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Magnetic Resonance Imaging in Brainstem Ischemic Stroke

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To evaluate the efficacy of magnetic resonance imaging in brainstem stroke, we studied 21 cases of clinically definite brainstem ischemic stroke with brain magnetic resonance imaging (MRI) and conventional computed tomography (CT). MRI demonstrated brainstem lesions in 79% of the cases (16.5 out of 21), while CT revealed 33% (7 out of 21) when cases with suspicious lesions counted as 0.5. Although MRI was done a few days later than CT in most cases, MRI was superior to CT in detecting the number and the size of ischemic lesions, with clear delineation of anatomy and visualization of the status of the blood flow in the vertebral-basilar artery. Disappearance of the flow signal void in the basilar artery can be an important clue in diagnosing occlusion or thrombus of the basilar artery. By delineating the extent and the location of the infarction, MRI findings allowed an interpretation of whether the ischemic vessel is a small basilar branch or a large vessel vertebral or basilar artery.

Key Words: *MRI, Brainstem ischemia, Stroke, CT*

INTRODUCTION

Ischemic stroke in the brainstem and cerebellum often presents various combinations of clinical symptoms and signs (Caplan, 1986). Neurologists determine the pathophysiologic nature of strokes or transient ischemic attack (TIA) based on its temporal course and clinical features. The conventional computerized tomography (CT) does not show the precise location of ischemic lesions in most instances of brainstem stroke because of bone artifact, computer ripple artifact, partial volume effect, or a suboptimal choice of the horizontal plane. CT scanning does not satisfactorily detect the early phases of the ischemic process. Therefore, brainstem infarction cannot be ascribed confidently to either of its probable causes, atherothrombotic large vessel disease of the vertebral or basilar artery, or small vessel disease such as lacunar stroke, when using this method. Clinically, we suspect lacunar stroke when the features are related to only one small lesion in the paramedian area of the brainstem with a relatively well-preserved consciousness. Because the therapeutic options are different for a small vessel stroke compared to a large vessel stroke such as basilar artery thrombosis (Kistler et al., 1984a; Grotta, 1987), precise documentation of the extent and the location of the ischemic lesion is important.

Magnetic resonance imaging (MRI) can show the ischemic lesion during the early phase of infarction by a marked change in proton T1 and T2 relaxation times (Buonanno et al., 1983; Sipponen et al., 1983).

Because of the wide distribution of MRI scanners and the growing knowledge of its sensitivity to brain ischemic lesion, there have been many clinical studies on MRI findings of ischemic stroke (Kistler et al., 1984b; Kertesz et al., 1987; Rothrock et al., 1987; Hershey et al., 1987). Our study represents a systematic study of brainstem strokes completed with more cases and more advanced MRI techniques than those previously reported.

SUBJECTS AND METHODS

We studied 21 patients with ischemic brainstem

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stroke who were admitted to the Neurological Department of Seoul National University Hospital from March to September, 1988. The cases were selected from acute stroke patients who visited this hospital within 72 hours after the onset of their symptoms and who showed brainstem signs. They underwent CT and MRI scans as early as possible. But some of cases were excluded because they did not have both images or because the time lag between the 2 images was too long. The brainstem strokes were diagnosed by 2 neurologists who made detailed examinations with special reference to brainstem symptoms and signs such as alternating hemiplegia or hemianesthesia, Horner's sign, internuclear ophthalmoplegia, horizontal gaze palsy, vertigo, dizziness, depressed alertness, and ataxia.

For comparison with MRI and better detection of possible hemorrhagic stroke in its early phase, all cases underwent brain CT initially wiith a GE 8800 or 9800 scanner. The image was obtained by an axial scan of 13-15 degrees from the orbito-meatal line in 10mm thickness. The MRI scanner (SPECTRO-20000), developed by the Korean Advanced Institute of Science and Technology and Goldstar Company, has a 2.0 Tesla superconducting magnet system. Spin-echo pulse sequences were used for the image, separated into T1-weighted as TR, 500 msec and TE, 30msec [SE (500/30)] and T2-weighted as TR, 2500-3000 msec and TE, 30 msec or 90 msec [SE (2500 or 3000/30, 90)] sequences. The MRI was obtained axially in 5 mm thickness, so that MR and CT images were not from the same plane or slice thickness.

Brain CT was performed within 0-5 days after the onset of symptoms, and MRI followed 0-5 days after the scan. Two neuroradiologists, who were blinded to the clinical information, read CT and MRI films and agreed upon the presence and the size of the lesions.

