Clinico-Topographic Correlation of Small Vertebrobasilar Infarct Using Magnetic Resonance Imaging

J. Bogoousslavsky, M.D.,* † A.J. Fox, M.D.,† H.J.M. Barnett, M.D.,* V.C. Hachinski, M.D.,*
S. Vinitski, Ph.D.,† and L.S. Carey, M.D.†

SUMMARY Neurological, CT and high-field (1.5T) magnetic resonance imaging (MRI) data were correlated in 6 patients previously clinically diagnosed as having suffered small vertebrobasilar infarct. MRI demonstrated infarcts in areas where CT was nearly always negative. MRI allowed very precise clinico-topographic correlations and appears to be the preferred imaging technique in vertebrobasilar infarcts.

Stroke Vol 17, No 5, 1986

WHEN PATIENTS WITH VERTEBROBASILAR STROKES make a reasonable recovery, no neuropathological verification is available. In such instances, clinico-topographic correlation cannot usually be made, because CT evaluation of small brainstem lesions is usually poor or negative. Previous studies1,2 have shown that magnetic resonance imaging (MRI) may be better than CT in evaluating posterior fossa lesions, because of the lack of streak artifacts and because of better contrast discrimination. Vertebrobasilar infarcts have been demonstrated by using low-field magnets,3 with a rather poor contrast resolution, rendering precise clinico-topographic correlation impossible. We present high-field MRI (1.5 Tesla) studies of 6 patients with small brainstem or cerebellar infarcts, with a precise topographic analysis.

Methods and Patients

Six patients with non-acute (1–18 months) brainstem (in 5) and cerebellar (in 1) infarcts, with complete or good recovery, have had their lesions demonstrated using a 1.5 Tesla GE MR prototype scanner. Single or multislice partial saturation recovery (PS) (TR in range of 300–600 ms; TE = 20 ms) scans were done at 5 mm intervals from the medulla to the midbrain, in axial projections. For the region(s) of interest, single slice multiple spin-echo (MSE) sequences (TR = 2s, TE = 25/50/75/100 ms) were also obtained. PS is a T1 weighted image in which areas with prolonged T1 produce a low signal. Third and fourth echoes of MSE are T2 dependent, thus areas with prolonged T2 will be characterized by high signal intensity. MRI signal also depends on proton density, flow and experimental parameters. Sagittal and coronal projections were also performed selectively. A 128 × 256 matrix was used in most instances. Sagittal and coronal projections were also performed selectively. A 128 × 256 matrix was used in most instances. Five patients had a CT study (GE 8800 CT scanner) during the acute phase and at the time of MRI study (after 1–18 months, mean interval: 13 months). The aim of this study was to compare the neurological, CT and MRI data in order to try to establish precise clinico-topographic correlations.

Case 1

Lateral Medullary Infarct (classical type)

A 50-year-old woman with common migraine suffered recurrent episodes of rotatory vertigo associated with gait instability. Seven months later, rotatory vertigo and ataxia recurred, with transient bright spots and persisting numbness of the right upper and lower lips and hoarseness of the voice. On admission, she was alert and complained of diplopia as well as of oblique tilt of the visual images. Skew deviation was present, with the right eye lower than the left eye. A primary position counterclockwise (when looking at the patient) rotatory nystagmus was present and increased on rightward gaze. The right pupil was smaller than the left, with symmetrical reaction to light, and a slight right-sided piosis was observed. A mild right-sided inferior facial paresis was present and the tongue slightly deviated to the right on protrusion. Pinprick and temperature sensation was decreased on the right side of the face, with a diminished corneal reflex. Moderate ataxia and hypermetria was noted in the right limbs, with a questionable slight weakness of the right leg. Decreased pinprick and temperature sensation was present in the left arm and leg. A CT scan did not show any abnormality. Cerebral angiography showed occlusion of the right vertebral artery at the level of the PICA. The patient progressively improved. Seventeen months later, she was still complaining of oscillopsia, related to persisting counterclockwise rotatory primary position nystagmus, which increased during lateral gaze. On extreme rightward gaze, a monocular down-beating nystagmus of the right eye was elicited. A conjugate deviation of the eyes to the right was present in the dark (Cogan's phenomenon). No Horner syndrome or facial sensation abnormality was noted. Mild dysmetria persisted in the right leg. The gait was broad-based with a tendency to sway to the right. Pain and temperature appreciation was decreased on the left side of the body, with facial sparing. MRI scan (fig. 1b, 1c) showed a focal area of prolonged T1 and T2 in
FIGURE 1. Patient 1. (A — top left) CT: inconclusive image at the medullary level. (B — top right) MRI (PS: TR 300): focal area of prolonged T1 in the right lateral part of the medulla (arrow). (C — bottom left) MRI (SE: TR 2, TE 75): focal area of prolonged T2 in the same area (arrow).

