Ipsilateral hemiparesis after a supratentorial stroke is rare. However, the role of the reorganization of the unaffected hemisphere in recovery after a stroke is poorly understood. Two patients developed ipsilateral hemiparesis after a left corona radiata infarct. Both of these patients had previously experienced contralateral hemiparesis after a right-sided supratentorial stroke. Functional magnetic resonance imaging demonstrated bilateral motor area activation during paretic left hand movement. This finding suggests that the ipsilateral hemiparesis was caused by a new stroke in the ipsilateral motor system that was functionally reorganized after the previous stroke.

**REPORT OF CASES**

**PATIENT 1**

A 62-year-old right-handed woman with a history of stroke and hypertension was first seen with sudden worsening of left-sided hemiparesis. She had previously experienced 2 episodes of stroke. She first developed mild left-sided hemiparesis after a right-sided corona radiata infarct 9 years previously. Subsequently, she almost completely recovered in several months. One month ago, left-sided hemiparesis recurred. Brain computed tomography showed the presence of an acute hematoma in the right thalamus and a subacute hematoma in the right temporoparietal and occipital lobe (Figure 1A). She made a good recovery, with only mild hemiparesis remaining when she was discharged from the hospital. However, her left-sided hemiparesis suddenly worsened, and she returned to the hospital. On neurologic examination, her eye movements were found to be full, without nystagmus. Her speech was mildly slurred. There was facial weakness and moderate arm and leg weakness (Medical Research Council scale score, 3 for arms and 4 for legs), with exaggerated deep tendon reflexes and the Babinski sign on the left side. Sensation to pain and touch was impaired on the left side. There was no visual field defect or hemineglect.

Brain MRI, including diffusion-weighted imaging, was performed using a 1.5-T MRI system to localize any acute lesions and the old lesions. Diffusion-weighted imaging was performed in the transverse plane using a single-shot, echo-planar, spin-echo pulse sequence with a repetition time of 6500 milliseconds, an echo time of 107 milliseconds, 1 excitation, and 2 $b$ values (0 and 1000 s/mm$^2$). Diffusion-weighted imaging revealed the presence of an acute infarct in the left corona radiata (Figure 1D). The T2-weighted image showed the old infarct in the right corona radiata and the old hemorrhage in the right thalamus and temporoparietal lobe, in addition to the acute lesion in the left corona radiata, which was observed on the diffusion-weighted image (Figure 1B and C).
Functional MRI was performed using a 3.0-T scanner 3 days after stroke onset. The T1-weighted axial images were included as anatomical images. The blood oxygen level–dependent contrast images consisted of single-shot echo-planar imaging gradient-echo images. Imaging variables included an interscan interval of 3000 milliseconds, an echo time of 40 milliseconds, a matrix of $64 \times 64$, a field of view of $24 \times 24$ cm, a flip angle of 90°, and a slice thickness of 6 mm for a total of 20 contiguous slices covering the brain. The motor task for imaging was repetitive thumb-index tapping. The paradigms comprised 3 trials, each consisting of rest–right-hand task–rest–left-hand task, with each rest or task lasting 24 seconds. Visual cues guided the patient through the series of successive tasks and rest periods. There were 3 right-hand task periods, 3 left-hand task periods, and 6 rest periods. The total duration of a run was 384 seconds. Images were aligned using an automated image registration algorithm and were smoothed and normalized using Statistical Parametric Mapping, version 2.0 (University College London, London, England). Statistical analysis was then performed on the pooled data using Statistical Parametric Mapping by setting up a contrast between the rest and the task conditions. The resulting z-maps were thresholded using the criteria of z-score height ($P < .001$) and cluster size ($P < .05$). Functional MRI demonstrated that the left sensorimotor cortex was activated during right-hand movement, whereas the bilateral sensorimotor cortex and the right supplementary motor area were activated during left-hand movement (Figure 2).

A 41-year-old right-handed man was first seen with left-sided hemiparesis. He had a history of right-sided corona radiata infarct with left-sided hemiparesis that had developed 1 month earlier. He had made a full recovery before noting newly developed weakness of the left arm and leg. On neurologic examination, he was found to have mild hemiparesis (Medical Research Council scale score, 4+ for arms and 4+ for legs), with increased deep tendon reflexes and the Babinski sign on the left side. There was no facial palsy or dysarthria. Motor function on the right side was normal. Sensation was intact on both sides.

