# Pure Motor Hemiplegia Secondary to a Saccular Basilar Artery Aneurysm

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Pure motor hemiplegia is the most commonly encountered lacunar syndrome and is classically associated with small infarctions in the contralateral internal capsule or basis pontis. Pure motor hemiplegia has also been observed secondary to a wide variety of other vascular and nonvascular focal central nervous system processes. We describe a patient with pure motor hemiplegia associated with a saccular basilar artery aneurysm causing a lacunar infarction of the cerebral peduncle. (*Stroke* 1988;19:104-107)

ure motor hemiparesis (PMH) refers to a pure motor stroke that produces weakness of the contralateral face, arm, and leg without associated sensory deficits, homonymous hemianopsia, aphasia, or agnosia.<sup>1</sup> The syndrome was initially reported by Fisher and Curry<sup>2</sup> who observed, pathologically, lacunar infarcts in the contralateral internal capsule or basis pontis. Subsequently, PMH has been described in association with infarction of the cerebral cortex, centrum semiovale, medullary pyramid, and the cerebral peduncle.<sup>3-6</sup> Small intracerebral hemorrhages involving the internal capsule, putamen, and basis pontis have also been observed in occasional patients with the clinical syndrome of PMH.7 A saccular basilar artery associated with lacunar infarction of the cerebral peduncle has not previously been described with PMH. We have recently seen a PMH patient with a lacunar infarction of the left cerebral peduncle and a large basilar artery aneurysm who had no other potential explanation for the syndrome.

#### **Case Report**

An 81-year-old woman was hospitalized with increasing dyspnea of 2 weeks duration. The patient had a medical history of coronary artery disease and cardiomyopathy with biventricular failure. There was no history of diabetes mellitus, hypertension, hyperlipidemia, or stroke. General physical examination on admission demonstrated an irregularly irregular pulse and marked jugular venous distention. A grade 2/6 systolic ejection murmur was heard at the cardiac apex. Examination of the lungs demonstrated percussion dullness and decreased breath sounds to the midlung fields bilaterally. Substantial pitting edema was present at both ankles. The neurologic examination was unremarkable. Electrocardiography demonstrated atrial fibrillation with a ventricular response of 100 and evidence of an old anterior wall myocardial infarction.

Cardiomegaly and bilateral pleural effusions were seen on chest x-ray.

A thoracentesis was performed, and 650 ml bloodtinged fluid was removed. The atrial fibrillation spontaneously reverted to normal sinus rhythm on the third hospital day. On the fourth hospital day the patient awoke complaining of right-sided weakness. Neurologic examination demonstrated the cranial nerves to be normal aside from a mild right central facial weakness with associated flattening of the nasolabial fold. The patient had a moderate right hemiparesis with slightly greater involvement of the arm than the leg. Deep tendon reflexes were normally active and symmetrical aside from the knee reflex, which was greater on the right side. The right plantar response was extensor, the left was flexor. Sensory examination was intact to pinprick, light touch, vibration, joint position, and stereognosis. There was no evidence of cerebellar ataxia or aphasia. Two days after the onset of symptoms a computed tomogram (CT scan) was performed on a GE 9800 (Milwaukee, Wisconsin) scanner with a  $512 \times 512$  matrix. The CT scan demonstrated a rounded area of increased density in the region of the interpeduncular fossa, compressing the left cerebral peduncle, and extending from the upper pons to the inferior aspect of the third ventricle (Figure 1). With contrast enhancement, this area of abnormality showed a further increase in density. There were no other abnormalities of a focal nature in the brainstem, internal capsule, basal ganglia, or cerebral hemispheres. A repeat CT scan was obtained 6 weeks after the ictus and again demonstrated the enhancing rounded mesencephalic lesion and no other area of focal abnormality. Follow-up neurologic examination at that time disclosed a residual but substantially improved right hemiparesis. Six months after the ictus, the patient died as a consequence of increasing congestive heart failure.

At autopsy, the brain weighed 1,280 g and was unremarkable externally. At the base of the brain, a saccular (berry) aneurysm 0.8 cm in diameter originated from the rostral basilar artery at its bifurcation into the posterior cerebral arteries, occupying virtually the entire interpeduncular fossa (Figure 2). The wall of the aneurysm contained focal atheromata but was other-

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Received April 8, 1987; accepted August 10, 1987.

