery and left vertebral artery and normal in the right vertebral artery. Angiography of the posterior circulation revealed a tight stenosis at the origin of the right vertebral artery. The right PICA was not well seen.

Neurologically the patient improved gradually in terms of her ataxia. She was able to ambulate very well with the use of a cane. It was felt that an embolism from a tight stenosis at the origin of the right vertebral artery was the etiology of her bilateral cerebellar strokes. She was placed on Coumadin and was also encouraged to stop smoking. She was ambulatory with the help of a cane at the time of discharge.

## Discussion

The clinical syndromes are different according

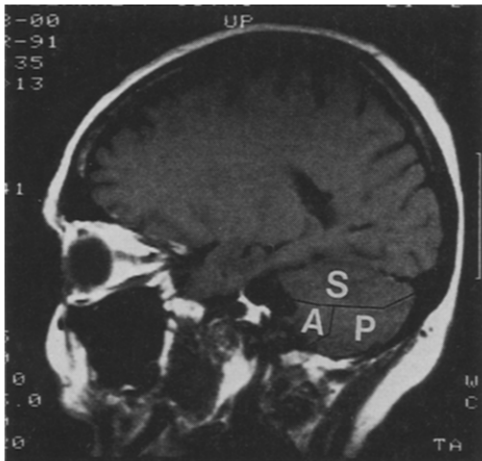


Fig. 2. Diagrammed vascular territories of the cerebellum on the parasagittal section of the T<sub>1</sub>-weighted MRI scan (2 cm from the midline). A: anterior inferior cerebellar artery. S: superior cerebellar artery. P: posterior inferior cerebellar artery.

to the vascular territory of cerebellar infarcts.<sup>1</sup> Cerebellar infarcts caused by the occlusion of the SCA are commonly associated with dysarthria, headache, dizziness, vomiting, ataxia, dysmetria, axial lateropulsion, and delayed coma. Superior cerebellar infarcts can cause the rapid onset of cerebellar swelling or hematoma producing compression of the brainstem, hydrocephalus, upward herniation of the cerebellum compressing the tectum at the diencephalic/mesencephalic junction, thus impairing upward gaze, or downward herniation of the cerebellar tonsils. Acute neurosurgical intervention to treat the hydrocephalus or evacuate the clot can be life-saving. In comparison to the other cerebellar arteries SCA infarction has the lowest incidence of specific neuro-ophthalmological findings.

By comparison, AICA infarction has the most frequent and specific pattern of neuro-ophthalmological findings and perhaps the fewest cerebellar hemisphere symptoms unless the ipsilateral PICA happens to be hypoplastic. Occlusion of the anterior inferior cerebellar artery (AICA) usually affects the lateral area of the lower pons, causing ipsilateral cranial nerve dysfunctions (trigeminal, abducens, facial and vestibular cranial nerves), ipsilateral Horner's syndrome, and contralateral pain and temperature sensory loss of the body regions below the neck. When the pons is not involved, the AICA infarct causes a pure vestibular syndrome.

Caudal cerebellar, or PICA territory, infarcts cause vertigo, headache, vomiting, ataxia, and

delayed coma. Occlusion of the medial branch of the PICA occasionally involves the dorsolateral medullary area and causes a modified Wallenberg syndrome with symptoms such as lateropulsion of saccadic eye movements, oblique saccades on attempted vertical gaze, impaired smooth pursuit, nystagmus, eyelid nystagmus, and skew deviation.

Stroke mechanisms of cerebellar infarcts also differ according to the vascular territories.<sup>2,4-6</sup> SCA and PICA territory infarctions are most frequently caused by embolic mechanisms, either intraarterial (artery to artery) or cardiac. AICA territory infarcts are usually caused by *in situ* thrombosis of the atherosclerotic intracranial vessels.<sup>2,3</sup> Thus, precise localization of the vascular territory is now one of the essential steps in evaluation of patients with cerebellar infarction. We always refer to the parasagittal images to confirm the exact vascular territories, which vary according to the angle of scanning on the conventional axial scans. It sometimes is difficult to tell the exact vascular territory if only the axial scan is available, as shown in Fig. 1A. The vascular distribution of lesions is often more apparent on images obtained in the parasagittal or coronal planes. For imaging of patients with suspected cerebellar infarcts, we always obtain parasagittal and coronal views as well as axial images to identify the vascular territory. Parasagittal sections are better for the vertical distribution (superior versus inferior cerebellar arteries) of the vascular territory of cerebellar infarction and coronal sections are better for visualizing the medial and lateral branches of the SCA and PICA. T<sub>2</sub>-weighted images are particularly helpful in identifying the affected vascular territories (Figs. 1B and C).

On the parasagittal plane, the SCA territory comprises the superior aspect of the cerebellar hemisphere, the ipsilateral superior vermis, and the largest part of the deep white matter, including most or all of the dentate nucleus (Figs. 1B and 2).<sup>8,10</sup> The typical PICA territory includes the entire inferoposterior surface of the cerebellar hemisphere and the ipsilateral part of the inferior vermis. The posterior extent of the PICA distribution is the great horizontal fissure (or slightly above it in the semilunar lobule) near the posterolateral margin of the cerebellum (Figs. 1C and 2).<sup>9</sup> The penetrating branches of PICA reach only the inferior part of the deep cerebellar white matter, most of which is supplied by the SCA. The course and vascular supply of the AICA is quite variable,<sup>9,11</sup> but it usually supplies

the lateroinferior part of the pons, the middle cerebellar peduncle, and the floccular region. The AICA also supplies the whole anterior surface of the cerebellar hemisphere.

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Corresponding authors: Chin-Sang Chung, MD, Neurology, Chungnam National Univ. Hospital, 640 Daesa-dong, Joong-ku, Taejeon, Korea 301-040, or Louis R. Caplan, MD, Tufts-England Medical Center, 750 Washington St., Boston, MA 02111.