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Case Report

Freezing of Gait Following Hypoxic Brain Injury
- Two Cases Reports -

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Freezing of gait (FOG), which is the most common symptoms in Parkinson's disease, is a unique gait disorder that patients are unable to initiate or continue locomotion. However, the pathophysiology of FOG has been poorly understood. We report two cases, one case is a 26-year old man and the second case is a 65-year old man, who showed FOG following hypoxic brain injuries caused by sudden cardiac arrest and hypovolemic shock, respectively. Brain F-18 FDG-PET images demonstrated the diffuse cortical hypometabolism in case 1 patient, and the decreased metabolism of the subcortical structures in case 2 patient. Two patients showed the typical features of FOG (turning, destination, and tight quarter hesitations combined with kinesia paradoxa) and the abnormal patterns of temporospatial data in kinematic gait analysis. We present two cases of FOG following hypoxic brain injury with reviewing of some literatures.

Key Words Freezing of gait, Hypoxic brain injury

INTRODUCTION

Freezing of gait (FOG) is a typical symptom of degenerative central nervous system (CNS) diseases, which indicates a short and sudden gait disorder that occurs when one starts walking or turns in another direction while walking or gets to one's destination after walking. Most patients with FOG feel as if their feet are glued to the ground, and in serious cases it may block locomotion. FOG, which blunts the sense of balance in the process of ambulation, is an important factor of a hurt from a fall. Nevertheless, its pathogenesis has not been clearly unraveled and also any effective treatments have not been developed yet. In particular, there have been few reports of FOG in patients with hypoxic brain injury (HBI). This paper is to report 2 cases of FOG in patients after HBI.

CASE REPORT

Case 1

A 26-year-old male, who had no significant medical history and neurologic abnormalities, was admitted to the emergency room due to sudden cardiac arrest. After undergoing cardiopulmonary resuscitation, he was transferred to the cardiology ward. The pharmacotherapy was performed with diagnosis of idiopathic ventricular fibrillation, J-wave syndrome and HBI. On a fluid-attenuated inversion recovery-magnetic resonance imaging (FLAIR-MRI) scan performed on the brain 1 month after hospitalization, the diffuse cerebral edema was observed (Fig. 1-A). A cardiac pacemaker was set in the patient 2 months after hospitalization. One month later, the patient was transferred to the rehabilitation department, where he underwent comprehensive treatments for cognitive dysfunction and gait disturbance.

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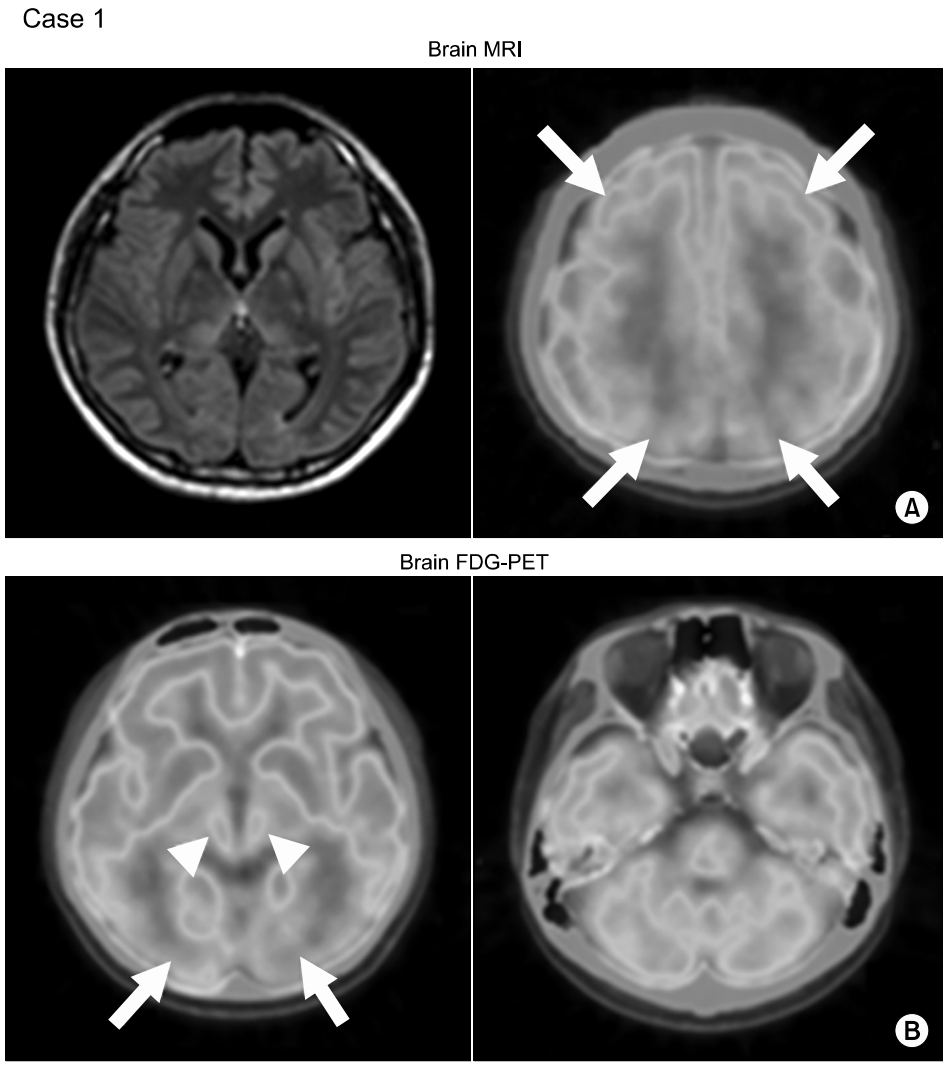

