
A Case of Post-stroke Rhythmic Akathisia

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Dyskinesia can occur as a neurological abnormality due to stroke, and its incidence in stroke patients is reported to be about 1%. It is possible to classify dyskinesia into one of the morphologic types already classified clinically. However, a specific type of dyskinesia can occur; one which does not fall into the existing morphologic types. We experienced such a case of specific type dyskinesia, which couldn't be classified into the existing classification system. A 50-year-old man visited our hospital due to rhythmic dyskinesia of the right hand, which appeared during the resting state, and had developed one month after left subcortical infarction. Flexion and extension movements of the fingers at 3Hz appeared due to the impatient impulse to move. However, this abnormal movement could be easily suppressed under the patients will. We suggested that the abnormal movement was similar to akathisia from the fact that it occurred due to the internal desire to move and that the patient could suppress dyskinesia. However, the rhythmic tendency and lack of medication history of antipsychotics suggested that the movement was not the typical form of akathisia. The present case may represent a new clinical type of movement disorder developed after stroke. Considering the clinical pattern of the present case and following a review of the literature, we believe that it can be labeled, post-stroke rhythmic akathisia.

Key Words : Rhythmicity, Akathisia, Stroke

Various reports have suggested that hyperkinetic movement disorders are uncommon in acute stroke. Ghika Schmid et al.¹ have identified 29 patients with acute or delayed movement disorders among 2500 acute stroke patients. Recently, we have experienced 34 post-stroke tremor patients among approximately 2000 acute stroke patients over a 5-year period. Our results suggested that post-stroke dyskinesia are not very rare symptoms of stroke. Furthermore, it is suspected that clinical patterns are also diverse. We have recently experienced a new form of post-stroke dyskinesia which was fairly rhythmic. As

far as we know, only one case of post-stroke akathisia has been reported until now.¹ The symptoms and signs are partly compatible with akathisia. However, the rhythmic tendency and lack of any medication history of antipsychotics suggested that dyskinesia were not the typical form of akathisia. The present case may represent a new clinical type of movement disorder developed after stroke. Therefore, we report the case and discuss with reference to related articles.

Case Report

A 50 year-old man was admitted to our hospital with chief complaints of altered mentality and dysarthria. Neurologic examination showed right hemiparesis and right hemifacial palsy. Brain CT showed multiple subcortical infarct(Fig. 1). Two months later, when he visited NR OPD, unvolun-

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Figure 1. Brain CT finding. Brain CT showed multiple subcortical infarct. Right subcortical lesion was related to the dyskinesia.

tary movements in the right hand were noticed (Fig. 2). These dyskinesia, which didn't seem to cause any stress to the patient, appeared during the resting state and were fairly rhythmic. Flexion and extension movements of the fingers at 3Hz appeared due to the impatient impulse to move (Fig. 3, 4). The dyskinesia were not purposeful, and could be easily suppressed under the patient's will. However, whenever concentration was disturbed, they reappeared. There was no previous medication history of antipsychotics. Furthermore, there were no combined extrapyramidal signs such as rigidity, akinesia, or dystonia, nor any strong affective components of

