

Case Reports

Acute Bilateral Cerebellar Infarction in the Territory of the Medial Branches of Posterior Inferior Cerebellar Arteries

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Background We describe the first clinoradiological report of acute bilateral cerebellar infarction confined to the territory of the medial branches of the posterior inferior cerebellar arteries.

Case Description A 65-year-old man with atrial fibrillation and hypertension had sudden onset of vertigo, followed by brief loss of consciousness. Three days later a cranial computed tomographic scan showed acute hydrocephalus and low-density areas in the cerebellar vermis on both sides. On transfer the patient showed mild dysarthria, dysequilibrium with retropulsion, symmetrical bilateral horizontal gaze-evoked nystagmus on lateral gaze, and marked gait ataxia without brain stem signs, followed by marked vertigo that was induced by motion. Cranial magnetic resonance imaging revealed abnormalities consistent with fairly symmetrical bilateral cerebellar hemorrhagic infarction that was confined to the territory of the medial branches of the posterior inferior

cerebellar arteries, in addition to minimal high-intensity areas in the pons on T₂-weighted images. The patient improved with conservative therapy, including intravenous administration of glycerol.

Conclusions We speculate that our patient likely had initial transient occlusion of the right vertebral artery at the origin of the right posterior inferior cerebellar artery, which probably gave rise to the bilateral medial branches of posterior inferior cerebellar arteries. This caused infarction in the territory of the medial branches on both sides without remaining brain stem signs. Such an unusual pattern of cerebellar infarction accompanied by acute hydrocephalus posed a diagnostic challenge at the time of transfer to our care, and correct diagnosis was facilitated by cranial magnetic resonance imaging. (*Stroke*. 1994;25:686-688.)

Key Words • cerebellar infarction • hemorrhage • hydrocephalus • magnetic resonance imaging

Cerebellar infarction is a common autopsy finding, the frequency of which varies from 1.1% to 4.2% in autopsy series.¹ The majority of infarcts occur in the territories of the posterior inferior cerebellar artery (PICA) and superior cerebellar artery.¹ Regarding PICA infarct, its clinical spectrum¹ shows the following patterns: (1) an asymptomatic form; (2) Wallenberg's syndrome caused by the involvement of the lateral medulla oblongata; (3) an infarct in the PICA territory distal to its medullary branches,² including the medial branch of the PICA,³ causing vertigo with or without ipsilateral axial lateropulsion and other types of cerebellar ataxia; and (4) a massive, often fatal cerebellar infarct with brain stem compression and acute hydrocephalus,^{2,4-7} which may require neurosurgical intervention to relieve increased intracranial pressure. Among these PICA infarcts, only a few clinicopathological^{2,7} and clinoradiological³ studies of infarcts in the PICA territory distal to its medullary branches have been reported, and patients with bilateral cerebellar infarcts primarily confined to the territory of the medial branches of both PICAs have not been previously described. Further, cerebellar infarcts with such an unusual distribution of lesions and acute

hydrocephalus may cause diagnostic problems, and this may delay appropriate management in those patients without prompt recognition of the infarcts.⁶ We describe a clinoradiological study of a patient with such bilateral cerebellar infarcts followed by acute hydrocephalus, the diagnosis of which was facilitated by magnetic resonance imaging (MRI) of the brain.

