

Case Report

Ischemic stroke cases presenting with hand weakness mimicking peripheral neuropathy

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ABSTRACT

Pure motor monoparesis due to ischemic stroke involving a single extremity is a rare condition that can be easily misdiagnosed. Herein, we present three cases with isolated upper extremity monoparesis. All of our patients had weakness in the left hand. They were previously evaluated in other centers and were diagnosed with peripheral neuropathy by electromyography. When patients whose complaints did not resolve were admitted to our clinic, we performed electromyography again and observed that it was normal. Hereupon, we detected ischemic infarctions in cranial magnetic resonance imaging that would explain the patients' complaints. Isolated monoparesis mimicking peripheral neuropathy is a rare symptom in stroke patients. Pure motor monoparesis should be kept in mind in every patient presenting with acute-isolated monoparesis, and neuroradiological imaging should be performed.

Keywords: Hand weakness, ischemic stroke, lacunar infarct, peripheral neuropathy, pure motor monoparesis.

Pure motor monoparesis (PMM) involving a single arm or leg due to ischemic stroke is a rare condition that can be easily misdiagnosed.^[1] It was first described by Lhermitte^[2] in 1909 and named pseudoperipheral palsy. According to the criteria determined in the studies conducted, PMM is diagnosed by an isolated motor deficit in the leg or arm without a sensory impairment, lack of coordination, or significant involvement in speech or ipsilateral face, and ischemic stroke must be confirmed radiologically. It has been reported that hemorrhagic stroke can also cause PMM, but it is seen less frequently than ischemic stroke. An ischemic infarction in the cortical region is more likely to cause PMM than deep structures since descending motor axons are compressed into a small area in the subcortical region, capsula interna, and brain stem. Pure motor monoparesis is less likely to be seen in the lesions involving these areas.^[3,4] There might be finger

involvement mimicking ulnar and radial paralysis in upper extremity distal motor involvement due to cortical infarction.^[5,6] Surprisingly, even a precentral knob infarction that mimics anterior interosseous nerve involvement has been reported.^[7] This report aimed to raise awareness on this issue by presenting three patients with PMM who were first thought to have peripheral nerve pathology and then found to have an ischemic stroke.

CASE REPORT

Case 1- A 77-year-old female patient presented with painless weakness in the left hand, which they had noticed six weeks ago. The patient stated that an EMG was performed in another center, and a diagnosis of brachial plexopathy was made. The neurological examination conducted in our clinic revealed weakness

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in the distal left upper extremity. Wrist extensors were markedly weak. Sensory examination was normal. Deep tendon reflexes (DTRs) were brisk in the left upper limb, and there was no pathological reflex. Left lower extremity motor examination was normal. The patient was under follow-up due to rheumatoid arthritis, and there were joint deformities in the elbows and wrists. An EMG was performed, revealing no signs of disease. Cranial magnetic resonance imaging (MRI) was requested, which revealed subacute ischemia areas in the right centrum semiovale and parietal region (Figure 1). The patient was started 100 mg acetylsalicylic acid. After the diagnosis, the patient received physical therapy for three weeks. Muscle strength was normal at the control examination three months later. However, fine motor skill impairment of the hand persisted.

Case 2- A 67-year-old male patient, who used medications due to diabetes mellitus, hypertension, and hyperlipidemia, presented with painless weakness in their left hand, which had started five months ago. The patient described that an EMG was performed in another center, and a diagnosis of left ulnar/median neuropathy was made. The neurological examination revealed mild weakness in the distal muscles (hypotenar, thenar, interosseous, and finger

extensors), and the sensory examination was normal. Deep tendon reflexes were normal in the upper extremities and could not be evaluated in the lower extremities. There was no pathological reflex. A mild sensorimotor axonal polyneuropathy was detected in the EMG, but no sign of isolated neuropathy was detected in the left upper extremity. Afterward, a brain MRI was performed, and multiple hyperintense ischemic gliotic foci and acute ischemic lesions in the right frontal and centrum semiovale were detected. On MRI angiography, stenosis of more than 80% in the intracranial part of the right internal carotid artery was detected, and stenting was performed. Acetylsalicylic acid and clopidogrel were started after stenting. After six weeks, the patient's complaints improved almost completely.

Case 3- A 52-year-old male patient presented with painless weakness in their left hand, which had started 20 days ago. The patient revealed that they were evaluated by EMG and cervical MRI, which were inconclusive. In the neurological examination, the patient's left-hand distal muscles were plegic. Proximal muscles of the left upper extremity, right upper extremity, and bilateral lower extremity muscles were normal. There was no pathological reflex, and DTRs were normal. The patient's blood pressure



Figure 1. Subacute ischemia areas in the right centrum semiovale in the magnetic resonance imaging fluid-attenuated inversion recovery sequence.