RESULTS

We evaluated 21 patients with radiologically-verified (16 patients) or clinically definite (5 patients) brainstem ischemic strokes. Diagnosis of brainstem ischemic stroke was based initially on characteristic brainstem symptoms and signs and finally verified by CT and MRI results. Other possible brainstem pathological factors such as hemorrhagic stroke, demyelinating disease, tumor, and inflammatory disease were carefully excluded.

Fourteen patients were men and 7 were women. Past medical histories showed 15 cases of hypertension (71.4%), 8 cases (38.1%) with previous stroke history including TIA, 8 cases (38.1%) of diabetes mellitus, 2 cases (9.5%) of rheumatoid arthritis, and 1 case each of valvular heart disease, angina, asthma, and hyperlipidemia. Among the 8 patients with previous stroke histories, 4 had lacunes in the subcortical structures, 2 infarctions in the left middle cerebral artery territory (Cases 1 and 15), and 2 with possible vertebrobasilar insufficiency (Cases 8 and 10). Their clinical symptoms and signs, which are summarized in Table

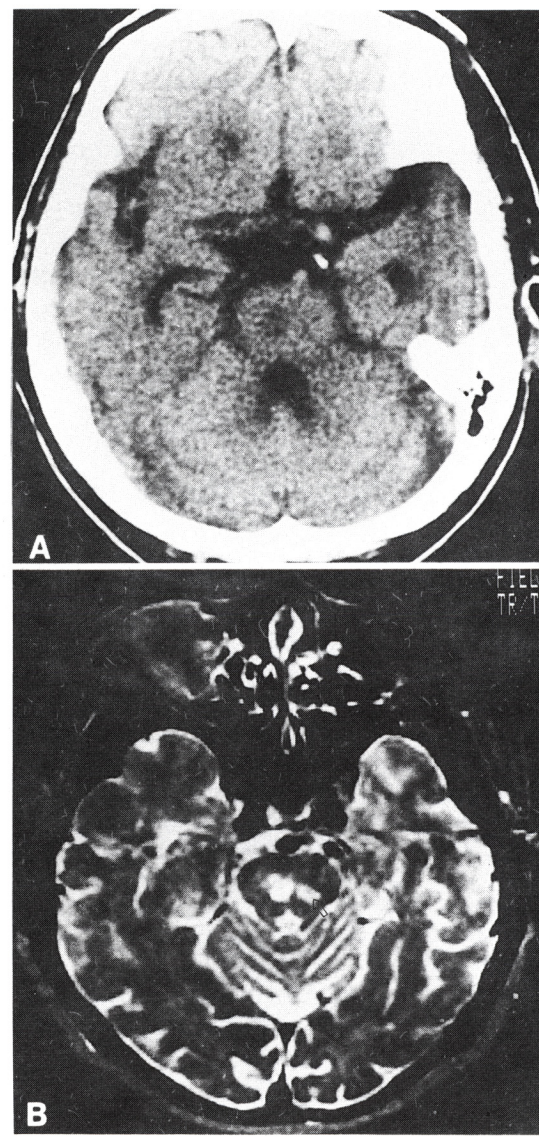

Fig. 1. Brain CT of Case 3 shows a lacune in the right pons, while MRI reveals an additional ischemic lesion (arrow) in the left.

