Isolated Hand or Finger Paresis Caused By Discrete Brain Pathologies

Jin Jun Luo, MD, PhD,1,2* and Ausim S. Azizi, MD, PhD1.

Abstract— Acute isolated hand or finger paresis can be caused by stroke. However, early diagnosis of the acute isolated hand or finger paresis caused by stroke can be a challenge because it may bear peripheral features. We presented five cases of acute isolated hand or finger paresis caused by a brain lesion involving discrete locations. Brain MRI showed a pathology including the precentral gyrus, namely the ‘cortical hand area’, or in the subcortical area in the centrum semiovale of the corona radiata beneath the precentral gyrus, the posteriolateral thalamus and the posterior limb of internal capsule. The etiologies included acute ischemic infarct and brain tumor.

Keywords — CIHOP, central isolated hand or finger paresis, cortical hand, IHOP, precentral gyrus, stroke.

I. INTRODUCTION

ACUTE isolated hand or finger paresis (IHOP) is frequently caused by a peripheral pathology such as a compression of, or traumatic injury to, the root, plexus, or the individual peripheral nerves in the arm. However, IHOP can also be caused by a central pathology. Recent clinical observations showed that an ischemic infarct in the precentral gyrus causes IHOP and mimics peripheral nerve damage.1-10 In this study we presented five patients with IHOP caused by a brain pathology in discrete locations. We named this entity as central IHOP (CIHOP).

II. METHODS

Patients with acute IHOP were collected for this study. The isolated hand or finger paresis was defined as the weakness in the extension or flexion of the hand and/or intrinsic hand muscles. Patients with a sole presentation of IHOP were included. All patients underwent a standard stroke protocol including: 1) Laboratory studies including hemetogram, chemistry, rapid plasma reagin, erythrocyte sedimentation rate, vitamin B12, homocysteine, lipids, and thyroid stimulating hormone. When indicated, antinuclear antibody, rheumatic factors, protein C, protein S, anti-thrombin III, and lupus anti-cardiolipin antibody were investigated. 2) Neuroimaging studies including a brain CT and MRI with diffusion weighted image (DWI) and co-efficiency factor, proton-density, T1 and T2 weighted and Fluid attenuated inversion recovery (FLAIR) imagings, and MR angiography of the brain and neck. 3) Transthoracic 2-D M-mode spectral and color Doppler echocardiography. Additional transesophageal echocardiography was performed if the transthoracic echocardiography was inconclusive when a cardiogenic etiology was suspected. 4) Doppler and Duplex sonography of the carotid arteries. Conventional angiography of the cerebral vessels nerve conduction study and EMG were performed when indicated. Patients with a history of a previous stroke or concomitant other neurological symptoms or signs, such as cognitive or language dysfunction, cranial nerve deficits, or long-tract signs, were excluded. Patients without an MRI study or with only negative MRI findings were also excluded.

III. RESULTS

A. Demographic data and clinical features

Five patients with IHOP were studied (age: 62.4±8.8 years old, mean±SD; range of 55 to 76). They all presented with an acute onset of isolated hand or finger weakness (Table 1). Two of them complained of
transient local paresthesia, however, without objective sensory deficits on examination. Tendon reflexes were symmetric and normal in 3 patients, while 1 increased in the affected arm and 1 diffusely decreased.

B. Risk factors

Two patients were found to have no risk factors for stroke. Three patients had multiple risk factors including hypertension in 3, cardiac disorders in 2, smoking in 2, previous transient ischemia attack (TIA) in 1, and diabetes mellitus in 1 (Table 2).

C. Stroke workup results

Brain MRI with DWI revealed a small ischemic lesion in the ‘cortical hand area’ of the precentral gyrus in 2 patients, white matter lesion in the centrum semiiovale of the corona radiata in one, the posteriolateral thalamus and the posterior limb of internal capsule in one, and a brain tumor immediately beneath the precentral cortical gyrus in one patient (Table 3, Figure 1).

Two-D echocardiography revealed only one patient with a mildly decreased left cardiac ventricular ejection fraction (40-45%). No atrial fibrillation was observed. Doppler and Duplex sonography of the carotid arteries and MRA of the cerebral vessels disclosed normal findings (Table 3).

IV. DISCUSSION

In the current study, we reported five patients with IHOP resulting from a pathology in a discrete brain location either in the cortical or subcortical regions. This pathology selectively affected the corticospinal motor pathway causing contralateral IHOP, constituting CIHOP.

It has been recognized that ischemic stroke is the major etiology of the brain for CIHOP. Abundant evidence from clinical observations suggests that ischemic infarct to the cortical hand area in the precentral gyrus is responsible for the development of the contralateral IHOP. This claim was supported by our clinical observation (cases 1 and 3). In agreement with the previous reports, our study confirmed that brain MRI with DWI is critical in disclosing the pathology for the diagnosis, because small pathologies were not shown on CT or T2 and FLAIR MRI images (data not shown).

Functional brain MRI and cortical magnetic stimulation demonstrates a reliable landmark for the cortical motor hand area in the precentral gyrus. It is a knob-like structure, shaped like an omega or epsilon in the axial plane and like a hook in the sagital plane. The radial digits to some extent are heavily represented laterally and the ulnar digits more heavily represented.

