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Radiologic-Clinical Correlation

Isolated Third Nerve Palsy Caused by Midbrain Hemorrhage

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Isolated major dysfunction of a single third nerve is usually caused by diabetes mellitus, an aneurysm, or basal meningitis. Occasionally, focal, ventral, paramedian midbrain lesions, usually infarcts, produce isolated third nerve palsy (1–4). We present the unusual case of a patient in whom an isolated complete third nerve palsy followed a midbrain hemorrhage.

Case Report

This 70-year-old Japanese woman suddenly presented with double vision and right ptosis. On admission, her blood pressure was 139/67 mm Hg, and the pulse rate 100/min and regular. She was mentally alert and exhibited no motor or sensory disturbances. Hemiparesis, cerebellar ataxia, and involuntary movements were absent. The right pupil measured 5 mm in diameter and was not reactive to light. The left pupil measured 3 mm in diameter and was reactive to light. Adduction, elevation, depression, and convergence of the right eye were limited, as was elevation of the left eye (Fig 1). Computed tomography of the brain on admission revealed a high-density lesion (72.8 Hounsfield units) in the anterior tegmentum of the right midbrain (Fig 2), most likely involving the distal portion of the oculomotor fascicles.

The patient had a history of chronic hepatitis and diabetes mellitus. Laboratory findings showed mild anemia (red blood cells, $296 \times 10^4/mm^3$; hemoglobin, 10.1 g/dL; hematocrit, 30.5%), leukopenia (white blood cells, $3600/mm^3$, and thrombocytopenia (platelets, $5.0 \times 10^4/mm^3$). Blood chemistry revealed hepatic dysfunction: glutamic-oxaloacetic transaminase, 58 IU/L (normal, 5 to 40 IU/L); glutamic-pyruvic transaminase, 45 IU/L (normal, 0 to 35 IU/L); lactate dehydrogenase, 524 IU/L (normal, 150 to 450 IU/L); total bilirubin, 0.9 mg/dL (normal, 0.2 to 1.1 mg/dL); albumin, 2.5 g/dL (normal 3.9 to 5.0 g/dL); and blood glucose, 185 mg/dL.

Coagulation tests revealed a slightly prolonged prothrombin time of 13.3 seconds (normal, less than 12.5 seconds) and decreased ratios in the thrombo test of 68% (normal, 70% to 130%) and the hepaplastin test of 57% (normal, 70% to 130%), but the activated partial thromboplastin time was 32.9 seconds (normal, less than 35 seconds), and the fibrinogen was 228 mg/dL (normal, 160 to 400 mg/dL).

Nine days after bleeding, magnetic resonance (MR) revealed a slight reduction in size of the lesion on a T1-weighted image (380/15/2 [repetition time/echo time/excitations]) (Fig 3A) compared with the computed tomographic findings (Fig 2). However, a T2-weighted image (2000/100/1) revealed that the high-intensity abnormality, probably including brain edema, extended around and partially involved the red nucleus, cerebral peduncle, and oculomotor nucleus (Fig 3B).

The third nerve palsy improved slowly, and the patient was discharged in 18 days with moderately improved ptosis and extraocular movements of the right eye. A repeat MR study 3 months after the hemorrhage revealed a small low-intensity lesion with a high-intensity rim in a T1-weighted image (380/15) (Fig 4A). A T2-weighted image (2000/100) also showed a low-intensity lesion without a high-intensity rim in the midbrain, which suggested hemosiderin deposition (Fig 4B).

