ISOLATED LEFT HAND WEAKNESS DUE TO CORTICAL INFARCTION

ABSTRACT

Isolated pure motor weakness of a hand or particular group of fingers usually results from a peripheral neurological disease, although, rarely, it can be caused by central neurological disease, such as cortical infarcts. Weakness of a hand or a particular group of fingers due to cortical cerebral infarction has been described as “pseudo-peripheral palsy.” The motor representation of the hand in the brain is localized to a knob-like structure in the precentral gyrus. A lesion localized in a region of the hand knob could affect all the muscles of the hand or the muscles innervated by the radial, median, and ulnar nerves. Infarctions in the parietal lobe or white matter of the angular gyrus, postcentral gyrus, ventroposterior thalamus, corona radiate, basal ganglia, and posterior limb of the internal capsule can also mimic peripheral nerve lesions. In patients with monoparesis caused by stroke, cerebral computed tomography can miss infarct lesions. Using magnetic resonance imaging can allow the identification of small vascular lesions that cause monoparesis. Early diagnosis of “pseudo-peripheral palsy” is important for adopting a suitable therapeutic approach to prevent a recurrent or larger cortical infarction. We describe 65-year-old patient who presented with an acute paresis of her left hand, suggesting a peripheral nerve lesion. However, on clinical examination, a peripheral origin could not be verified, prompting further investigation. Diffusion-weighted magnetic resonance imaging revealed an acute hemorrhagic lesion. The peripheral nerve pattern of lesions caused by cortical infarcts should be taken into consideration in the differential diagnosis of isolated hand monoparesis.

Key Words: Aged; Monoparesis; Hand.

OLGU SUNUMU

KORTİKAL İNFARKTA BAĞLI OLARAK GELİŞEN İZOLE SOL ELDE GÜÇSÜZLİĞÜ

Öz


Anahtar Sözcükler: Yaşlı; Monoparezi; El.
INTRODUCTION

Isolated pure motor weakness of the hand or a particular group of fingers can be the result of a peripheral or central neurological disease (1). Focal hand weakness can be related to peripheral neurological diseases and, in rare cases, central neurological diseases, such as cortical infarcts, can cause isolated hand weakness (2). Weakness of a hand or a particular group of fingers due to cortical cerebral infarction was first described by Lhermitte in early 1900s and called “pseudo-peripheral palsy” (3). This can mimic peripheral nerve palsy (4).

The motor representation of the hand in the brain is localized to a knob-like structure in the precentral gyrus shaped like an omega or epsilon in the axial plane and a hook in the sagittal plane. “Pseudo-peripheral palsy” is usually caused by cortical infarction within this structure (5). Infarction in the parietal lobe or white matter of the angular gyrus, postcentral gyrus, ventroposterior thalamus, corona radiate, basal ganglia, and posterior limb of the internal capsule also can mimic peripheral nerve lesions (6, 7).

We describe a patient who presented with an acute paresis of her left hand related to acute right hemorrhagic centrum ovale, corona radiate, parietal lobe, and parieto-occipital brain infarction.

CASE

A 65-year-old male patient presented to the Physical Medicine and Rehabilitation Department with difficulty in moving the thumb, index, and middle finger of her left hand. He had noticed the symptoms two days earlier. He had not taken alcohol or hypnotics before the event, and she felt no sensory disturbances. He had no history of arterial hypertension or other disease. He had presented at the neurology clinic with the same symptoms one day before, and had undertaken a brain computed tomography (CT) scan that revealed increased third lateral ventricle compartment, increased cortical sulcal length consistent with senile atrophy, right centrum ovale chronic infarct, and periventricular ischemic microangiopathic changes. The neurology clinic transferred the patient to the outpatient clinic. Clinical examination of her left hand revealed a paresis (strength 3–4 on the Medical Research Council scale for muscle strength) of Mm. flexor pollicis, Mm. flexor abductor pollicis brevis, Mm. opponens pollicis, Mm. flexor digitorum superficialis 2–3, and Mm. flexor digitorum profundus 2–3. Ulnar-radial and dorsal wrist flexions and abduction of fingers II–V were normal. Similarly, proximal arm muscles, somatosensory perception, muscle tone, and tendon reflexes were unaffected. Her bilateral Babinski sign was absent. Further clinical examination, including language function, cranial nerves, and plantar reflex, showed no abnormalities. Electroneuromyography (EMG) was performed. Nerve conduction studies were normal. In the needle EMG, although there was activation deficiency with muscles innervated by C8–T1, acute denervation was not observed. The needle EMG suggested weak chronic C7 radix involvement. Magnetic resonance imaging (MRI) of the cervical spine revealed multiple prolapsed discs at the C3–4 and C4–5 levels, with cord compression at the C3–4 and C5–6 levels. There was also diffuse bulging at the C6–7 level; the cord and radix touched at the same level. Peripheral nerve weakness origin could not be detected, and brain and contrast-enhanced brain MRI scans were performed to look for a central cause. The brain MRI revealed right centrum ovale, corona radiata, parietal lobe, and parieto-occipital brain hypertensive lesions in T1 weighted, T2 weighted, and fluid attenuated inversion recovery (FLAIR) sequences consistent with acute hemorrhagic infarction. The contrast-enhanced brain MRI revealed an acute right sulcal hemorrhagic hypertensive lesion in the T1 sequence and a heterogeneous hypertensive lesion in the T2 sequence in the centrum semiovale, corona radiata, parietal lobe at the precentral-postcentral gyrus level, and parieto-occipital region developed from venous angioma-arteriovenous malformation (Figure 1).