<table>
<thead>
<tr>
<th>#/S/age</th>
<th>Clinic presentations</th>
<th>Finger extension</th>
<th>Finger flexion</th>
<th>Sensory</th>
<th>Tendon reflexes</th>
<th>Babinski (Rt/Lt)</th>
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<tr>
<td>1F76</td>
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<tr>
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<td>Rt hand weakness, tingling, numbness</td>
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<td>4/5</td>
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<tr>
<td>5M66</td>
<td>Lt hand weakness</td>
<td>4/5</td>
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<td>↓ Lt arm</td>
<td>-/-</td>
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|.Collections of Risk Factors

Table II

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<th>#/S/age</th>
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<tr>
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<tr>
<td>3M60</td>
<td>HTN, CAD, CHF</td>
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<tr>
<td>5M66</td>
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Table I

DEMOGRAPHIC DATA AND CLINICAL FEATURES

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Damage to this area results in contralateral IHOP mimicking pseudoulnar, pseudoradial or pseudomedian neuropathy, or in combination. A small size of acute infarct in the cortical hand area with contralateral IHOP observed in our patients confirmed this claim (Table 3 and Figure 1). The pathogenesis for ischemic stroke for CIHOP may include artery to artery embolic infarction from a plaque of the internal carotid artery or cardiac resources. The prognosis of CIHOP caused by an ischemic infarct is generally favorable.

Interestingly, CIHOP may be caused by an ischemic infarct in the subcortical area, such as thalamus and corona radiata, and may mimic peripheral nerve lesions which was also seen in our study (case 2 and 4). Theoretically, any damage in the corticospinal motor pathway between the primary cortical hand knob area and the contralateral anterior horn of the cervical spinal cord may cause the hand weakness. Fisher reported that over 70 different neurological pictures could be caused by lacunar infarction including pure motor paralysis and IHOP, however, IHOP caused by subcortical infarction is at times clinically indistinguishable from that caused by cortical hand area infarct.

Hypertension is one of the most important modifiable risk factors for stroke and was seen in three of five of our IHOP patients. Additional risk factors include cardiac disturbances, smoking, TIA, diabetes mellitus and hypercholesterolemia (Table 2). Concomitant large artery (such as internal carotid artery) stenosis also contributes to the modifiable risk factors. Cortical stroke caused by internal carotid artery stenosis appears more likely to be artery-artery emboli involving the distal artery, which may include the Rolandic artery in the cortical hand area. Experimental evidence from animal studies suggests that separate emboli from carotid artery may enter the same cerebral artery territory.

Intermittent or prolonged decrease in the cerebral perfusion caused by systemic hypotension or decompensated cardiac function may cause either reversible or irreversable ischemic damage in the cortical hand area. It decreases brain perfusion, alters blood currents, and causes a state of hypercoagulability, which may promote a thrombosis at a terminal branch of the Rolandic artery and cause a stroke.

Additionally, our clinical observation also suggested that IHOP may be caused by a mass lesion (case 5 and Figure 1) or a small hematoma in the cortical hand area after a traumatic injury. A mass lesion in the cortical hand area can be an important etiology for IHOP when it compromises the hemodynamic circulation locally in the precentral gyrus or subcortical structures involving the hand and finger movement. The symptoms may worsen rapidly when vasogenic edema around the mass occurs. An intracerebral hematoma under the cortical hand area caused by traumatic brain injury could be an additional rare etiology for IHOP, whose clinical presentation was clinically indistinguishable from that caused by ischemic stroke.
V. CONCLUSIONS

In summary, IHOP due to central pathology, or CIHOP, may become a clinically diagnostic challenge because it may bear the features of peripheral pathology, particularly in patients with pre-existing peripheral neuropathy. Differential diagnosis of CIHOP should be taken into consideration if an asymmetric or increased tendon reflex in the affected arm is identified. Findings from detailed history, scrutinized physical and neurological examinations are critical. The distribution of the intrinsic hand muscle weakness beyond the territory of one single peripheral nerve may be another valuable token in the differential diagnosis of CIHOP. Brain MRI with DWI is essential in confirming the diagnosis. The prognosis of CIHOP caused by an ischemic infarct is generally favorable.

References

QUESTIONS:

1. The following statements are all correct except:
   
   A: Acute isolated hand or finger paresis (IHOP) can be caused by stroke.
   
   B: IHOP is frequently caused by a peripheral pathology such as a compression of, or traumatic injury to, the root, plexus, or the individual peripheral nerves in the arm.
   
   C: Ischemic infarct in the precentral gyrus causes CIHOP.
   
   D: Ischemic infarct in the medioposterior thalamus causes CIHOP.
   
   E: CIHOP may become a clinically diagnostic challenge because it may bear the features of peripheral pathology

2. Ischemic infarct in the following location(s) may cause CIHOP:
   
   A: Precentral gyrus.
   
   B: Centrum semiovale of the corona radiata
   
   C: Both A and B.
   
   D: Neither A or B.

3. CIHOP may manifest as following except:
   
   A: Pseudomedian neuropathy.
   
   B: Pseudoradial neuropathy.
   
   C: Pseudoulnar neuropathy.
   
   D: Pseudoulnar and pseudomedian neuropathy.
   
   E: Peudo musculacutaneous neuropathy.

4. The modifiable risk factor for CIHOP including all of the following except:
   
   A: Aging.
   
   B: Atrial fibrillation.
   
   C: Hypertension.
   
   D: Smoking.
   
   E: Diabetes mellitus

ANSWERS:

1. D; 2. C; 3. E; 4. A.