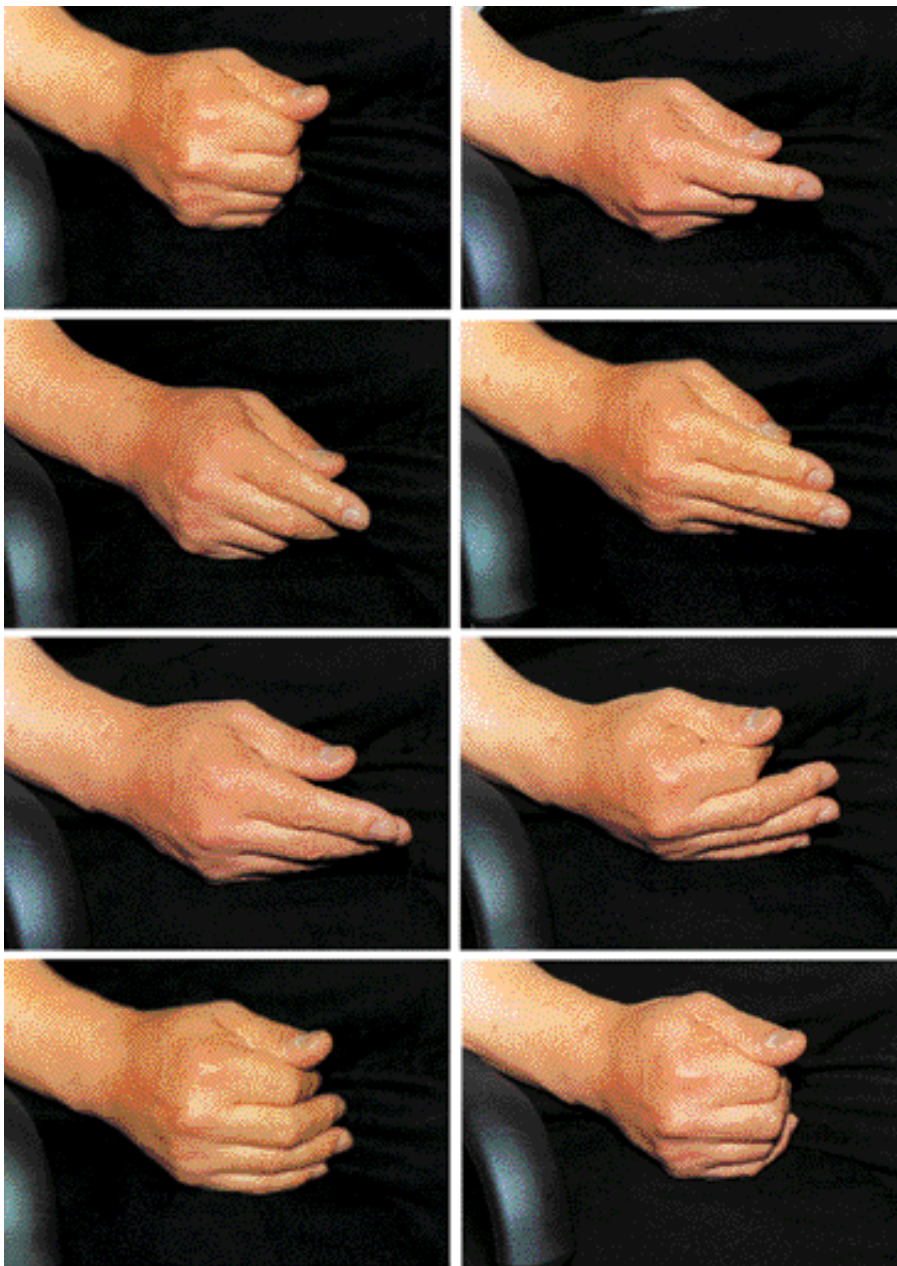


Figure 2. A series of movements. Adduction of thumb was followed by subsequent movements, such as extension of index finger, extension of 3rd, 4th, 5th fingers, abduction of thumb, flexion of index finger, flexion of 3rd, 4th, 5th fingers.

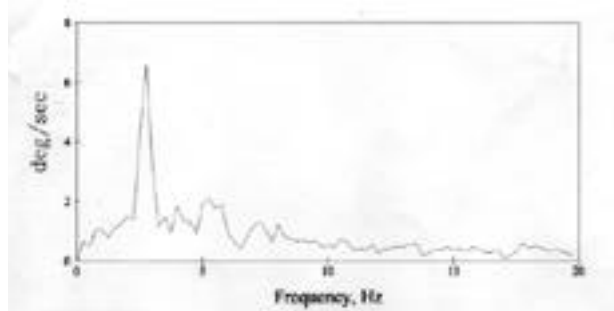


Figure 3. Spectral analysis of involuntary movements. Spectra show angular rate RMS for the frequency components of abnormal movements plotted against frequency. 3 Hz involuntary movement component was most frequent.

fright, terror, anger, rage or anxiety. He showed additional kinetic, 10 Hz tremors in the right hand. Bzotropine was prescribed, but did not prove effective.

Discussion

The term akathisia (not sitting) was first introduced by Haskovec in 1901 to describe individuals unable to remain in a seated position. In consideration of the combined occurrence of akathisia with extrapyramidal signs of parkinsonism, dystonia, and choreoathetosis, and of the improvement of neuroleptic akathisia with anticholinergic drugs, akathisia can be regarded as an extrapyramidal phenomenon. Patients with akathisia usually have strong subjective feelings of internal discomfort and restlessness, with a secondary need to move in order to relieve the uncomfortable sensation. We believe that the abnormal movements of our patient were similar to akathisia, given that they were present due to the internal desire to move and that the patient could suppress them. However, the dyskinesia showed strong rhythmicity, and there was no medication history of antipsychotics. Usually, strong affective components of fright, terror, anger, rage and anxiety may be included in acute akathisia. Our patient, nevertheless, had no such strong affective symptoms except for an unstable feeling of the desire to move. The movement was partly compatible with akathisia. However, some other previously described characteristics suggested that the movement was not the typical form of akathisia. The term, rhythmic akathisia, has not been used in the medical field yet. As far

