# Case Report

## A CASE OF PURE MOTOR MONOPARESIS DUE TO PONTINE INFARCTION

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(Received July 26, 1990)

Pure motor hemiplegia (PMH) is a common manifestation of lacunar infarction. However, pure motor monoparesis (PMM), isolated corticospinal tract deficits confined to one limb, have received little attention. We observed a patients in whom the clinical feature was compatible with PMM and discussed the possible pathomechanism of PMM.

Keywords: Pure motor monoparesis, pure motor hemiplegia, lacunar stroke

Pure motor hemiparesis (PMH) is one of the syndromes of lacunar stroke, defined by Fisher et al. (1965) as complete or incomplete paralysis of the face, arm, and leg on one side, unaccompanied by sensory signs, visual field defects, aphasia, or apractognosia. In most cases, it is caused by a lacunar infarction in the internal capsule, basis pontis, rarely the medullary pyramid or cerebral peduncle (Donnan et al., 1982). There are few reports of isolated corticospinal tract deficits confined to one limb, i.e., pure motor monoparesis (PMM). Ashizawa et al. reported 5 patients with PMM and in each patient the case had a space-occupying lesion in the high cortical surface of the cerebral hemiphere contralateral to the PMM (Ashizawa et al., 1986). Finally, they emphasized that PMM is seldom due to lacunar or other vascular disease but rather is frequently caused by a mass lesion superficially in the cerebral hemisphere. We had an opportunity to observe a patient in whom the clinical feature was compatible with PMM, and the responsible lesion causing PMM was considered to be located in the basis pontis.

## CASE REPORT

A 65-year-old right-handed man with a history of poorly controlled hypertension had a sudden onset of weakness in the left arm, and then he was seen at our department. The patient denied any sensory symptoms on admission. On admission, his blood pressure was 180/102 mm Hg and general examination was normal.

On neurological examination the patient was alert and intelligent. The neck was supple, and the cranial nerves were normal. Both sensations and cerebellar functions were normal. Manual muscle testing showed 3/5 to 4/5 weakness in the left upper extremity. Deep tendon reflexes were brisk throughout but symmetrical. The Babinski signs were negative. The skull X-ray was normal as were the X-ray of cervical and lumbar spine. Chest X-ray and EKG showed left ventricular hypertrophy. The CBC



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FIGURE 1 CT scan 5 days after the onset showed a nonenhancing, density lesion in the right basis pontis

and blood chemistry were normal. Computed tomography (CT) of the brain 5 days after the onset showed a low density area in the right basis pontis, which did not enhance and was felt to be consistent with an infarction (Figure 1). The remainder of the CT was normal, with no evidence of hematoma or space-occupying lesions. His condition improved gradually during the succeeding weeks, with absolute bed rest. At the time of discharge from hospital no neurological deficits were noted.

#### DISCUSSION

Fisher et al. (1965) defined PMH as a complete or incomplete paralysis of the face, arm, and leg on one side unaccompanied by sensory signs, visual field defect, dysphasia, or apractognosia. PMH is also a common manifestation of lacunar infarction, consisting of 60% of lacunar stroke (Iwasaki & Kinoshita, 1988; Mohr et al., 1977). Although it should be clarified whether PMM is a mild form of PMH or whether these two syndromes have to be regarded as two separate syndromes, it has only rarely been seen in lacunar infarction. Donnan et al. reported that a total of 58 patients with pure

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motor deficits due to lacunar infarcts, only 1 had PMM (Donnan et al., 1982). Nelson et al. reported that 1 of 26 patients had PMM (Nelson et al., 1980). The clinical features of our case were consistent with PMM. In the present case, the lacunar infarction of the right basis pontis was noted. The basis pontis is across both the corticospinal and corticobulbar tracts and it is unknown why both tracts, corresponding to the lower limb were not involved. PMM theoretically can be caused by any condition that damages the corticospinal tract. Although PMM has been reported in brainstem glioma (Rothma & Olanow, 1981) and lenticocapsular hemorrhage (Soisson et al., 1982), it rapidly progressed to PMH in these patients, suggesting that a discrete lesion small enough to cause PMM may be rare in the small motor pathway. Interestingly, our case did not evolve to PMH. Ashizawa et al. (Ashizawa et al., 1986) concluded that acute PMM is often caused by a space-occupying lesion in the contralateral superficial frontoparietal cortex rather than by lacunar infarction, although our case emphasized that acute PMM may be caused by lacunar infarction and the physician should recognize that acute PMM can be caused not only spaceoccupying lesions in the contralateral superficial cortex but also lacunar infarction in the brainstem.

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