Fig. 1. A 26 year-old man associated with hypoxic brain injury following sudden cardiac arrest. (A) T2-flair MR axial image showed diffuse brain swelling. (B) Positron emission tomography (PET) axial views showed decreased FDG uptake in the bilateral frontal, parietal, and occipital lobes (white arrow) and metabolic defect in the bilateral thalamus (white arrow head).

The neurologic examinations found that the patient was alert with the JFK coma recovery scale (JFK-CRS) score of 23. But on the Korean mini-mental state examination (K-MMSE), the patient showed the cognitive dysfunction with the score of 21. On positron emission tomography (PET) performed on the brain, the cerebral hypometabolism was observed on both frontal, both parietal, and both occipital lobes and both sides of the thalami (Fig. 1-B).

The patient could walk by himself when he was transferred to the rehabilitation ward, but showed gait disturbance. An inquiry, made of the patient's care-giver, found that the patient showed gait disturbance from early ambulatory phase after cardiac arrest. On physical examinations, he showed a shuffling gait when turning, hence the time up and go test (TUG) was performed under suspicion of FOG. On TUG, turning hesitation and destination hesitation were observed. Also, tight-quarter hesitation was observed whenever the patient passed through the room door. Moreover, the patient showed kinesia paradoxa whenever crossing a line (Table 1). On a FOG questionnaire (FOG-Q)[1] performed to measure the severity, the patient was classified as moderate level of FOG with the score of 14 out of 24. A gait analysis, performed prior to rehabilitation, showed the delay of cadence and double support time, but the shortage of step length, step time, walking speed and single support time (Table 2).

The patient underwent balance and gait training, and comprehensive rehabilitation for 2 months, but did not show significant changes in clinical characteristics and gait (Table 1). Afterwards, the patient moved to another hospital so as to continue to undergo rehabilitation.

Table 1. Subtypes of Freezing of Gait and Occurrence of Kinesia Paradoxa in Two Patients with Hypoxic Brain Injury before and after Rehabilitative Treatment

	Case 1		Case 2	
	Initial	Follow up	Initial	Follow up
Subtypes of freezing of gait				
Start hesitation	−	−	−	−
Turning hesitation	+	+	+	+
Destination hesitation	+	+	+	−
Tight quarter hesitation	+	+	+	+
Kinesia paradoxa	+	+	+	+
Freezing of gait questionnaire (total 24)	14	14	19	17

Table 2. Spatiotemporal Data of Kinematic Gait Analysis in Two Patients with Freezing of Gait after Hypoxic Brain Injury

	Case 1	Case 2	Normal adults
Cadence (steps/min)	128	203	106
Walking speed (m/s)	0.42	0.09	1.16
Step length (m)	0.17	0.02	0.61
Step time (sec)	0.49	0.34	0.51
Single support time (%)	30.6	21.8	38.4
Double support time (%)	31.1	57.1	23.2

Case 2

A 65-year-old male, who had no significant medical history and neurologic abnormalities, had gone into hypovolemic shock as he had been stabbed in the abdomen, and as a result sustained cardiac arrest and HBI. Four months later, he had been able to walk by himself but showed gait disturbance. Six months after the accident, he was hospitalized and underwent rehabilitation. The neurologic examinations, performed immediately after hospitalization, found that the patient was alert with the JKF-CRS score of 23. But on K-MMSE, the patient showed the manifestation of cognitive dysfunction with the score of 25.