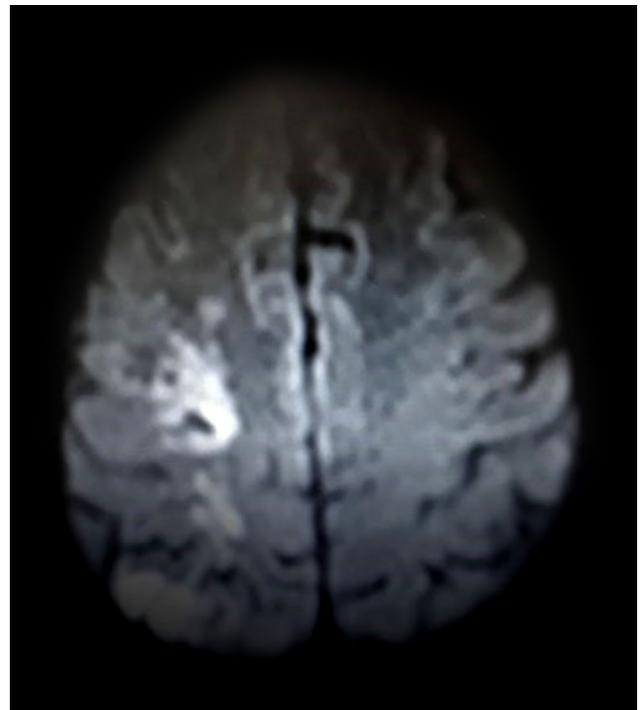


Figure 2. Acute ischemia with the omega sign in the axial plane in the magnetic resonance imaging diffusion sequence.

was measured as 140/90 mmHg. Cranial MRI was performed for the patient whose EMG was normal. In MRI, acute ischemia was observed in the right centrum semiovale and frontoparietal region (Figure 2). The patient was started 100 mg acetylsalicylic acid. After the diagnosis, the patient received physical therapy for three weeks. Muscle strength was normal at the control examination one month later; however, fine motor skill impairment of the hand persisted.

DISCUSSION

Isolated monoparesis, which can mimic peripheral neuropathy, is a rare symptom in stroke patients. The prevalence of PMM has been reported as between 2 and 13% in different studies.^[3,8] Cortical infarcts that cause PMM in the upper extremity have been reported as mid-rolandic region lesions, anterior superficial middle cerebral artery (MCA) infarcts, posterior superficial MCA infarcts, and pial branch infarcts of MCA.^[4] The precentral gyrus or parietal lobe has often been reported as the region responsible for upper extremity monoparesis or PMM due to cortical infarction.^[1,7,8] Parietal lobe lesions are known to be associated with motor disorders in animal models as well as in humans.^[4] Timsit et al.^[7] described a nonpyramidal motor deficit that develops in association with an inferior parietal lobe lesion. A large study conducted showed that 10 patients with subcortical (corona radiata or centrum semiovale) ischemia had PMM, and the majority were in the upper extremity.^[4] In all three of our cases, there was an infarction in the centrum semiovale, and it was observed that all of them had upper extremity monoparesis. In another study conducted in 2005, it was reported that facial deficits were mostly associated with subcortical lesions, arm deficits with MCA lesions, and leg deficits with anterior cerebral artery lesions.^[3]

In PMM patients with upper extremity involvement, the neurological deficit is limited to only the hand or fingers, and this condition can be misdiagnosed as radial neuropathy, ulnar neuropathy, and plexopathy since there are no cortical, pyramidal, and cerebellar findings.^[9] All three of our patients were primarily considered to have peripheral nerve pathology and were investigated. Similarly, ischemic infarcts in the centrum semiovale, frontal, or parietal cortical regions were detected in all patients in the brain MRI performed since no abnormality was present in the history, neurological examination, and EMGs. More importantly, right carotid stenosis was

detected in the etiology of ischemic infarction in our second case, and stenting was performed, which was vital for the patient.

It has also been reported that in PMM, wrist extensors are affected more than wrist flexors; however, there is no significant difference in finger flexors and extensors.^[1,5] In our first case, significant wrist extensor weakness was observed. Normal or symmetrical DTR findings are more prominent in patients with weakness in only one finger or certain fingers.^[1]

Pure motor monoparesis should be kept in mind in patients above a certain age, with risk factors such as hypertension, diabetes, hyperlipidemia, and smoking, particularly those with isolated monoparesis. However, the patient's young age and the absence of any known risk factors should not mean that PMM will not occur. Our third case was a 52-year-old patient with no known risk factors. The detailed neurological examination, the patient's blood pressure, measured as 140/90 mmHg, and rationalizing PMM on the differential diagnosis were what guided us in this case.

It is crucial to perform radiological imaging after a detailed history and neurological examination in cases suspected of PMM. Diffusion MRI should be performed as a neuroimaging method. In computed tomography scans performed without diffusion-weighted imaging, acute infarction and minor cortical infarctions may be overlooked or misidentified. Diffusion MRI was performed in all of our cases, and acute/subacute infarcts were detected. As a result, the patients were diagnosed with PMM. In a study published in 2020, a region in the motor function of the hand, extending from the precentral gyrus into the central sulcus, was defined.^[10] Lesions in this region were identified as the "omega" sign in the axial plane of MRI and the "hook" sign in the sagittal plane. There was an image similar to the omega sign in the MRI of our third case (Figure 2).

Among PMMs, the prognosis of isolated upper extremity monoparesis is generally good, and the patients recover completely or almost completely.^[3,6]

Studies on ischemic stroke localization have shown that lesions of the corticospinal tract and the opercular cortex around the insula are associated with higher functional impairment.^[11,12] It has also been reported that there is a relationship between the subcortical location of the stroke and the presence and severity of long-term functional impairment.^[13] It is a known fact that functional improvement is related to lesion

size, lesion location, and age of the patient, and the effect of early physical therapy on functional recovery cannot be denied.

In conclusion, isolated monoparesis mimicking peripheral neuropathy is a rare symptom in stroke patients. Pure motor monoparesis should be kept in mind in every patient presenting with isolated monoparesis, with or without vascular risk factors, and a detailed neurological examination and neuroradiological imaging should be performed.

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