Dear Editor,

The symptoms of pontine infarction include focal deficits such as hemiparesis, facial palsy, dysarthria, disorders of eye movements or vertigo. However, involuntary abnormal movements, such as dystonia, chorea, tremor, periodic limb movements in sleep (PLMS) or restless leg syndrome, due to pontine infarction have been reported, albeit rarely. Here, we report one such rare case of a patient who developed involuntary abnormal leg movements after pontine infarction.

A 58-year-old man developed an acute onset right upper and lower extremity weakness along with complaints of dysarthria. His symptoms lasted approximately 15 min with a spontaneous resolution. Upon arrival at our hospital, he was asymptomatic. His medical history included significant hypertension requiring medication. He had a 40 pack-year history of smoking. He was alert and oriented, and had no cranial nerve function deficits. The rest of his neurological examination was unremarkable with a normal motor and sensory examination, and normal deep tendon reflexes. However, a diffusion-weighted magnetic resonance imaging (MRI) revealed an acute infarction in the left anterior pons (Figure 1A). In addition, a computed tomographic angiography revealed an apparent focal stenosis in the left middle cerebral artery. He was then admitted to our hospital.

On the day after admission, he once again developed motor weakness of the right arm and leg including a right facial palsy. His neurologic exam demonstrated grade IV/V muscle strength along with increased deep tendon reflexes and a positive Babinski’s sign in the right side. The patient was prophylactically treated with aspirin and clopidogrel for the prevention of further vascular events. However, seven days after admission, his strength was found to be decreased to a grade III/V. A follow-up diffusion-weighted MRI of his brain was performed which demonstrated an increased involvement of the left pons, now including the tegmentum (Figure 1B). At this time, he began to complain of abnormal movements in his right leg which when examined were rhythmic repetitive stereotypic dorsiflexion of the big toe, dorsiflexion at the ankle and flexion at the knee and hip joints. These movements were involuntary lasting approximately one to three seconds about every 5 to 10 minutes during the day and occurring more frequently at night. He exhibited no involuntary movements in any other parts of his body including the left leg. Furthermore, electroencephalography performed at this time revealed no epileptiform discharges. He was started on ropinirole and clonazepam, and the symptoms began to gradually improve after the 5th day of treatment.

Post-stroke movement disorders are well-known, and their prevalence is estimated to be approximately 1%. The most common sites of lesions that induce movement disorders are the basal ganglia and thalamus. However, involuntary abnormal movements are rare following pontine infarctions. We encountered a patient who exhibited involuntary leg movements following a pontine infarction. The temporal relationship between the pontine infarction and the newly developed involuntary leg movements favored the existence of a causal relationship. Additionally, the observation that the involuntary movements only involved in the paretic right leg also supports the association between these movements and the pontine infarction.

Although a polysomnography was not performed, the kinesiethics of his movements were relatively compatible with PLMS. PLMS is characterized by recurrent episodes of repetitive and highly stereotyped limb movements that predominately occur during deep sleep. The patient was admitted to our hospital on the fifth day of treatment, and we performed a follow-up diffusion-weighted MRI of his brain, which revealed an increased involvement of the left pons, now including the tegmentum (Figure 1B). At this time, the patient began to complain of abnormal movements in his right leg, which were rhythmic repetitive stereotypic dorsiflexion of the big toe, dorsiflexion at the ankle, and flexion at the knee and hip joints. These movements were involuntary, lasting approximately one to three seconds, about every 5 to 10 minutes during the day, and occurring more frequently at night. He exhibited no involuntary movements in any other parts of his body, including the left leg. Further, electroencephalography performed at this time revealed no epileptiform discharges. He was started on ropinirole and clonazepam, and the symptoms began to gradually improve after the 5th day of treatment.

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nantly occur in the legs.\(^3\) The movements observed in our case were similar to those of PLMS and consisted of dorsiflexion of the ankle and flexion of the hip and knee.\(^3\) Additionally, the involuntary movements occurred periodically with an increase in frequency at nighttime, which is consistent with the patterns seen in PLMS. Although various conditions, such as peripheral neuropathy, uremia, anemia and chronic pulmonary disease, are known to cause PLMS, pontine infarction-induced PLMS is rare, and only a few such cases have been reported.\(^3\) The exact pathogenesis of pontine infarction-related PLMS is unknown. One plausible explanation is related to the association with reticular formation lesions. Due to the relationship between sleep and PLMS, the generator of PLMS is presumed to be associated with sleep-related structures, such as the reticular formation in the brainstem.\(^3\) The findings of our case support such a hypothesis. Initially, the anterior pontine infarction produced a right hemiparesis, and the involuntary movements appeared only after the progression of the stroke with tegmentum involvement, where the reticular formation was presumably located. Another hypothesis is that the PLMS originates from a lesion in the pyramidal tract. The leg movements observed in PLMS are similar to the Babinski's sign, i.e., dorsiflexion of the big toe and ankle joint, which is typically observed in cases of pyramidal tract lesions. In addition, the movements in PLMS are seen more often during sleep when the Babinski's sign can sometimes be observed as a normal variation.\(^3\) Our patient's symptomology also supports this hypothesis. However, he denied any history of sleep disorders, including PLMS, prior to his pontine infarction, and his involuntary leg movements developed only after the development of hemiparesis. One previous report of involuntary movements following ischemic stroke in the corona radiate also supports our hypothesis that the loss of cortical or subcortical inhibition resulting from a pyramidal tract lesion might cause some cases of PLMS.\(^6\)

Another possible explanation for the involuntary movements observed in our patient with a pontine infarction is a releasing phenomenon of a rhythmic generator. There is some evidence that a central rhythm generator for locomotion exists at the lumbar level of the spinal cord and generates rhythmic and coordinated locomotion in the absence of external drive.\(^7\) This locomotion center might be influenced at the supraspinal level, such as pontine tegmentum which is known to be one of the sites that suppress this center through the enhancement of GABA (gamma aminobutyric acid) ergic action. Thus, destructive lesions, such as ischemic infarctions in the pontine tegmentum, might decrease the GABA mediated inhibition on this motor rhythmic generator, allowing for it to operate without regulation leading to abnormal movements.

In conclusion, pontine infarctions can produce involuntary leg movements requiring careful neurologic examinations specifically for deficits associated with pontine lesions should be performed in all patients with new-onset involuntary leg movements.

**Conflicts of Interest**

The authors have no financial conflicts of interest.

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