the right lateral aspect of the medulla, without any other abnormality. CT scan (fig. 1a) was inconclusive, because of streak artifacts.

Case 2
Lateral Medullary Infarct (Ventral Type)

A 64-year-old hypertensive woman suddenly suffered a right frontal headache with veering to the right when walking. Over the following hours, hoarseness of voice, mild drooping of the right eyelid and decreased sensation of temperature on the left side of the body developed. On neurological examination, she was alert and had normal ocular movements. She had a moderate ptosis of the right eyelid with a reactive miosis on the same side. Pinprick sensation was reduced in the right forehead without clear asymmetry of the corneal reflex. Her voice was hoarse, but without demonstrable palatal or pharyngeal weakness. Motor function of the limbs was normal, without demonstrable dysmetria, but tandem gait was poor, with swaying to the left. Light touch, pinprick and temperature sensations were decreased on the left side of the body, with sparing of the face. The patient progressively im-
FIGURE 2. Patient 2. (A — top left) CT: inconclusive image at the medullary level. (B — top right) MRI (PS·TR 300): focal area of prolonged T1 in the ventral part of the lateral medulla on the right side (arrow). (C — bottom left) MRI (SE·TR 2, TE 100): focal area of prolonged T2 in the same area (arrow).

proved but six months later, she started complaining of episodic burning jabs in the eye and forehead on the right side, lasting a few seconds without triggering events. These episodes spontaneously disappeared over a few weeks, but the patient described a feeling of hyperesthesia in the left arm and leg, without spontaneous pain. On examination, fourteen months after the stroke, a miosis and a mild right-sided palpebral ptosis without were still present. Light touch sensation was decreased in the right forehead and on the left side of the body with sparing of the other modalities of sensation. The gait was normal. MRI scan (fig. 2b, 2c) showed a small area of prolonged T1 and T2 in the intermediate part of the lateral medulla on the right side, probably not larger than 3 mm. in cross sectional diameter. Bony artifacts made CT scan images (fig. 2a) inconclusive at this level.

Case 3
Medial Medullary Infarct (ventral type)
A 51-year-old hypertensive man suddenly experienced lightheadedness, followed a few hours later by
left-sided weakness, vomiting and somnolence. On admission the same day, he was drowsy and had an irregular (ataxic) respiration. He showed spontaneous rotatory nystagmus, with a horizontal component on lateral gaze, more marked to the right than to the left. He had severe dysarthria and this tongue markedly deviated to the right on attempted protrusion. The remainder of the cranial nerves were normal. He had left flaccid hemiplegia sparing the face and decrease of vibratory and postural sensation in the left limbs. The tendon reflexes were decreased on the left side and a left Babinski’s sign was present. A CT scan performed the same day showed only mild atrophy. A DIVA did not show any conspicuous abnormality but was of poor quality because of lack of cooperation. Over the following two weeks, the spontaneous nystagmus disappeared and the patient progressively developed spasticity and increased tendon reflexes on the left side. One year later, he still showed horizonto-rotatory nystagmus on lateral gaze, predominating during rightward gaze. Dysarthria and swallowing disturbances were present without evident palatal or pharyngeal weakness. The tongue was atrophic on the right side and deviated to the right on protrusion. Left-sided spastic hemiparesis persisted but the patient was able to walk with a cane. Sensation was no longer impaired. MRI scan (fig. 3b, 3c) showed a focal area of prolonged T1 and T2 in the right medial part of the ventral aspect of the medulla. In addition, a small round area of increased signal in both T1 and T2 weighted images (shortened T1 and prolonged T2) situated just below the ventral medulla suggested occlusion of the right vertebral artery. CT (fig. 3a) was unable to demonstrate the medial medullary infarct, because of bony artifacts.

