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Pure Motor Hemiplegia with Conjugate Lateral Gaze Palsy in Pontine Lacunar Infarction

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The combination of pure motor hemiplegia and horizontal gaze palsy is a rare but identifiable lacunar syndrome. Among horizontal gaze palsies, one-and-a-half syndrome and abducens nerve palsy are reported to be associated with pure motor hemiplegia in pontine lacunar infarction. Although conjugate lateral gaze palsy is also hypothesized, pure motor hemiplegia with conjugate lateral gaze palsy has never been reported. We present a 75-year-old man who showed right hemiparesis and impaired left horizontal conjugate eyeball movement. Both the findings of the brain CT scan and those of the MRI study were consistent with a small infarction in the left midpontine tegmentum. Magnetic resonance angiography revealed no stenotic narrowing of the vertebrobasilar artery. Radiological findings suggested that pure motor hemiplegia with conjugate lateral gaze palsy, in our patient, might have been produced by the occlusion of a single penetrating branch of the basilar artery.

Key Words: Cerebrovascular disorders, cerebral infarction, hemiplegia, oculomotor palsy, pons

Since the concept of lacunar syndromes was established in the 1960s by Fisher and his colleagues, many new syndromes have been identified (Fisher, 1991). Pure motor hemiplegia (PMH), in pontine lacunar infarction, is described as being associated with such oculomotor disturbances as abducens nerve palsy and one-and-a-half syndrome (Fisher and Caplan, 1971; Hommel *et al.* 1990; Besson and Hommel, 1993). However, to the best of our knowledge, the combination of PMH and conjugate lateral gaze palsy has never been reported. We present a patient who showed PMH and transient conjugate lateral gaze palsy from pontine lacunar infarction. The probable pathogenic mechanism is also discussed.

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CASE REPORT

A 75 year-old right-handed diabetic man with a history of cigarette smoking and alcohol drinking suddenly developed weakness in his right extremities, along with dysarthria. Three days later, on admission, his blood pressure was 120/80 mmHg, and his pulse was regular at 72 beats per minute. The general physical examination was normal. He was alert, well oriented, and dysarthric. The pupils were isocoric and reactive to light. Horizontal conjugate eyeball movement was impaired. He was unable to move his eyes to the left past the midline. However, both right lateral conjugate and vertical eyeball movements were normal and no nystagmus was observed. Moderate weakness was present in his right upper and lower extremities. There were no additional neurological abnormalities. His hori-

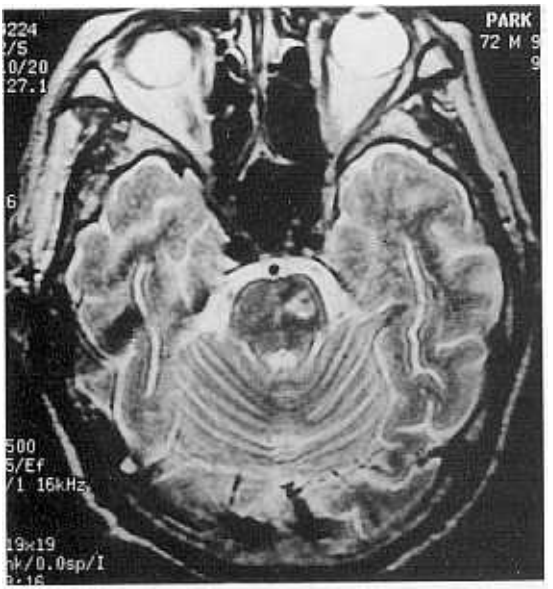

Fig. 1. Axial T_2-weighted magnetic resonance imaging of the brain shows a lesion of small high signal intensity in the left midpontine tegmentum, consistent with an infarction.

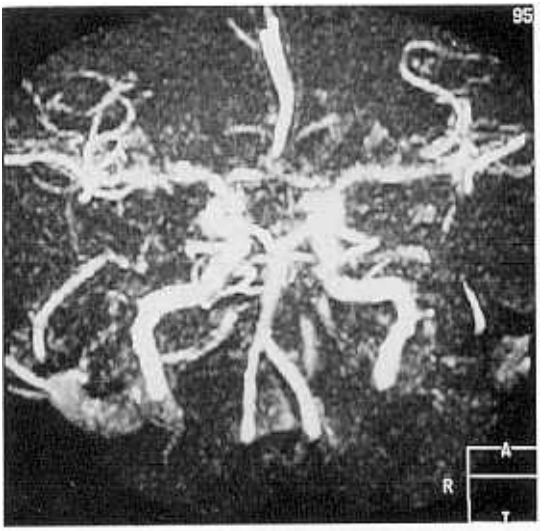

Fig. 2. Magnetic resonance angiography shows neither occlusion nor stenotic narrowing of the vertebrobasilar artery.

zontal gaze palsy improved after a two week follow-up, when no gaze palsy was observed. The brain CT scan obtained immediately after admission and the follow-up MRI performed with a 1.5-T Signa (General Electrics) after 1.5 months showed a finding consistent with a small infarction in the left midpontine tegmentum (Fig. 1). Magnetic resonance angiography revealed no stenotic narrowing of the vertebrobasilar artery (Fig. 2).

DISCUSSION

The association of PMH and oculomotor palsy has rarely been reported in pontine lacunar infarction. Since Fisher and Caplan (1971) reported a pathologically proven patient who showed PMH with one-and-a-half syndrome in pontine lacunar infarction, the association of PMH and horizontal gaze palsy has been recognized as a form of lacunar syndrome (Fisher, 1982). However, few patients afflicted with PMH and abducens nerve palsy, and few with PMH and one-and-a-half syndrome were described (Fisher and Caplan, 1971; Hommel *et al.* 1990; Besson and Hommel, 1993). Although it is suggested that PMH with conjugate lateral gaze palsy is a possible form of lacunar syndrome in pontine infarction (Fisher, 1991), this exact combination has never been reported. Our patient is the first case to present as PMH with conjugate lateral gaze palsy from pontine lacunar infarction as demonstrated by CT scan and MRI.

An infarct affecting the pontine gaze center as well as the pyramidal tract may produce a combination of PMH and conjugate lateral gaze palsy. A lesion, demonstrated by MRI in our patient, was in the left midpontine tegmental area involving part of the pyramidal tract. The lesion was presumed to be just ventral to the nucleus reticularis tegmenti pontis to where efferent fibers from the cortical eye field project (Leichnetz *et al.* 1984). The transient nature of conjugate left lateral gaze palsy in our patient may be explained by bilateral cortical projections to the nucleus reticularis tegmenti pontis, or by an indirect

involvement of the nucleus reticularis tegmenti pontis, or both.

Besson and Hommel (1993) suggest that an infarct involving the pontine gaze center as well as the pyramidal tract is usually larger than a lacune and is usually caused by an occlusion either of the basilar artery or of the ostia of several paramedian arteries. However, in our patient, the size of the lesion was less than 1 cm in diameter, and located in the territory of an anterolateral penetrating pontine artery (Pullicino, 1993). Neither an occlusion nor a stenotic narrowing of the basilar artery was found. These radiological findings suggest that PMH with conjugate lateral gaze palsy in our patient was due to the occlusion of a single penetrating branch of the basilar artery, and this is consistent with the findings in the only pathologically proven case with PMH and one-and-a-half syndrome (Fisher and Caplan, 1971).

In conclusion, we demonstrate that pontine lacunar infarction may manifest with PMH with conjugate lateral gaze palsy by an occlusion of a single penetrating branch of the basilar artery.

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