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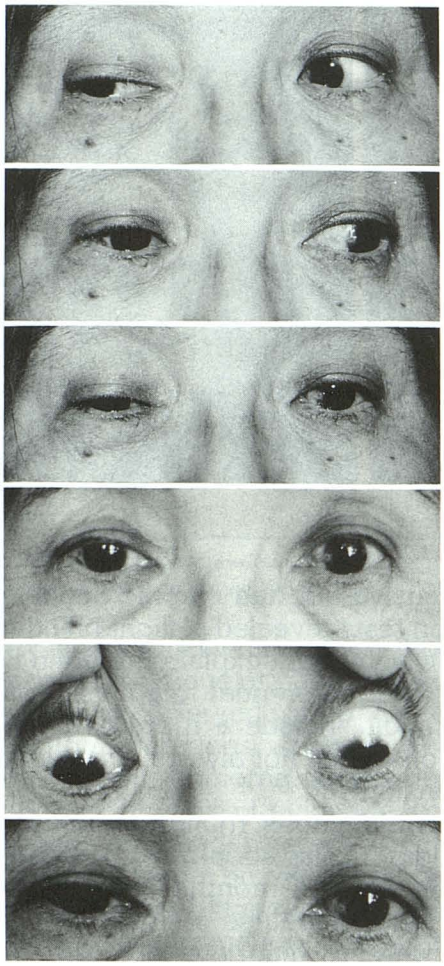

Fig 1. Photograph illustrates right third nerve palsy on admission, February 25, 1992. *Top* to *bottom*, right gaze, left gaze, forward gaze (illustrating ptosis), upward gaze, downward gaze, and convergence. The right eye has limited adduction, elevation, depression, and convergence. Elevation of the left eye is also limited.

Discussion

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 (Fig 5). Widespread involvement of the third nerve fascicles within the midbrain is associated with a variety of neurologic symptoms. Damage to the ventral fascicles and adjacent cerebral peduncle produces ipsilateral third nerve palsy and contralateral hemiparesis (Weber syndrome) (5). Involvement of fascicles within the red nucleus leads to oculomotor palsy with contralateral involuntary movement (Benedikt syndrome). Extension of such a lesion to the brachium conjunctivum produces additional contralateral ataxia (Claude syndrome) (5). In our patient, the hemorrhage in the anterior tegmentum of the right midbrain involved exactly the distal portion of the oculomotor fascicles (Fig 5). Because it did not include the other regions of the midbrain, our patient exhibited an isolated right third nerve palsy. Focal midbrain lesions, usually infarcts, occasionally involve a few third nerve fascicles and produce isolated third nerve palsy (1–4). However, isolated third nerve palsy caused by brain hemorrhage is rare (6, 7).

Keane reported the case of a 38-year-old man with isolated brain stem third nerve palsy caused by midbrain hemorrhage (6). This patient exhibited a right third nerve palsy that was complete except for slight pupillary reactivity, and the contralateral left eye had full extraocular movements. In our patient, the light reaction of the right pupil was absent, and elevation of the contralateral left eye was limited (Fig 1). The subnucleus for superior rectus function on either side of the brain stem gives rise to fibers that innervate the contralateral (and to a lesser extent, the ipsilateral) superior rectus muscle (5). Lesions that affect this region, while causing

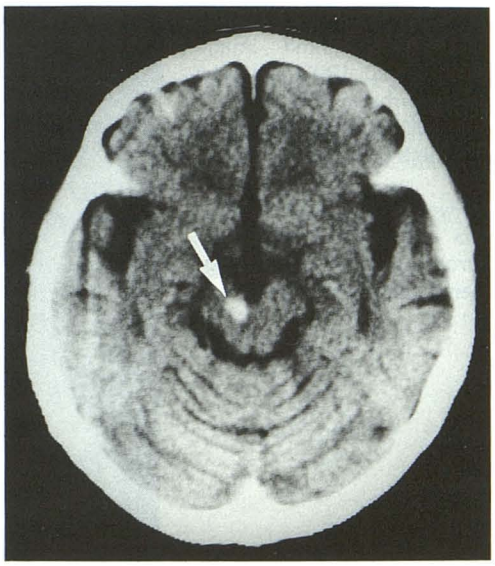

Fig 2. Computed tomography on admission (February 25, 1992) revealed a lesion of high density (72.8 Hounsfield units; *arrow*) in the right midbrain. The abnormality lay anteriorly in the tegmentum, most likely affecting the distal portion of the oculomotor fascicles.