Case 4
Paramedian Infarct of the Tegmentum Pontis (sixth and seventh nerve palsy)
A 67-year-old hypertensive man with a prior history of coronary bypass one year before suddenly experienced nausea, right facial numbness, slurred speech and tendency to deviate to the left, which improved over the following hours. One year later, he was admitted after he suffered bifrontal headache, blurred vision and ataxia. Oculomotor examination showed esotropia of the right eye with a marked paresis of abduction in this eye. The horizontal movements of the left eye were normal, without nystagmus. A right superior and inferior facial paresis was present. The remainder of the cranial nerves were normal (taste was not tested). Motor strength, tone and sensation were normal and symmetrical. Tendon reflexes were slightly increased on the left, without Babinski’s sign. No frank dysmetria of the limbs was observed, but the tandem gait showed swaying to the right. A CT scan showed cerebral atrophy without focal abnormalities. Fourteen months later, neurological examination only showed persistence of a tendency to deviate to the right on tandem gait, with normal ocular movements and facial contraction. Taste was unimpaired. MRI scan (fig. 4b, 4c, 4d) showed a focal area of prolonged T1 and T2 on the right side of the tegmental region of the pons. No evidence of brainstem infarct could be demonstrated on CT (fig. 4a).

Case 5
Upper Basal Pontis Infarct (ataxic hemiparesis)
A 71-year-old man with hypertension, chronic atrial fibrillation and intermittent claudication was admitted because of confusion, dizziness and right-sided weakness. On examination, the patient was oriented in time and place, but showed moderate amnesic difficulties. A right-sided hemiparesis with inferior facial involvement was present, with increased tendon reflexes and bilateral Babinski’s sign. Moderate incoordination with dysmetria and hypermetria was present as well in the right arm and leg. Ocular movements, the lower cranial nerves and sensation were normal. The gait was moderately impaired by the right-sided weakness and ataxia. Over the following two weeks, marked improvement was noted, and only slight residual right-sided weakness remained present. A CT scan showed multiple lucencies suggesting infarcts involving the white matter of the right frontal lobe, the left internal capsule and the left side of the pons (fig. 5a). MRI scan (fig. 5b, 5c, 5d) showed a focal area of prolonged T1 and T2 in the left paramedian part of the rostral pons, and confirmed the supratentorial infarcts seen on CT.

Case 6
Hemispheric Cerebellar Infarct (downbeat nystagmus)
A 48-year-old hypertensive man who smoked, experienced recurrent episodes of gait staggering to the left, lasting a few minutes. Eight months later he had a transient episode of blurred vision and dysarthria, followed two months later by a transient clumsiness of the left arm and leg. He was referred to us one month later because he was complaining of persisting unsteadiness and blurred vision. On examination, a primary position downbeat nystagmus was present, with increase on lateral gaze. The remainder of the cranial nerves were normal but mild dysmetria and hypermetria of the left arm and leg were noted, with unsteady tandem gait and a tendency to deviate to the left. Motor strength, reflexes and sensation were normal. A CT scan was normal. Two months later, spontaneous downbeat nystagmus was still present, and mild left-sided incoordination persisted. Eighteen months after the stroke, neurological examination showed microsaccadic smooth pursuit ocular movements in both horizontal directions. On extreme leftward gaze, a down-beating nystagmus was elicited, but no downbeat jerks were noted in the primary position of gaze. A very mild dysmetria of the left arm was still present, but the gait was normal. MRI scan (fig. 6b, 6c) showed a focal area of prolonged T1 and shortened T2 in the left cerebellar hemisphere close to the midline, without associated brainstem lesion. CT showed a questionable lucency in this area (fig. 6a).
FIGURE 3.  Patient 3. (A — top left) CT: inconclusive image at the medullary level. (B — top right) MRI (PS:TR 470): focal area of prolonged T1 in the ventral part of the medullar medulla on the right side (arrow). (C — bottom left) MRI (SE:TR 2, TE 100): focal area of prolonged T2 in the same area (arrow).

