Benign Brainstem Hemorrhage Simulating Transient Ischemic Attack

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A 48-year-old hypertensive man had sudden onset of symptoms suggesting vertebrobasilar insufficiency, which were transient in nature lasting for only 4 hours. Brain computed tomography revealed a small hematoma in the pontomedullary junction. This is an uncommon presentation of benign brainstem hemorrhage simulating transient ischemic attack. We propose that computed tomographic scan using thin slices of 3mm to 5mm thickness at the level of brainstem is required before starting anticoagulation therapy for vertebrobasilar transient ischemic attack.

Key Words: Cerebral hemorrhage, brainstem, transient ischemic attack, computed tomography

Transient ischemic attack (TIA) is defined as focal neurologic symptoms lasting less than 24 hours caused by cerebrovascular ischemia (Ross Russell 1983). However, it has been reported that TIA-like symptoms can be resulted from various causes other than ischemia, such as brain tumor, chronic subdural hematoma as well as intracranial hemorrhage (Moster et al. 1983; Weissberg and Lacorte 1985; Kase and Caplan 1986), but all of cases reported were located in the cerebral hemisphere or cerebellum, not in the brainstem.

Most spontaneous brainstem hemorrhages occur in pons usually with poor prognoses resulting in severe neurologic deficits or death (Silverstein 1972). With the introduction of computed tomography (CT), however, it has been clearly demonstrated that a small hemorrhage occasionally develops in the brainstem with complete recovery or benign courses (Payne et al. 1978; Lavi et al. 1981; Morel-Maroger et al. 1982; Hommel et al. 1985), although none of those cases reported were appropriate to be called as TIA at our knowledge. We report a case who presented with transient brainstem dys-

function simulating TIA caused by a small brainstem hemorrhage.

CASE REPORT

A known hypertensive 48-year-old man developed sudden onset of dizziness, diplopia, paresthesia on the right face and the left hand, and gait difficulties. These symptoms spontaneously disappeared within 30 minutes, but mild gait disturbance with subjective dizzy feeling persisted. He visited emergency room approximately one hour after the symptom onset.

At the emergency room, the blood pressure was 190/100 mmHg, the pulse rate 104/minute and the body temperature 36.5°C. General physical examination did not reveal any abnormalities. On neurological examination, he was alert and oriented. The pupils were round and isocoric with prompt responses to light and accommodation. The eye movement appeared normal without nystagmus and diplopia. Corneal reflexes and gag reflexes were intact and symmetric. No facial weakness was found and the tongue was midline. Speech was normal. Perceptions of pinprick, touch, position and vibration were intact. Muscle tone and strength appeared normal. The tendon reflexes were active and symmetrical, and the plantar responses were flexor bilaterally. On the examination of cerebellar functions, he did
not show any difficulties on rapid alternating movement test and finger-to-nose test, but showed mild ataxia on tandem gait with swaying bilaterally. Romberg test was negative. On repeated examination three hours after his admission, his gait ataxia as well as subjective feeling of dizziness were completely recovered.

All performed laboratory examinations including blood cell count, urinalysis, serum electrolytes, serum lipid level, serum creatinine, blood urea nitrogen, serum transaminases were normal. However, the blood sugar was increased up to 165mg/dl in fasting and 236mg/dl at 2 hours after meal. Brain computed tomography using 5mm slice thickness at the level of the brainstem revealed a small hematoma, about 5mm in diameter, in the right paramedian portion of the dorsal pontomedullary junction. The hematoma was seen only in one slice. In addition to that, a small contrast enhancing lesion was found in the deep portion of left cerebellar hemisphere, which was suggestive of a vascular malformation (Fig. 1). Vertebral angiography taken on the 13th hospital day revealed a spider-like venous structure in the deep portion of left cerebellar hemisphere, which was consistent with a venous angioma.

He was discharged at the 15th hospital day without any residual symptoms and signs. During one year follow up, no further attacks recurred.

DISCUSSION

The initial symptoms of this patient compelled us to make the diagnosis of transient ischemic attack in the vertebrobasilar arterial territory until the immediately performed CT scan revealed a small hematoma in the pontomedullary junction. Although a venous angioma confined to the cerebellum, confirmed by angiography in our case, may occasionally cause subarachnoid as well as intraparenchymal hemorrhages (Biller et al. 1985), the hematoma causing the transient brainstem dysfunction seemed to be resulted from a hypertensive vasculopathy rather than from the venous angioma because most cerebellar venous angiomas bleed within the cerebellum, adjacent to the venous angioma (Nishizaki et al. 1986). However, considering the proposal that venous angiomas of the posterior fossa may be represented as anomalous venous drainage compensating lack of normally developed venous drainage systems in that area (Senegor et al. 1983), we cannot completely exclude the possibility of the hemorrhage being caused by a cryptic venous anomaly extended from the venous angioma.

Although the prognoses of brainstem hemorrhag-
es are known to be primarily affected by their size (Masiyama et al. 1985), their good recoveries seem to be also related to their location, dorsal paramedi-an portion of the lower pons and upper medulla in which most previously reported benign brainstem hemorrhages with good recoveries (Payne et al. 1978; Lavi et al. 1981; Morel-Maroger et al. 1982; Hommel et al. 1985) were chiefly located including the present case. The reason of their better recoveries may be explained by sparing of major motor and sensory pathways which located ventrally and laterally at the pontomedullary level. The transient neurologic dysfunctions caused by hemorrhage are thought to be due to its pressure effects and/or secondary ischemia rather than direct tissue dam-age (Moonis et al. 1988).

The rarity of brainstem hemorrhage reported as the cause of TIA might be related the fact of major neural structures being compactly organized in the brainstem, thus the size of hematoma should be very small. As demonstrated in our case, the size of hematoma was only 5mm in diameter, which might be easily missed if one used conventional CT slices of 10 mm thickness. Considering that an anticoagulation therapy is often used in cases of brainstem ischemia, using thin slices of 3 to 5mm thickness at the level of posterior fossa during CT scanning in patients with vertebrobasilar TIA certainly cannot be overemphasized.

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