Table 1. Summary of 21 cases of brainstem ischemic stroke

Case/Sex/Age	PMH	Symptoms and Signs	CT	MRI	Final Probable Diagnosis
1/M/52	Stroke, DM	Dizziness, altered conscionsness, dysarthria, dysphagia, L facial palsy, R hemiparesis	Old L MCA infarct L pontine lacune	L old MCA infarct L pons lacune, PVWM	L pontine lacune
2/F/50	HT, DM	Vertigo, vomiting, R clumsiness, paraparesis, L hypesthesia, R facial hypesthesia	WNL	R pons, R midbrain, R thalamus	V-BBS
*3/F/73	HT, stroke	Dysarthria, ataxia, L hemiparesis, facial diplegia (R>L), dysmetria	L pontine lacune SAE	Upper pontine lacune (B) PVWM	Pontine lacune
4/M/45	HT, DM	Dysarthria, ataxia (L>R), L INO,	L pons lacune (?)	L pons lacune	L pontine lacune
*5/M/25	VHD	Anarthria, facial diplegia, R hemiplegia, one-and-half syndrome	WNL	Pons (B), midbrain (B) FSV (−)	Basilar embolism from heart
6/M/55	Angina	Dysphagia, anarthria, ataxia, tongue deviation	Lacunes in R pons, BG (B)	Lacunes R pons, BG (B) PVWM	R pontine lacune
7/M/67	HT, DM	Dysarthria, R hemiparesis, nystagmus	Diffuse brain atrophy	Midbrain (L>R), L pons, L cbll BG (B), PVWM	V-BBS
*8/M/73	HT, TIA, MI	Altered consciousness, facial diplegia, limited ocular movement, locked-in state	Obliteration of quadrigeminal cistern	Pons (B), midbrain (B), R cbll FSV (−)	Basilar thrombosis
*9/M/48		Altered consciousness, INO (B), somnolent, dysarthria, ataxia	Pontine lacune (?)	Midbrain, pons, L occipital lobe	Top of basilar embolism
10/M/73	HT, TIA	Confusion, dysphagia, dysarthria, ataxia	R Cbll	L pons, Cbll vermis, FSV (−)	Severe basilar stenosis
11/M/51	HT	Vertigo, vomiting, headache, hearing loss (R)	R Cbll	R Cbll, L midbrain	BBS including
12/F/75	HT, stroke, CHF, RA	Altered consciousness, quadriparesis (L>R), facial diplegia	Lacunes in BG Diffuse brain atrophy	L pons, BG lacunes PVWM	R AICA
13/M/72	HT, DM stroke	Vertigo, diplopia, R hemiparesis,	WNL	L pons lacune, lacunes in BG (B), deep WM	L pontine lacune
*14/M/62	HT, asthma, stroke	L hemiparesis, dysarthria, ataxia	Diffuse brain atrophy	R pons lacune, L deep WM lacune	R pontine lacune
15/M/69	HT, DM, stroke	Vertigo, dysarthria, L arm weakness	Old L MCA infarct R pons lacune	L parieto-occipital infarct R pons lacune	R pontine lacune
16/F/46		R Horner, R facial hypesthesia, L hypesthesia, dysphagia	WNL	R Cbll, R medulla	Wallenberg's syndrome
17/M/60	HT, DM	L facial palsy, R hemiparesis, dysarthria, ataxia	WNL	PVWM	(?) L pontine lacune
18/F/59	HT, hyperlipidemia	R facial palsy, L hemiparesis, dizziness	WNL	PVWM	(?) R pontine lacune
19/F/56	RA	Dysarthria, R facial hypesthesia, vertigo, L hemiparesis, L paresthesia	WNL	WNL	(?) Wallenberg's syndrome
20/F/64	HT, DM	Dizziness, L facial numbness, nystagmus, L soft palate sagging, R hypesthesia	WNL	SAE L medulla (?)	(?) Wallenberg's syndrome
21/M/54	HT	Diplopia, ataxia, R INO	WNL	WNL	(?) pontine lacune

TIA: transient ischemic attack, HT: hypertension, DM: diabetes mellitus, VHD: valvular heart disease, MI: myocardial infarction, CHF: chronic heart failure, PMH: past medical history, RA: rheumatoid arthritis, L: left, R: right, B: both, INO: internuclear ophthalmoplegia, WNL: within normal limit, MCA: middle cerebral artery territory, Cbll: cerebellum, BG: basil ganglia, PVWM: periventricular white matter increased signal, SAE: subcortical arteriosclerotic encephalopathy, BBS: thrombotic basilar branch syndrome, V-BBS: thrombotic vertebro-vasilar branch syndrome, AICA: anterior inferior cerebellar artery, (?): questionable or presumed, FSV (−): disappearance of flow signal void, *: illustrated cases.

1, were compatible with those of brainstem stroke as described before.

CT revealed 4 definite and 2 suspicious pontine lesions and 2 definite cerebellar lesions; 33% (7 our of 21) were thought to have positive results with 2 suspicious cases counted as 1. MRIs performed 0 to 5 days after CT scans demonstrated definite lesions in 16 cases and 1 case of suspicious medullary lesion; 79% (16.5 out of 21) had positive results with 1 suspicious case counted as 0.5. Brief radiological results are summarized in Table 2.