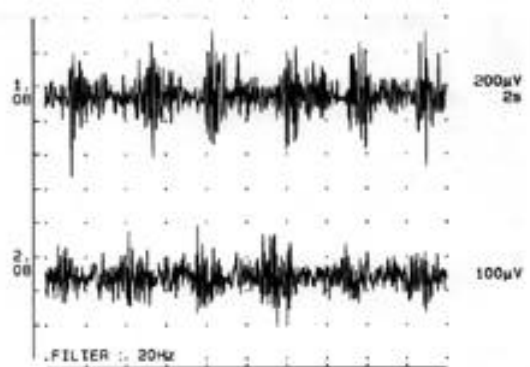


Figure 4. Alternating (60%) and co-contracting (40%) EMG activities at 3 Hz frequency were recorded from both finger flexor and extensor muscle groups of the right upper extremity.

as we know, only one case of post-stroke akathisia has been reported until now. Therefore, in consideration for the clinical patterns, the condition of our patient was termed, post-stroke rhythmic akathisia.

According to the literature, rhythmic movement is seen rarely in akathisia. Even though akathisia appears as repetitive movements, it does not persist. The repetitive movement usually occurs in bursts of varying duration. Periods without repetitive movements are interspersed with bursts during which the movements occur with great regularity.² These burst-like temporal patterns of repetitive movements are different from the dyskinesia of our case. Our patient showed slow, persistent rhythmic movements. Braude et al.³ identified a "coarse tremor" phenomenon in akathisia patients. It is a low frequency, large amplitude rhythmic movement in the feet of patients with akathisia. They insisted that this phenomenon could facilitate the clinical identification of akathisia. Although the body portion is different, this phenomenon is similar to the dyskinesia of our case. In accordance with their suggestion, our case could be regarded as a variant of coarse tremor phenomenon. Further observations and studies are needed for the verification of this "coarse tremor" phenomenon.

The onset of dyskinesia after stroke was remarkably varied. Some patients showed dyskinesia at stroke onset time while others exhibited delayed-onset dyskinesia 10 years after the stroke. The underlying mechanisms for the delayed onset of dyskinesia are poorly understood. The latency between stroke and onset of dyskinesia may reflect the time required for

occurrence of remyelination, inflammatory changes, ephaptic transmission, oxidative reactions, central synaptic reorganization, trans-synaptic neuronal degeneration, and diaschisis mediated by collateral sprouting and denervation supersensitivity. It is suspected that incorrect recovery of the damaged neural circuit may be responsible for delayed onset.

Kim⁵ suggested that persistent failure of the proprioceptive sensory and cerebellar inputs, in addition to successful, but unbalanced, recovery of motor dysfunction, seemed to result in a pathological motor integrative system and consequent dyskinesia in stroke patients. His proposition could be applicable to most patterns of dyskinesia. Although the involved arm was weak in our case, dyskinesia could be suppressed completely with the patient's will. Furthermore, dyskinesia were not lessened by sensory stimuli and there were no geste antagonistique. Therefore, it is suspected that the underlying mechanisms of our case might be different from Kim's suggestion. Marsden and Jenner(1980) have proposed that akathisia may be a result of post-synaptic dopamine receptor blockade in cerebral dopamine-containing regions of the brain other than the corpus striatum. Tassin et al.⁷ insisted that destruction of mesocortical dopamine neurons is critical for the development of locomotor hyperactivity. The mesocortical dopamine system projects from the ventral tegmental area of the midbrain to the neocortex. Therefore it was suspected that this system might have been injured at the subcortical level in our case. Moreover, it was also suspected that underlying mechanisms of rhythmicity might have been related to the olivo-cerebellar-thalamo-cortical circuit.

Rhythmic akathisia should be differentiated from similar diseases such as pseudoakathisia, psychic agitation, tardive dyskinesia, secondary akathisia, malingering, etc. "Pseudoakathisia" refers to motor manifestations of akathisia without subjective experiences of restlessness. Patients with "pseudoakathisia" have a high incidence of orofacial and limb dyskinesia. Our

patient had no previous medication history of antipsychotics, nor any orofacial or limb dyskinesia. Patients with psychotic agitation may exhibit excessive movement, but usually in the form of semi-purposeful movements of the upper extremities, such as handwriting and repetitive activities.⁸ The movement pattern of our patient was not semi-purposeful. Furthermore, our patient did not experience internal restlessness. As previously mentioned, there was no medication history of antipsychotics. Our patient could suppress dyskinesia completely as well. Therefore tardive dyskinesia could be differentiated. Our patient showed neither signs of Parkinsonism nor encephalitis. Secondary akathisia also could be differentiated. When our patient was disturbed, dyskinesia reappeared. So malingering was less likely.

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