Although stroke has not been recognized as an important cause of isolated ocular motor nerve palsy in the previous review articles, recent reports suggest that intrinsic brain stem lesions including strokes may be an important cause of isolated ocular nerve palsy. Reports of strokes that produce isolated oculomotor palsy without significant nonocular signs have been rare. Biller et al described a patient with brain stem infarction identified by computed tomographic (CT) scan who showed bilateral oculomotor paresis and transient Babinski signs. More recently, a series of reports described patients with midbrain hemorrhage or infarction who exhibited oculomotor palsies and transient Babinski signs. We studied seven patients with brain stem stroke who had ocular motor palsies as the only or predominant neurological manifestation, and we correlated their clinical features with radiological findings. Five patients had small strokes in the area of oculomotor nuclei or fascicles and showed various combinations of oculomotor disturbances. Three of these patients showed vertical gaze paresis of the opposite eye. One patient with a small hemorrhage near the aqueduct of Sylvius showed fourth nerve palsy on the contralateral side, and a patient with a small infarct in the pontine tegmentum showed isolated sixth nerve palsy. Nonocular minor neurological signs included trigeminal sensory changes in four patients and clumsy arm in two. Ocular and nonocular dysfunctions generally improved within several months. Radiological findings of the brain stem correlated well with the ocular signs.

Conclusions: We conclude that acute brain stem stroke should be included in the differential diagnosis of isolated ocular motor nerve palsies and that appropriate diagnostic investigations should be performed in these cases. (Stroke 1993;24:581–586)

KEY WORDS • brain stem • cerebrovascular disorders • oculomotor nerve

Subjects and Methods

We studied seven patients with acute brain stem stroke with ocular motor nerve palsy as the sole or a predominant neurological sign and correlated their clinical features with radiological findings. One patient was evaluated by CT (GE/9800) scan and six by magnetic resonance imaging (MRI) with a 1.5-T superconducting unit (GE). Axial T2- (repetition time [TR], 2,500 msec; echo time [TE], 80 msec) and T1- (TR, 600 msec; TE, 20 msec) weighted and sagittal T1-weighted scans were generated with a slice thickness of 5 mm. Angiography was performed in two patients.

Case Reports

Third nerve palsy. Patient 1, a 60-year-old hypertensive male smoker, developed sudden dizziness and diplopia. Examination showed right oculomotor nerve palsy without definite ptosis or pupillary changes. In primary position, his right eye deviated outward and downward; in the opposite eye, upward and downward gaze pareses were noted. In addition, he exhibited a clumsy left hand and dysesthesia on pinprick and touch on the left side of his face and left hand. Results of routine laboratory tests were normal. Brain MRI showed a small infarct in the right midbrain (Figure 1). The patient's oculomotor function normalized 6 months after onset.

Patient 2, a 58-year-old hypertensive woman, became drowsy and complained of diplopia and numbness in the right upper peribuccal area. Examination showed left oculomotor nerve palsy, with bilateral ptosis worse in her left eye. Attempted adduction of the left eye was accompanied by transient abducting nystagmus of the
right eye. The pupil size was bigger in the left eye (6 mm) than the right (3 mm), and the light reflex was sluggish in the left. There was upward gaze limitation of the right eye, although it was in the neutral position on straight gaze. Neurological examination showed dysesthesia in the right upper peribuccal area and a mildly weak and ataxic right arm. On gait, she tended to veer to the right. Brain MRI showed a small infarct in the left midbrain (Figure 2). Her right-arm weakness resolved within 2 days, and oculomotor abnormalities were normalized 5 months after onset.

Patient 3, a 53-year-old man with a history of angina and hyperlipidemia, had sudden diplopia and painful left facial numbness. Examination showed right-sided oculomotor nerve palsy with ptosis. His right eye was mildly deviated downward and outward on primary gaze. Pupils were equal and reactive on both sides. Ocular movements of his left eye were normal. Sensory examination revealed facial hyperalgesia in the left V1 and V2 areas. Brain MRI showed ischemic changes in the right midbrain (Figure 3). Three months later, the patient's oculomotor function was fully recovered.