Discussion

This study of 6 patients with small brainstem or cerebellar infarcts shows that high-field MRI can improve considerably the imaging correlation of vertebrobasilar syndromes. The small size of the brainstem and the dense surrounding bony structures render CT imaging usually poor, especially in the lower pons and medulla. Only large brainstem infarcts with very severe neurological dysfunction are commonly visualized by CT, and the less severe but more frequent small infarcts are usually diagnosed on clinical grounds only, in the absence of CT confirmation. In 5 of our patients, CT was negative a few days after the stroke and/or 12–18 months later. In all but one, MRI showed a focal area of prolonged T1 and T2 suggesting infarction, in the region appropriate to the clinical picture. In the last patient, the association of a prolonged T1 and a shortened T2 is an unusual finding, which may suggest a previously hemorrhagic lesion, although the first CT was negative. The small infarcts (3–8 mm. in diameter) were readily imaged by a fairly rapid (approximately 1 min. scan time) partial saturation recovery
Patients 1 and 2 suffered Wallenberg’s syndrome and demonstrated a small lateral medullary infarct on MRI. Both of them had a good recovery and MRI did not show evidence of an associated cerebellar infarct. This should be emphasized, as most of the usually severe cases which have come to autopsy also demonstrate cerebellar infarction.

In case 1, Wallenberg’s syndrome was more complete than in case 2, and corresponded to a larger and more dorsally situated infarct on MRI. In fact, patient 1 showed the association of nearly all the oculomotor disturbances reported in Wallenberg’s syndrome, with tilt of the visual field, skew deviation with vertical diplopia, primary position rotatory nystagmus, monocular downbeat nystagmus on lateral gaze, ipsilateral ocular lateropulsion and overshooting saccades. These disturbances have been attributed to involvement of the restiform body with subsequent isolation of the vestibular nuclei from the flocculus which was suggested by MRI in our patient, whose infarct spared the cerebellum and the most dorsal medulla, but involved the region of the restiform body. This patient also showed a moderate upper motor neuron facial paresis; MRI did not show pontine involvement, which confirms that isolated medullary infarcts may produce some degree of facial weakness, probably by involving aberrant corticopontine fibers descending as low as at the pyramid level before reascending toward the facial nucleus. In patient 2, MRI showed a more ventrally situated infarct, at the level of the ventral spinocerebellar and lateral spinothalamic tracts, with sparing of the restiform body. This patient did not show significant cerebellar dysfunction, confirming that isolated involvement of the ventral spinocerebellar tract in the medulla does not markedly impair coordination, unless the more dorsally situated restiform body is involved.

The occurrence of spontaneous downbeat nystagmus in patient 6, in relation to the cerebellar (hemorrhagic?) infarct shown on MRI, must be emphasized, because isolated focal lesions very rarely produce downbeat nystagmus. Downbeat nystagmus may be related to lower brainstem dysfunction, as in Arnold-Chiari malformation, multiple sclerosis or brainstem-cerebellum degeneration. To our knowledge, it has not been reported in documented brainstem or cerebellar stroke, although a pontine infarct has occasionally been suspected on clinical ground. Experimentally, it can be produced by floccullectomy in the monkey. In man, a focal lesion of the nodulus has also been hypothesized. In our patient, most of the lesion was deep in the cerebellum and rather close to the midline on MRI; it certainly could have involved fibers from the flocculo-nodular lobe. This patient thus provides good evidence that downbeat nystagmus can be produced by a small stroke limited to the cerebellum, without significant associated neurological disturbances.

In these 6 cases of small vertebrobasilar infarcts, MRI studies allowed very precise clinico-topographic correlations, which were impossible with CT. These patients usually make a good recovery and neuropathological study would be useless.

