Periodic limb movement during sleep (PLMS) is defined as repetitive and regular involuntary movement. It occurs primarily in the legs during sleep and usually consists of foot dorsiflexion and less often flexion at the knee or the hip. PLMS occurs 10-20% of general population and most prevalently in restless legs syndrome (RLS), which accounts for 80-90% of cases, but it can also co-occur with various medical and neurological disorders, including cerebrovascular stroke. Previously reported cases of PLMS following ischemic stroke exhibited different clinical manifestations according to the lesion site. These cases of PLMS were associated with motor weakness with or without RLS. A 53-year-old male patient presented with dysarthria without other limb motor weakness after the right globus pallidus infarction. Overnight video-polysomnography revealed newly developed PLMS in the left leg without symptoms of RLS. This case indicates that the globus pallidus could be involved in the pathophysiology of PLMS without RLS.

**Key Words:** Periodic limb movement, Stroke, Basal ganglia.

**Case Report**

A 53-year-old right-handed man was admitted to our hospital complaining of dysarthria, which had started approximately 2 days prior to admission. There was no previous history of medical conditions or drug use. The patient had no other symptoms besides dysarthria. On neurological examination, the patient was found to be alert, and there were no changes in motor strength, sensory abilities, pathologic reflexes, or cortical function. The brain 3.0-tesla magnetic resonance imaging (Achieva 3.0T; Philips, Eindhoven, Netherlands), including diffusion-weighted imaging, revealed an acute ischemic stroke in the right globus pallidus along with a tiny portion of adjacent area (Fig. 1). Besides snoring, the patient had no history of abnormal movement or sensation in the legs, insomnia, daytime sleepiness, or other abnormal sleep-related behavior or movement before the ischemic stroke. During his hospital stay, repetitive movement in the left leg during sleep was observed by chance. Video-polysomnography (COMET PSG; Grass Technologies, Twin 4.5.2 Software, Warwick, RI, USA) was performed, and it revealed regular and repetitive movement in the left leg, with an electroencephalographic microarousal following periodic movements (Fig. 2). The polysomnography began at 09:12:53 PM and ended at 04:08:48 AM the next day. Total sleep time was 303 min with sleep efficiency of 73.0%. Total periodic limb movements during sleep were 83 (left, 72; right, 11) with a PLMS-index of 16.4 (left, 14.3; right, 2.1). The apnea-hypopnea index was 17.6 with a minimum oxygen saturation of 92%. The patient had new onset of leg movements. He was treated with an antiplatelet agent and...
PLMS Following Stroke

Figure 1. Axial and coronal diffusion-weighted imaging reveals acute infarction in the right globus pallidus and adjacent area.

Figure 2. Polysomnographic recording shows 5 periodic leg movements (a-e) in a 120-s epoch. The movements of left leg are observed at 12- to 45-s intervals, which persist for 0.5-1 s. These movements are associated with electroencephalographic microarousal (*). LOC-M2 and ROC-M1 are 2 channels used for electrooculography. Chin-Chin 1 and Chin 1-Chin 2 are 2 channels used for chin electromyography, F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, and O2-M1 are 6 channels used for electroencephalography. LAT: left anterior tibialis electromyogram, RAT: right anterior tibialis electromyogram, Snore: snore sensor, CHEST: chest respiratory movement, ABD: abdomen respiratory movement, PTAFlower: pressure transducer air-flower, Thermister: oro-nasal thermal sensor, EKG: electrocardiography, SaO2: oxyhemoglobin saturation by pulse oximetry.
the risk factor for ischemic stroke was assessed. Since the patient had no accompanying subjective sleep disturbance due to PLMS, he was discharged without medication for PLMS.

**Discussion**

Our patient predominantly experienced asymptomatic PLMS in the left leg, as indicated by video-polysomnography. On admission to hospital, the periodic movement was observed incidentally by his wife who was the caregiver. Based on the previous history, imaging studies, and polysomnographic findings, the patient was diagnosed with PLMS following stroke. The reported prevalence of PLMS in the population is 7.6%, and it is common even in the absence of other sleep complaints, particularly in elderly people. Therefore, PLMS is often discovered coincidentally on polysomnography. However, both the patient and his wife denied that there was any abnormal limb movement during sleep or that he experienced any sleep disturbance before the ischemic stroke. Hence, we concluded that the ischemic stroke in the right globus pallidus was possibly responsible for the contralateral PLMS without RLS.

There have been several previous reports of PLMS following ischemic stroke corresponding to the lesion site, which demonstrated different clinical manifestations (Table 1). In a prospective study, 54.3% of patients with supratentorial infarction had PLMS. The anatomical sites of PLMS were middle cerebral artery territory, anterior cerebral artery territory, corona radiata, basal ganglia, thalamus, and centrum semiovale. However, this study questioned the possibility of premorbid PLMS before stroke. Although there was no detailed clinico-anatomical correlation in each case, most patients demonstrated PLMS contralateral to the lesion. The majority of PLMS cases following ischemic stroke was observed along with motor weakness, or during recovery periods. In the case of accompanying pyramidal tract damage, there was no clear RLS. However, when damage to the extrapyramidal tract occurred, RLS was also observed. Unlike the case of PLMS following a lesion to the lenticulostriate region, no accompanying symptoms related to RLS were found in our patient.

Periodic limb movement during sleep is similar to the Babinski sign and occurs without plantar stimulation, which suggests a central origin. It is known that damage to the pyramidal tract in the cortex or subcortex leads to a loss of suppression of the reticular structures and, consequently, may lead to the symptoms commonly seen as a result of supratentorial lesion.

In conclusion, our case presented PLMS without RLS, which developed following a globus pallidus infarction with no accompanying motor weakness. We suggest that the globus pallidus may be a neuroanatomical site within the basal ganglia involved in the pathophysiology of PLMS without RLS.

**REFERENCES**