

Pure Motor Hemiplegia Due to Cerebral Cortical Infarction

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• Although pure motor hemiplegia has not been reported after cerebral cortical infarction, occasional exceptions may occur. We provide three such examples. Necropsy study confirmed the site of lesion in one patient, and laboratory results (EEG and computerized axial tomography) suggested cortical involvement in the other two patients.

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In previous publications^{1,2} we reported that pure motor hemiplegia may result from lesions at sites other than the internal capsule or basis pontis. While it is true that cerebral cortical infarction does not usually cause pure motor hemiplegia without other associated defects, such as language dysfunction or sensory impairment, occasional exceptions may occur, as speculated by Fisher and Curry.³ Theoretically, it is possible for an appropriately placed infarction in the cerebral cortex to give rise to pure motor hemiplegia, but to our knowledge this has not yet been recorded. We now provide three such examples. Necropsy study confirmed the site of lesion in one and laboratory results suggested cerebral cortical involvement in the other two patients.

REPORT OF CASES

CASE 1.—On March 4, 1975, an 83-year-old man had transient weakness of the right

side of his body lasting for a few minutes. In the course of the next hour the weakness returned and progressed to complete paralysis. He did not have a headache, convulsion, impairment of consciousness, paresthesia, vertigo, or visual or language dysfunction. General physical examination included a blood pressure of 210/100 mm Hg and a pulse rate of 80 beats per minute. On neurological examination his mental functions were appropriate for his age and there was no evidence of dysphasia. He had undergone an operation for removal of a cataract in the right eye. The left pupil was reactive to light. Facial sensation was normal, but he had mild right central facial paresis. He was very deaf in both ears. There was no palatal or lingual weakness. He had complete paralysis of the right upper extremity and severe diffuse weakness of the right leg. The hemiplegic limbs were flaccid, but the muscle stretch reflexes were greater on the affected than on the unaffected side. Plantar response was extensor on the right, but flexor on the left side. Sensory examination was entirely normal. Laboratory data did not show any significant abnormalities except slight enlargement of the heart on the chest roentgenogram.

The strength of the right leg improved considerably after physical rehabilitation, but the right arm remained functionless. He was discharged to a nursing home on April 28, 1975, but was readmitted on Aug 11, 1975, in a dehydrated and confused state. He died the next day from cardiopulmonary arrest.

General postmortem findings consisted of severe generalized and moderate coronary atherosclerosis, obstructive pulmonary emphysema, occlusion of the left renal artery, and atrophy of the left kidney. The cerebral hemispheres were symmetrical but somewhat atrophic. The major cerebral arteries showed atheromatous plaques. Serial coronal sections of the cerebral hemispheres showed thinning and yellowish discoloration of the left frontal cortex involving the precentral region, the superior frontal gyrus, and the crown of the left inferior frontal gyrus (Fig 1). The immediate subcortical white matter was retracted and discolored. In the crown of the left middle frontal gyrus there was a very small area of thinning. The opercular, triangular, and orbital portions of the left inferior frontal gyrus were normal. There were small areas of softening (0.5 to 1 cm in diameter) in the left superior and right

Fig 1.—Coronal section of brain showing softening and discoloration of left frontal cortex.



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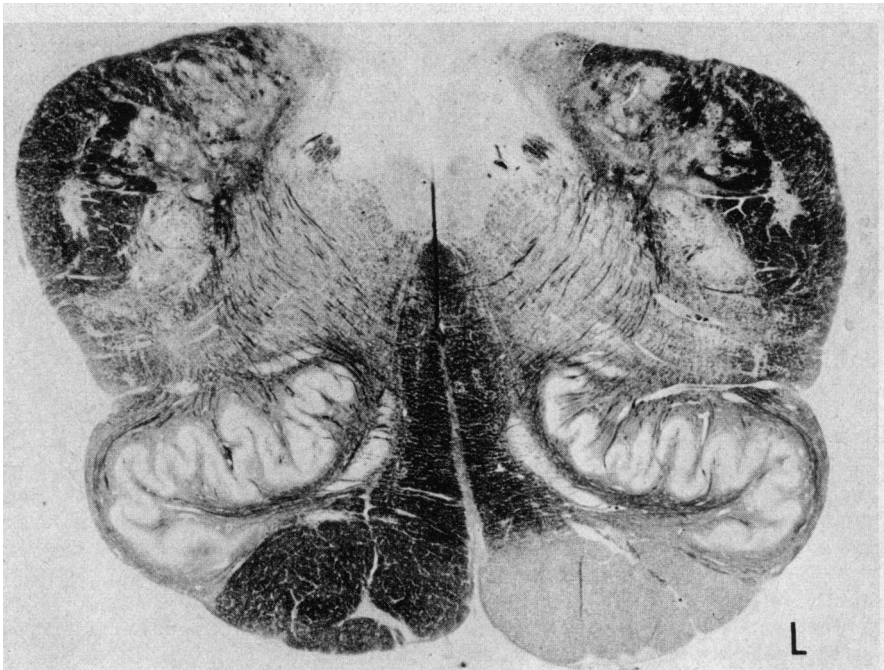