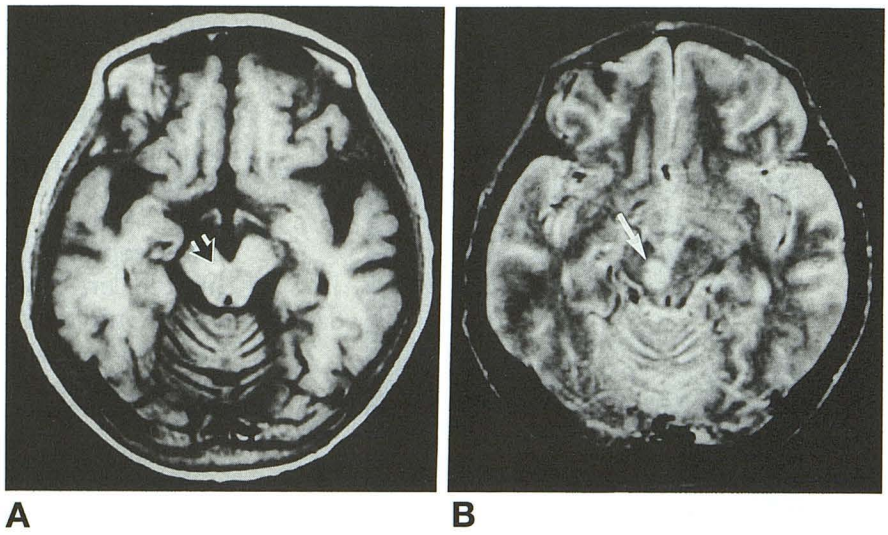

Fig 3. *A*, MR on March 5, 1992, 9 days after the onset of bleeding. A T1-weighted image (380/15/2) reveals a slight reduction in size from that on computed tomography at admission.

B, In the T2-weighted image (2000/100/1), the high-intensity lesion (*arrow*), probably brain edema, partially involves the red nucleus, cerebral peduncle, and oculomotor nucleus.

ipsilateral deficiencies of superior rectus, medial rectus, inferior rectus, and/or inferior oblique function, thus also produce a limitation of elevation in the contralateral eye from an impairment of superior rectus function (5). As shown in Figure 3B, edema around the brain hemorrhage involved the oculomotor complex; the function of the left superior rectus muscle, innervated by the right subnucleus, was impaired in our patient.

The pathogenesis of the hemorrhage remains unclear, because cerebral angiography was not performed. The patient was normotensive but had chronic hepatitis. Coagulation tests revealed a slight bleeding tendency, which might explain the brain hemorrhage. The differential diagnosis of such cases should include a ruptured perforating artery (often, but not always, associated with hypertension).

An arteriovenous malformation or a venous angioma also should be considered. Repeat MR study 3 months after the hemorrhage (Fig 4) revealed a small low-intensity lesion with a high-intensity rim in T1- and T2-weighted images. The central low-

