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Case Report

Unusual MR Findings of the Brain Stem in Arterial Hypertension

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Summary: MR imaging findings have been reported in only a few cases of severe arterial hypertension. We report two cases of severe paroxysmal arterial hypertension associated with unusual brain stem hyperintensity. The lesions improved dramatically after stabilization of blood pressure, suggesting that edema could be the main cause of the MR imaging–observed hyperintensity.

Hypertensive encephalopathy is an acute disorder that occurs in patients with dramatic rise in blood pressure associated with CNS signs such as headache, seizures, visual disturbances, and altered mental status (1, 2). The clinical findings are sufficiently nonspecific to make diagnosis difficult. Neuroimaging features in hypertensive encephalopathy include diffuse or focal hyperintensity on T2-weighted images, predominately in the supratentorial white matter, especially in occipital lobes (3−5). Basal ganglia, brain stem, and cerebellum hyperintensities have rarely been reported and were always associated with cortical lesions (5). The pathophysiologic changes in hypertensive encephalopathy are still unclear, being described as edema associated with marked vasodilation or microinfarcts in the arterial territories (1−5). We report two cases of unusual MR findings (extensive brain stem hyperintensity), without mental status alteration, caused by paroxysmal hypertension secondary to a pheochromocytoma in one case and renovascular hypertension in the other. We discuss a pathophysiologic hypothesis for these MR findings.

Case Reports

Case 1

A 41-year-old man presented with blurred vision and headache. His neurologic history was unremarkable. He had a recent history of hypertensive episodes (160/90) that were treated by Ramipril with good results. The results of his neurologic and general examinations were normal. Arterial blood pressure was 140/70 at admission, but a few weeks later, severe paroxysmal hypertensive episodes (up to 220/120) were noted.

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Visual acuity was normal. An ophthalmologic examination showed bilateral papillary edema and severe hypertensive retinopathy. Visual evoked potentials and other evoked potentials (somatosensory, auditory, and motor) were normal. The results of routine laboratory and CSF examinations, including isoelectrophoresis, were normal. The electroencephalographic results were normal.

MR images showed an extensive increased signal in the brain stem on T2-weighted and fast fluid-attenuated inversion-recovery (fast-FLAIR) sequences (Fig 1A). We also observed alternating bright and low signals in the pons. This last pattern was absent in the tegmentum of the pons and in the superior cerebellar peduncles (Fig 1A). Mild periventricular hypersignals were also present (Fig 1C). A T1-weighted sequence confirmed that the pons was swollen (Fig 1E). There was no enhancement after the injection of contrast material. Urinary and blood catecholamines were increased: plasma noradrenaline = 5 μg/L (normal = 0.1−0.6 μg/L), urinary noradrenaline = 599.2 μg/24 hr (normal < 75 μg/24 hr), total urinary normetanephrine = 3618 μg/24 hr (normal < 350 μg/24 hr), total urinary methoxytyramine = 1684 μg/24 hr (normal < 280 μg/24 hr). ^{31}I-Metaiodobenzyl-guanidine scanning was performed for suspicion of pheochromocytoma and showed an abnormal uptake. CT of the thorax confirmed a mediastinal tumor without adenopathy. MR imaging findings of the adrenal glands were normal. At surgery, a thoracic pheochromocytoma was removed. One month after surgery, the brain stem and periventricular lesions had completely resolved (Fig 1B and D). The pons returned to a normal size (Fig 1F).

Case 2

A 52-year-old woman presented with progressive headaches. One month after the onset of symptoms, she developed bilateral blurred vision. Her mental status was unaltered. A neurologic examination showed a static and kinetic cerebellar syndrome and a vestibular syndrome with a mutidirectional nystagmus. Arterial blood pressure at rest was 170/90. During the first 3 days after admission, arterial blood pressure was between 120/70 and 180/100, with a peak at 220/150. Hypertension improved after 2 days of antihypertensive medication. Fundoscopy showed severe hypertensive retinopathy. Multitechnique evoked potentials and the results of CSF analysis were normal.

MR imaging showed extensive brain stem hyperintensity on T2-weighted and fast-FLAIR sequences (Fig 2A). We observed the same alternating bright and low signal as seen in case 1 (Fig 2A [*left image*]). There were also a few periventricular hyperintensities, with only mild occipital lobe lesions (Fig 2B [*left image*]). On the T1-weighted sequence, the pons looked swollen, as in case 1. After 3 months of hypotensive treatment, the headaches and blurred vision improved and the results of a neurologic examination remained normal. MR images showed a clear improvement, but mild brain stem and periventricular lesions were still observed (Fig 2A [*right image*] and 2B [*right image*]).