On the brain FLAIR-MRI scan performed 3 months after the accident, high-signal intensities were observed on both basal ganglia and both sides of the subcortical white matter (Fig. 2-A). On brain PET performed 6 months after the accident, the cerebral hypometabolism was observed on both basal ganglia and both thalami (Fig. 2-B).

The patient could walk by himself before hospitalization, but showed a shuffling gait when turning, hence TUG was performed under suspicion of FOG. On TUG, turning hesitation and destination hesitation were observed. Also, tight-quarter hesitation was observed when the patient passed through the room door. Moreover, the patient showed kinesia paradoxa whenever crossing a line (Table 1). On FOG-Q before treatment, the patient was classified as severe FOG with the score of 19. A gait analysis, performed prior to rehabilitation, showed that the decrease of cadence and double support time but the shortage of step length, step time, walking speed and single support time (Table 2).

The patient underwent comprehensive rehabilitation with balance and gait training for 6 weeks. As a result, the problem of destination-hesitation was completely resolved, but in turning and tight-quarter hesitations, significant changes were not observed. The severity was slightly lowered with the FOQ-Q score of 17 (Table 1). Afterwards, the patient periodically underwent out-patient treatment.

DISCUSSION

FOG is a gait disturbance caused by a CNS injury, which precludes one from starting ambulation or inhibits the movements during locomotion. Ordinarily, it occurs unpredictably, and in serious cases it may cause abasia.[2] In common with Parkinson disease and Parkinson plus syndromes (progressive supranuclear palsy, multiple system atrophy, etc.), FOG is known to be closely related with degenerative CNS diseases, and in rare cases it is reported to be related with cerebrovascular disease.[2] As regards FOG in patients with HBI, however, there have been no reports other than that of Feve et al.[3]

The pathophysiology of FOG had been reported to be based upon the damage to the basal ganglia, the core