Brain MRI showed high signal intensity in the left corona radiata, which was shown to correspond to an acute lesion by diffusion-weighted imaging. The old lesion was observed in the right corona radiata (Figure 3). Functional MRI was performed 7 days after onset and demonstrated the existence of left sensorimotor cortex activation during nonparetic right-hand movement. Conversely, the bilateral sensorimotor cortex and the right supplementary motor area were activated during paretic left-hand movement (Figure 4).

Ipsilateral hemiparesis after a cerebral lesion has rarely been reported. It could result from a congenital uncrossed pyramidal tract or from a lesion that affects the secondary motor area in the precentral insular cortex bilaterally in-
nervating the face and limbs. However, our patients had a history of contralateral hemiparesis after a stroke, suggesting that the current ipsilateral hemiparesis was unlikely to be caused by a congenital uncrossed pyramidal tract. Moreover, in neither patient did the lesion correspond to the recent infarct in the insular cortex.

Fisher described 2 patients who both had 2 successive hemiplegias, the first involving the limbs on the left side, which recovered some function. A second hemiplegia involving the right side resulted in bilateral paralysis. Pathological studies revealed a bilateral cerebral infarct. A case involving the deterioration of preexisting hemiparesis brought about by a subsequent ipsilateral corona radiata infarction was also reported, and was similar to our case. Patient 1 had a history of recurrent infarct and hemorrhage in the right cerebral hemisphere. She had 2 hemorrhagic lesions—a right thalamic hemorrhage and a temporo-occipital hemorrhage—when the second hemiparesis occurred. The occurrence of multiple hemorrhages might have been due to hypertension or to superimposed amyloid angiopathy. The most plausible lesion that could have contributed to the second hemiparesis is the thalamic one, which extended to the internal capsule. The temporo-occipital hemorrhage is thought to be an asymptomatic subacute lesion.

Ago et al. performed MRI, which showed that the paretic left hand grip activated the ipsilateral left motor areas but not the right hemispheric motor areas. The activation pattern in MRI was different from that in our case. In our patients, MRI showed activation of the left and right motor areas during paretic left hand movement. This difference in activation patterns may be due to the use of different MRI protocols or to interindividual variation in brain reorganization. The activation patterns were different, but ipsilateral motor area activation was a common essential finding. Activation of the ipsilateral motor area during paretic hand movement indicated that the reorganization of the unaffected hemisphere was involved in the process of recovery after a previous stroke and that a new lesion in the reorganized area resulted in deterioration of hemiparesis.

The activation pattern in fMRI or positron emission tomography after stroke includes enlarged activation of the ipsilesional motor cortex, activation of the contralesional motor cortex, and bilateral activation of the primary motor cortex or secondary motor areas, such as the premotor cortex and the supplementary motor area. The activation patterns of our patients belong to the third pattern. Although the precise role of the reorganized ipsilesional and contralesional motor areas is largely unknown, recent studies suggest that ipsilesional extended activation of the motor area correlates more with functional recovery than does contralesional motor area activation and that the shift toward the affected hemisphere is related to recovery. In these studies, the contribution of the contralesional motor area to motor recovery seemed to be less efficient. However, our cases suggest that contralesional motor area activation reflects the important functional role of the unaffected hemisphere in recovery after a stroke because both patients made a good recovery after a previous stroke, whereas a new stroke in the contralesional area resulted in re paralysis of the ipsilateral limbs. The unaffected hemisphere might contribute to recovery from hemiparesis through the activation of the latent ipsilateral motor pathway.

Both patients had normal motor function on the right side when ipsilateral hemiparesis occurred, which suggests that the lesion spared the pathway projecting to the contralateral limbs and affected only the pathway projecting to the ipsilateral limbs. We speculate that the newly activated ipsilateral pathway produced by the functional reorganization did not share the preexisting crossed pathway.

In conclusion, ipsilateral hemiparesis can develop as a result of a new stroke after a previous stroke on the opposite side. The mechanism involved is thought to be functional reorganization of the ipsilateral hemisphere.

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