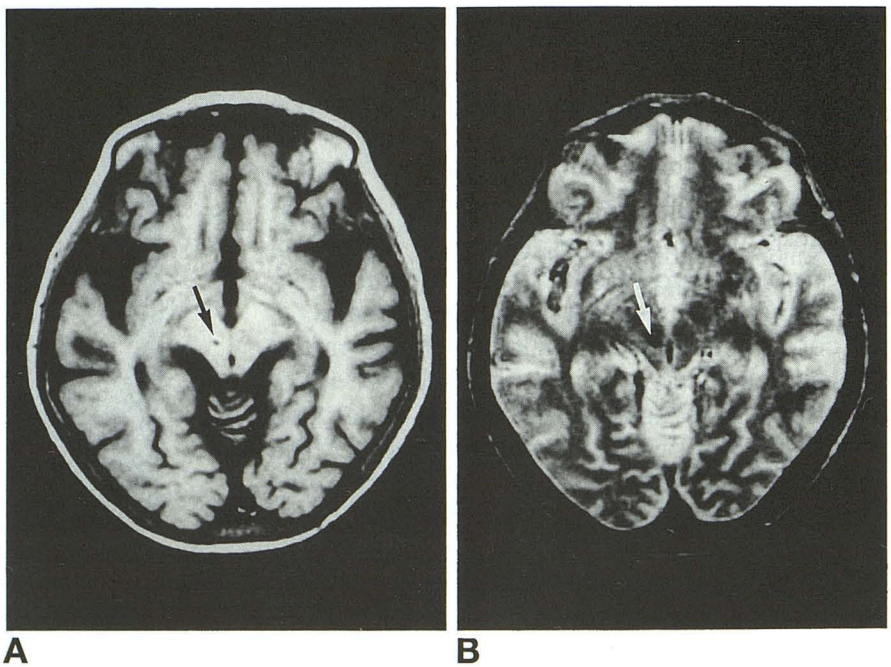

Fig 4. *A*, T1-weighted image (380/15) on May 20, 1992, 3 months after the hemorrhage. MR shows a small lesion of low intensity with a rim of slight high intensity in the midbrain.

B, T2-weighted image (2000/100) also shows a low-intensity lesion (*arrow*) in the midbrain, suggesting hemosiderin deposition.

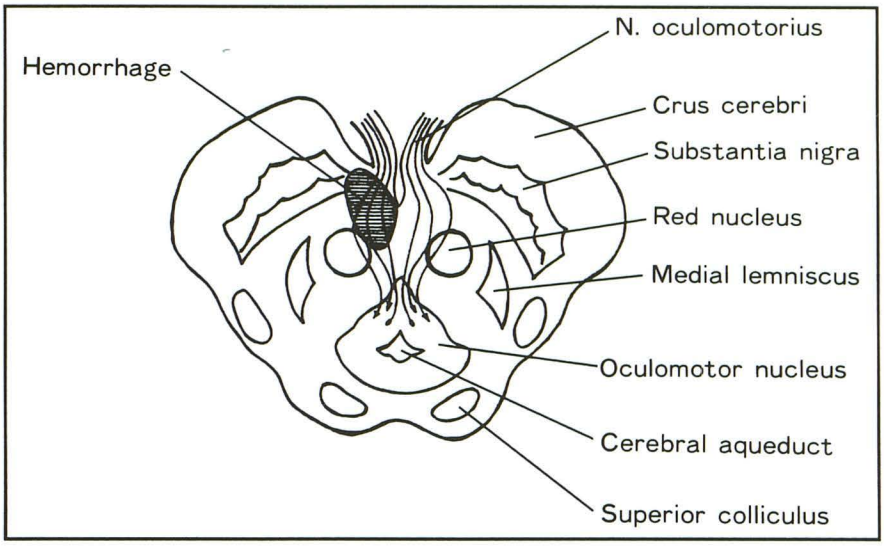

Fig 5. Diagram of an axial section through the midbrain. The lesion is on the *right* side, and normal anatomy of the region and relevant structures are on the *left* side.

intensity area suggested the deposition of hemosiderin, not a nidus or calcification. These results may be observed with occult cerebral vascular malformations, such as cavernous angioma, venous angioma, and capillary telangiectasia. The lack of evidence for an enlarged nidus and feeder vessel, which would produce a flow signal on the T2-weighted image, excludes the diagnosis of a nonoccult arteriovenous malformation. The patient may have bled from an occult cerebral vascular malformation.

Finally, although the patient had a history of diabetes mellitus, neuroradiologic findings in computed tomography and MR revealed that her isolated third nerve palsy was not caused by that disease alone.

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