Case Report

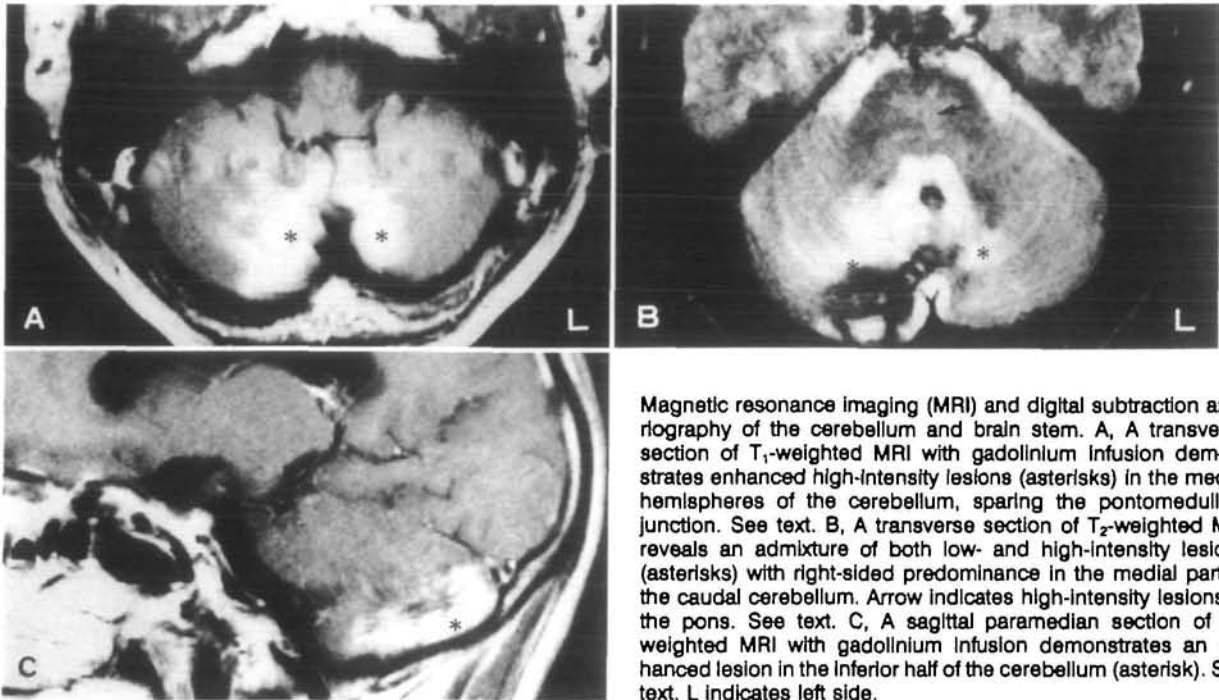
A 65-year-old man with atrial fibrillation, long-standing hypertension, and a previous left parietal cerebral infarct had sudden onset of rotatory vertigo on February 17, 1992, while he was watering his garden, followed by an inability to keep standing and then loss of consciousness. On admission to a local hospital the same day, he was disoriented in regard to time, place, and person but became mentally alert the next day. A cranial computed tomographic scan (CT) showed a moderate-sized low-density area in the left parietal cortex and subcortical white matter, consistent with an old infarct. Three days later he had decreased level of consciousness, which varied between disorientation and moderate lethargy. On February 24 a cranial CT scan showed moderate to marked dilatation of the lateral and third ventricles as well as low-density areas in the cerebellar vermis on both sides. The patient was transferred to our care on February 25 because of the acute hydrocephalus. Family history was noncontributory.

The pertinent findings on general physical examination consisted of blood pressure of 166/90 mm Hg, irregular pulse rate of 70 beats per minute, and a

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Magnetic resonance imaging (MRI) and digital subtraction arteriography of the cerebellum and brain stem. A, A transverse section of T₁-weighted MRI with gadolinium infusion demonstrates enhanced high-intensity lesions (asterisks) in the medial hemispheres of the cerebellum, sparing the pontomedullary junction. See text. B, A transverse section of T₂-weighted MRI reveals an admixture of both low- and high-intensity lesions (asterisks) with right-sided predominance in the medial part of the caudal cerebellum. Arrow indicates high-intensity lesions in the pons. See text. C, A sagittal paramedian section of T₁-weighted MRI with gadolinium infusion demonstrates an enhanced lesion in the inferior half of the cerebellum (asterisk). See text. L indicates left side.

diastolic murmur with opening snap at the apex, consistent with mitral stenosis. Neurologically the patient was awake but was mentally slow, disoriented in time, and mildly dysarthric. Extraocular movements were full, and symmetrical bilateral horizontal gaze-evoked nystagmus on lateral gaze was observed without diplopia. Dysdiadochokinesis was mild in the right hand and moderate in the left hand. Muscle strength, deep tendon reflexes, and sensation were preserved. Babinski's sign was not present. Gait tested 7 days after admission revealed that the patient was unable to keep standing with his feet together, instead falling backward. He walked with feet 15 cm apart and was unable to perform a tandem gait.

Laboratory data on admission showed normal values of blood cell count, blood chemistry, and urine, except for hemoglobin of 18.3 g/dL, white blood cell count of 12 300/mm³, C-reactive protein of 2.7 mg/dL, and serum lactic dehydrogenase of 641 IU/L. An electrocardiogram revealed atrial fibrillation. Chest roentgenogram demonstrated moderate cardiomegaly, and an echocardiogram revealed a markedly dilated left atrium without thrombi and moderate mitral stenosis. A cranial CT scan on March 4 demonstrated the development of high-density areas in the vermis, which were mixed with the preexisting low-density areas. Transverse sections of cranial MRI taken on March 11 revealed abnormalities consistent with hemorrhagic infarction¹ in the vermis and the adjacent areas in the hemispheres of the cerebellum on both sides (Figure, panels A and B), a distribution consistent with the territory of the medial branches of the PICAs^{1,3} as well as ill-defined minimal high-intensity lesions in the pons on T₂-weighted images (Figure, panel B). Sagittal sections of MRI confirmed that the abnormalities were confined to the bilateral PICA territory (Figure, panel C).^{1,8} After this MRI, digital subtraction arteriography (DSA) was performed on March 23. The frontal views of the DSA via transfemoral approach with contrast medium injected into