Patient 4, a hypertensive 38-year-old man with a history of congestive heart failure, developed sudden headache and diplopia. Examination showed left-sided complete oculomotor nerve palsy, including ptosis, and dilated, fixed pupil. On primary gaze, his left eye deviated outward and downward; in addition, upward gaze limitation was noted in his right eye. Brain CT scan showed a small hemorrhage in the left midbrain (Figure 4). An angiogram showed normal findings. During 7 days of admission, there was a slight improvement in his oculomotor function. After discharge, however, he was lost to follow-up.
Patient 5, a previously healthy 42-year-old woman, developed sudden dizziness and diplopia on downward gaze. Examination showed isolated inferior rectus paresis of her right eye, with a mild hypertropia of the right eye in primary gaze. Pupils were equal, and ptosis was absent in both eyes. Results of laboratory tests were normal except for positive lupus anticoagulant. Brain MRI showed a small infarct in the right midbrain (Figure 5). Two months later her ocular motor function had improved, but she still had diplopia on looking downward.

Fourth nerve palsy. Patient 6 (previously described), a 38-year-old man, developed a dull headache and dizziness associated with diplopia on looking downward. He also experienced a tingling sensation in his right cheek. Three weeks later, when he came to Asan Medical Center, examination showed isolated right superior oblique palsy: limitation of his right eye in the left downward direction and 10 prism diopters of right hypertropia with his head tilted to the right. Pupil sizes and light reflexes were normal in both eyes. In addition there was decreased facial sensation over the right V2 and V3 areas. T1- and T2-weighted MRI showed a linear low-intensity signal in the left midbrain (Figure 6). An angiogram showed normal findings. One month later his facial paresthesia improved, and ocular movements were completely recovered 4 months later.

Sixth nerve palsy. Patient 7, a hypertensive 76-year-old man, developed dizziness and diplopia. He exhibited a right lateral rectus paresis with a slight esotropia of the right eye on straight gaze. Otherwise, his neurological examination was normal. Brain MRI revealed a small infarct in the right pontine tegmentum (Figure 7). His oculomotor dysfunction fully improved 3 months later.

Results

In this series, we studied seven patients (five men and two women, 38–76 [mean, 52] years of age). Five had small infarcts, and two had hemorrhages. The risk

![Figure 4](image4.png)

**Figure 4.** Patient 4. Computed tomographic scan shows a hemorrhage in the left paramedian midbrain probably involving the oculomotor fascicles (arrows, left panel). Note the extension of the hemorrhage in the most rostral midbrain that may involve the pupillary fibers (arrows, right panel).

![Figure 5](image5.png)

**Figure 5.** Patient 5. Magnetic resonance image shows a small infarct (arrows) in the left paramedian rostral midbrain that may selectively involve the subnucleus of the inferior rectus.
factors for stroke included hypertension in four, smoking in one, hyperlipidemia in one, and coronary heart disease in one. One patient showed a positive result for lupus anticoagulant. One young man (patient 6) suffered small hemorrhage probably due to vascular malformation. Five patients showed third nerve palsies; ptosis was noted in three, one of them bilaterally.

Pupillary changes were observed in two patients. Four patients had weakness of all muscles innervated by the oculomotor nerve, whereas one showed a selective paresis of the inferior rectus. Three patients showed vertical gaze palsy on the opposite eye (upward gaze palsy in two and upward and downward gaze palsy in one). One patient had trochlear nerve palsy, and one showed isolated lateral rectus palsy. In all patients, diplopia secondary to ocular paresis was the only symptom or the major symptom. Minor nonocular neurological manifestations included facial numbness in four (with additional sensory change in the hand of one patient) and ataxic and clumsy arm in two. One patient was drowsy initially. In all, ocular motor dysfunctions improved within 6 months, although one patient was not adequately followed up. Radiological evaluation showed lesions in the brain stem (midbrain in six and pontine tegmentum in one) that correlated well with clinical manifestations (see “Discussion”).

Discussion

MRI findings in our patients suggest that patients 2 and 5 had lesions in the third nerve nucleus complex; therefore, the bilateral ptosis of patient 2 was probably due to the involvement of the central caudal nucleus, which is known to bilaterally innervate the levator palpebrae. There have been occasional reports of a single nuclear lesion causing bilateral ptosis, and Conway et al reported an unusual patient with isolated bilateral complete ptosis secondary to lesions confined to the central caudal nuclear complex.

It is of interest that patient 5 had isolated inferior rectus paresis. Although rare, there have been reports of isolated inferior rectus paresis due to intra-axial brain stem lesions. It is not clear whether the responsible lesion in these cases is in the fascicles or nucleus. Recently, Pusateri et al described a patient with isolated inferior rectus paresis in whom MRI findings correlated with the autopsy findings of metastasis on the third nerve nucleus. According to Warwick’s scheme, the inferior rectus subnucleus is isolated from other subnuclei at the most rostral part of the oculomotor complex. Therefore, selective involvement of this area may cause the isolated inferior rectus paresis.

