IMAGING OF OCULOMOTOR NERVE PALSY : LESIONS AT MEDIOVENTRAL PART OF THE THALAMUS AND MIDBRAIN

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ABSTRACT

Two patients presented with third nerve palsy. One patient had an ischemic focus at anteromedial part of the thalamus and another patient had a subacute hematoma at anteromedial part of the thalamus, upper pons and midbrain. The images were demonstrated by MRI study. Discussion concerning the path of the oculomotor nerves and the pathologic processes were performed.

INTRODUCTION

The third cranial nerve palsy was second in frequency in 4000 cases of patients with ocular motility palsies (1,2). Patients may present with an isolated or nonisolated, pupil-sparing or nonpupil-sparing cranial nerve III palsy. Isolated major dysfunction of a single third nerve palsy is demonstrated by MRI study to be caused by brain stem lesions (ischemia, mass, cryptic vascular malformation, hemorrhagic shearing injury, and hemorrhagic infarct), subarachnoid space lesion (aneurysm, lymphoma, ophthalmoplegic migraine, viral meningitis, coccidioidomycosis. trauma, syphilis, Miller-Fisher syndrome) and cavernous sinus/superior orbital fissure lesions (lymphoma, carcinoma, Tolosa-Hunt syndrome, cavernous carotid aneurysm, pituitary apoplexy, and craniopharyngioma) (1).

Two cases presented by us, the third cranial nerve palsy was found in the patients who had medioventral thalamic and midbrain lesions.

CASE REPORT

CASE 1

A 50-year-old male patient had double vision for one week prior to searching for the medical advice. The visual symptom was preceded by severe vertigo. His blood pressure was 210/120 mgHg. Left lateral rectus palsy, impaired upward gaze of left side and downward gaze on the right side. Clinical impression was left cranial nerve III and VI palsy. MRI study (Fig.1) showed a subacute ischemic focus at ventromedial part of right thalamus and smaller ischemic foci at left dorsomedial part of thalamus and dorsolateral part of right thalamus. The patient had also hypercholesterolemia. He was treated medically and the eye symptoms improved after two weeks.

CASE 2

A 17-year-old man had diplopia for twenty days prior to admission. He developed ptosis for

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one day. Numbness of left face and left hemiparesis was also present. Right pupil was 8 mm diameter non-reacted to light, there was no reaction to consentual light reflex. Impaired upward gaze of both sides. Palsy of right medial rectus muscle, superior rectus muscle, inferior oblique muscle was observed. The tongue was deviated to the left. MRI study (Fig.2) showed small intracerebral hematomas at ventromedial aspect of right upper pons and midbrain-right thalamus, and superficial white matter of posterior right parietal lobe. Evidence of previous small hematomas are noted at posterior aspect of left pons, right deep parietal white matter, left deep parietal white matter, high right deep parietal white matter. Bleeding from the parasitic tract was suspected. The serum antibody and CSF antibody was negative of Gnathostomiasis; and the antibody-serum was positive for Angiostrongyliasis but was negative in CSF.

DISCUSSION

The oculomotor nucleus extends through the entire dorsal portion of the midbrain. Nerve fibers that supply the levator palpebrae superioris divide into right and left bundles from one central caudal nucleus. The other nuclei are represented bilaterally, with the superior rectus being the only subnucleus with contralateral representation. This subnucleus is located in the more caudal portion of the nuclear complex. These fibers cross intraparenchymally, and all fibers run ventrally and slightly rostrally to exit through the interpeduncular fossa. The oculomotor nerve then courses anteriorly within the subarachnoid space until it pierces the dura covering the roof of the cavernous sinus. In the anterior cavernous sinus the oculomotor nerve divides into superior and inferior divisions. The superior division innervates the levator palpebrae superioris and the superior rectus muscles, whereas the inferior division innervates the inferior and medial rectus muscles, the inferior oblique muscle, and the iris sphincter (3,4). Traditionally, divisional oculomotor pareses are localized to the anterior cavernous sinus or posterior orbit. Aneurysms of the internal carotid artery, diabetes mellitus, enlargement of the third ventricle, and presumed viral syndromes have all been reported to cause superior division paresis (5-9). Inferior division third nerve palsies have been ascribed to viral disorders, trauma, or local orbital disease (10-12).

