

Isolated Weakness of Middle, Ring, and Little Fingers due to a Small Cortical Infarction in the Medial Precentral Gyrus

Young-Su Han, M.D., Sang-Won Ha, M.D., Jeong-Seon Cho, M.D., Sang-Eun Park, M.D.,
Jung-Mee Kim, M.D., Jeong-Ho Han, M.D., Eun-Kyoung Cho, M.D., Doo-Eung Kim, M.D.

Department of Neurology, Seoul Veterans Hospital, Seoul, Korea

Small cortical strokes can produce predominant isolated weakness in a particular group of fingers: radial or ulnar. The traditional views are of point-to-point representations of each finger to neurons located in the precentral gyrus of the motor cortex such that the neurons of the radial fingers are located laterally and those of the ulnar fingers are located medially. We present a case of isolated weakness of middle, ring, and little fingers due to a small cortical infarction in the medial precentral gyrus.

J Clin Neurol 2(2):146-148, 2006

Key Words : Cortical infarction, Precentral gyrus, Finger weakness

A discrete stroke may produce symptoms mimicking those from peripheral nerve diseases.¹ The predominant involvement of a particular group of fingers due to brain lesion, or "pseudoperipheral palsy," was described as early as 1909.² Since the development of the concept of the homunculus, it has been shown that the cortical representation of motor hand function is located in the superior part of the precentral gyrus.³ Small cortical strokes in the precentral gyrus can produce isolated weakness of the fingers, which may be prominent in ulnar fingers⁴ or radial fingers.^{5,6} The results from a previous study suggest that the ulnar fingers are located medially and radial ones laterally in the knob of the precentral gyrus.⁷ We present a case of isolated weakness of middle, ring, and little fingers due to a small cortical infarction in the medial precentral gyrus.

A 58-year-old right-handed man was admitted because of sudden weakness in his right middle, ring, and little fingers. He had had hypertension, diabetes mellitus, and hyperlipidemia since undergoing percutaneous transluminal coronary angioplasty with stenting for angina pectoris 8 years previously. However, he had never experienced paresthesia or weakness in the extremities. On admission, he was alert without dysarthria, aphasia, apraxia, or other higher cortical dysfunction. Cranial nerve examinations were normal. Motor examination revealed mild weakness (grade IV by manual muscle testing) of his right middle, ring, and little fingers. The weakness was severest at the distal interphalangeal joint. Flexion and extension movements were equally impaired. There was no significant difference in muscle power among the affected fingers. The strength of the other fingers, wrist, elbow, shoulder, and leg were normal. Sensations, including cortical sensation, were normally perceived in all modalities in his fingers as

CASE REPORT

Received : November 24, 2005 / Accepted : February 13, 2006 / Address for correspondence : Sang-Won Ha, M.D.
Department of Neurology, Seoul Veterans Hospital, 6-2 Duncheon 2-dong, Gangdong-gu, Seoul, 134-791, Korea
Tel: +82-2-2225-1324, Fax: +82-2-2225-1327, E-mail: hippocam@naver.com

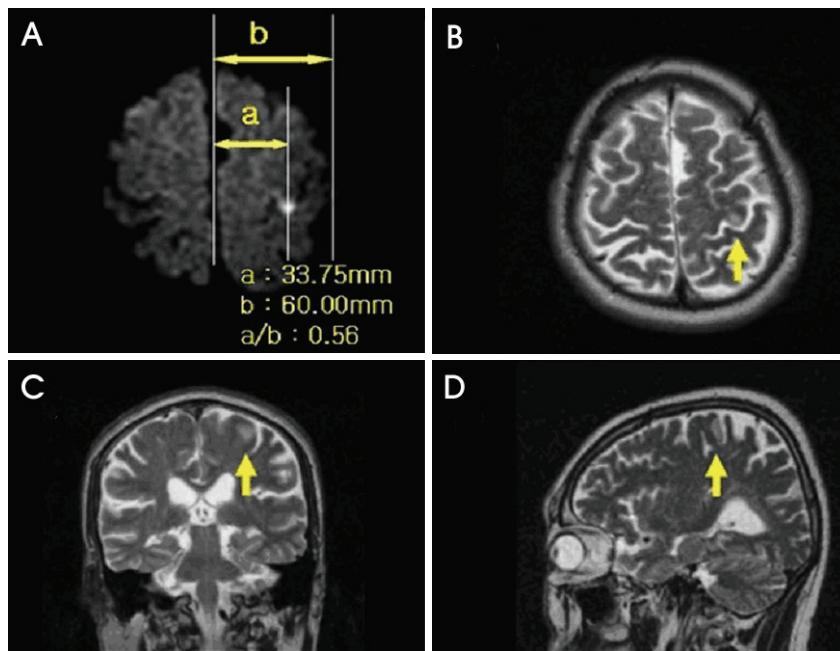


Figure 1. Brain MRI of the patient. (A) Diffusion-weighted image shows an infarct in the medial precentral gyrus. Distances from the falx to the center of the lesion (a) and to the lateral surface of brain (b) are indicated. (B) The lesion (arrow) is located medially in the precentral hand knob in T2 axial view. In coronal (C) and sagittal (D) views the lesion (arrows) shows high signal intensity at the left precentral gyrus.

well as in other body parts.