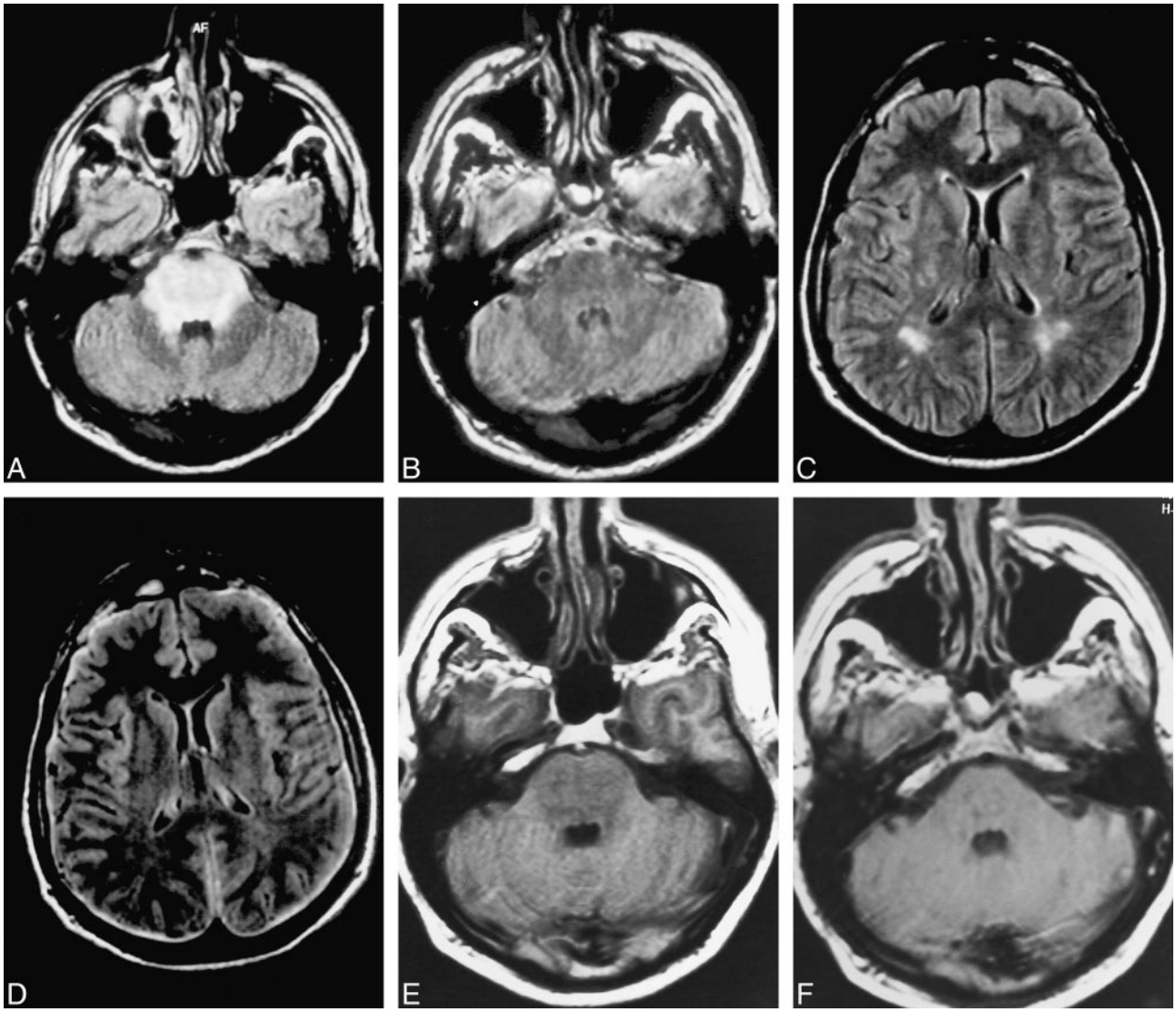

FIG 1. Images of a 41-year-old man who presented with blurred vision and headache. The fast-FLAIR imaging parameters were as follows: 9000/119/1 (TR/TE/excitations); acquisition time, 3 min 27 s; field of view, 175 × 280; matrix, 154 × 256. The T1-weighted imaging parameters were as follows: 540/12/1; acquisition time, 2 min 39 s; field of view, 188 × 250; matrix, 144 × 256.

A, Fast-FLAIR image shows an extensive brain stem lesion.

B, Fast-FLAIR image shows that the lesion dramatically improved after surgical treatment of pheochromocytoma.

C, Fast-FLAIR image shows periventricular hyperintensities.

D, Fast-FLAIR image shows that the periventricular hyperintensities also totally resolved after surgery.

E, T1-weighted image shows that the pons was swollen at the begining of the symptoms.

F, T1-weighted image shows that the pons returned to normal size and configuration after surgery.