The oculomotor fascicles sweep ventrally and laterally from the oculomotor complex, pass through and medial to the red nucleus, and converge to exit the brain stem medial to the cerebral peduncles (13,14). The clinical localization of lesions along the dorsal-ventral course of the oculomotor fascicles relies on associated neurologic findings. Damage to the ventral fascicles and the nearby cerebral peduncle causes ipsilateral third-nerve palsy and contralateral hemiparesis (Weber's syndrome)(15). Involvement of fascicles within the red nucleus leads to oculomotor palsy with contralateral involuntary movements (Benedikt's syndrome). Extension of such a lesion to the brachium conjunctivum produces additional contralateral ataxia (Claude's syndrome)(15). When sufficiently small, fascicular lesions can mimic complete or partial extra-axial third nerve palsies (16-21). Most of these have been located in the ventral midbrain, sparing the red nucleus and cerebral peduncles. Small lesion of the proximal oculomotor fascicles have been reported only rarely. Extension of such a lesion would involve the oculomotor subnuclei, the medial longitudinal fasciculus, and the mesencephalic reticular formation, causing ipsilateral oculomotor paresis and combinations of bilateral ptosis, bilateral superior rectus palsy, internuclear ophthalmoplegia, and lethargy (13).

Bithalamic hyperintensity on T2WI has been described in a relatively small number of pathologic states. Causes include birth asphyxia, bithalamic glioma, bilateral germ cell tumors, carbon monoxide poisoning, Wernicke encephalopathy, and vascular events such as "top of the basilar" syndrome and deep cerebral vein thrombosis (22). T2 hyperintensity in the mesodiencephalic region, occipital lobes and posterior fossa is well described in cases of basilar artery occlusion and strongly suggests this diagnosis (23). Changes characteristic of infarction by MR are not present for approximately 12 hours, potentially delaying diagnosis and intervention. T2 hyperintensity develops in the thalami and basal ganglia in patients with deep cerebral vein thrombosis, corresponding to the venous vascular territories, and infarctions that are usually hemorrhagic develop (24). MR alone is not sufficient for evaluation of the venous system. It cannot consistently distinguish between flow and

occlusion, given the phenomenon of flow-related enhancement and the different signal characteristics of blood products within a thrombus (25). Two-dimensional phase-contrast MR angiography is the optimal technique for evaluating occlusion of cerebral veins (26).

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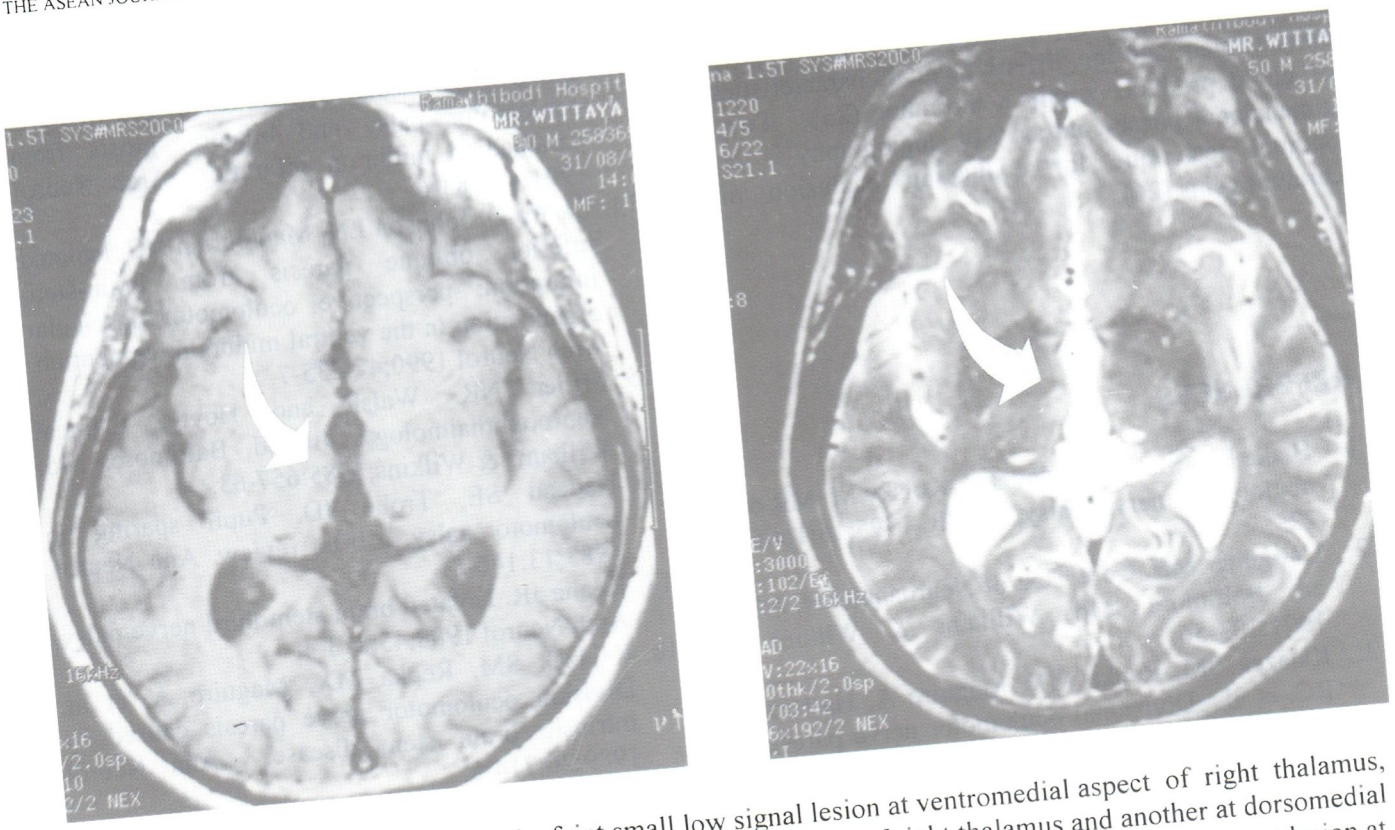