Compared to CT, MRI was more sensitive in showing the number of lesions as well as the size of ischemic lesions. In Cases 5, 8, and 10, it gave additional infor-

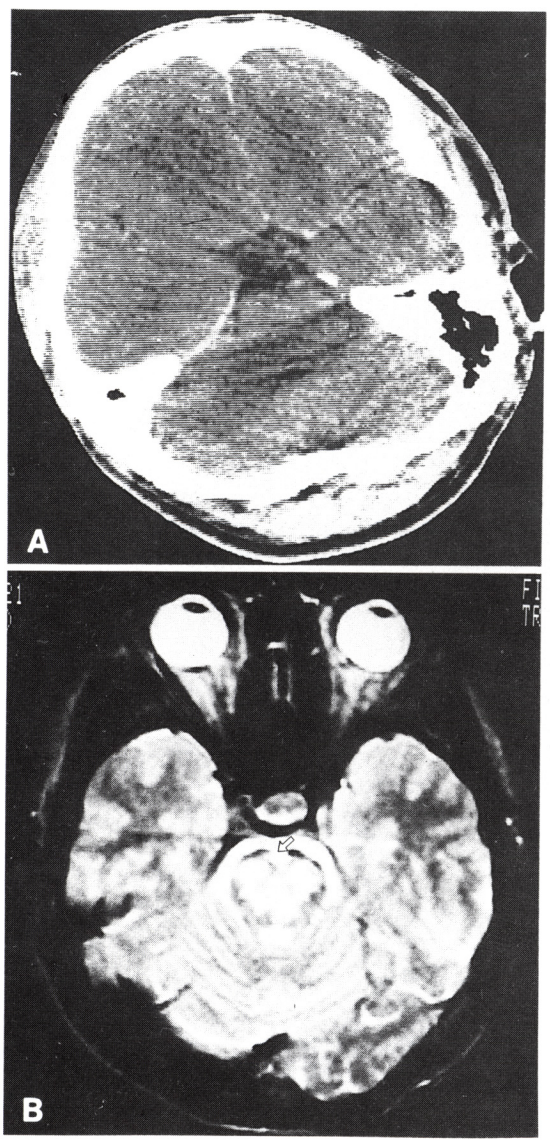

Fig. 2. Brain CT of Case 5 has no definite lesion in the brainstem, while MRI shows a massive infarction in the midbrain and disappearance of flow signal void in the basilar top area (arrow).

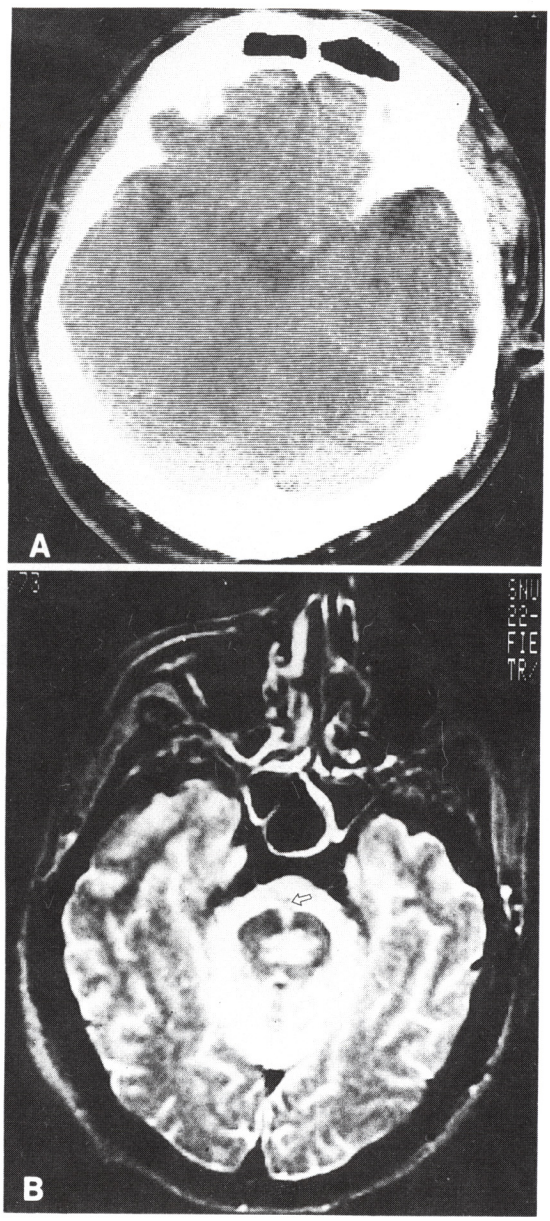

Fig. 3. Brain CT of Case 8 shows obliteration of the quadrigeminal cistern, while MRI demonstrates pontine infarction and disappearance of flow signal void in the basilar artery (arrow).