Fig 2.—Transverse section of medulla showing Wallerian degeneration in left pyramid (myelin, original magnification $\times 5$).

posterior parietal lobes and both occipital lobes. The thalami had lacunae bilaterally. Transverse section of the medulla disclosed Wallerian degeneration of the left pyramid (Fig 2). Microscopically, the discolored areas in the cerebral cortex and the thalamic lacunae were consistent with old cystic infarctions, and the left thalamic lacuna involved the medial edge of the anteriormost portion of the posterior limb of the left internal capsule. The capsular lesion measured 5 mm in the anteroposterior and 1 mm in the transverse direction.

CASE 2.—A 66-year-old man had sudden weakness of the left side of his body and slurring of speech without loss of consciousness. He denied visual or language difficulties, vertigo, paresthesia, or convulsion. Past history included diabetes mellitus and hypertension of two years' duration. In the last three months he had had four to five episodes of transient vertigo, oscillopsia, and incoordination with a tendency to fall to the right, lasting for three to five minutes on each occasion. His blood pressure was 170/100 mm Hg. Neurological examination showed normal mental and cranial nerve functions except for left central facial weakness, mild dysarthria, and evidence of diabetic retinopathy. He had a dense left hemiplegia with only a

Fig 3.—Electroencephalogram (case 2) shows focal spikes and delta waves in right central and parietal regions. International 10-20 electrode placement system was used (timer, one second; calibration, 50 μV).

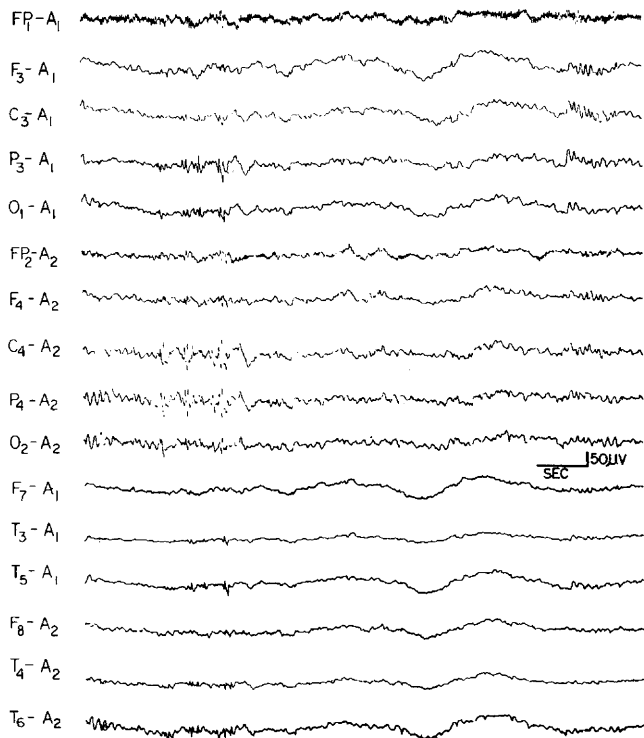
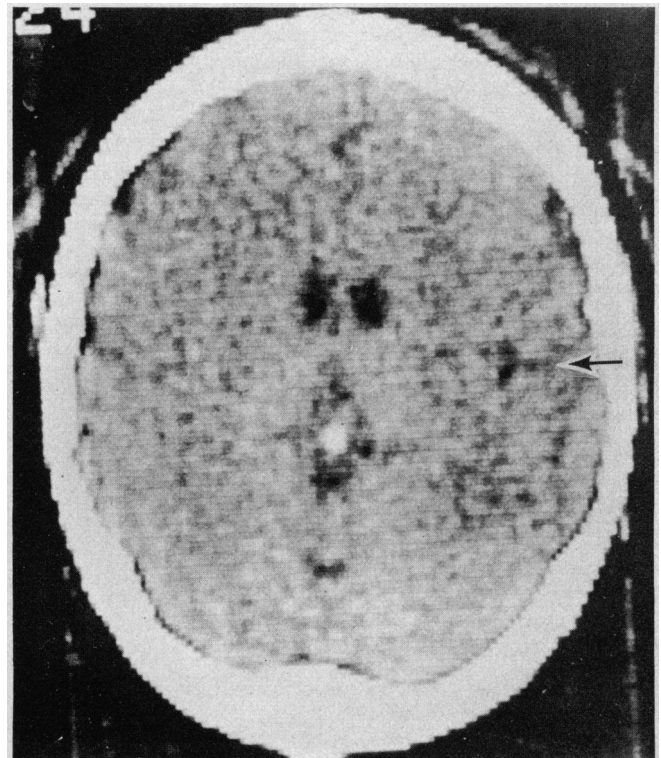


Fig 4.—Computerized axial tomography (case 3). Arrow points to area of infarct in right posterior temporal region.



trace of movement in the leg. There was marked wasting of the muscles of the left arm, left thigh, and left gluteal region on the second day of the illness. Muscle tone in the affected limbs was reduced for the first two to three days and then increased. Muscle stretch reflexes were brisker on the left than on the right side. Plantar response was extensor on the left side but flexor on the right. All sensory modalities, including cortical sensation, were normal. There was no cerebellar ataxia of the right side.