Fig. 1A. Axial T1WI, showed a faint small low signal lesion at ventromedial aspect of right thalamus, one smaller low signal area at dorsoloteral aspect of right thalamus and another at dorsomedial aspect of left thalamus. These lesions appeared bright on T2WI, however, the largest lesion at the ventromedial aspect showed less bright signal because the lesion is probably newer than the more posteriorly lesions.

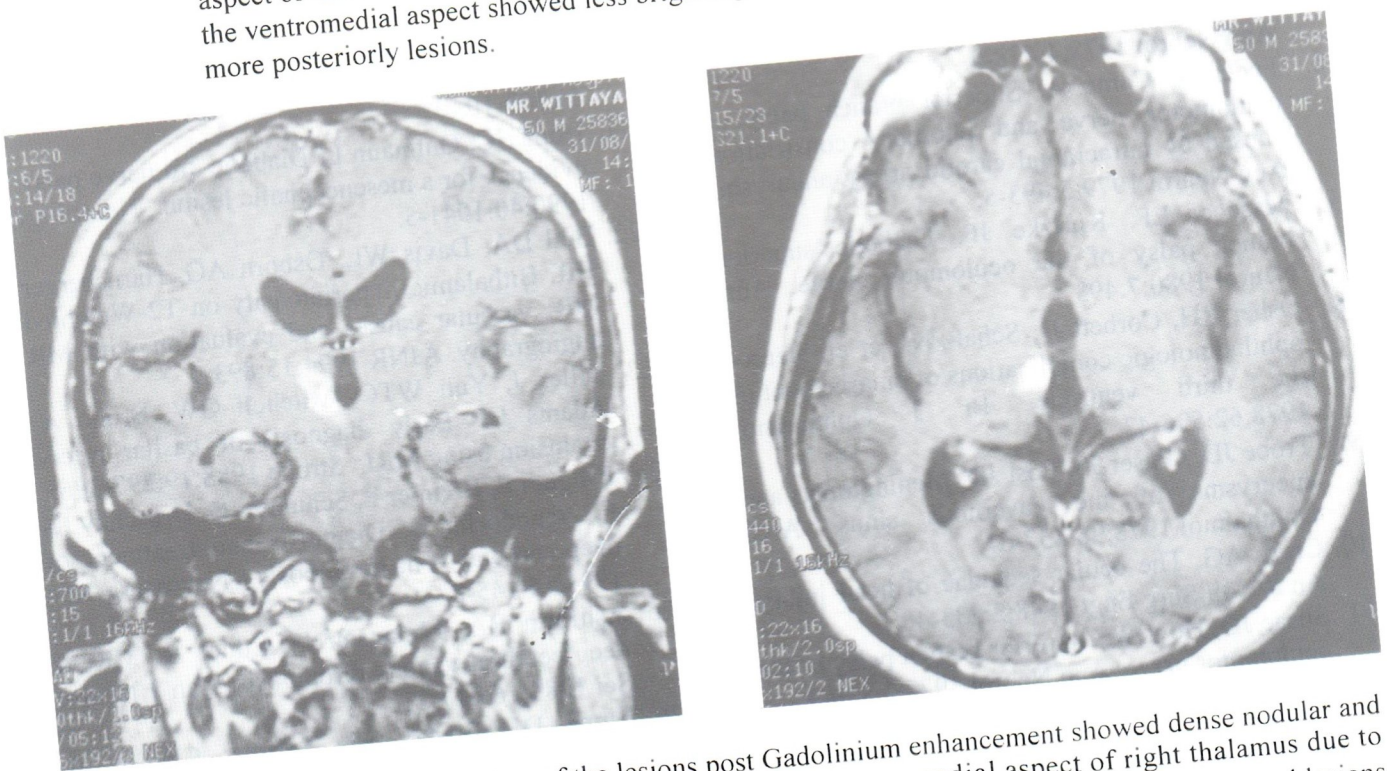