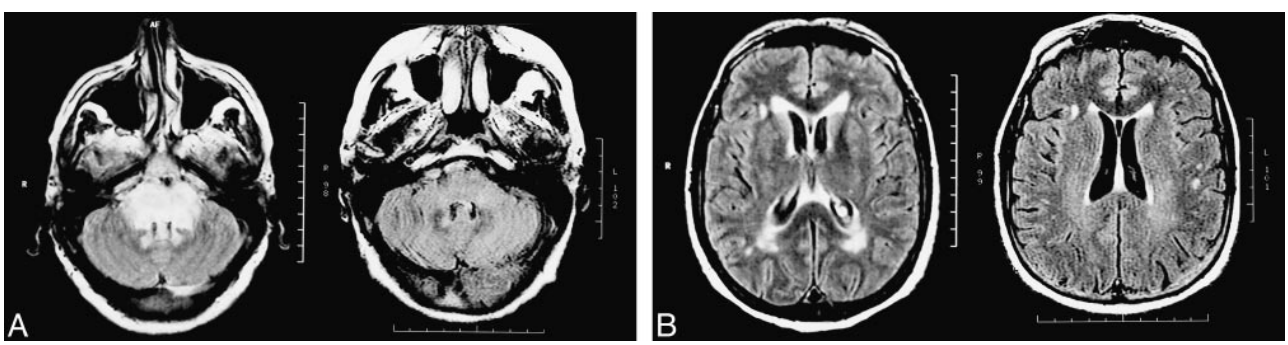

FIG 2. Images of a 52-year-old woman who presented with progressive headaches.

A, Fast-FLAIR image shows a brain stem lesion (*left*) and that the brain stem lesion resolved after treatment for hypertension (*right*).

B, Fast-FLAIR image shows periventricular hyperintensities (*left*) and that the periventricular hyperintensities decreased considerably after treatment (*right*).

Discussion

Brain stem MR hyperintensity has not been described as sign of paroxysmal hypertension. In our two cases, clinical signs and CT findings first suggested glioma. Major retinal hypertensive signs were of particular importance for the diagnostic workup leading to a diagnosis of CNS manifestations of severe paroxysmal hypertension. The MR findings with alternating bright and low signals, probably signifying edema/fluid between the transverse pontine bundles, could also help to distinguish edema secondary to paroxysmal hypertension from brain stem tumor.

In two studies of acute hypertensive encephalopathy, the main MR finding was hyperintensity in the supratentorial white matter (5, 6). Brain stem hyperintensities have also been reported, but they are associated with extensive cortical lesions and clinical signs such as altered mental status (5, 6). In the brain stem, neuropathologic changes have been variously described as fibrinoid necrosis, thrombosis of arterioles and microvessels, and petechial hemorrhages (3, 5, 7, 8).

Several mechanisms have been discussed to try to account for MR-revealed abnormalities. The first hypothesis was edema, as in eclamptic encephalopathy (7). In such cases, edema, involving mainly the posterior brain areas, is frequently bilateral and reversible, allowing the diagnosis to be confirmed. The rapid increase in blood pressure could affect the blood-brain barrier, leading to increased permeability and extravasation of proteins and fluid, resulting in cerebral edema (1, 7, 9). On the other hand, abnormal increased sensitivity of vessels to circulating vasoactive agents or endothelial cell dysfunction has also been suggested (5, 10, 11). An alternative mechanism has been suggested to explain the pathogenesis of hypertensive MR lesions, namely that autoregulatory vasoconstriction in the brain, in response to a severe rise in blood pressure, could induce hypoperfusion, leading to focal and diffuse areas of ischemia and infarction (8−12). Whatever the case, sequential MR examinations should help to determine the mechanism involved. We did not obtain diffusion-weighted images, but such a sequence could be important in distinguishing between interstitial and cytotoxic edema. Diffusion-weighted images would not show hyperintense signal because of the presence of interstitial rather than cytotoxic edema.

In our two cases, edema seems to have been the major pathophysiologic mechanism because MR imaging findings after treatment were normal or nearly normal. In one case, the diagnostic workup showed a thoracic pheochromocytoma. Pheochromocytomas occur extra-adrenally in only 10% to 20% of the cases (13). Cases with CNS symptoms, such as headache and blurred vision, and an MR study of the brain have been rarely reported (14). Eclevea et al (14) reported a case of hypertensive encephalopathy with white matter changes shown on MR images, but the lesions were mainly in the supratentorial regions. Although there are a few reports of cerebral hemorrhage and stroke associated with pheochromocytoma (14), brain stem lesions have not been previously described as being shown on MR images. In a non-imaging report of pheochromocytoma-related hypertensive encephalopathy, infarcts were found to involve the pons, basal ganglia, cerebellum, and periventricular white matter (15). It has been suggested that direct catecholamine toxicity might also have a role in the generation of brain lesions (16). That study reported a high concentration of neuropeptide Y around cerebral and coronary vessels, which could have contributed to a vasospasm and thus induced a stroke. In our case, however, MR imaging–shown abnormalities were probably secondary to edema, as evidenced by the complete disappearance of the lesions after surgery and arterial blood pressure stabilization. Further reports of MR imaging of the brain or neuropathologic analysis of the brain in cases of pheochromocytoma should allow a better understanding of the pathophysiologic pathways of CNS manifestations and the role of endocrine substances. To our knowledge, there is no clear explanation for the prominence of brain stem changes compared with the other brain territories, especially in occipital lobes.

Our cases show a particular MR pattern secondary to severe paroxysmal hypertension, thus allowing a better understanding of the change associated with arterial hypertension. The dramatic improvement of the lesions suggest that they should be considered secondary to edema rather than to ischemic episodes.

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