Figure 4. Patient 4. (A — top left) CT: inconclusive image at the pontine level. (B — top right) MRI (PS: TR 600): focal area of prolonged T1 in the tegmental part of the midpons on the right side (arrow). (C — bottom left) MRI (PS: TR 600): same area of prolonged T1 in the coronal plane (arrow). (D — bottom right) MRI (SE: TR 2, TE 100): coronal slice showing prolonged T2 in the same areas as (C) (arrow).
FIGURE 5. Patient 5. (A — top left) CT: Lucency in the paramedian part of the pons on the left side (arrow). (B — top right) MRI (PS: TR 600): same area, with prolonged T1 (arrow). (C — bottom left) MRI (SE: TR 2, TE 100): prolonged T2 in the same area (arrow). (D — bottom right) MRI (PS: TR 300): sagittal view showing the same area (arrow).
FIGURE 6. Patient 6. (A — top left) CT shows a questionable low density among artefacts. (B — top right) MRI (MSR): focal area of shortened $T_2$ just on the left side of the midline of the cerebellum, adjacent to the fourth ventricle (arrow). (C — bottom left) MRI (PS: TR 600): focal area of prolonged $T_1$ in the same area (arrow).

Logical confirmation is rarely provided; thus, MRI appears to be the best technique for in vivo topographic correlation of localized vertebrobasilar stroke syndromes.

Editor’s Note: In accordance with Stroke policy, this article was guest edited by Dr. JP Mohr.

References

7. Hornsten G: Wallenberg’s syndrome. I. General symptomatology,
THE PRIMARY OBJECTIVE of this study was to determine the stroke and survival rates in a large group of patients with angiographically documented distal vertebral artery (VA) and/or basilar artery (BA) stenosis. Our second objective was to compare the clinical course of patients with proximal VA occlusive disease, distal VB occlusive disease appears to carry a higher risk for brainstem infarctions, one intraventricular hemorrhage). The observed 5 year survival rate was 78% compared to 90% in a matched normal population. Eight patients died during follow up, three patients due to stroke (2 sustained a stroke, 5 of which were in the VB territory. The observed stroke rate was 17 times the expected rate for a matched normal population. Eight patients (18%) had definite VB TIA and 3 patients had possible VB TIA. Eight patients died during follow up, three patients due to stroke (2 brainstem infarctions, one intraventricular hemorrhage). The observed 5 year survival rate was 78% compared to 90% in a matched normal population. In comparing this data with our previous study of 93 patients with proximal VA occlusive disease, distal VB occlusive disease appears to carry a higher risk for brainstem ischemia.

THE PRIMARY OBJECTIVE of this study was to determine the stroke and survival rates in a large group of patients with angiographically documented distal vertebral artery (VA) and/or basilar artery (BA) stenosis. Our second objective was to compare the clinical course of patients with proximal VA and/or BA stenosis to that of a group of patients with proximal vertebral artery stenosis which we have previously reported.1

Methods
The records of patients who underwent vertebral angiography at the Cleveland Clinic between 1974 and 1984 were reviewed. The angiographic films of all patients with reported stenosis of at least one VA distal to the second cervical vertebra (C2) and/or of the BA were examined. Forty-four patients with greater than 50% stenosis of at least one distal VA or the BA were selected for follow-up. Clinical follow-up after angiographic demonstration of the stenotic lesion was accomplished using a standardized questionnaire and telephone interviews. End points were death or stroke. Vertebrobasilar transient ischemic attacks (VB TIA) were noted but were not sufficient reason for stopping follow-up. The recommendations of the ad hoc Committee of the National Institute of Neurological and Communicative Diseases were followed in defining VB TIA.2 Only crossed or bilateral symptoms or combinations of symptoms localized to the posterior circulation were considered definite evidence of VB TIA. Transient unsteadiness, blurred vision, lightheaded-
Clinico-topographic correlation of small vertebrobasilar infarct using magnetic resonance imaging.

J Bogousslavsky, A J Fox, H J Barnett, V C Hachinski, S Vinitski and L S Carey

Stroke. 1986;17:929-938
doi: 10.1161/01.STR.17.5.929

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/17/5/929

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/