Table 2. Comparison of detected brainstem lesions between MRI and CT

Location Case	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Midbrain		○			○		○	○	○	○	○										
Pons	■	○	■	□	○	■	○	○	□			○	○	○	■						
Medulla																○					
Cerebellum							○	○		■	■					○				•	
FSV (−)					○			○		○											

■ lesion detected on both images, □ suspicious lesion on CT with definite MRI lesion
○ lesion detected on MRI only, • suspicious lesion on MRI only,
FSV (−): disappearance of flow signal void

mation on absent or slow blood flow movement through the disappearance of the normal arterial flow signal void. Together with the clinical course and features, MRI findings pointed out responsible causal vessel in the majority of cases. The final possible diagnoses were pontine lacune in 10 cases (Cases 1,3,4,6,13,14,15,17,18, and 21), occlusion of the circumferential branch of the vertebral-basilar artery in 4 cases (Cases 2,7,11, and 12). Wallenberg's syndrome in 3 cases (Cases 16,19, and 20), basilar thrombosis in 2 cases (Cases 8 and 10), and embolism to the basilar artery in 2 cases (Cases 5 and 9). Figures 1-5 show Cases 3, 5, 8, 14, and 16, respectively.

DISCUSSION

It is widely accepted that brain MRI is superior to CT in early detection of acute ischmic infarction and in visualizing ischemic lesions of any age (Dewitt, 1986; Kertesz et al., 1987). Brain CT appears to be equally or more sensitive for detecting acute cerebral hemorrhages, but MRI is thought to have the capability of defining the age of the hematoma through the differentiation of oxyhemoglobin, deoxyhemoglobin, methemoglobin, and hemosiderin by various pulse sequences (DeLapaz, et al., 1984; Elster, 1988).

As described in the introduction, brainstem stroke has a wide array of symptoms and signs so that its confirmation through a neuroimaging technique is very important to clinicians. Bonafe et al. (1985) tried to document vertebro-basilar ischemia through brain CT but failed to delineate lesions smaller than 2 cm in diameter or lesions located in the lower brainstem. One study on vertebrobasilar ischemia with MRI was reported in the early 1980s (Young et al., 1981). Kistler et al. (1984b) describe the usefulness of proton NMR images in studying ischemic stroke in the territory of vertebrobasilar and posterior cerebral circulation without direct comparison with CT.

In this present study, we tried to compare MRI findings of brainstem ischemic stroke with those of CT, although the images were not done on the same day and were not obtained from the same brain plane. Because MRI was done somewhat later than CT in most cases, its sensitivity might be higher than MRI done at the same time. Nonetheless, MRI is more sensitive, shows larger and additional lesions, and reflects the blood flow effect through flow void phenomenon.

Some authors (Axel, 1984; Bradley et al., 1984) have reported on blood flow effect since the early 1980s. Disappearance of the blood flow effect (flow signal void) is quite important in confirmation of the occlusion of large vessels such as the carotids, the vertebro-basilar arteries, and the venous sinuses. In this study, 3 cases showed the disappearance of the flow signal void in the basilar artery, suggesting occlusion or severe stenosis of the artery.

MRI is also useful in that it can detect small lesions like lacunes. Rothrock et al. (1987) found lacunar lesions on MRI which match symptoms of patients to MRI findings in 74% of their cases with lacunes. Moreover, Gd-DTPA-enhanced MRI can differentiate recent lacunes among the different stages of a multiple small infarct in lacunar stroke patients within 4 weeks after ictus (Miyashita et al., 1888). We included 10 cases of probable brainstem lacune in this study, and 8 of them (80%) were clearly identified by MRI.

Ross et al. (1986) report 4 cases of Wallenberg's lateral medullary syndrome whose lesions were demonstrated with MRI but not with CT. In our series, we included 3 cases of possible lateral medullary syndrome, but MRI showed 1 definite lesion and 1 sus-

picious brainstem lesion, giving this method a 50% detection rate (1.5 out of 3). The reason for the low sensitivity in lateral medullary syndrome in our series is that 2 of the cases may have had mild ischemic changes in the lateral medulla only. Also, our experience after this study showed that lesions detected by MRI are more clearly shown often in the cerebellum or in the cerebellar peduncle than in the lateral medulla itself.

Through this study, we believe MRI is more useful than CT in diagnosing brainstem strokes and can almost pinpoint the culprit vessel along with clinical findings.