Fig. 1B. Coronal and axial view of the lesions post Gadolinium enhancement showed dense nodular and ring enhancement of the larger lesion at the ventromedial aspect of right thalamus due to subacute process with loss of blood brain barrier. The smaller more posteriorly located lesions were not enhanced and were probably old lesions.

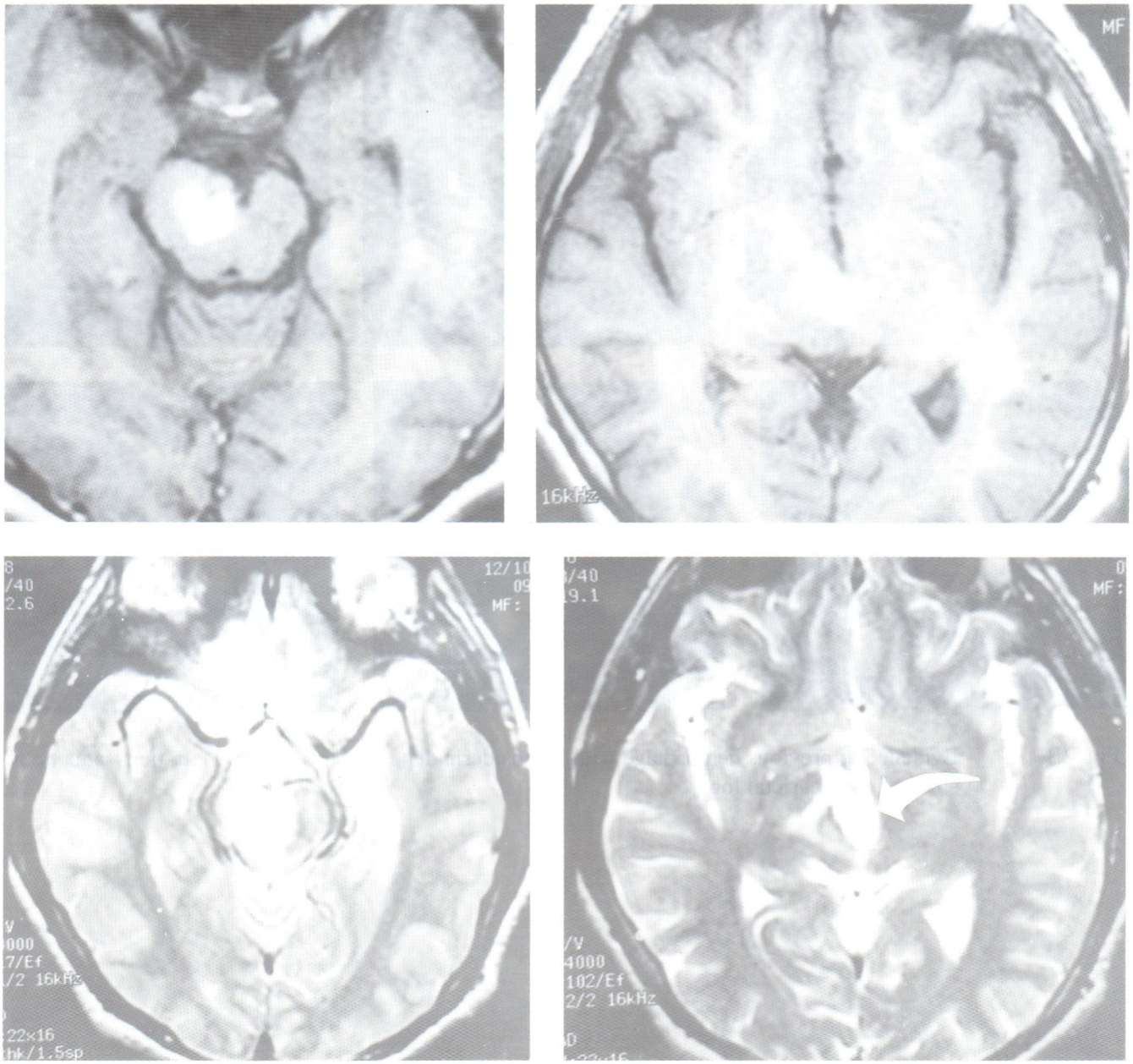

Fig. 2A. Axial T1WI and T2WI showed small subacute hematoma at right parietal lobe, right ventroparamedian area of upper pons, midbrain and thalamus.