DISCUSSION

Focal and isolated extremity monoparesis is a rare presentation of stroke, often misdiagnosed as peripheral nerve lesion (6, 8). “Pseudo-peripheral palsy” is a term used to describe a rare clinical condition associated with cerebral infarction consisting of weakness of the hand or a particular group of fingers (3). Isolated hand weakness due to cortical infarction has been reported to mimic ulnar, median, and radial nerve entrapment neuropathies (1-8). In the case reported here, the patient’s muscle innervated by the median nerve was weakened. Muscle groups innervated by the radial and ulnar nerves were at normal strength. There were no clinical findings of central origin lesions, such as abnormal muscle tone, symmetric deep tendon reflexes, or extensor Babinski reflex. In differential diagnosis, non-traumatic entrapment neuropathies such as carpal tunnel syndrome and anterior interosseous syndrome were investigated. Peripheral nerve origin hand we-
akness could not be detected in an EMG but brain MRI revealed acute right hemorrhagic centrum ovale, corona radiata, parietal lobe, and parieto-occipital brain infarction. The cortical “hand knob” in the precentral gyrus has been referred to as the site of hand motor function, although the control center of hand movement is not limited to this area alone. Infarctions in the parietal lobe are also related to isolated hand motor disorders. In human and animal models it has been shown that the inferior parietal lobe includes somatotopic representation of the hand. Additionally, infarction of the white matter of the angular gyrus, postcentral gyrus, ventro-posterior thalamus, corona radiate, basal ganglia, and posterior limb of the internal capsule can cause hand weakness and can mimic peripheral nerve lesions (6, 7). Timsit et al. first reported isolated hand palsy as a parietal lobe syndrome in six patients. In their series, all patients had pure motor pseudounar deficits (8). In a recent article by Kim concerning stroke patients with either a predominant radial or an ulnar-sided finger disturbance, infarct localization was in the hand-representation area of the motor cortex (9). Chen et al. presented a case series study with four patients with uniform hand weakness and two patients with radial and ulnar weakness. The lesion localizations were at the hand knob area in five patients and the hand knob area plus the postcentral gyrus in one patient. The two cases with uniform digit weakness had additional involvement of the inferior parietal lobule (10). Çelebisoy et al. described pure motor hand monoparesis in eight patients who had a precentral gyrus lesion (11). Manjaly et al. presented a case of acute paresis of the distal right hand suggesting a peripheral median nerve lesion. The case they described had an acute ischemic lesion in the hand knob area of the motor cortex (12). Ueno et al. presented a case with a small infarct of the left precentral knob area, which induced both motor and sensory impairments that were similar to right ulnar

Figure 1—The contrast-enhanced brain MRI shows an acute right sulcal hemorrhagic hyperintense lesion in the T1 sequence and a heterogeneous hyperintense lesion in the T2 sequence in the centrum semiovale, corona radiata, parietal lobe at the precentral-poscentral gyrus level, and parieto-occipital region.
In the present case, the lesion localization was in the centrum ovale, corona radiate, parietal lobe at precentral and postcentral gyrus level, and parieto-occipital brain. In previous studies of patients with hand monoparesis caused by stroke, cerebral CT showed no lesions in approximately 40% of the patients. According to these studies, CT can miss some small infarcts. Using functional MRI, transcranial magnetic stimulation and fiber tracking studies could identify anatomic localization of lesions causing monoparesis (1,14). In the present case report, although the brain CT conducted by the Neurology Department did not reveal acute hemorrhagic infarction, diffusion and contrast-enhanced MRI revealed a hemorrhagic infarction.

The prognosis of cortical infarcts related to the isolated hand weakness seems to be favorable, with the patients usually recovering (12). Early diagnosis of “pseudo-peripheral palsies” is important for adopting a suitable therapeutic approach for preventing recurrent or larger cortical infarctions.

In conclusion, isolated hand weakness can be due to cortical infarction mimicking peripheral nerve disease. Such cortical infarctions are often misdiagnosed, and, consequently, the opportunities to identify and address stroke risk factors are missed. Early recognition of a small cortical stroke in patients with sudden onset hand weakness can lead to appropriate stroke management.

REFERENCES