Response

The authors report yet another cause of direct trauma to the vertebral artery with resultant occlusion. While claims of primacy in observation and reporting are subject to contradiction (and frequently are), their case may well be the first such instance to be reported. The authors do not comment on the presumed cause of the prolonged initial period of unconsciousness, a most unusual feature in patients with acute ischemic lateral medullary syndrome. The occlusion of the PICA at some distance from the site of vertebral artery occlusion suggests that embolism to the PICA may have occurred. This case sheds no further light on the continuing debate over the relative roles of occlusion of the vertebral artery and the PICA as the cause of this syndrome. It also sheds no light on the long-standing dispute as to the role of anticoagulant drugs in acute vertebral basilar ischemic disease; one aspirin per day was associated with a good clinical outcome.

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Pure Motor Monoparesis Due to Intracerebral Hemorrhage

Pure motor monoparesis (PMM), characterized by motor involvement limited to one limb without sensory deficits, is a rare symptom in central nervous system diseases. Recently, we had a chance to treat a patient with persistent PMM caused by an intracerebral hemorrhage whose lesion (confirmed by magnetic resonance imaging [MRI]) was restricted to the primary motor area in the opposite hemisphere.

The patient was a 49-year-old right-handed man. He suddenly realized that he was unable to move his left arm and was immediately admitted to our hospital. His history included aortic valvuloplasty for aortic regurgitation (AR) 10 years earlier and chronic hepatitis. On admission, he was alert and oriented. Cranial nerves, including the facial nerve, were normal. His left upper limb was flaccid, with 0/5 strength by manual muscle testing. However, the left lower limb retained completely normal strength. Sensation was normal in all modalities. Deep tendon reflexes were weak in the left upper limb. Babinski sign was negative bilaterally. Although slight liver dysfunction was noted on laboratory examinations, results of the coagulation tests were within normal limits. An echocardiogram revealed mild AR but no embolic sources. A brain computed tomographic (CT) scan demonstrated a high-density lesion in the right frontoparietal region, suggesting hemorrhage. A right carotid arteriogram was normal (including the venous phase). Somatosensory evoked responses were normal. Deep tendon reflexes in the left upper limb became hyperactive in a few days. The left arm weakness improved gradually. One month after the onset, the proximal strength of the left arm improved to 4/5 and those of the distal muscles to 3/5. Another CT scan performed at this time showed a shrinkage of the high-density area. A T1-weighted MRI at this time demonstrated a small circumscribed high signal intensity area in the upper portion of the right precentral gyrus (Figure 1). The lesion was restricted to the cortical and partially subcortical regions in the primary motor area. After the rehabilitation the patient was discharged with mild residual forearm weakness.

Reported etiologies of PMM originating from intracranial lesions include brain tumor,1-2 brain abscess,3 and ischemic stroke.3-7 Although Sossin and colleagues,8 in their series of patients with pure motor hemiplegia, described a patient showing PMM caused by an intracerebral hemorrhage in the internal capsule, the symptom extended to hemiparesis in a few hours. Thus, to our knowledge, our patient is the first case of stable PMM due to intracerebral hemorrhage. Theoretically, any suitably placed lesions along the course of the corticospinal tract could produce PMM. However, the lesion most likely to produce this sign would be in the cortical or near-cortical area because somatomotor motor representation is most widely separated at this level. At the origin of motor innervation, i.e., in the primary motor cortex, the pattern of motor representation has been well known as the ‘motor homunculus’.9 The T1-weighted MRI of our patient obtained 1 month after stroke onset demonstrated a restricted lesion in the upper portion of the right precentral gyrus. This lesion location corresponded well with the site of the contralateral upper limb in the motor homunculus. The nature of hemorrhage in our patient was different from the usual hemorrhagic stroke in its location and size. The results of the neuroradiological and other examinations could not disclose the exact causes of the unusual hemorrhage. However, we considered the possibility of the hemorrhage following the rupture of the small angioma.

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References


Figure 1. T1-weighted magnetic resonance image (1.0-T; spin-echo; repetition time, 500 msec; echo time, 34 msec) obtained 1 month after stroke onset demonstrating a small high signal intensity area in upper portion of right precentral gyrus. Arrow 1, right precentral sulcus joining to superior frontal sulcus; arrow 2, right central sulcus.
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