The electrocardiogram findings were normal. Trans-thoracic and transesophageal echocardiograms revealed severe atherosclerotic changes with mobile atheroma at the aortic arch area. Chest CT with enhancement showed multifocal atheromatous plaque in the thoracic and abdominal aorta. A nerve conduction study showed no evidence of peripheral neuropathy. Brain MRI performed on the onset day showed a small, discrete infarct in the medial precentral gyrus of the left frontal cortex (Fig. 1). MR angiogram showed hypoplastic changes in A1 of the right anterior cerebral artery, and focal luminal narrowing in the cavernous segment of the right internal carotid artery and the knee segment of the right middle cerebral artery. The patient was immediately treated with heparin and antiplatelet agents. He showed gradual improvement and regained his normal power 3 months later.

DISCUSSION

The motor topography of human fingers remains controversial. Traditional views have suggested point-to-point representations of each finger to neurons located in

the motor cortex such that the radial fingers are located laterally and the ulnar fingers are located medially in the hand knob.⁷ Previous studies^{3,8} have calculated the ratio of the distance from the falx to the center of the stroke lesion in relation to that from the falx to the most lateral margin of the brain. The mean value in patients with predominant involvement of radial fingers (0.71) was greater than that in patients with predominant involvement of ulnar fingers (0.58). Therefore, lesions related to predominant involvement of ulnar fingers are located significantly more medially than those associated with predominant involvement of radial fingers in the presumed hand representation area of the motor cortex. These authors further suggested that the medial portion of the precentral knob representing topographically ulnar fingers correspond to the border-zone area associated with a hemodynamic mechanism, whereas the lateral portion representing radial fingers are supplied by distal middle cerebral artery branches associated with artery-to-artery embolism or cardiogenic embolism.⁸

In our patient, the location of the lesion was medially in the precentral hand knob with a ratio of 0.56 (Fig. 1-A). Therefore, MRI of our patient suggests that the medial portion of the precentral knob, representing topographically predominant involvement of ulnar fingers,

corresponds to the situation reported previously.⁷ Some authors advocate multiple representation or spatially overlapping patterns of the cortical motor hand area with more sophisticated methods using intracortical microstimulation, or functional MRI.^{5,9,10}

Regarding the pathogenesis of stroke, our patient had mobile aortic atheroma and normal MR angiogram findings except for the right anterior circulation. Therefore, artery-to-artery embolism from the aorta is a likely pathogenic mechanism. This observation appears to be inconsistent with the previous observation that medially located lesion was more often associated with a hemodynamic mechanism.⁸ Our case therefore suggests that the variable pathogenesis of stroke-induced weakness in some of the fingers.

REFERENCES

1. Lampl Y, Gilad R, Eshel Y, Sarova-Pinhas I. Strokes mimicking peripheral nerve lesions. *Clin Neurol Neurosurg* 1995;97:203-207.
2. Lhermitte J. De la valeur sémiologique des troubles de la sensibilité à disposition radicaire dans les lésions de l'encephale. *Sem Med* 1909;24:277.
3. Yousry TA, Schmid UD, Alkadhi H, Schmidt D, Peraud A, Buettner A, et al. Localization of the motor and hand area to a knob on the precentral gyrus. A new landmark. *Brain* 1997;120:141-157.
4. Phan TG, Evans BA, Huston J. Pseudoulnar palsy from a small infarct of the precentral knob. *Neurology* 2000;54:2185.
5. Lee PH, Han SW, Heo JH. Isolated weakness of the fingers in cortical infarction. *Neurology* 1998;50:823-824.
6. Kim JS, Chung JP, Ha SW. Isolated weakness of index finger due to small cortical infarction. *Neurology* 2002;58:985-986.
7. Penfield W, Boldrey E. Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain* 1937;60:389-443.
8. Kim JS. Predominant involvement of a particular group of fingers due to small, cortical infarction. *Neurology* 2001;56:1677-1682.
9. Kwan HC, MacKay WA, Murphy JT, Wong YC. Spatial organization of precentral cortex in awake primates. II Motor outputs. *J Neurophysiol* 1978;41:1120-1131.
10. Sanes JN, Donoghue JP, Thangaraj V, Edelman RR, Warach S. Shared neural substrates controlling hand movements in human motor cortex. *Science* 1995;268:1775-1777.