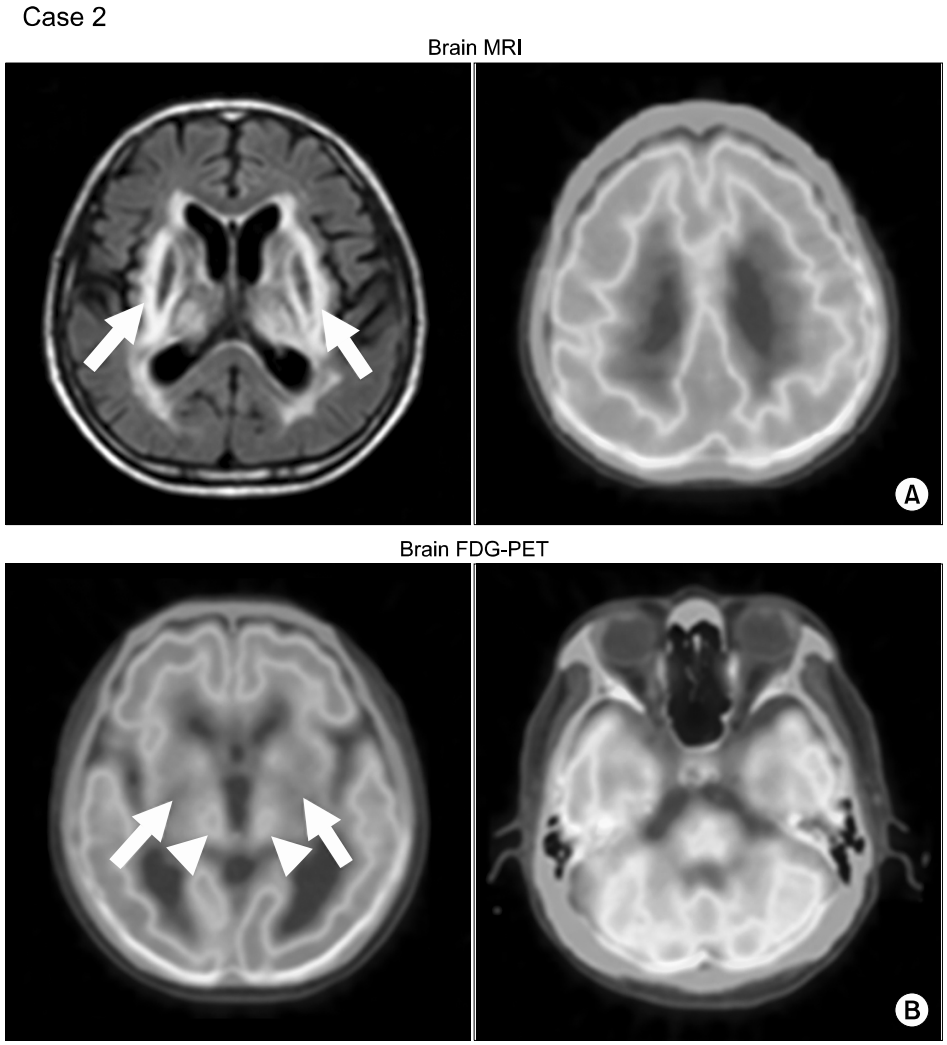

Fig. 2. A 65 year-old man associated with hypoxic brain injury following hypovolemic shock. (A) Brain MR T2-flair axial image showed high signal intensity in the bilateral thalamus and deep white matter (yellow arrow) (B) Positron emission tomography (PET) axial views showed diffusely decreased FDG uptake in the bilateral basal ganglia (white arrow) and thalamus (white arrow head).

part of motor control. However, recent neuroimaging studies have revealed that FOG is related with cerebral regions other than the basal ganglia. Matsui et al.[4] reported that the decreased cerebral blood flow of the orbitofrontal cortex is associated with FOG. Bartels et al.[5] reported that FOG is related with the hypofunction of the parietal lobe and the putamen. The neural correlates for FOG have not been clearly understood, but it has been reported that the input of wrong signals into the occipitoparietal area that integrates sensory and visual information or the malfunction of the frontostriatal pathway that controls gait by processing inputted signals may disrupt basal ganglia coordination of gait and cause a sudden distruption in gait programming, leading to FOG.[6] According to a report,[3] HBI-related FOG occurs after the injury of the basal ganglia. However, conclusive results have not been brought about as of now. Meanwhile, the above-mentioned two cases show that the hypofunction of frontostriatal pathway or occipitoparietal area may cause FOG even in patients with HBI. (In Case 1, dysfunction was observed in the diffuse cerebral cortex including the frontal lobe, the parietal lobe and the occipital lobe. In Case 2, dysfunction was observed in the subcortical area including the basal ganglia and the thalamus.) The two cases did not show significant differences in the clinical characteristics of FOG.

The clinical characteristics of FOG are largely divided into four according to situations, start hesitation, turning hesitation, destination hesitation just before reaching destination, and tight-quarter hesitation when passing through a narrow space.[2] In many patients, at least two hesitations arise simultaneously with each other. Also in the two cases presented in this report, TUG before and after rehabilitation session showed that various hesitations arose concurrently.

The FOG-Q is to measure the severity of FOG sensed by patients.[1] In Case 1, the FOG-Q score was 14 even after rehabilitation. But in Case 2, it was slightly lowered from 19 to 17 after rehabilitation due to resolution of destination hesitation. In the case of Parkinson disease, FOG ensures on its progress. But in the case of HBI-related FOG, there have been no studies on clinical characteristics. In Case 2, slight improvements were observed after rehabilitation, but checks were not made of other improvements in connection with the results, which was a limitation of this study. Thus, there is a need to conduct a further study on the clinical symptoms related with the improvement of FOG.

Kinesia paradoxa means the phenomenon that if a line is drawn on the ground in front of the foot of a patient, the patient can usually step over it without FOG.[7] In the two cases, kinesia paradoxa was observed not only before but after rehabilitation. It has been reported that the mechanism of kinesia paradoxa might be explained by the activation of the premotor cortex; to be specific, the premotor cortex is activated when the patient crosses a line drawn on the floor, and its activation heightens the accuracy of visuomotor control, and as a result the patient may be able to walk normally without FOG.[8] But, there have no few studies on the mechanism of kinesia paradoxa. In this connection, further studies need to be conducted.

FOG-related motion analysis has been mostly performed on patients with Parkinson disease. Alice et al.[9] performed motion analysis on normal adults and 10 patients who showed the FOG, and compared them in visuospatial indexes. The results showed the decrease of cadence and double support time, but the shortage of walking speed, step length and single support time, which were statistically significant. Likewise, the two cases, presented in this report, showed the decrease of cadence and double support time but the shortage of step length, step time, walking speed and single support time. In Case 2, gait disturbance was more serious in company with a subcortical injury. It may be because gait was more affected by the injury of the basal ganglia, a subcortical area, rather than by the cerebral cortex.

It is very difficult to treat FOG, but clinically balance and gait training, visual or auditory stimulation, pharmacotherapy and surgical operations are tried. The problem is that there is still controversy over such methods.[10] Particularly in relation to FOG caused by HBI, the mechanism has not been unraveled yet and also there have been few scientific reports. Such being the case, there have been no reports on therapeutic methods and effects. It is imperative to study them for the future.

This paper is to report 2 cases of FOG following HBI with literature review.

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