In the present case as well as in those reported by Linden et al\(^1\) and Youl et al.,\(^2\) a pain in one arm is the initial symptom of extracranial vertebral artery dissection. In our case and in that of Linden et al, a transient ST elevation was observed on ECG. This cardiac abnormality together with the fact that pain was in the left arm led Linden et al\(^1\) to presume a cardiac origin, possibly a concomitant coronary artery dissection. The quality of the pain and its right-sided topography in our case makes this explanation unlikely. Another mechanism could be an upper cervical radicular ischemia because the first radicular arteries are branches of vertebral arteries. The absence of sensorimotor deficit in the C5–C6 territory and the normality of the bicipital reflex do not rule out this hypothesis, which might be confirmed by electrophysiological studies. The ST elevation might be of cardiac origin, but it can also be hypothesized that it is secondary to the brain stem injury.\(^3\) Whatever its mechanism, such an observation illustrates that unilateral brachial pain can be the initial symptom of a vertebral artery dissection. Such a misleading presentation should prompt not only radiological investigation of cervical vertebrae but also ultrasound and MRI investigation of vertebral arteries.

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Bilateral Internuclear Ophthalmoplegia, Ataxia, and Tremor from a Midbrain Infarct

Bilateral internuclear ophthalmoplegia (INO) and ataxia are rarely associated with brainstem infarction.\(^1\),\(^2\) Moreover, a combination of bilateral INO, ataxia, and tremor from a focal infarction is very rare.\(^3\) We describe a patient with bilateral INO, ataxia, and tremor due to a focal infarction in the paramedian midbrain tegmentum. To our knowledge, this is the first report in which a focal lesion responsible for the above clinical manifestations is detectable.

A 57-year-old woman suddenly developed dysarthria and gait disturbance. She had undergone mastectomy due to breast cancer at age 52, and had had diabetic mellitus since age 55. Eight days after the onset, the patient entered the Fifth Department of Internal Medicine, Hyogo College of Medicine. On admission, her blood pressure was 116/74 mm Hg, and her pulse was regular at a rate of 72 beats per minute. General physical examination was negative. She was drowsy and dysarthric, but correctly responsive to verbal commands. Neurological examination revealed bilateral INO. The primary position of the right eye was in the midline, but the left eye was extropic. Adduction of both eyes was impaired, whereas abduction of both eyes was preserved with abducting nystagmus. Upward gaze was mildly limited on both sides. Downward gaze was preserved with downbeat nystagmus. Convergence was impaired. Caloric stimulation evoked only abduction of each eye. Pupils were 2 mm, round, and isocoric, and reacted to light. The eyelids were not ptotic. The other cranial nerves were spared. Muscle tone and power were normal. Deep tendon reflexes were normal in the upper limbs and diminished in the lower limbs. No pathological reflexes were present. Limb coordination and equilibrium were severely disturbed. The patient was quite atactic on bilateral finger-to-nose and heel-to-knee testing. Sitting was difficult without support. Superficial and deep sensations were preserved.

Computed tomographic (CT) scan showed a low-density area in the paramedian midbrain tegmentum extending to the left cerebral peduncle (Figure 1, top panel). Vertebral angiography disclosed complete obstruction of the left vertebral artery without stenosis in the basilar and posterior cerebral arteries. T2-weighted magnetic resonance imaging (MRI) revealed a high signal intensity in the paramedian midbrain tegmentum and left cerebral peduncle (Figure 1, bottom panel). Blood chemical examinations were normal except that fasting blood sugar was 160 mg/dl. The cerebrospinal fluid contained 3 mononuclear cells/mm\(^3\), 68 mg/dl protein, and 123 mg/dl glucose. Immunoglobulin G oligoclonal band was negative. The patient's condition remained unchanged for 5 weeks, and thereafter rhythmic tremors appeared in the head and limbs at rest and during action. The coarse tremor at 3–4 Hz was almost symmetric and predominant in the proximal portions (Figure 2). The rhythmic movements contained bidirectional rotation of the head and supination–pronation, abduction–adduction, and internal–lateral rotation of the upper and lower limbs. The tremor was intensified by voluntary motions and disappeared during sleep. Muscle rigidity was absent in the neck and limbs.

FIGURE 1. X-ray computed tomographic scan (top panel) and T2-weighted magnetic resonance imaging (repetition time, 2,000 msec; echo time, 90 msec; bottom panel) showing a midbrain infarct in the paramedian tegmentum extending to the left cerebral peduncle.
Subsequent CT scan and MRI demonstrated the midbrain lesion similar to that previously noted.

On admittance, our patient had a left exotropia and impaired convergence as well as paretic adduction of each eye. Exotropia and loss of convergence are due to bilateral medial longitudinal fasciculus lesions and do not involve the oculomotor nucleus. Furthermore, gaze-evoked abducting nystagmus and downward nystagmus suggest INO rather than oculomotor nuclear lesions. Bilateral INO has been mostly observed in multiple sclerosis, but has been infrequently encountered in cerebral infarction. A combination of bilateral INO and ataxia has been rarely documented in brainstem infarction. In the present case, the paramedian midbrain tegmentum lesion could involve the bilateral medial longitudinal fasciculus and the decussation of the superior cerebellar peduncle, resulting in bilateral INO and ataxia. In addition, the patient exhibited rhythmic tremor in the head and limbs on both sides. The tremor differs from parkinsonian tremor in frequency and exacerbation on voluntary movements, but the features resemble involuntary movements called midbrain tremor or rubral tremor. The tremor may possibly be caused by a combined disturbance of the nigrostriatal pathway and the rubro-olivo-cerebello-rubral loop.

As mentioned above, the paramedian midbrain tegmentum lesion accounts for the unique clinical manifestations. The midbrain infarction is presumably ascribed to an embolus arising from the occluded vertebral artery. The present case suggests that a focal midbrain infarction causes bilateral involvement of the oculomotor, cerebellar, and extrapyramidal systems.

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