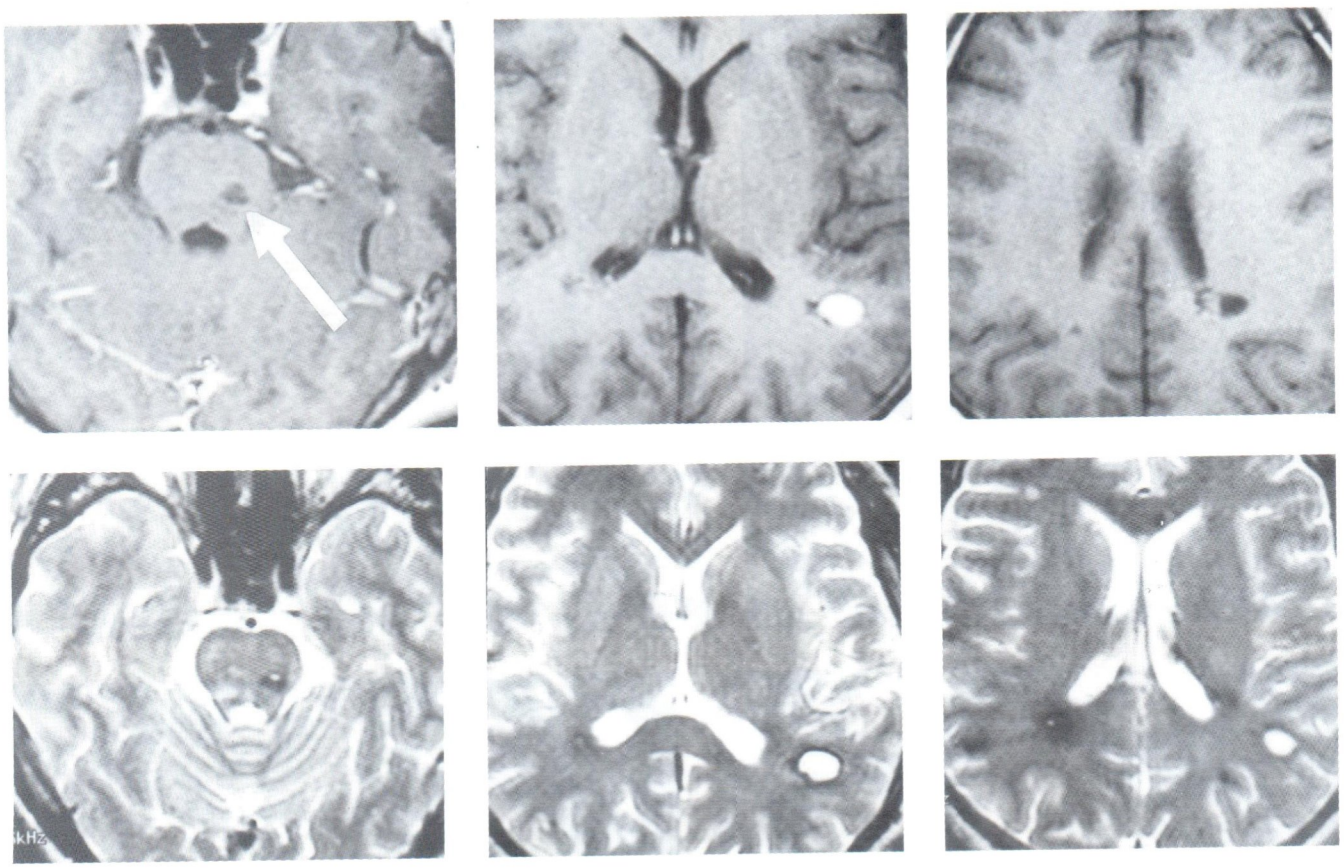

Fig. 2B. Evidence of previous hemorrhage with hemosiderin deposit was noted at left pons and both sides of deep parietal lobe.