the aorta showed that the basilar artery and both vertebral arteries and the right PICA were all visualized. The PICA, which was well developed, arose from the right vertebral artery, and its medial branch appeared to cross the midline and extend to the left side of the cerebellum. Although the left PICA was not visualized in the frontal views, selective left vertebral arteriography by DSA demonstrated that the left PICA arose from the left vertebral artery and was hypoplastic. In addition, the lower half of the cerebellum lacked vascularity in the arterial phase, and the medial branch of the left PICA was not visualized, whereas the lateral branch of the left PICA was demonstrated. However, it was not possible to identify whether the medial branch of the left PICA originated from the contralateral right PICA because lateral views were not taken when contrast medium was injected into the aorta. The vertebral arteries showed mild stenosis, but the sites of their origin from the subclavian arteries appeared normal, although the latter arteries showed mild irregularities in their walls. The common carotid arteries and their bifurcations appeared normal.

After admission the hydrocephalus improved with intravenous administration of glycerol. The patient became able to ambulate with a markedly wide-based gait, complicated by frequent episodes of nausea, vomiting, and vertigo associated with movement of his body, and both horizontal and rotatory gaze-evoked nystagmus on lateral gaze to the right side. He was discharged 7 weeks after admission with a mildly wide-based gait and without other signs.

Discussion

Cranial MRI demonstrated that the bilateral cerebellar infarct observed in our patient was confined to the territory of the medial branches of the bilateral PICAs.^{1,3} The neurological symptomatology of our patient was also consistent with involvement of this PICA

territory, which contains the vestibulocerebellum, consisting of vertigo, dysequilibrium with retropulsion, horizontal gaze-evoked nystagmus, gait ataxia, and vertigo induced by motion.^{1,3} Although several patients with infarction in the bilateral PICA territory^{7,9-12} have been described, no details of their neurological histories and abnormalities, histopathological findings, or both were given, so that it is unclear whether these patients developed PICA infarction on both sides simultaneously or only on one side at one time. In addition, all of the cerebellar infarcts reported to date that were confined to the territory of the medial branches of the PICA were unilateral.³ Thus, the occurrence of cerebellar infarcts confined to this arterial territory on both sides as observed in our patient is unusual and has not been well documented previously.

Regarding the pathogenesis of this unusual cerebellar infarct, several possibilities were considered, as follows: (1) both PICAs arose from the basilar artery; (2) both medial branches of the PICAs arose from the PICA on one side; (3) two emboli occurred in the PICAs, one to each side; and (4) pressure effects caused by a large PICA infarct on one side compressed arteries in the cerebellar cistern between the two sides, inducing a smaller infarct on the other side. Based on the DSA findings of our patient, the first possibility was excluded, whereas the second possibility appeared most likely, although we could not prove this PICA variation because lateral views were not taken when the contrast medium was injected into the aorta. Regarding the third possibility, two emboli to the PICAs must have occurred almost simultaneously in our patient to produce hydrocephalus, because an infarct in the territory of the medial branches of the PICA on one side is usually benign and does not cause hydrocephalus.³ We therefore considered this possibility to be probably less likely, although we could not exclude it. The fourth possibility appeared unlikely because of the symmetry of the infarct observed in our patient. The initial occlusion of the PICA was probably caused by an embolus because the cerebellar infarction was hemorrhagic and DSA revealed that the vertebrobasilar arteries and both PICAs were patent. We speculated that the most likely event that occurred in our patient was the initial occlusion of the right vertebral artery by an embolus, which caused infarction in the territory of bilateral medial branches supplied by the PICA on the right side and later also caused basilar artery ischemia after the embolus became fragmented. It is not clear whether the embolus originated from an artery or from the heart. Both possibilities are likely because this patient had several risk factors for arteriosclerosis as well as for cardiac embolism, such as atrial fibrillation and a previous moderate-sized infarct in the parietal lobe.

Our patient did not have brain stem signs on transfer to our care, although he showed involvement of the cerebellum in the bilateral PICA territory. This unusual distribution and presentation of lesions as a result of vertebrobasilar ischemia initially made us consider the diagnosis of vascular malformation or brain tumor rather than infarct. However, both horizontal and sagittal sections of the cerebellum by MRI clearly demonstrated that the lesions were primarily confined to the territory of the medial branches of both PICAs. This led us to consider that infarction was a more likely possibility, which was also consistent with the sudden onset of symptoms and the gradual improvement of both the hydrocephalus and neurological abnormalities in this patient.

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