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FIGURE 1. Left: 1.6 cm highdensity vascular lesion in region of left cerebral peduncle. Right: Lesion shows marked enhancement and is contiguous with circle of Willis. Bottom: Sagittal reconstruction demonstrating basilar artery aneurysm extending from upper pons to inferior aspect of third ventricle.

wise intact, without evidence of prior hemorrhage. The lumen of the aneurysm contained a dark red (postmortem) thrombus. The remainder of the circle of Willis was normal except for mild focal atherosclerosis. Coronal sections of the cerebrum were unremarkable. Horizontal sections of the brainstem revealed, at the level of the midbrain, a partly cavitary  $0.2 \times 0.4$  cm lacunar infarct within the middle third of the left cerebral peduncle (Figure 3). Histologically, this infarct contained numerous lipid-laden macrophages and was surrounded by reactive astrocytes and scattered axonal swellings. A few hemosiderin-infarcts were present in the right paramedian basis pontis. Microscopic sections of the pons and medulla showed wallerian degen-

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FIGURE 2. Transverse section of midbrain showing large saccular aneurysm within interpeduncular fossa.

eration of the descending corticospinal fibers within the left basis pontis and pyramid. No abnormalities were present in the cerebellum.

### Discussion

The syndrome of PMH is the most common lacunar syndrome and may account for up to 60% of all lacunar strokes.<sup>8</sup> Capsular or pontine infarcts are the most common pathologic findings associated with PMH, and these anatomic loci produce an almost identical clinical picture.<sup>2</sup> Lacunar infarcts of the cerebral peduncle producing PMH have been reported only rarely.<sup>6</sup> Nonlacunar PMH has also been described with a large variety of other lesions including nocardial abscess of the motor cortex, cortical cerebral infarction, subdural hematoma, and demyelinating disease.<sup>9,10</sup> Small primary intracerebral hemorrhages in the capsule, putamen, and basis pontis have also been associated with PMH.<sup>7</sup> Although the most commonly encountered lacunar syndromes with small primary intracerebral hemorrhages are ataxic hemiparesis and sensorimotor stroke, the present patient appears to be the first PMH case occurring secondary to a saccular basilar artery aneurysm. The patient had a clinical syndrome compatible with acute PMH and pathologic evidence of a lacunar infarction in the appropriate cerebral peduncle. No other focal pathologic abnormalities were observed that could have explained the clinical symptoms.

There are several potential pathogenetic mechanisms for the lacunar infarction in this case. The lacunar infarction observed in the left cerebral peduncle was close to the large saccular basilar artery aneurysm, which appeared to compress that cerebral peduncle. Focal neurologic events have been described as a complication of the compressive effects of large intracranial aneurysms.<sup>11</sup> The abrupt development of symptoms may have occurred because the enlarging aneurysm acutely obstructed a penetrating branch of the basilar artery within the confines of the aneurysm. Aneurysmto-artery embolization has been described as an uncommon etiology for completed stroke and transient ischemic attacks, which is another possible cause for our patient's lacunar infarction.<sup>12,13</sup> This mechanism of aneurysm-induced cerebral ischemia has been proven by angiographic documentation of aneurysmal thrombus and distal arterial emboli in the appropriate vascular territory in a few cases. It is uncertain which mechanism was most likely causative in our case. This patient demonstrates that PMH can occur secondary to a basilar artery saccular aneurysm and provides an-



FIGURE 3. Histologic section of midbrain showing lacunar infarction in middle third of left cerebral peduncle (Woelcke,  $\times$ 11).

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other example of the many potential etiologies for this common stroke syndrome.

# Acknowledgments

We thank Dr. Alexander H. Moore for referring the patient and Shirley Felix for her help in preparing the manuscript.

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KEY WORDS • cerebral aneurysm • cerebral infarction • basilar artery





# **Pure motor hemiplegia secondary to a saccular basilar artery aneurysm.** M Fisher, T W Smith and R Jacobs

Stroke. 1988;19:104-107 doi: 10.1161/01.STR.19.1.104 Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231 Copyright © 1988 American Heart Association, Inc. All rights reserved. Print ISSN: 0039-2499. Online ISSN: 1524-4628

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