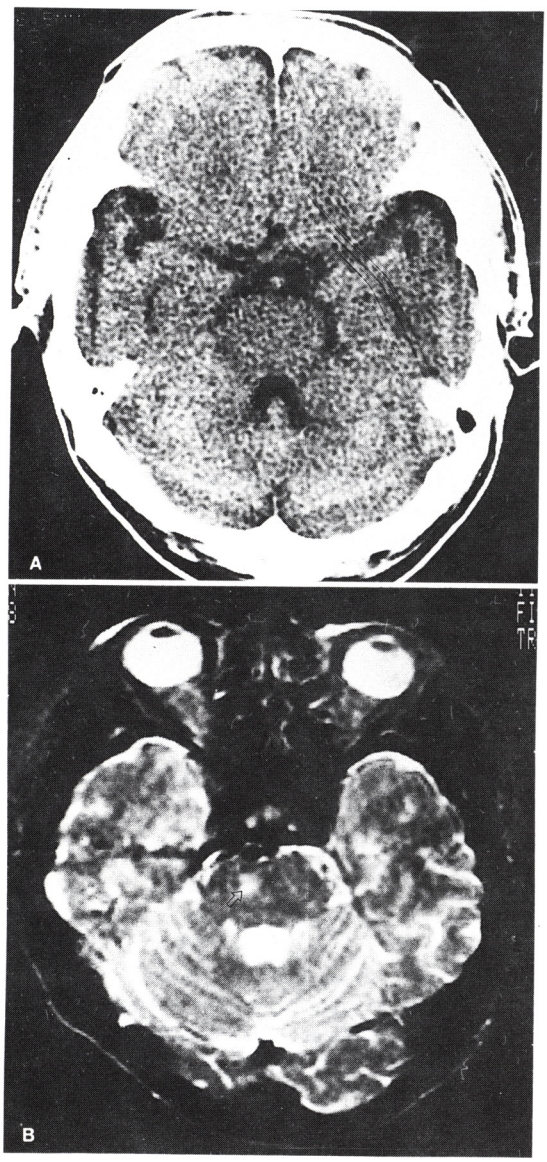

Fig. 4. Brain CT of Case 14 shows a normally appearing pons, while MRI visualizes a lacune (arrow) in the right pons.

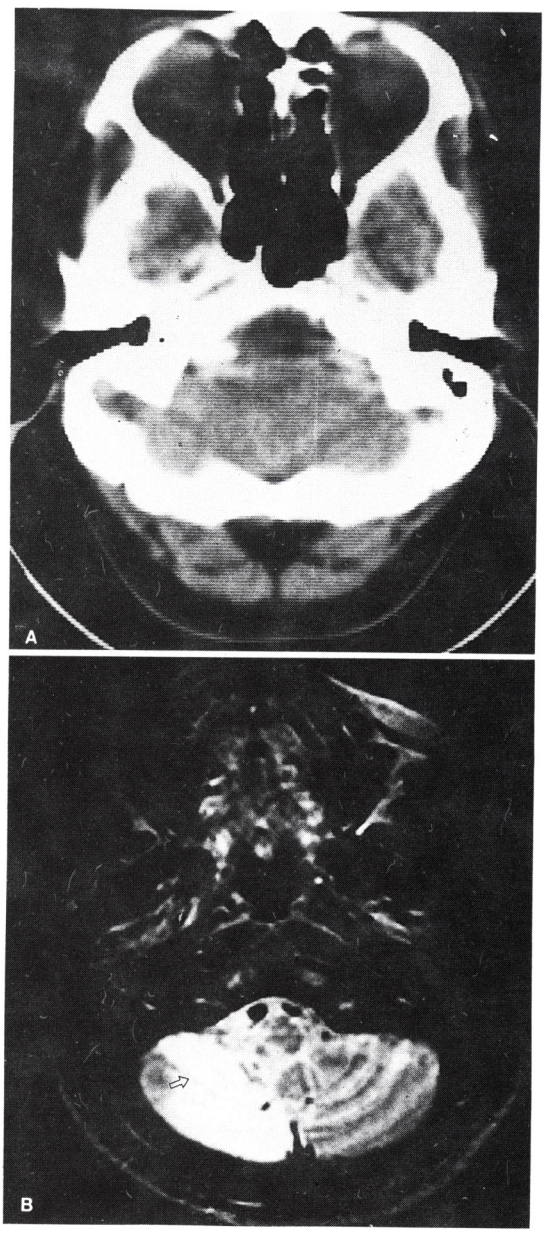

Fig. 5. Brain CT of Case 16 has no lesion in the medullocerebellum, while MRI demonstrates a right lateral medullary infarction (arrow) and a large infarction in the right posterior inferior cerebellar artery territory.

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