Clinical and MRI findings of patients 1, 3, and 4 suggest lesions are in the oculomotor nerve fascicles. Fascicular lesions were reported to cause various combinations of signs such as isolated inferior oblique paresis, isolated pupillary changes, or divisional nerve paresis that suggests intra-axial divisional differentiation. However, Castro et al suggested topographical arrangement of the fibers from lateral to medial as follows: inferior oblique, superior rectus, medial rectus, levator palpebrae, inferior rectus, and pupillary fibers.

Our patients with fascicular lesion showed diffuse involvement of the fibers, which did not support any particular topographical arrangement. However, the spared pupillary dysfunction in two cases (patients 1 and 3) deserves mention. Fascicular lesions sparing the pupil have been described by a number of authors, whereas patients described by others showed pupillary involvement; the patients described by Keane and one (case 2) reported by Shuaib and Murphy had paramedian midbrain hemi-
orrhage extending rostrally to the hypothalamus. Shuaib et al described an unusual patient with isolated pupillary deficit who had a small hemorrhage in the paramedian, rostral midbrain. In our series, patient 4 (the only one with a fasicular lesion who showed pupillary involvement) had a hemorrhage extending into the most central—rostral portion of the midbrain (Figure 4, right panel), whereas patients 1 and 3 showed lesions situated more caudally. Therefore, within widespread fascicles, the pupillary fibers seem to run in the most rostral and central portion.

It is noteworthy that three patients with third nerve palsy showed vertical gaze palsy in the opposite eye. Vertical saccade for both upward and downward gaze has been known to be related to the cell groups of the rostral interstitial nucleus of medial longitudinal fasciculus (RiMLF), which is located in the anterosuperior portion of the oculomotor complex. It has recently been documented that a unilateral lesion of RiMLF can result in bilateral vertical gaze paresis. Our patients 1, 2, and 4 probably had lesions involving the RiMLF. In patient 2, the possibility of contralateral superior rectus paresis secondary to nuclear involvement cannot be ruled out. However, the patient's contralateral eye was not hypotropic on straight gaze, and there was a limitation of the eye movements in the inferior oblique direction, both of which support a diagnosis of supranuclear upward gaze defect. Patients with nuclear lesions with or without involvement of the contralateral superior rectus have been reported, and Bogousslavsky et al described a patient with midbrain infarction that caused contralateral superior rectus paresis, probably due to the involvement of the prenuclear fibers.

There have been only a few reports in which fourth nerve palsy was an isolated or predominant manifestation of stroke. Patient 6 showed isolated trochlear nerve palsy and trigeminal sensory change in the contralateral side of the lesion, probably due to involvement of the trochlear fascicles and ascending trigeminothalamic fibers. Including patient 6, four of our patients showed trigeminal sensory changes, sometimes in the restricted area of the face. This can be explained by the close association of the trigeminothalamic second-order neurons with the oculomotor or trochlear nuclei in the human brain stem. The patients described by Growdon et al and Breen et al also showed facial sensory changes on the side contralateral to the lesion. Therefore, careful examination of facial sensation may help localize the lesion in patients with oculomotor nerve palsy. Patient 1 had additional numbness in the hand, thus demonstrating a choro-oral sensory syndrome. Midbrain lesion as a cause of choro-oral syndrome has been previously described.

To our knowledge, there have been three reports that described small pontine infarcts as a cause of isolated sixth nerve palsy. MRI of our patient 7 revealed a tiny pontine infarct impinging on the abducens fascicles but probably sparing the corticospinal tracts and the medial lemniscus. Our patient and previously reported cases did not show any signs or symptoms of brain stem dysfunction, and their sixth nerve palsies improved completely within several months. Therefore, it seems impossible to distinguish a central from a peripheral cause of sixth nerve palsy in these cases. It is suggested that pontine infarcts be considered as a possible cause of isolated sixth nerve palsy in elderly patients with hypertension or diabetes mellitus. However, it remains to be determined whether every patient should be evaluated by MRI.

In conclusion, clinical findings in our patients suggest that brain stem stroke can manifest itself with isolated or predominant oculomotor nerve palsy, which is sometimes indistinguishable from peripheral nerve lesions (patients 3, 4, and 7). As with peripheral lesions, the prognosis for these patients is generally good, and ocular motor dysfunction tends to improve or resolve within several months. Subtle signs and symptoms such as trigeminal sensory change, vertical paresis of the contralateral eye, clumsy hands, or transient drowsiness may help localize the brain stem lesion and warrant MRI evaluation, which usually correlates well with ocular and nonocular signs in these patients.

References


Isolated or predominant ocular motor nerve palsy as a manifestation of brain stem stroke.

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