Laboratory studies showed a fasting blood glucose level of 200 to 300 mg/100 ml. An EEG initially showed focal epileptiform activities accompanied by 2 to 3 Hz slow waves in the right central and parietal areas (Fig 3). A second EEG a week later was normal. Aortic arch angiogram showed atheromatous plaques in the left internal carotid artery 4 to 5 cm distal to its origin, and in the upper portion of the basilar artery. The rest of the laboratory data, including somatosensory cerebral evoked potential study and brain scan, were normal.

The patient remained totally paralyzed for two days and then began to regain strength, first in the leg and then in the arm. One week later he was walking without a cane. He was discharged from the hospital a month after admission.

CASE 3.—A 54-year-old man had sudden onset of weakness of the left side of the body and a transient feeling of numbness in the left side of the face. In the course of the next 12 hours the strength in the left leg improved, but the weakness in the left arm progressed to complete paralysis. He did not have language or visual difficulties, convulsion, loss of consciousness, or vertigo. There was no history of hypertension, and blood pressure was 150/80 mm Hg. On neurological examination his mental functions were normal and there was no dysphasia. The visual fields were intact. Pupils were equal and reactive to light. The extraocular movements were full. Facial sensation was not impaired. There was mild left central facial paresis, but no weakness of the palate or tongue. Motor examination showed only a trace of movement in the left upper extremity but mild diffuse weakness of the left leg. Muscle stretch reflexes were brisker on the left than on the right side, and there was an unsustained left ankle clonus. The plantar response was extensor in the left foot and flexor in the right. Tests for coordination and sensation including all cortical sensory

modalities were normal. Computerized axial tomography of the skull showed an area of lucency in the right posterior temporal region suggesting a small infarction (Fig 4). The result of the laboratory tests, including brain scan, EEG, and somatosensory cerebral evoked potential study, did not show any abnormalities. The patient regained normal strength in the leg and had considerable improvement in the left arm after physical rehabilitation. He was discharged home three weeks after admission to the hospital.

COMMENT

In patient 1, there was unequivocal necropsy evidence of cerebral cortical infarction that was large enough to involve the face, arm, and leg areas of the precentral cortex, yet it spared the inferiormost part of the posterior frontal and most of the parietal cortices. The net result was a pure motor paralysis of one half of the body without associated language dysfunction or sensory impairment. The question we have to decide next is whether the small lacunae in the thalami and in the anteromedial part of the posterior limb of the left internal capsule were incidental findings, or were in some way responsible for the patient's clinical features. The thalamic lacunae were obviously not sufficient to cause any sensory impairment. Similarly small infarctions in the left superior and right posterior parietal lobes and both occipital lobes remained asymptomatic. It is extremely unlikely that an infarct of 5 mm × 1 mm in dimension in the medial and anteriormost part of the posterior limb of the internal capsule will cause paralysis of the entire one half of the body. According to the standard textbooks of neuroanatomy, the corticospinal fibers are located in the anterior two thirds of the posterior limb of the internal capsule with face, arm, and leg in an anteroposterior distribution. The lesion in patient 1 was not extensive enough to involve all the corticospinal fibers. Furthermore, Englander et al⁴ recently challenged the traditional concept of the location of the corticospinal fibers in the internal capsule, and they concluded from their

careful necropsy study of a patient that the corticospinal fibers occupied the third quarter of the posterior limb of the internal capsule. Therefore, the above-mentioned lesion in our patient could not involve all the corticospinal fibers.

In the second patient, clear documentation of focal delta waves and epileptiform activities in the initial EEG indicated that the responsible lesion for pure motor hemiplegia was in the cerebral cortex. The other notable feature in this patient was the presence of marked muscle atrophy in the hemiplegic limbs on the second day of the illness. Hemiplegic amyotrophy has recently been reinforced by us.⁵

In patient 3, computerized axial tomography suggested a cortical lesion in the appropriate side for pure motor hemiplegia, although it did not show involvement of the motor cortex. We believe that the technique of computerized tomography is not yet sufficiently accurate to outline the total extent of the lesion in all cases. Complete paralysis of the arm in the presence of mild leg weakness might have indicated that the lesion possibly originated in an area where the corticospinal fibers did not form a compact bundle, such as in the cerebral cortex.

The above three examples show that infarction of the cerebral cortex may cause pure motor hemiplegia, and one should be aware of this possibility in an occasional patient and employ appropriate investigations, including computerized axial tomography.

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